ISSN 1727-818X

International Journal of Interventional Cardioangiology

№26
2011

Read in the journal:

RUSSIAN SOCIETY OF INTERVENTIONAL CARDIOANGIOLOGY

ISSN 1727-818X

INTERNATIONAL JOURNAL OF INTERVENTIONAL CARDIOANGIOLOGY

Quarterly Journal of the Russian Scientific Society of Interventional Cardioangiology

№ 26, 2010

"International Journal of Interventional Cardioangiology" peer-reviewed scientific and practical journal. Founded in 2002

Website: www.ijic.ru

Address of the Editions:
101000, Moscow,
Sverchkov per., 5
Phone: (+ 7 495) 624 96 36
Fax: (+7 495) 624 67 33

Head of Editorial Office:
E.D. Bogatyrenko

Scientific editors of translation:
V.S. Chekanov

Translation:
Translation bureau MEDTRAN

Original layout prepared by:
A.Aksuk, V.Shelepukhin

Computer type-setting and makeup:
E. Fedorov

Corrector:
N. Sheludiakova

Special gratitude to
George Gigineishvili,
doctor and artist, for the offered opportunity to put the photocopy of his painting
"Interventional Cardioangiology" on the cover of the magazine

ISSN 1727-818X
9 771727 818001

Instructions for authors

The International Journal of Interventional Cardioangiology (IJIC) publishes peer-reviewed articles on all aspects of cardiovascular disease, as well as the abstracts of communications, presented at the scientific congresses, sessions and conferences, held by the Russian Scientific Society of Interventional Cardioangiology.

All manuscripts should be addressed to:
Prof. David G. Iosseliani, Editor-in-Chief, International Journal of Interventional Cardioangiology, Sverchkov per., 5, Moscow, 101000, Russia.
Fax: (7 495) 624 67 33
e-mail: davigdi@mail.ru

Manuscripts are considered for review only under the conditions that they are not under consideration elsewhere and that the data presented have not appeared on the Internet or have not been previously published. On acceptance, written transfer of copyright to the IJIC, signed by all authors, will be required. The IJIC will maintain copyright records. The publication of manuscript is free.

No part of materials published in IJIC may be reproduced without written permission of the publisher.

Address permission requests to:
Prof. David G. Iosseliani, Editor-in-Chief, International Journal of Interventional Cardioangiology, Sverchkov per., 5, Moscow, 101000, Russia. Fax: (7 495) 624 67 33
e-mail: davigdi@mail.ru

The Editors require authors to disclose any financial associations that might pose a conflict of interest in connection with the submitted article. If no conflict of interest exists, please state this in the cover letter.

Along with a cover letter, submit two complete copies of the manuscript, two sets of figures and tables, and two copies of the cover letter. If supplementary materials such as “in press” references are included, provide two copies.
The manuscript should be typed double-spaced throughout, on one side only, on 22 x 28 cm (8.5 x II”) white paper with 3-cm margin on all sides (8-cm at bottom of tide page). Please use a standard 10 cpi font or a laser printer font no smaller than 12 points.

The manuscript should be typed double-spaced throughout, on one side only, on 22×28 cm (8.5×II”) white paper with 3-cm margin on all sides (8-cm at bottom of tide page). Please use a standard 10 cpi font or a laser printer font no smaller than 12 points.

TITLE PAGE

Include the tittle, authors’ names (including full first name and middle initial, degrees and, where applicable, SICA), and a brief title of no more than 45 characters. List the departments and institutions with which the authors are affiliated, and indicate the specific affiliations if the work is generated from more than one institution (use the footnote symbols). Also provide information on grants, contracts and other forms of financial support, and list the cities and states of all foundations, funds and institutions involved in the work. Under the heading, “Address for correspondence,” give the full name and complete postal address of the author to whom communications, printer” s proofs and reprint requests should be sent. Also provide telephone and fax numbers and E-mail address.

STRUCTURED ABSTRACT

Provide a structured abstract of no more than 250 words, presenting essential data in five paragraphs introduced by separate headings

in the following order: Objectives, Background, Methods, Results, Conclusions. Use complete sentences. All data in the abstract must also appear in the manuscript text or tablesin the following order: Objectives, Background, Methods, Results, Conclusions. Use complete sentences. All data in the abstract must also appear in the manuscript text or tables.

CONDENSED ABSTRACT (for the summary)

Provide a condensed abstract of no more than 100 words, stressing clinical implications, for the expanded table of contents. Include no data that do not also appear in the manuscript text or tables.

TEXT

To save space in the Journal, up to 10 abbreviations of common terms may be used throughout the manuscript. On a separate page following the condensed abstract, list the selected abbreviations and their definitions. Editors will determine which lesser known terms should not be abbreviated. Use headings and subheadings in the Methods, Results and, particularly, Discussion sections. Every reference, figure and table should be cited in the text in numerical order according to order of mention. The list of keywords is mandatory.

STATISTICS

All publishable manuscripts will be reviewed for appropriate accuracy of statistical methods and statistical interpretation of results. Provide in the Methods a subsection detailing the statistical methods, including specific methods used to summarize the data, method for hypothesis testing (if any) and the level of significance r hypothesis testing. When using more sophisticated statistical methods (beyond t tests, chi-square, simple linear regression), specify statistical package used.

REFERENCES

Identity references in the text by Arabic numerals in parentheses on the line. The reference list should be typed double-spaced (separate from the text; references must be numbered consececutively in the order in which they are mentioned in the text.

Do not cite personal communications, manuscripts in prepation or other unpublished data in the references; these may be cited mi in parentheses.

Use Index Medicus (National Library of Medicine) abbreviations for journal titles. Use the following style and punctuation for references:

Periodical

List all authors if six or fewer, otherwise list the first three and add the et al.; do not use periods after the authors' initials. Provide inclusive page numbers.

Chapter in book

Provide inclusive page numbers, authors, chapter titles, book title, editor, publisher and year.

Book (personal author or authors)

Provide a specific (not inclusive) page number.

FIGURE LEGENDS

Figure legends should be typed double-spaced on pages separate from the text; figure numbers must correspond with the order in which they are mentioned in the text.

All abbreviations used in the figure should be identified either after their first mention in the legend or in alphabetical order at the end of each legend. All symbols used (arrows, circles, etc.) must be explained

If previously published figures are used, written permission from original publisher and author is required. Cite the source of the figure in the legend.

FIGURES

Submit two sets of laser prints or clean photocopies in two separate envelopes. Two sets of glossy prints should be provided for all half-tone or color illustrations. Note: The artwork of published articles will not be returned to authors.

Figures, particularly graphs, should be designed to take as little space as possible. Lettering should be of sufficient size to be legible after reduction for publication. The opti-

mal size after reduction is 8 points. Symbols should be of a similar size. All graphs and line drawings must be professionally prepared or done on a computer and reproduced as high quality laser prints. Decimals, lines and other details must be strong enough for reproduction. Use only black and white, not gray, in charts and graphs.

The first author's last name, the figure number, and the top location

should be indicated on the back of each figure, preferably on an adhesive label. Figure title and caption material must appear in the legend, not on the figure.

TABLES

Tables should be typed double-spaced on separate sheets, with the table number and tide centered above the table and explanatory notes below the table. Use Arabic numbers. Table numbers must correspond with the order cited in the text.

Abbreviations should be listed in a footnote under the table in alphabetical order. Tables should be self-explanatory, and the data presented in them should not be duplicated in the text or figures. If previously published tables are used, written permission from the original publisher and author is required. Cite the source of the table in the footnote.

OTHER PAPER CATEGORIES

Special materials will be considered by the Editors. In order to avoid any conflict of interests the authors should follow the recommendations:

State-of-the-Art Papers. The Editors will consider both invited and uninvited review articles. Such manuscripts must adhere to preferred length guidelines. Authors should detail in their cover letters how their submission differs from existing reviews on the subject.

Editorials and Viewpoints. Succinct opinion pieces will also be considered. These papers should have a brief unstructured abstract.

Editorial Comments. The editors invite all Editorial Comments published in the Journal.

Letters to the Editor. A limited number of letters will be published. They should not exceed 500 words and should focus on a specific article appearing in IJIC. Type letters double-spaced and include the cited article as a reference. Provide a title page that includes authors' names and institutional affiliations and a complete address for correspondence. E-mail (davigdi@mail.ru) or Mail two copies. Replies will generally be solicited by the Editors.

Board of the Russian Society of Interventional Cardioangiology

President
Kokov L. (Moscow)

Vice-Presidents
Semitko S. (Moscow)
Demin V. (Orenburg)
Iosseliani D. (Moscow)

Board Members
Arablinsky A. (Moscow)
Ardashev A. (Moscow)
Babunashvili A. (Moscow)
Biriukov A. (Riazan)
Bobkov Yu. (Moscow)
Bondar V. (Khabarovsk)
Buzaev V. (Ufa)
Chebotar E. (Nijny Novgorod)
Chernyshov S. (Yekaterinburg)
Chestukhin V. (Moscow)
Dolgushin B. (Moscow)
Dundua D. (Moscow)
Eroshkin I. (Moscow)
Fedorchenko A. (Krasnodar)
Filatov A. (Moscow)
Ganiukov V. (Novosibirsk)
Gromov D. (Moscow)
Ilyin V. (Moscow)
Ivanov V. (Krasnogorsk)
Kapranov S. (Moscow)
Karakulov O. (Perm)
Kavteladze Z. (Moscow)
Khamidullin A. (Kazan)
Kislukhin T. (Samara)
Koledinsky A. (Moscow)
Kozlov S. (Yekaterinburg)
Krylov A. (Tomsk)
Kucherov V. (Moscow)
Kuzmenko V. (Kaliningrad)
Lopotovsky P. (Moscow)
Maltzev A. (Moscow)
Mazaev V. (Moscow)
Melnik A. (Irkutsk)
Mironkov A. (Moscow)
Mironkov B. (Moscow)
Mizin A. (Khanty-Mansisk)
Morozova E. (Penza)
Osiev A. (Novosibirsk)
Perevalov A. (Ijevsk)
Pershukov I. (Voronezh)
Plekhanov V. (Ivanovo)
Poliaev Yu. (Moscow)
Prokubovsky V. (Moscow)
Protopopov A. (Krasnoyarsk)
Samko A. (Moscow)
Shakhov B. (Nijny Novgorod)
Sharabrin E. (Nijny Novgorod)
Shebriakov V. (Kupavna)
Shipovsky V. (Moscow)
Shukurov B. (Volgograd)
Sukhorukov O. (Moscow)
Sukhov V. (St. Petersburg)
Tarazov P. (St. Petersburg)
Terekhin S (Krasnogorsk)
Tibilov A. (Vladikavkaz)
Volkov S. (Moscow)
Volynsky Yu. (Moscow)
Yarkov S. (Moscow)
Zakharov S. (Moscow)
Zyrianov I. (Tiumen)

Russia, 101000, Moscow, Sverchkov per., 5
Moscow City Center of Interventional Cardioangiology
(for the Secretary of RSICA)
Phone: +7 (495) 624 96 36
President of RSICA: +7 (915) 301-00-67, +7 (985) 233-62-02
Fax+7 (495) 624-67-33
e-mail : lskokov@mail.ru
Website: www.rnoik.ru

HONORARY MEMBERS
of Russian Society of Interventional Cardioangiology

COLOMBO Antonio	Milan, Italy
CONTI, C.Richard	Gainesville, USA
DORROS Gerald	Phoenix, Arizona, USA
FAJADET Jean	Toulouse, France
HOLMES David R., Jr.	Rochester, Minnesota, USA
IOSSELIANI David	Moscow, Russian Federation
KATZEN, Barry T.	Miami, USA
KING Spencer B., III	Atlanta, Georgia, USA
LUDWIG Josef	Erlangen, Germany
MEIER Bernhard	Bern, Switzerland
PROKUBOVSKY Vladimir	Moscow, Russian Federation
RIENMULLER Rainer	Graz, Austria
SERRUYS Patrick W.	Rotterdam, Netherlands
SHAKNOVICH Alexander	New York, New York, USA
SIGWART Ulrich	Geneva, Switzerland
SIMON R diger	Kiel, Germany
SUKHOV Valentin	St.Petersburg, Russian Federation
VAHANIAN Alec	Paris, France
VOLINSKY Youry	Moscow, Russian Federation

Contents

Simultaneous Endovascular Coronary Angioplasty (Direct Stenting of the Marginal Branch, Recanalization and Stenting of "Chronic" Occlusion of the LAD) and Closure of Secundum Atrial Septal Defect

D.G. Iosseliani[1], A.G. Koledinsky, E.Yu.Danilov, I.A. Kovalchuk, V.A. Kriukov, A.Zh. Abildinova, A.N.Rogatova, I.G. Kibizova, A.V. Stepanov, A.V. Novachly, P.S. Vasiliev, V.S. Chekanov
Moscow City Center of Interventional Cardioangiology, Moscow, Russia

The authors present a clinical case of successful simultaneous endovascular myocardial revascularization for stenotic and occlusive coronary lesion and closure of ASD secundum. The dilatation of the LAD and the OMB with balloon catheter has been performed followed by DES implantation. During the second stage endovascular closure of the ASD using Amplatzer occluder has been performed. 16 similar cases have been described in the literature. The issue of reasonability of simultaneous performance of two independent endovascular procedures remains open. The necessity of ASD closure is dictated by the possibility of serious complications, such as ischemic stroke and migraine, in the presence of an open interatrial communication.
Keywords: *ASD, coronary stenting, occluder, coronary artery disease.*

Atrial septal defect comprises 5 to 15% of al congenital heart defects (3). In the absence of radical correction 30% of patients with this pathology die within the first 20 years of life (4). However the reasonability of this defect correction in patients who survived this age is not evident. In some patients the atrial septal defect (ASD) does not produce a significant impact on intracardiac hemodynamics (1), while in others the ASD can lead to progressive volume overload and right heart dilatation, development of arrhythmias and persistent pulmonary hypertension (6). Moreover, independently of the state of intracardiac hemodynamics, in all patients with ASD pathological interatrial communications can cause such serious complications as migraine and ischemic stroke (13). Also with the course of time such patients can develop coronary artery disease (CAD). In case of AMI and postinfarction cardiosclerosis the presence of pathological blood stream within the heart chambers can contribute to a more marked postinfarction remodeling of the left ventricle, volume overload, right heart dilatation and development of significant circulatory insufficiency. If such patients develop clinical signs of coronary artery disease, they should be examined for the detection of coronary pathology, and if it is revealed, a special treatment is mandatory. Thus, all grown-up patients with ASD should be continuously followed in order to receive timely medical care, including endovascular and surgical treatment.

Moscow City Center of Interventional Cardioangiology has an experience with simultaneous treatment of ASD and CAD in three patients. We describe one clinical case of successful simultaneous endovascular myocardial revascularization for stenotic and occlusive lesions of the coronary arteries and closure of a secundum ASD.

The patient P., 72 years old, was admitted to Moscow City Center of Interventional Cardioangiology with the diagnosis: CAD; angina of effort class II; postinfarction cardiosclerosis; 3rd degree arterial hypertension; circulatory insufficiency class I; congenital heart defect: secundum ASD; dysplasia of the interatrial septum.

The analysis of medical history revealed a long-standing arterial hypertension (max. to 200/110 mm Hg). The patient did not follow a regular hypotensive therapy. In 2004, without prior angina, he had a Q-wave anterior myocardial infarction, and received systemic thrombolysis with Alteplasa. Transesophageal EchoCG and MRI performed on the same occasion revealed for the first time a congenital heart defect — secundum atrial septal defect (ASD) with volume overload of the right heart. Endovascular ASD closure was suggested, but the patient refused this procedure. After that he started complaining of angina pain and heart rhythm disturbances (extrasystoles) and had several hospital stays. Selective coronary angiography performed in 2007 revealed an occlusion in the proximal segment of the LAD and a stenotic lesion in the marginal branch. The patient refused special treatment and continued conservative therapy. In October 2010 he applied to the consultative and diagnostic department of Moscow City Center of Interventional Cardioangiology complaining of decreased physical tolerance, increased fatigue, dyspnea of effort and heart rhythm disturbances. Multihelix computed tomography confirmed the diagnosis of an oval-shaped ASD secundum located in the upper part of the septum, measuring 1.07 x 1.25 cm and surrounded by an aplastic interatrial septum (IAS) with marked aneurysmatic bulging into the right atrium (Fig. 1).

[1] Address for correspondence:
Prof. David Iosseliani
Moscow City Center of uinterventional Cardioangiology
5, Sverchkov per., Moscow, 101000, Russia
Tel. +7 495 624 96 36
Fax +7 495 624 67 33
e-mail: davidgi@mail.ru
Manuscript received on July 19, 2011
Accepted for publication on September 05, 2011

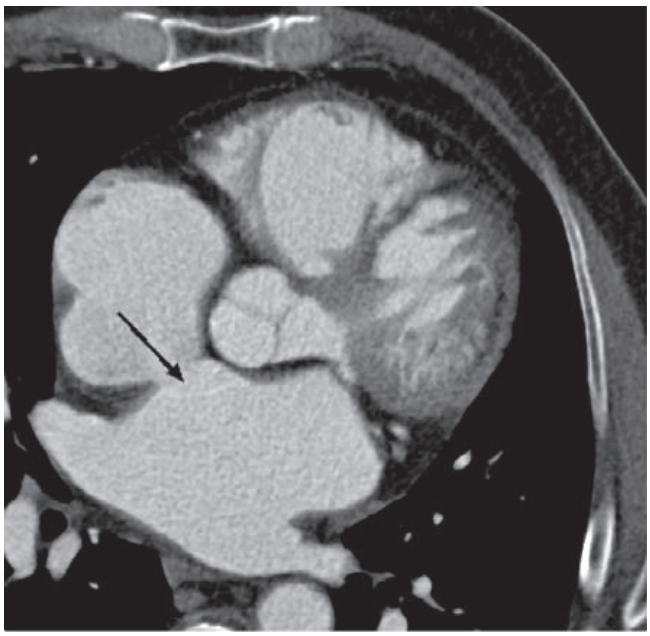


Fig. 1. Axial heart section at the ASD level. The arrow marks the defect

A pathological left to right shunt was seen an the level of the middle segment of the IAS (assumed size >0.6 cm), a moderate dilatation of heart chambers was noted: LVEDD — 5.46 cm, RVEDD –4.86 cm, LAEDD — 6.94 cm, RAEDD — 5.81 cm.

After a necessary out-hospital examination the patient was admitetd to the Center with the diagnosis: CAD; postinfarction cardiosclerosis; angina of effort and at rest; arterial hypertension; circulatory insufficiency class I; congenital heart defect: secundum atrial septal defect (ASD). At admission the patient's condition was satisfactory, with skin and mucosa of normal color and humidity. Pitting lower leg edema was present. The chest at the heart area looked normal, the borders of relative heart dulness were enlarged to the right by 1 cm from the right parasternal line at the level of the 3rd intercostal space. A soft systolic murmur was heard, maximal in the 3rd -4th right intercostal space. Heart rate — 84 b/min., blood pressure — 150/80 mm Hg.

ECG revealed sinus rhythm, 77 b/min. Transmural scars were present at the anterolateral segment of the left ventricle. 24-hour ECG monitoring revealed mean heart rate of 62 beats per minute. Several episodes of 1st degree AV block were registered. 428 isolated atrial extrasystoles were revealed. During the monitoring no ischemic changes of ST segment were registered. Transthoracic EchoCG revealed dilated LV cavity: end-diastolic dimension (EDD) — 6,0 cm; end-systolic dimension (ESD) — 4,8 cm. The thickness of interventiricular septum (IVS) was 8 mm, of left ventricular posterior wall — 9 mm. Left ventricular ejection fraction was 41%. The disturbances of local left ventricular contractility included apical akynesia, marked hypokynesia of the apical and middle segments of the anterior wall and interventricular septum. A large aneurysm measuring up to 40 mm with a central 9 mm defect resulting in the left-to-right shunt was located in the IAS area. Transesophageal EchoCG performed in order to precise the defect's details and determine its edges' dimensions showed a markedly dilated (up to 7,0 cm) right atrium with a surface area of 32 cm2. The right ventricle was moderately enlarged, RV EDD — 4,3 cm. Tricuspid regurgitation of the 2nd degree and moderate pulmonary hypertension (mean pulmonary arterial pressure —

65 mm Hg) were also revealed. An IAS aneurysm measuring 40 mm with a 20 mm bulging into the RA cavity was seen. Marked bulging into the RA caused a moderate obstruction to the blood flow. An ASD measuring 9 x 6 mm, with left-to-right blood shunting and a peak gradient of 21 mm Hg was located in the superoanterior part of the aneurysm (Fig. 2).

Taking into account the history of myocardial infarction, clinical presentation of angina, significant size of atrial septal defect and the signs of volume overload of the right heart, we decided to perform endovascular ASD closure as well as diagnostic left ventriculography and selective coronary angiography. Contrast left ventriculography (right oblique view) showed LV EF — 49%, moderate dilatation of LV cavity (EDV–202 ml, ESV — 104 ml). Hypokynesia of the anterolateral and apical segments of the LV were revealed. Diagnostic coronary angiography revealed left type of coronary circulation, an occlusion of the proximal segment of the LAD. There was over 75% stenosis in the obtuse margin branch (Fig. 3). The RCA was moderately changed without significant stenosis.

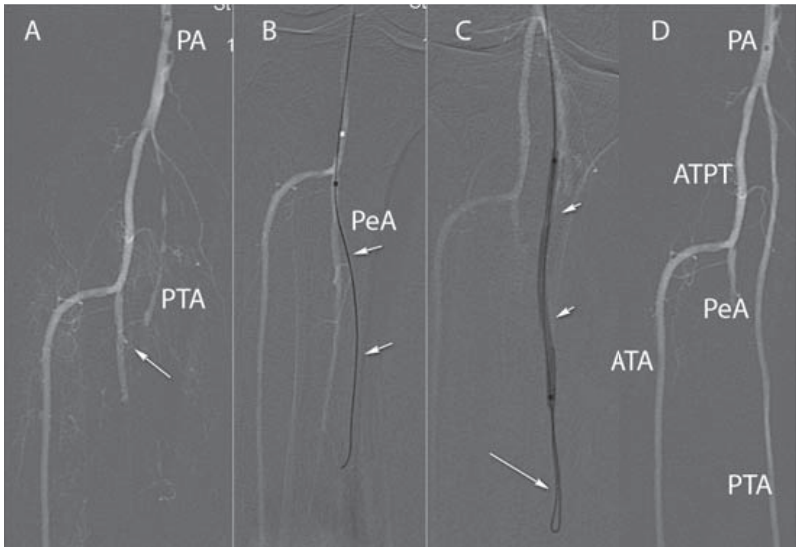


Fig. 2. Aneurysm of the IAS

On the base of CAG data (occlusion of the LAD and >75% stenosis of the OMB) multiple coronary angioplasty was performed before ASD closure. The left coro-

nary artery was catheterized with the guiding catheter Mach 3.5 SH (Boston Scientific), mechanical recanalization of the occluded LAD was performed using hydrophyle Shinobi guidewire (Cordis). The LAD was dilated using balloon catheter 1,5 x 20mm (Cordis), and then stented with a DES Taxus express 3,0 x 20 mm (Boston Scientific) (Fig. 4 a,b). The stent was completely deployed under 14 Atm. pressure.

After that we performed direct stenting of the OMB using Promus Element stent 3,5 x 28 mm (Boston Scientific) (Fig. 5 a,b) with good angiographic results.

At the second stage of the procedure endovascular ASD closure was performed. The right femoral vein was punctured under local anesthesia and a 6F introducer was inserted. The catheterization of the left upper lobe pulmonary vein was performed using multipurpose 6F catheter. Then a 34-mm sizing balloon was advanced using Amplatzer guide 0,035 x 260 cm and the defect size was determined. As the defect's edges were dysplastic, we have chosen 33 mm Figula Flex occluder. The occluder was placed under fluoroscopic guidance using generally adopted technique. At first the left occluder branch was deployed into the left atrium and the safety of occluder fixation at the defect's edges was checked with recurrent tractions of the delivery system. Only after that and after echocardiographic control of distal occluder branch position relative to the defect and the valvular apparatus of the LV, the proximal branch was deployed in the right atrium (Fig. 6).

Control transthoracic EchoCG revealed adequate positioning of the occluder and complete deployment of both discs, there was no shunt through the ASD. After the confirmation of secure occluder fixation the delivery system was removed. In our opinion, the process of this delivery system detachment — the removal of fittings' block — is simple and more convenient in comparison with the widely used Amplatz ocluders. The hemostasis was achieved using manual compression. We did not observe any complications during and early after the procedure. Control transthoracic EchoCG carried out in 4 days after ASD closure confirmed good effect of the procedure, the shunt through the defect was absent. In-hospital period was eventless and the patient was discharged at day 6 in a satisfactory condition. After the angioplasty the angina attacks stopped and did not recur thereafter. There were no changes on the ECG.

At the examination performed in one month the patient did not present any complaint, his condition was satisfactory. The results of performed

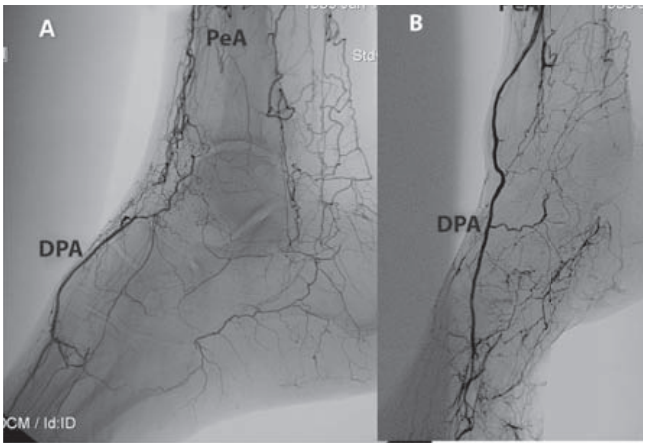


Fig. 3. Selective coronary angiogram of the left coronary artery: the LAD cannot be visualized. The middle segment of the OMB is stenotic (>75%)

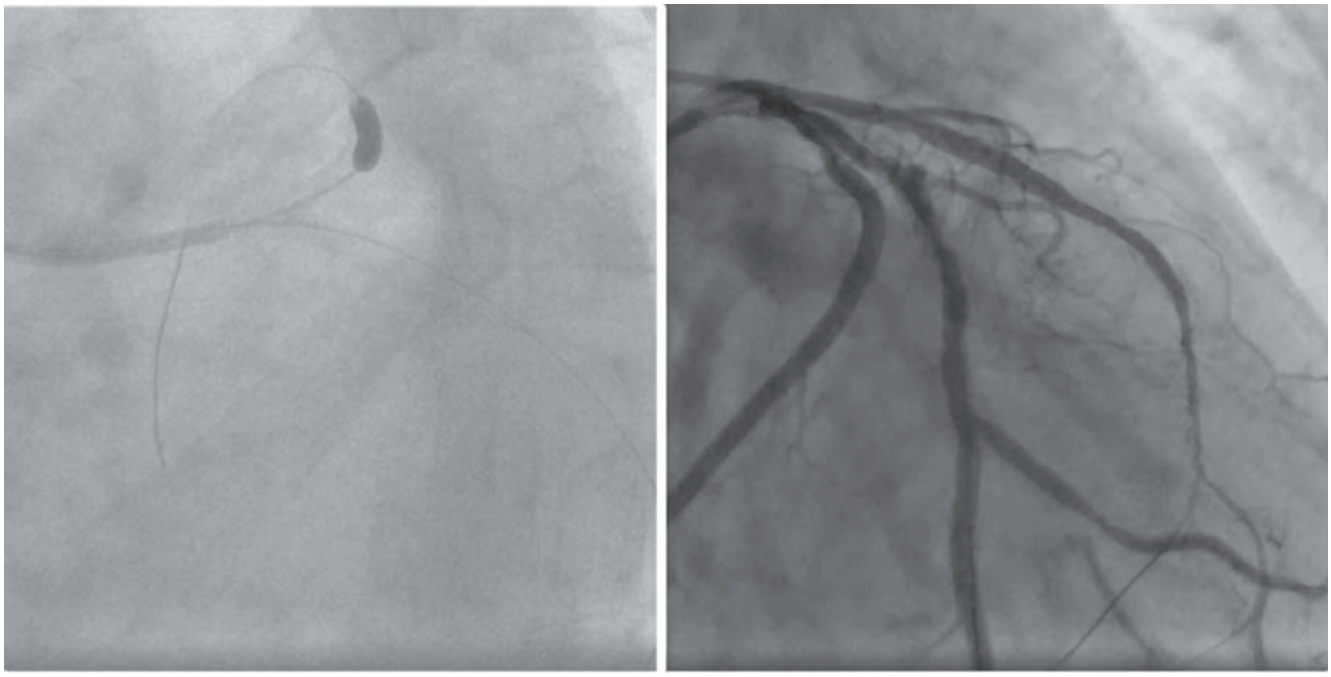


Fig. 4. a) Stenting of the proximal segment of the LAD using Taxus express DES (3x20mm); b) Immediate result of stenting

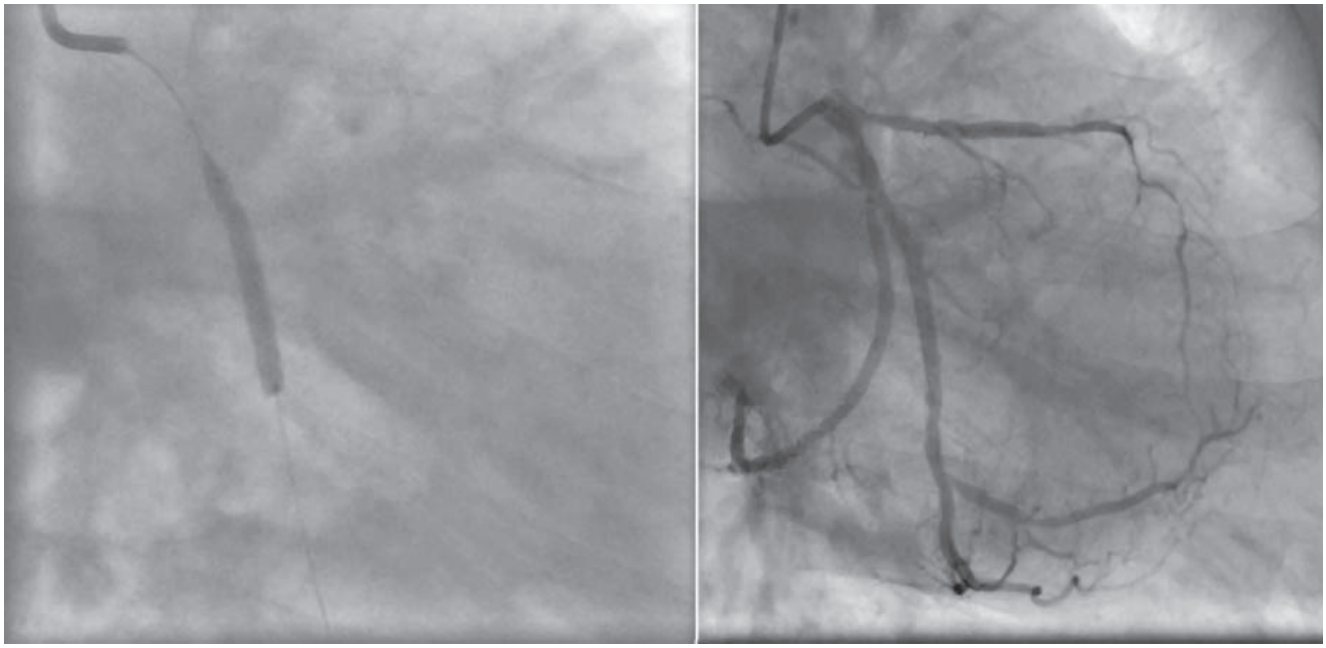


Fig. 5. a) Stenting of the OMB using Promus Element stent (3.5x28mm); b) Immediate results of OMB stenting

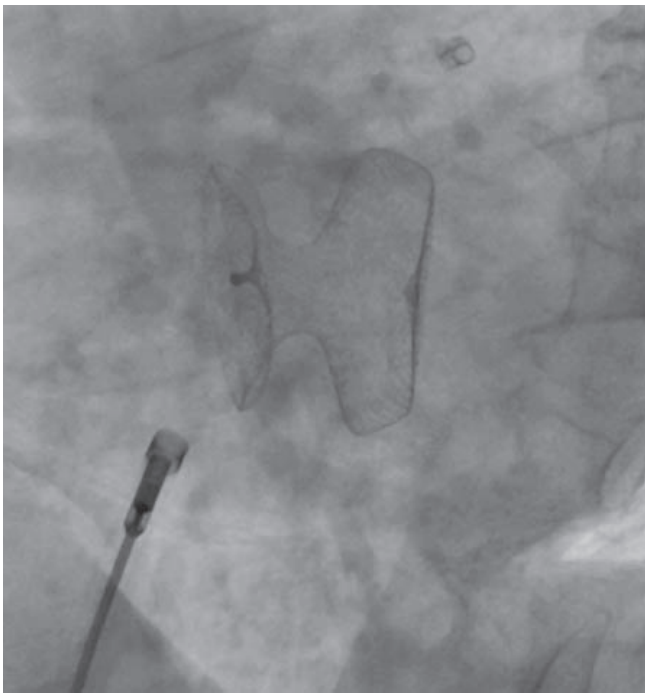


Fig. 6. Figula Flex ASD occluder (33 mm) is placed in the defect

endovascular procedures were assessed using control EchoCG as well as repeated MHCT, including of the coronary arteries with opacification. Transesophageal EchoCG showed the occluder with the right atrial disc closely adjacent to the IAS. The left atrial disc was fixed at the isthmus area, located at a 6,5–7 mm distance from the septum and separated from it by the aneurysmatic tissue. No echo-signals suspicious for thrombi or vegetations were located. No signs of residual shunt were seen (Fig. 7).

MHCT of the heart showed the occluder with signs of epithelization along its contours in the projection of the middle third of the IAS. No signs of transseptal shunt were revealed (Fig. 8). In the projection of the proximal third of the LAD a stent without restenosis signs was seen, the artery beyond the stent was moderately changed without sites of significant stenosis. The OMB and the RCA were without changes, the stent in the OMB — without restenosis.

Thus, the patient underwent successful combined procedure of ASD closure and multiple stenting of the coronary arteries. The experience with such combined procedures in adults, especially in elderly patients, is very small. While the number of such patients in the world amounts to many thousands, only 16 similar procedures have been described (14, 15, 16). Probably, the lack of clear indications inhibits wider spreading of such procedures. The procedure of coronary stenting does not raise any questions, as this procedure became a usual, even routine method for the treatment of CAD. The indications for it are also well elaborated. However the correction of ASD, especially in combination with angioplasty, is still subject to many questions. They concern mainly two moments: 1) the reasonability of congenital heart defect correction in elderly; 2) the reasonability of simultaneous performance of two independent endovascular procedures.

The problem of correction of the congenital heart disease — the ASD — in adults is still subject to discussions and not fully elucidated. Formerly it was usual to refrain from surgical intervention in adults. On the one hand, this was due to relatively high morbidity of surgical intervention with extracorporeal circulation, and on the other hand — to the paucity of clinical manifestations of the disease

and the absence of clear indications for surgery. Broad clinical introduction of endovascular methods of closure of pathological intracardiac communications, not requiring sternotomy and extracorporeal circulation and associated with an extremely low traumatism and high effectiveness again placed this problem in the agenda (2,5,9,10,11,12). While determining the tactics of treatment one should remember that despite left-to-right blood shunting within the heart — leading to: (a) diastolic overload of the RV and the RA; (b) increase of pulmonary blood flow sometimes by several times over the systemic flow; (c) decrease of blood input into the left heart and the aorta, — significant changes of intracardiac hemodynamics, including marked pulmonary hypertension, can become evident not immediately, but after a certain, sometimes a long time, for example, after 20 years and more. Hence, the absence of significant changes of intracardiac hemodynamics at the moment of examination should not be considered as a reason to refrain from ASD closure, as one has to consider this problem at a long term. In particular, this is true for the patients who already have some signs of CAD or a history of MI. This disease can enhance the appearance of significant disturbances of intracardiac hemodynamics due to the progressive LV failure. In adults this can be also influenced by the detraining of functionally underloaded left ventricle, the presence of arterial hypertension and other acquired heart diseases. Associated LV failure in patients with ASD can result in the increase of blood congestion in the right ventricular inflow tract. The more pronounced is left ventricular damage, the more significant will be these changes. In particular, they can be enhanced in the presence of postinfarction LV cardiosclerosis.

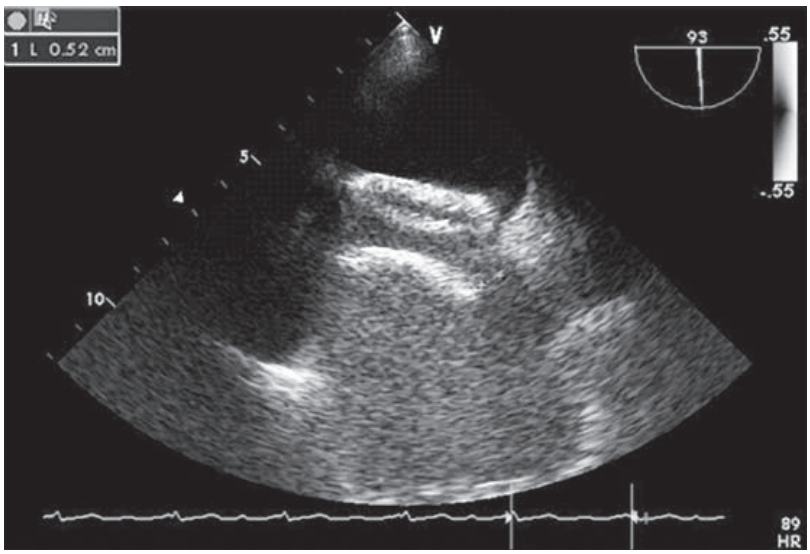


Fig. 7. Control TE-EchoCG in 1 month after the procedure

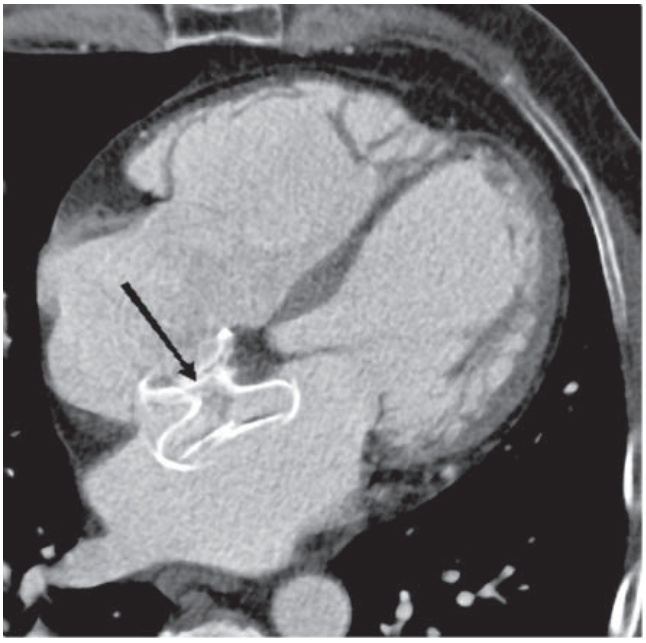


Fig. 8. Axial heart section at the level of the ASD. The arrow indicates the occluder

One also has to remember that the communication between the left and the right heart enhances the probability of thromboembolic complications in different organs, including the brain and the coronary arteries. Hence, the closure of the defect contributes to the decrease of the probability of thromboembolic complications.

Finally, there are evidences of the role played by ASD in the pathogenesis of migraine (13). The authors from Utah have shown that 25% of patients with ordinary migraine and 50% with migraine with aura had ASD. Certainly, this problem necessitates further investigation, however the presence of migraine in patients with ASD should be considered as an additional factor for positive solution of the problem of endovascular ASD closure.

Thus, while solving the problem of the choice of tactics of treatment in adults with ASD one has to take into account the probability of association of other cardiovascular conditions to this disease. With the course of time these conditions can significantly worsen the prognosis in such patients. For this reason we consider reasonable to eliminate pathological communication between the heart chambers being a probable cause of heart failure.

As to the simultaneous performance of two endovascular procedures in this contingent of patients, an extremely low number of cases makes it difficult to maintain pro or contra positions. Today we can only confirm the feasibility of such concomitant procedures without any serious complications (14,15,16). Surely it is a positive moment for patients who can avoid repeated emotional stress related to the procedure and repeated hospitalization. Also the overall in-hospital stay is reduced (17). However in order to

clarify this question we need to accumulate additional experience and perform a meticulous analysis of the obtained data.

References

1. King T.D., et al., Secundum atrial septal defect. Nonoperative closure during cardiac catheterization. JAMA, 1976, 235, 2506-9.

2. Holzer R., Hijazi Z.M. Interventional approach to congenital heart disease. Curr. Opin. Cardiol., 2004, 19(2), 84-90.

3. Bankle G. Congenital diseases of the heart and great vessels. Moscow, Meditzina, 1980, 52-65.

4. Belokon N.A., Podzolkov V.P. Congenital heart diseases. Moscow, Meditzina,1991, 69-79.

5. Hessling G., Hyca S., Brockmeier K., et al. Cardiac dysrhythmias in pediatric patient before and 1 year after transcatheter closure of atrial septal defects using the Amplatzer septal occluder. Pediatr. Cardiol., 2003, 24(3), 259 -62.

6. Oliver J.M., Gallego P., Gonzalez A.E., et al. Surgical closure of atrial septal defect before or after the age of 25 years. Comparison with the natural history of unoperated patients. Rev. Esp. Cardiol., 2002, 55(9), 953 -61.

7. Medeiros A., Iturrald P., Marques M, et al. Permanent rhythm and conduction disorders in patients surgically treated for atrial septal defect. Arch. Inst. Cardiol. Mex., 2000, 70(1), 46-54.

8. Meijboom F., Hess J., Szatmari A., et al. Long-term follow-up (9 to 20 years) after surgical closure of atrial septal defect at a young age. Am. J. Cardiol., 1993, 15, 72(18), 1431-34.

9. Chan K.C., Goodman M.J., Walsh K. et al. Transcatheter closure of atrial septa defects and interatrial communications with a new self expanding nitinol double disc device (Amplatzer septal occluder): multicenter UK experience. Heart, 1999, 82, 300-6.

10. Berger F., Ewert P., Bjornstadt P.G. et al. Transcatheter closure as standard treatment for most interatrial defects: experience in 200 patients treated with Amplatzer septal occluder. Cardiol. Young, 1999,9, 468-73.

11. Fisher G., Stieh J., Uebing A. et al. Experience with transcatheter closure of secundum atrial septal defects using the Amplatzer septal occluder: a single center study in 236 consecutive patients. Heart, 2003,89, 1999-2004.

12. Masura J. Endovascular Correction of Congenital Pathological Communications between Heart Chambers with Amplatzer Occluders. International Journal of Interventional Cardioangiology, 2006, 11, 17-26.

13. McCandless R.T., Aringtou C.B., D.C., Nielsen D.C., et al. Patent Foramen Ovale in Children with Migraine Headaches. J.of Pediatrics, D.0I.10.1016/j. jpeds, 2011.01.062.

14. Chessa M., Bossone E., Bini R. Et al. Transcatheter closure of an atrial septal defect within a giant aneurysm of the fossa ovalis. Echocardiography, 2003, 20, 297-9

15. Onorato E., Pera I., Lanzone A. et al. Transcatheter treatment of coronary artery disease and atrial septal defect with sequential implantation of coronary atent and Amplatzer septal occluder: preliminary results. Catheter. Cardiovasc. Interv., 2001, 54, 454-8

16. Yalonetsky S., Schwarts Y., Roguin A., Lorber A. Combined percutaneous coronary intervention and atrial septal defect closure in an adult patient. Acute Card. Care, 2007, 9 (4), 254-6

17. Calver A., Dawkins K., Salmon A. Combined percutaneous management of an atrial septal defect and coronary artery disease. J. Intervent. Cardiol., 2000, 13, 35-37.

Results of Coronary Stenting using the Stents with Biodegradable Polymer and Antiproliferative (Biolimus A9) Coating

R.V. Zeynalov[1], D.A. Asadov, M.B. Matini, V.V. Mazurova., D.G. Gromov, O.V. Zakharova, A.G. Koledinsky, D.G. Iosseliani.
Moscow City Center of Interventional Cardioangiology, Moscow, Russia

The study comprised two groups of patients with coronary artery disease. Then patients from Group 1 received DES with biodegradable coating, and the patients from Group 2 – DES with permanent polymer coating. Baseline clinical, historic and angiographic characteristics were comparable in both groups. The rate of restenosis in the mid-term follow up was similar in both groups. The rate of thrombosis was reliably lower in the group of DES with biodegradable polymer coating.
Keywords: *DES, stents with biodegradable polymer coating, Biomatrix stents.*

Purpose of study: to determine the effectiveness of the use of drug-eluting stents (DES) with biodegradable polymer coating (Biomatrix) in comparison with DES with permanent polymer coating («Cypher») in patients with coronary artery disease (CAD).

Material and methods: the study comprised 117 patients in whom a total of 235 stents have been implanted. The patients were divided into 2 groups. The patients from Group 1 (n=51) received 110 Biomatrix stents. The patients from Group 2 (n= 66) received 125 Cypher stents. The final purpose of the study consisted in the comparison the rate of restenosis and thrombosis of the above stents in the mid-term follow-up.

Results: the rate of restenosis in the mid-term was 2,72% in the group with Biomatrix stents and 3,2% — in the group with Cypher ($p>0.05$). The rate of thrombosis in the studied groups was significantly different — 0% and 3,2%, respectively ($p< 0.05$)

Conclusions: the effectiveness of the stents with bidgradable polymer coating is comparable with the effectiveness of the stents with permanent polymer coating. At the same time the rate of thrombosis after the implantation of Biomatrix stents is significantly lower than with Cypher stents.

Introduction

Large-scale implementation of DES into the clinical practice has allowed to improve the results of stenting due to the reduction of the rate of restenosis. The first clinical trials demonstrated significant advantages of first-generation stents in comparison with balloon angioplasty and stenting with bare metal stents. However after mass introduction of DES into the practice, in particular, in multiple extended coronary lesions as well as in the presence of several risk factors, the increase of the rate of late restenoses and thromboses became evident. One of the main causes of these serious cardiac complications consist in the presence of permanent (non-soluble) polymer stent coating. The impact of this coating on the vascular wall contributes to the development of stent restenosis and thrombosis after the end of the drug action (1,2,3).

The introduction of DES with biodegradable (soluble) polymer coating represents an attempt to eliminate this side effect, thus decreasing the rate of unfavorable results of treatment. Such stents are made of a steel frame and polylactic biodegradable polymer coating impregnated with a medication — Biolimus A9. Due to the abluminal coating as well as to the polymer's capacity to get degraded within 6-9 months with the formation of water and carbon dioxide, these stents decrease the rate of late restenosis and thrombosis, thus contributing to full endothelization of the inner surface (4).

Methods

In conformity with the purpose and the tasks we present the data of examination and treatment of patients with coronary artery disease (CAD) in the Moscow City Center of Interventional Cardioangiology during the period from 2008 to 2010.

The patients have been selected for the study on the base of the following criteria:

– CAD diagnosed on the base of an extensive clinical and instrumental examination;

– documented painless ischemia or stable angina of effort assessed in conformity with ACC/AHA classification;

– primary, not restenotic character of coronary arterial narrowing without the signs of fresh parietal thrombi;

[1] Address for correspondence :
Dr. Rufat Zeynalov,
Moscow City center of Interventional Cardioangiology
Russia, 101000, Moscow,
Sverchkov per., 5
Phone: +7 495 624 96 36
Fax: +7 495 624 67 33
e-mail: zeynalovrufat@hotmail.com
Manuscript received on September 17, 2011
Accepted for publication on September 29, 2011

– lesions allowing for coronary stenting;

– hemodynamically significant (≥ 60% of the diameter) arterial lesion;

– diameter of the involved vascular segment 2,25 — 4 mm.

The trial comprised the patients with angina of effort of class I-IV (AHA/ACC) and painless myocardial ischemia, independently of the number of the involved coronary arteries and the function of left ventricular myocardium, with coronary lesions of types A, B and C.

The patients with a history of balloon angioplasty or stenting , with concomitant cardiovascular pathology (aortic aneurysm, valvular diseases) requiring surgical correction were excluded from the study.

The patients included in this study received only Cypher or Biomatrix stents. Only one type of stents could be implanted in each patient.

The study comprised 117 patients in whom a total of 235 DES have been implanted. Depending on the type of drug and polymer coating of the implanted stents the patients have been divided into 2 groups. Group 1 comprised 51 patients with Biomatrix stents with biodegradable polymer and antiproliferative coating — Biolimus A9. Group 2 (control) comprised 66 patients with Cypher stents with permanent polymer and antiproliferative coating — Sirolimus.

Mean age of patients in Group 1 was 58 ±8,2 years, in Group 2 — 53 ±6,7 years. 44 of 51 patients in Group 1 (86,3%) were males. In Group 2 there were 48 (72,7 %) males, i.e. male patients prevailed in both groups.

The analysis of risk factors in Group 1 revealed smoking in 18 patients (35,3%), family predisposition to CAD — in 8 (15,7%), arterial hypertension — in 12 (23,5%), dislipidemia — in 15 (29,4%), obesity — in 3 (5,9%), diabetes mellitus — in 10 patients (19.6 %). In Group 2 these risk factors were met in 22 (33,3 %), 11 (16,7 %), 13 (19,7%), 16 (24,4 %), 4 (6,1 %) and 11 patients (16.7%), respectively (p>0,05) (Table № 1).

The combination of 3 and more risk factors was met in 11 (21,56 %) patients from Group 1 and in 15 (22,7 %) patients from Group 2 (p>0,05).

Thus, the comparison of risk factors prevalence in the studied groups (including smoking, family history and arterial hypertension) did not reveal any significant difference. Neither was the rate of diabetes mellitus statistically different. The Table №2 shows patients' distribution according to the forms of CAD.

7 patients from Group 1 (13,7%) and 7 patients from Group 2 (9,0%) had a history of myocardial infarction, that is postinfarction cardiosclerosis (PICS) (p<0,05).

In Group 1 painless ischemia was seen in 3 patients (5,9 %) and angina of effort — in 35 (67,7%). Among them 5 patients (9,8 %) had heart failure of functional class I, 13 (25,5%) — of class II, 11

Table № 1.

The prevalence of risk factors for CAD in the studied groups

Risk factors	Group 1, n=51		Group 2, n=66		P
	abs.	%	abs.	%	
Smoking	18	35.3	22	33.3	>0.05
Family history	8	15.7	11	16.7	>0.05
Arterial hypertension	12	23.5	13	19.7	>0.05
Dislipidemia	15	29.4	16	24.2	>0.05
Obesity	3	5.9	4	6.1	>0.05
Diabetes mellitus	10	19.6	11	16.7	>0.05

Table № 2.

Patients' distribution by the forms of CAD

Form of CAD	Group 1, n=51		Group 2, n=66		P
	abs.	%	abs.	%	
Painless ischemia	3	5.9	5	7.6	>0.05
Angina of effort	36	70.6	48	72.7	>0.05
Unstable angina	6	11.8	8	12.1	>0.05
Myocardial infarction	7	13.7	5	7.6	>0.05
Non ST-elevation myocardial infarction	1	2.0	2	3.0	>0.05
ST-elevation myocardial infarction	6	11.8	3	4.5	**<0.05**

(21,5 %) — of class III and 7 (13,7 %) — of functional class IV. Unstable angina was seen in 6 (11,7%) patients. In Group 2 painless ischemia was observed in 5 patients (7,6%). In total, 48 patients in the control group (72,7%) were in heart failure. Among them 9 (13,6 %) had heart failure of functional class I, 20 (30,3%) — of class II, 12 (18,2 %) — of class III and 7 (10,6%) — of functional class IV. That is, both groups were highly homogenous by their functional class of angina, and statistical analysis did not reveal significant differences. A statistically significant difference have been revealed among patients with ST-elevation myocardial infarction (STEMI). In Group 1 there were 7 patients admitted with the diagnosis of MI. Among them 1 patient had non ST-elevation MI and 6 had STEMI. In Group 2, MI has been diagnosed at admission in 5 patients, 2 of them had non ST-elevation MI and 3 had STEMI.

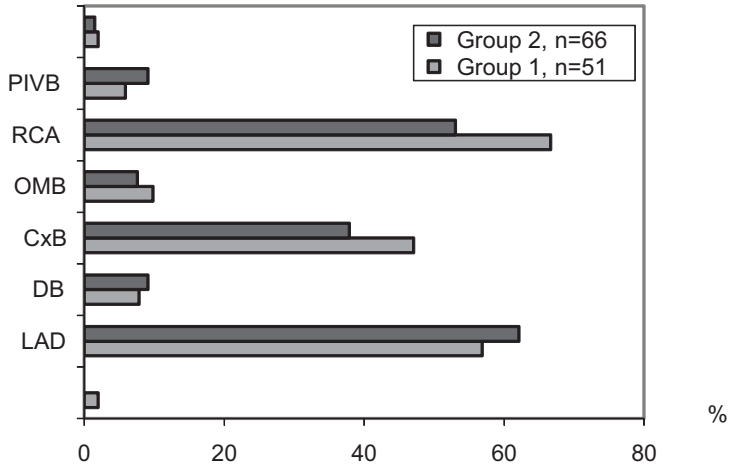


Diagram № 1. Involved (target) coronary arteries in the studied groups

The diagram № 1 shows the distribution of target arteries in the groups.

Statistical analysis

Statistical analysis of the quantitative data was carried out with the use of non-parametric methods — Wilcoxon-Mann-Whitney criterion and Wilcoxon signed-ranks test for matched pairs. If the amount of data in the compared groups was under 30 and in at least in one group it was under 5, the results have been compared using exact Fisher test (8, 9, 10).

Results

Early results. Final clinical success of the PCI as well as final optimal angiographic results have been obtained in 100 % of cases in both groups. In Group 1 (n=51) the complications occurred at different stages of the stenting procedure in 4 patients. In 1 case there was a dissection along the distal edge of the stent requiring an additional stent implantation. In another case the development of “no-reflow” phenomenon inhibited the antegrade filling of the vessel. «No-reflow» could be stopped by intracoronary nitroglycerin. The implantation of an additional stent was not necessary. Also 2 patients (3,92%) from this group had a subcutaneous hematoma at the puncture site, not requiring blood transfusion.

In Group 2 (n=66) the stenting-related complications developed in 3 patients. In one case the intimal damage distal to the target lesion in the territory of the stented artery, occurring during guidewire positioning, led to this artery thrombosis. After the administration of IIb/IIIa platelet receptors inhibitiors the thrombus was lysed and an additional stent has been implanted with good results (the MI did not develop). One patient had a subcutaneous hematoma with the signs of post-hemorrhagic anemia (not requiring blood transfusion). Another patient had a transient acute cerebrovascular accident, which was completely stopped in the intensive care unit in 2 hours after the procedure.

There were no other complications, including deaths, in the studied groups. The analysis of the immediate results revealed the absence of significant differences in the prevalence of complications between the groups (p>0,05) (table №3).

All patients were discharged in stable condition with the recommendations for further medical therapy.

Mid-term results of endovascular treatment.

The duration of mid-term follow-up in the studied groups was 8,4 ±1,4 months for Group 1 and 8,1 ±1,7 months for Group 2. The fate of all patients included into the study was known by the moment of control examination. According to the existing protocols the patients underwent exercise testing for the detection of eventual myocardial ischemia and coronary angiography (95 patients). The data are presented in Table № 4.

In the mid-term follow-up 51 patients (100%) from Group 1 and 65 patients (98,48%) from Group 2 underwent clinical examination. The evaluation of myocardial perfusion by the means of stress-testing did not reveal clinical signs of angina in 47 patients (92,15%) from Group 1 and in 61 (93,8 %) patients from Group 2. Clinical signs of angina with confirmed myocardial ischemia has been noted in 4 (7,84%) patients from the Group 1 and in 3 (4,54%) patients from the control group. All patients with clinical signs of angina underwent coronary angiography.

Control coronary angiography in the mid-term follow-up has been performed in 42 patients (82 %) from Group1, who received 94 stents (85,5%), and in 53 patients (80,3%) from Group 2, in whom 76 stents (79,2%) had been implanted. The results of coronary angiography allowed for the assessment of such pa-

rameters as restenosis, thrombosis and progressing of atherosclerotic process.

In-stent restenosis was determined as the lumen loss > 50%, and also at 5 mm distal and proximal to the stent

Restenosis was revealed in 2 patients from Group 1. In one case clinical picture of angina resumed within 5 months, in another — within 7 months after the discharge.

In Group 2 there were 4 patients with in-stent restenosis. One of then had painless ischemia, 2 had angina of effort of functional class II. Clinical signs of angina resumed at 5 and 6 months after the discharge. The 4th patient with in-stent restenosis had angina of the III functional class in 5 months after stenting. In all cases restenosis involved only one stent. The frequency of restenosis was not statistically different between the groups (p>0.05).

The progressing of atherosclerotic process was noted in 2 (3,92%) patients from Group 1. As their lesions were hemodynamically significant, both of them underwent coronary stent implantation. The progressing of atherosclerotic process in 1 (1,5%) patient from Group 2 also required coronary stent implantation. The evaluation of atherosclerosis progression in the coronary arteries during the period of follow-up did not reveal significant differences between the groups (p>0,05).

The frequency of stent thrombosis in the studied groups was statistically different. In Group 1 there were no cases of stent thrombosis during the whole period of the follow-up. In group 2 (control) thrombosis was revealed in 4 stents (3,2%) of 3 patients (4,54%) (Table №5).

One patient from Group 2 had a subacute thrombosis in the area of the implanted stent and developed a Q-wave MI at day 17 after stenting. The patient with subacute thrombosis has been brought to the Center of Interventional Cardioangiology and underwent mechanical recanalization and balloon angioplasty in the area of thrombotic stent (with good effect).

Both patients with late thromboses revealed in the mid-term follow-up were from the control group. Both of them were admitted in emergency. The first patient was admitted at day 45 after stenting with the diagnosis of a Q-wave MI. He underwent mechanical recanalization, thrombextraction and balloon angioplasty of the stent. The second patients was admitted in 8 months after the discharge with clinical picture of unstable angina. He underwent mechanical recanalization and balloon angioplasty in two stents.

The analysis of factors influencing the development of restenoses and thromboses in stents did not

Table №3.

Immediate clinical and angiographic results of stenting in the studied groups

	Group 1	Group 2	P
Successful procedure, n (%)	51 (100%)	66 (100%)	>0.05
Vessel's dissection, n (%)	1 (1,96%)	1 (1,5%)	>0.05
Myocardial infarction, n (%)	0	0	>0.05
Cerebrovascular complications, n (%)	—	1 (1,5%)	>0.05
Hematoma at the puncture site, n (%)	2 (3,92%)	1 (1,5%)	>0.05
Total number of patients with complications (%)	4 (7,84%)	3 (4,54%)	>0.05

p>0,05 non-significant differences; n – number of patients

Table № 4.

The number of patients examined in the mid-term follow-up

	Group 1 n=51 (100 %)	Group 2 n=66 (100%)	P
Mid-term follow-up, months	8,4 ± 1,4	8,2 ±1,7	>0.05
The patients with available information, n (%)	51 (100%)	66 (100 %)	>0.05
Examined in the mid-term follow-up, n(%)	51 (100%)	65 (98,48%)	>0.05
Control CAG in the mid-term follow-up, n (%)	42 (82 %)	53 (80,3%)	>0.05

p>0,05 non-significant differences; n – number of patients

reveal statistically reliable relation between any factor and the development of these complications.

The analysis of the influence of the stent length on the frequency of restenosis revealed statistically significant differences in Group2: the implantation of stents longer than 23 mm was related to higher frequency of restenosis in comparison with shorter stents ($p<0.05$).

The analysis of the influence of the stent diameter on the frequency of restenosis did not reveal any significant inter-, or intragroup differences. Small diameter of stent had no statistically reliable impact on the frequency of restenosis ($p>0.05$).

The study of the influence of stent's diameter and length on the development of late stent thrombosis has shown that in all three cases of stent thrombosis in the control group the diameter of endoprosthesis was < 2,75 mm, while its length was > 18 mm .

After stenting all patients received double antiplatelet therapy. The Table № 6 shows different regimens of antiplatelet therapy in the mid-term follow-up.

The Table shows that the number of patients receiving double atniplatelet therapy in the mid-term follow-up was not statistically significant between the studied groups. A patient from Group 2 with subacute stent thrombosis received Plavix (75 mg/day) by the moment of thrombus formation. One of two patients with late thrombosis has stopped to take Plavix in 4 months after the implantation of Cypher. Another patient received Plavix (75 mg/day) by the moment of late stent thrombosis development.

Thus, the comparison of the results revealed that in the mid-term follow-up the rate of restenosis was somewhat higher in the group of stents with permanent polymer coating. However this difference between the groups was statistically insignificant.

The analysis of the obtained results did not reveal any correlation between the rates of restenosis, progressing of atherosclerotic process and any clinical risk factor in the studied groups.

The analysis of the influence of the stent's diameter and length has shown that in Group 2 patients the rate of thrombosis was significantly higher in the presence of stents longer than 18 mm in comparison with Group1.

Total rate of thrombosis was statistically different between the groups ($p<0,05$). In Group 1 it was 0%, while in Group 2 — 3.2%. ($p<0,05$). The analysis of the obtained data revealed a correlation between the stent's dimensions and the rate of late thrombosis. Statistical analysis has shown that the probability of thrombus formation in Group 2 increased with the implantation of stents with diameter < 2.75 mm and length > 18 mm.

Discussion

The introduction of drug-eluting stents with biodegradable polymer coating into the practice has opened new possibilities of influencing the rate of stent restenosis and thrombosis. Herewith, due to drug-eluting coating these stents decrease the rate of restenosis, and their dissolving polymer coating contribute to the decrease of the rate of stent thrombosis.

The LEADERS trial comprised the patients with Biomatrix and Cypher stents. According to this trial's data, the rate of MACE in the mid-term follow-up

Table №5.

Analysis of the frequency of stent thrombosis in two groups

Type of thrombosis	Group 1, n=110 (100%)	Group 2, n=125 (100%)	P
Acute	—	—	—
Subacute	—	1 (0.8%)	>0.05
Late	—	3 (2,4%)	< 0.05
Thromboses, in total	—	4 (3,2%)	< 0.05

p>0,05 non-significant differences; n – number of stents

Table № 6.

Antiplatelet therapy in the studied groups in different periods of follow-up

Antiplatelet regimen	Up to 3 months			3 – 6 months			6 – 9 months			> 9 months		
	Gr.1	Gr.2	P	Gr.1	Gr.2	P	Gr.1	Gr.2	p	Gr.1	Gr.2	p
Plavix + Aspirin	51	66	p>0,05	49	63	p>0,05	45	57	p>0,05	38	49	p>0,05
Only Plavix	—	—		—	—		—	—	—	—	—	
Only Aspirin	—	—		2	3	p>0,05	4	6	p>0,05	10	13	p>0,05
No antiplatelet therapy	—	—	—	—	—		2	3	p>0,05	3	4	p>0,05

(9 months) was 9,2% and 10,5%, respectively. Herewith the indices of cardiac death (1,6% vs. 2,5%), Mi (5,7% vs.4,6%), target vessel revascularization (4.4% vs.5.5%), in-stent stenosis (20,9% vs.23,3%) were lower in the group with Biomatrix stents (5,6). The comparison of these data with our results reveals some divergences. Probably, the main cause of these divergences is a more complex contingent of patients included in the LEADERS trial. The rate of extended stenoses, as well as of small coronary arteries in this trial was higher (6). However, despite higher indices of restenosis in the trial, they are lower than in the group with Cypher stents, which was also confirmed by our study.

The thrombosis of the stented coronary arteries is still the main cause of cardiac complications, including cardiac death. Recent long-term trials conducted in large populations of patients have revealed the problem of late thrombosis, primarily related to the delay of vessel healing and to the inflammatory reaction of its wall to the polymer coating of DES.

Awata M. et al. (7) have evaluated the endothelization of stents with biodegradable and permanent coating using angioscopy. The coating of the stent's struts was evaluated with a special score, where 0 corresponded to a visible strut, and 3 — to a completely endothelized strut. After 9 months the surface of stents with biodegradable coating was more homogenous in comparison with the stents with permanent coating. (80% vs. 56% p = 0.035).

Conclusion

Optimal immediate angiographic results of PCI and absence of recurrent angina at in-hospital stage are seen in the vast majority of patients after the implantation of stents with biodegradable (Biolimus A9), as well as with permanent polymer coating (Sirolimus). The rate of restenosis in the mid-term follow-up in both groups was not statistically different. However the rate of stent thrombosis in the mid-term follow-up was significantly lower in the group of stents with biodegradable polymer coating in comparison with the stents with permanent coating. Also, in the group of stents with biodegradable polymer coating we did not reveal factors influencing the results of stenting in the mid-term, while the use of stents with diameter < 2,75 mm and length > 18 mm was a risk factor fo thrombosis in the group of stents with permanent coating.

References

1. Mauri L., O'Malley A.J., Popma J.J. et al. Comparison of thrombosis and restenosis risk from stent length of sirolimus-eluting stents versus bare metal stents. Am. J. Cardiol., 2005, 15, 95(10), 1140-5.

2. Hwang C.W., Levin A.D., Jonas M., et al. Thrombosis modulates arterial drug distribution for drug-eluting stents.. Circulation, 2005, 111 (13), 1619–26.

3. Virmani R., Guagliumi G., Farb A. et al. Localized hypersensitivity and late coronary thrombosis secondary to a sirolimus-eluting stent: should we be cautious? Circulation, 2004, 109 (6), 701–5.

4. Grube E., Buellesfeld E.L. BioMatrix Biolimus A9-eluting coronary stent: A next-generation drug-eluting stent for coronary artery disease. Expert. Rev..Med. Devices, 2006, 3, 731-41.

5. Wykrzykowska J.J., Ruber L., de Vries T. Biolimus-eluting biodegradable polymer versus sirolimus-eluting permanent polymer stent performance in long lesions: results from the LEADERS multicentre trial substudy. EuroIntervention, 2009, 5(3), 310-7.

6. Chevalier B., Serruys P.W., Garcia E. et al. Randomized comparison of Nobori™, Biolimus A9-eluting coronary stent with a Taxus®, paclitaxel-eluting coronary stent in patients with stenosis in native coronary arteries: the Nobori trial. EuroIntervention, 2007, 2, 426–34.

7. Awata M., Uematsu M., Sera F. Angioscopic assessment of arterial repair following biodegradable polymer-coated biolimus A9-eluting stent implantation. — Comparison with durable polymer-coated sirolimus-eluting stent. Circ. J., 2011, 25, 75(5), 1113-9.

8. Conover W. J. Practical Nonparametric Statistics, 2nd ed. Wiley, New York, 1980.

9. Glantz S. Medical and biological statistics (transl. From English). Moscow, Praktika, 1999, p.459.

10. Gubler E.V., Genkin A.A. Use of non-parametric statistical criteria in medical and biological studies. Leningrad, Meditzina, 1973, 140 p.

Comparative Effectiveness of Conservative and Invasive Treatment of Myocardial Infarction in Elderly and Old Patients

A.B. Shames[1], V.A. Ivanov
A.A. Vishnevsky 3rd Central Military Clinical Hospital, Krasnogorsk, Russia

Old age is one of the most important factors influencing the outcome of myocardial infarction (MI). According to several studies, over 50% of MI-related deaths occur in patients over 75 years old. The authors performed a comparative analysis of the effectiveness of conservative and invasive treatment in 656 patients with MI aged from 60 to > 90 years. It was found that emergency invasive tactics is a method of choice for the treatment of MI in high-risk patients, the majority of whom are elderly and old patients.
Keywords: *myocardial infarction, elderly and old patients, invasive tactics.*

Introduction

The 20th century has been marked by demographic aging of population in the industrial countries — the redistribution of age structure of population with the increase of the share of elderly and old people. According to the prognoses made by American researchers, by 2025 there will by over 1 billion of people over 60 in the world population (1). The increase of the number of older age groups is a challenge for modern medicine. It is related to the particularities of elderly and old age (involutional changes in the organism, polymorbidity, atypical course of the diseases, changes in social and psychological status), as such patients, as a rule, are at high risk of death. For this reason the choice of the method of treatment should be made with caution and based on the "risk / benefit" ratio.

Elderly and old age of patients is one of the most important risk factors influencing the outcome in myocardial infarction (MI). According to several authors, over 50% of death from MI occur in patients over 75 years old (1-3).

Purpose of study

To compare the effectiveness of conservative and invasive treatment of MI in elderly and old age.

Material and methods

We have compared the effectiveness of conservative and invasive treatment in 656 patients with MI. The patients have been assigned to age groups in accordance with the classification of the periods of aging and old age of the European regional bureau of the World Health Organization (WHO) (4). At first admission clinical characteristics of patients with MI have been determined with the use of the classification suggested by the All-Union Cardiological Scientific Center of the Academy of Medical Sciences of the USSR (1984), developed on the base of WHO expert guidelines (5).

370 patients were aged from 60 to 74 years, 284 — from 75 to 89 years, and there were 2 patients aged 90 and more. Non ST-elevation MI (nSTEMI) was diagnosed in 344 patients, while 312 had ST-elevation MI (STEMI). In 48,6% of cases MI involved the anterior left ventricular wall, in 43,5% — the inferior/posterior wall, in 7,9% of cases it had another localization. Primary MI was diagnosed in 449 (68,4%) patients: nSTEMI in 203 (30,9%), and STEMI in 246 (37,5%). 207 patients (31,6%) had repeated MI: nSTEMI in 141 (21,5%), and STEMI in 66 (10,1%). 16% of all patients were women. Comorbidities included: arterial hypertension in 73,8% of patients, diabetes mellitus in 20%, signs of chronic heart failure in 49,6%, signs of chronic renal failure in 3,1%; 6,7% of patients had a history of surgical myocardial revascularization.

Conservative treatment in conformity with national and international guidelines for the treatment of MI (6-9) was applied in 530 cases. 48,3% of patients were in the elderly age group (60-74 years), 51,7% — in the old age group (75-89 years). Primary MI was diagnosed in 369 patients (166 — nSTEMI, 203 — STEMI), repeated MI — in 161 patients (113 — nSTEMI, 48 — STEMI).

Invasive treatment (coronary angioplasty with stenting) was applied in 126 cases — among them 91% from the elderly and 9% from the old age group. Among these patients 65 had nSTEMI and 61 had STEMI. Primary MI was diagnosed in 80 (63,5%) patients: nSTEMI — 29,4%, STEMI — 34,1%; repeated MI — in 46 (36,5%): nSTEMI –22,2%, STEMI– 14,3%. Coronary angiography revealed significant (>79%) single vessel disease in 19,5% of patients, two-vessel disease in 32,5%, three-vessel disease — in 43,9% of patients; isolated lesion of the left main coronary artery was found in 4,1% of patients. In 41% of cases occlusive and stenotic lesions involved the LAD, in 31,4% — the right CA, in 26,8% — the

[1] Address for correspondence:
Dr. A. Shames
Russia, 143420, Moscow region, Krasnogorsk district.,
PO Arkhangelskoye, township Novy.
A.A. Vishnevsky 3rd Central Military Clinical Hospital»
Tel. 007 495 564-63-81.
E-mail: ashames@yandex.ru
Manuscript received on June 03, 2011.
Accepted for publication on September 21, 2011

circumflex artery. In the presence of the occlusion of the infarct-related artery (IRA) we have performed mechanical recanalization with a guidewire with subsequent predilatation of the occluded area and stenting of the residual stenosis. In cases of subtotal stenosis of the IRA also the predilatation of the involved area and stenting of the residual stenosis were performed. Direct stenting of the IRA was performed in cases of 70-90% stenosis. In 78% of cases we have used bare metal stents «Tsunami Gold» ("Terumo", Japan), «Multi-Link Vision» ("Abbot Vascular", USA), in 22% — drug-eluting stents: «Cypher» ("Johnson & Johnson", USA), «Xience V» ("Abbot Vascular", USA), «Taxus» ("Boston Scientific", USA). Not later than 6 hours before coronary angioplasty or no later than 2 hours before the intervention the patients received oral Plavix ("Sanofi-Synthelabo", France) — 300 mg or 600 mg, respectively. Patients who previously were on Aspirin, received 500 mg, and those who did not intake it — 250 mg of oral Aspirin ("Bayer", Germany) not later than 2-6 hours prior to the intervention. At the beginning of coronary angioplasty the patients received an intraarterial bolus of non-fractionated heparin (NFH) (70-100 MU/kg) with subsequent administration of additional dose of NFH to keep the activated clotting time (ACT) within the limits of 250-300 sec. An inhibitor of glycoprotein IIb/IIIa platelet receptors "Monafram" (Framon, Russia) was used during coronary angioplasty in 19,5% of cases. The dosage was calculated at the rate of 0,25 mg per 1 kg of body weight, and the medication was administered as intravenous bolus 10 — 30 minutes prior to the intervention on the coronary arteries. The NFH was administered before the start of coronary angioplasty as an intraarterial bolus (5000 U) and during the whole intervention the ACT was kept within the limits of 200-300 sec. After the intervention the patients received subcutaneously average doses of low-molecular heparin: enoxaparine sodium — «Klexan» ("Sanofi-Aventis", France) or nadroparin calcium — «Fraxiparine» (Glaxo Wellcome Production", USA) for 24 hours; after that the patients received the recommendations to continue oral Aspirin (100 mg) and Clopidogrel (75 mg) daily for 12 months.

Statistical analysis of variance was performed with the use of STATGRAPHICS software (Statistical Graphics System; Version: 2.6; Serial Number: 710240) on IBM/AT. The significance of difference of the studied parameters was assessed using Student test. $P<0,05$ was considered as statistically significant.

Results and discussion

Comparative characteristics of hospital mortality associated with conservative and invasive treatment of MI in elderly and old patients are presented in Table 1.

Of 530 patients receiving conservative therapy, 115 (21,7%) died during in-hospital stay. Herewith the mortality among elderly patients was 19,9%, and among old patients– 23,4%. Primary MI was associated with 15,4% mortality, repeated MI — with 36% mortality.

In the group of patients (n=126), who underwent coronary angioplasty with stenting, the mortality rate was 1,6% (2 patients). Both patients were elderly (1,7%), there were no deaths in the old age group. There were 10 (7,9%) complications related to endovascular intervention on the coronary arteries. Intraoperative MI developed in 1,6% of cases, bleeding from the access artery — in 6,5%, including 2 cases (1,6%) with mild anemia not requiring replacement transfusion.

Conclusions

1. Emergency invasive tactics of the treatment of myocardial infarction, contributing to the restoration

Table 1.

Comparative characteristics of hospital mortality associated with conservative and invasive treatment of myocardial infarction in elderly and old patients

Patients' age (years)	Number of patients (abs.)	Mortality distribution by the forms of MI Abs. (%)				Mortality in age groups	
		nSTEMI primary	nSTEMI repeated	STEMI promary	STEMI repeated	abs.	%
CONSERVATIVE THERAPY							
60 – 74	256	8 (3,1)	23 (9)	15 (5,8)	5 (1,9)	51	19,9
75 – 89	272	9 (3,3)	15 (5,5)	25 (9,2)	15 (5,5)	64	23,4
90 >	2	—	—	—	—	—	
CORONARY ANGIOPLASTY WITH STENTING							
60 – 74	115	—	—	—	2 (1,7)	2	1,7
75 – 89	11	—	—	—	—	—	—

of the infarct-related coronary artery, is the method of choice for high-risk patients, including the majority of elderly and old patients. The mortality associated with percutaneous coronary interventions is 13,5 fold lower than with conservative therapy (p<0,001).

2. The expansion of the network of the departments of endovascular diagnostics and treatment will allow to significantly decrease mortality from myocardial infarction in the population with this pathology in whole, including among elderly and old patients.

References

1. J.M. Guralnik, S.C. Fitzsimmons. Aging in America: a demographic perspective. Cardiol. Clin., 1986, 4, 175.

2. Himbert D., Juliard J.M., Steg P.G. et al. Acute myocardial infarction in patients over 70 years of age. Ann. Cardiol. Angeiol,. 1994, 43(2), 97-100.

3. M. Kotamski, T.E. Strandberg, M.S. Nieminen. Treatment of elderly patients with acute myocardial infarction. Eur. Heart J., 2000, 21, 362.

4. Manual on Gerontology. Ed. D.F. Chebotarev, N.V. Mankovsky, V.V. Frolkis. Moscow, Meditzina, 1978, 411-2.

5. Nomenclature and Criteria for Diagnosis of Ischemic Heart Disease. Report of the Joint Internacional Society and Federation of Cardiology/World Organization Tack Force on Standartization of Clinical Nomenclature. Circulation, 1979, 59, 607-9.

6. Russian Scientific Society of Cardiology. «Diagnostics and treatment of ST-elevation acute myocardial infarction». Russian guidelines. Cardiovasc. Terap. Profilakt., 2007, 8, 66 p. — Supplement

7. Russian Scientific Society of Cardiology. «Treatment of acute coronary syndrome without persistent ST-elevation on ECG». Russian guidelines. Moscow, 2006, 32.

8. ACC/AHA 2007 Guidelines for the Management of Patients With Unstable Angina/Non–ST-Elevation Myocardial Infarction. A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines for the Management of Patients With Unstable Angina/Non–ST-Elevation Myocardial Infarction). J. Amer. Coll. Cardiol., 2007, 50, 3–157.

9. ACC/AHA Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction. Circulation, 2004, 110, 5-292.

Coronary Stenting using XIENCE V DES: General Problems, Perspectives (a Review)

M. B. Matini[1], R.V. Zeynalov, O.E. Sukhorukov, V.V. Mazurova, O.V. Zakharova, I.A. Kovalchuk, A.G. Koledinsky.
Moscow City Center of Interventional Cardioangiology, Moscow, Russia

Endovascular methods of diagnostics and treatment have emerged already in mid-20th century, but only by the end of this century they rose to prominence in modern cardioangiology. With the advent of interventional methods of myocardial revascularization the possibilities of treatment of different forms of coronary artery disease were significantly extended (1). The development and the perfection of endovascular techniques allowed to replace one method of treatment by another due to the extension of indications for non-surgical treatment. From 1991 through 2001, the amount of interventional procedures for coronary artery disease in the USA has increased by 7 times, while of direct surgical interventions — by only 1,5 times(2).

Andreas R. Gruentzig can be named pioneer of angioplasty. In 1974 he was the first to invent a polymer balloon catheter with a fixed inflated diameter, and already by mid-1977 he has performed to first successful transluminal balloon angioplasty of a human coronary artery (3).

With the accumulation of experience, during the last decades, certain disadvantages of balloon angioplasty have been revealed. These disadvantages significantly influence the clinical course of the underlying disease.

In the last decade of the 20th century the leading place among all endovascular interventions on the coronary arteries went to coronary stenting.

The first implantation of an intracoronary stent in man was described by Sigwart et al. in 1987 (4). The use of high pressure during stent implantation in combination with antithrombotic pharmacological support has contributed to adequate blood flow restoration in the coronary arteries. Herewith the procedure of stenting was associated with low complication rate. Initially stenting was applied in cases with threatening coronary artery occlusion during transluminal balloon angioplasty (5). According to Garas S.M. et al. (6), the use of intracoronary stenting significantly decreased the rate of restenosis — from 50% to 20-30% — in comparison with balloon angioplasty. However it did not completely solve the problem of restenosis. Stent insertion has provided a high rate of immediate success and has allowed to avoid some serious complications proper to PTCA: marked dissections, acute coronary occlusions (7, 8, 9, 10). But the main advantage of stenting in comparison with balloon angioplasty consisted in a significant reduction of the rate of restenosis — recurrent narrowing of the lumen of a vessel previously subjected to angioplasty (11, 12). The reduction of restenosis rate by 10-15% after stenting as compared with balloon angioplasty was firstly proved in 1993-1994 in the STRESS (13) and BENESTENT (7) trials, that investigated the results of coronary stenting and balloon angioplasty. The trials have demonstrated an improvement of the results after stenting in comparison with balloon angioplasty. The authors concluded that after the procedure the diameter of a stented vessel was increased in a far greater degree than after PTCA, and in the long-term the stented coronary arteries preserved a bigger internal lumen; herewith the extension of their angiographic restenosis was decreased (31,6% vs. 42,1%, p=0,046 in the work of Fishman D., et al., and 26% p=0,02 — in the work of Serruys P. et al) (7, 13, 14).

Similar results were obtained by Rodriguez A. et al. (15), Versacci F. et al. (16) and Antoniocci D. et al. (17). The authors have demonstrated a decrease of the rate of late restenosis in the stented group, which, in its turn has contributed to a significant reduction of the need of repeated endovascular interventions (15, 16, 17).

Today coronary stenting is the most frequently used method of heart revascularization. However the damage of vascular endothelium and subsequent hyperplasia of the neointima developing mainly within the first 6 months after stent insertion often causes in-stent restenosis (18,19). According to most authors, a restenosis is considered hemodynamically significant if the vessel lumen in the site of dilatation is reduced by > 50% of the reference diameter or 75% of its surface area (20, 21, 22, 23).

In 1999, late in-stent stenosis was revealed in over 250.000 stented patients. For this reason worldwide trials are being conducted with the aim to study the causes of in-stent stenosis, the possibilities of its prevention and the development of an optimal tactics of treatment for the improvement of clinical prognosis (24, 25). The mechanism of restenosis is well known: it occurs due to a multitude of factors, such elastic recoil of the vessels, thrombosis, neointimal hyperplasia and negative remodeling of the vessels (26). Elastic recoil is caused to natural elastic properties of

[1] Address for correspondence:
Dr. Mohammad Matini,
Russia, 101000, Moscow, Sverchkov per., 5
Moscow City Center of Interventional Cardioangiology
Tel . 007 (495) 624 96 36
Fax 007 (495) 624 67 33
e-mail: zoliz@mail.ru
Manuscript received on June 20, 2011.
Accepted for publication of September 15, 2001

the blood vessels, manifested as a response to dilatation. It occurs immediately after percutaneous transluminal coronary angioplasty (PTCA). Thrombosis is a consequence of endothelial damage, rupture of the intima and damage of the middle layer of a vessel. These damages lead to the accumulation of platelets and the formation of a thrombus. The accumulated platelets represent a source of attractants and mitogens for smooth muscle cells (SMC). Besides, platelet-derived growth factor (PDGF), released by endothelial cells and macrophages, is considered as the main factor contributing to SMC migration. Inflammation plays an important role in restenosis, as leucocytes are found in the site of vascular damage early and in great amount (26). According to Hofma H. S., et al. (27), inflammation process plays a more important role in the healing of arterial wall after stenting, which can be explained by the presence of a foreign body (stent) in the arterial lumen (28). An important role is also played by neointimal hyperplasia. In the involved vessel, the SMC enter into the proliferation phase and move to the intima through the damaged internal elastic membrane. An important place in this process belongs to metalloproteinases. They continue to divide and to synthesize the extracellular matrix (ECM), which, In the end, forms the volume of restenotic lesion. The components of ECM — hyaluronan, fibronectin, osteopontin and vitronectin — also contribute to SMC migration. Moreover, the reorganization of ECM, as well as its replacement with collagen can lead to the contraction of the vascular wall. The adventitial myofibroblasts, playing an important role in intima supply with proliferative cell elements in restenosis, also divide and migrate in the neointima. The adventitial cells also participate in the process of vessel remodeling, as myofibroblasts are able to synthesize collagen and lead to tissue contraction (26). Rogers and Edelman (29), Edelman and Rogers (30) have demonstrated that the damage and, as a consequence, the interruption of the integrity of the internal elastic membrane is of key importance for intimal hyperplasia during the development of in-stent stenosis. During stent implantation, after primary balloon-induced injury of the vascular wall with the rupture of internal elastic membrane, one assists at an intensive proliferation of the intima. Its degree is proportional to the depth of stent insertion. Thus, it has been shown, that the hyperplasia of the intimal layer represents a response to an external impact (lumen dilatation with an inflated balloon and stent insertion) and is proportional to the degree of mechanical damage of the vascular wall's structures (29, 30). The feasibility of using stents as a reinforcing device became evident after experimental and clinical confirmation of an important role of early elastic recoil and negative remodeling of the vessels in the development of restenosis (31). Intravascular ultrasound (IVUS) allowed to understand that the late lumen loss and restenosis after PTCA are due not so much to intimal hyperplasia, as to negative remodeling of the vessel. Catala (32) has experimentally shown that the design and the material of stent produce a significant on the probability of restenosis development. An ideal stent design should maximally limit smoot muscle cells migration to the surface of the intima (32). Similarly, Topaz and Vetrovek (28) came to the conclusion that more dense, uniform reinforcement of the arterial lumen contributes to more effective prevention of neointimal growth.

Today two different tactics are being used for the fight against restenosis — it can be treated or prevented. The therapeutic tactics is based on the use of mechanical methods, while the prevention of in-stent stenosis formation is effectuated through the use of pharmacological agents and new technologies of stenting (including the use of new stents with antiproliferative properties) (33).

A drug-eluting stent (DES) usually contains a metal base, a polymer layer with a drug absorbed into or mixed with this layer, sometimes — a protective polymer layer preventing early drug washing out, and the drug, as such.

The pharmacological agent must be able to inhibit maximal amount of different components participating in complex process of restenosis (34, 35). All know anti-inflammatory and histochemical agents, immunomodulators, some antibiotics, as well as the medications used in oncology for the decrease of the intensity of cellular division in tumors, have been tested (36). The biggest effect was obtained with only two agents — Rapamycin (trade mark — Sirolimus) and Paclitaxel (trade mark — Taxol) (37, 38).

By its chemical composition, Rapamycin belongs to natural macrocyclic lactons and is a waste product of Streptomyces hydroscopicus bacteria. By its pharmacological properties Rapanycin is a cytostatic agent — immunosuppressor (39). Its action is based on the binding of cytozolic receptor FRBP 12, blocking of TOR enzyme, regulation of p27 level and deceleration of retinoblastoma protein phosphoration with the block of cell cycle development at G1-S transition (39). In vitro studies have demonstrated that Rapamycin can suppress the division and the migration of the SMC, and experimental models have proved its capacity to reduce neointima formation in the area of vascular wall damage (40, 41, 42).

Paclitaxel is an alkaloid derived from Taxus brevifolia, a well-known antitumoral agent producing a powerful antiproliferative effect. The combination of Paclitaxel and tubulin leads to cell blocking and division in the phases G0/G1 G2/M of the cellular cycle (43, 44). In vitro and animal studies have demonstrated the capacity of Paclitaxel to decelerate SMC division and migration, to prevent neointima formation after catheter angioplasty (45, 46). An active substance-containing polymer layer should possess an absolute biocompatibility, perform mechanical functions as well as provide necessary drug concentration. It means, that besides being non-toxic,

it should follow the changes of geometrical configuration during stent deployment and be resistant to mechanical influences caused by balloon inflation. Besides, local drug release must be maximally effective. The speed of drug release and its concentration in the due site should be predictable and controllable (41, 47). The first drug-eluting stents (DES) had been coated with a cytostatic agent only. Currently used DES possess a new important component — an additional coating made of biocompatible polymer. In the absence of polymer up to 40% of the drug is lost through the mechanical and chemical processes before stent implantation, and complete wash-out of the drug occurs before the occurrence of the expected restenosis; for this reason controlled endothelization does not occur and the rate of early thromboses increases (48). Biodegradable polymers are the most recent innovations in this field. Contacting with the biological environment of the living organism, these polymers can dissolve without changing their molecular mass, or get biodestructed under the impact of the following mechanisms: hydrolysis with the formation of oligomeric and monomeric products, enzymatic hydrolysis and cytophagous destruction (organism's protective cellular response) (49, 50). In first trials the stents with polymer coating did not only decrease proliferation, but even enhanced it due to their toxicity (51). Newer phosphoryl choline-based polymer coatings have demonstrated better biocompatibility and reduced the rate of DES thromboses (52), which allowed to consider these coatings as a transport mean for local delivery of drugs aimed to dosed release into the stented vascular wall area (53). Due to polymer coating, the drug is released in

Table 1.

Comparative charateritics of drug-eluting stents

Manufacturer	Boston Scientific	Cordis	Abbot	Biosensors	Medtronic
Article	Taxus Liberte	Cypher Select	Xience V.	BioMatrix	Endeavor Resolute
Platform	Taxus Express	Bx Sonic	Multi Link Vision	S-stent	Driver
Stent material	Stainless steel	Stainless steel	Cobalt-chromium	Stainless steel	Cobalt-chromium
Primary coating	No	Parylene c	No	Parylene c	Нет
Third layer	No	Polybutyl methacrylate	No	No	Polivinil pirrolidinone
Polymer	Polyvinylidine fluoride hexa fluropropylyene (PVDF-HFP)	Poly ethylene co-vinyl Acetate	Polyvinylidine fluoride hexa fluropropylyene (PVDF-HFP)	Polylactic acid (PLA)	Biolinx three-layer coating: upper layer — hydrophilic polivinil pirrolidinone, middle layer– hydrophobic hexil methacrylate, hedrophilic venyl pirrolidinone and venyl acetate- medicinal diffuse barrier; hydrophobic butyl methacrylate- drug carrier
Dissolving polymer, term	No	No	No	Yes, up to 6-9 months	No
Hydrophile coating	No	No	No	Yes	Yes/no
Type of coating	Coated from all sides (including the surface facing the arterial lumen)	Coated from all sides (including the surface facing the arterial lumen)	Coated from all sides (including the surface facing the arterial lumen)	Applied at the surface facing the arterial wall	Coated from all sides (including the surface facing the arterial lumen)
Agent	Paclitaxel	Sirolimus	Everolimus	Biolimus A9	Zotarolimus ABT-578
Dose, μg/mm^2	1	140	100	15,6	10
Thickness of coating, μm	17,8	13,7	7,8	11	4,8

a homogenously dosed concentration and during a pre-determined period of time corresponding to the phases of vessel's healing (41). The system of drug delivery preserves its properties after sterilization, is able to change its geometrical form and volume following stent implantation and deployment and is resistant to mechanical influence caused by balloon inflation (52). The first prospective double blind multi-center trial (RAVEL) compared the results of angioplasty with Sirolimus-eluting stents and with bare metal stents (Bx Velocity) in 238 randomized patients with primary coronary lesions. At six months the rate of restenoses in the Sirolimus group was 0% vs. 26,6% in the control group. According to angiography data, late decrease of arterial lumen (so-called late lumen loss), as well as the number of MACE in the Sirolimus group were significantly lower (54). SIRIUS trial was the largest-scale study of Sirolimus-eluting stents. It was conducted in 53 centers throughout the USA and comprised 1101 randomly selected patients with primary coronary lesions. The patients received Sirolimus-eluting stents and bare metal stents. Final results of the trial have demonstrated a significant decrease of the rate of restenoses, late lumen loss and necessity of repeated revascularization in the Sirolimus group (55). Primary results of the use of Sirolimus-eluting stents for the treatment of in-stent stenosis are equally encouraging. A non-randomized study has shown the rate of restenosis below 10%. In comparison with the standard coronary stent, a Sirolimus-eluting stent offers better perspectives for the prevention of proliferation of the neointima, of restenosis and restenosis-related unfavorable clinical events (26). At present there are many various drug-eluting stents. Antiproliferative agents used in most available stents are Rapamycin and Paclitaxel. All stents without distinction are balloon-deployable, processed with polymer coating containing a cytostatic agent. The analogues of Rapamycin available at the present time include: Everolimus (stent Xience V, Abbot), Zotarolimus (stent Endeavor, Medtronic), Sirolimus (stent Cypher select, Cordis), Biolimus-A9 (stent Biomatrix, Biosensors). These agents are used in the coatings of second-generation stents. Comparative characteristics of the most widely used DES are presented in Table 1.

While the use of stents for PCI can be compared to a breakthrough resulting in a significant decrease of the rate of restenosis (20), the advent of stents with drug-eluting coating became a true revolution in the treatment of the coronary artery disease (56).

The stent Xience V (Abbott Vascular, USA), is one of the best second-generation stents. The safety and the effectiveness of Xience V have been statistically confirmed in numerous clinical trials comparing this stent with bare metal stents as well as with other second-generation stents.

Here is a brief summary of these trials

1. SPIRIT I. A 6-months (5 years) follow-up of 60 patients in comparison with a bare metal stent (Multi Link Vision) revealed that the rate of MACE, including death, myocardial infarction, emergency and elective CABG was 7,14% for Xience V, and 18,75% for Multi Link Vision (57, 58, 59).

2. SPIRIT II. The data of a clinical trial conducted in a group of 300 patients and comparing the Xience V stent with Paclitaxel-eluting stent Taxus Express2 / Taxus Liberte, have been presented by P. Serruys at 58th Annual session of the American College of Cardiology (ACC-2009). The rate of MACE at 6 months was 2,7% for Xience V vs. 6,5% for Taxus; at 2 years– 6,6% and 11,0%, respectively. (60).

3. SPIRIT III. (the results have been presented at PCR-2008 in Barcelona, Spain, on May 13, 2008): In a group of 1002 patients the rate of MACE decreased at 2 years by 45% in comparison with Taxus stent (6,0% for Xience V vs. 10,3% for Taxus) (61, 62, 63).

4. Meta-analysis of the data of SPIRIT II + SPIRIT III trials has shown the following key results for Xience V stent at 2 years follow-up: clinically significant decrease of the risk of ischemia, caused by target vessel failure by 31% in comparison with Taxus stent (10,4 % for Xience V vs.14,7% for Taxus). The risk of death from all causes decreased by 28% in comparison with Taxus stent (2,4% for Xience V vs.3,3% for Taxus). The risk of cardiac death decreased by 28% in comparison with Taxus stent (0,9% for Xience V vs. 1,3% for Taxus). Besides, the authors have noted clinically significant decrease of the risk of myocardial infarction (MI) by 45% in comparison with Taxus (3,1% for Xience V vs. 5,6% for Taxus); clinically significant decrease of the risk of ischemia caused by target lesion revascularization (TLR) by 1% in comparison with TAXUS (4,1% for Xience V vs. 6,8% for Taxus). Also a low rate of stent thrombosis between the years 1 and 2 was noted (0,5% for Xience V and 0,8% for Taxus) (64).

5. SPIRIT IV. In September 2009, at TCT held in San-Francisco, G. W. Stone has presented the results of SPIRIT IV trial comprising 3690 patients, including 1185 patients with diabetes mellitus (32.2%). According to this study, the rate of MACE decreased by 39% in comparison with Taxus stent (4,2% for Xience V vs. 6,9% for Taxus). At 1 year there was a significant reduction of the target lesion failure in comparison with Taxus Express (Xience V 4.2% vs. Taxus 6.8%). Besides, with the use of Taxus the rate of ischemia-driven target lesion revascularizтion (ID-TLR), was higher than with the use of Xience V, with relative risk decrease by 46% (2,5% for Xience V vs. 4,6% for Taxus). Also the number of cases of cardiac death or target-vessel revascularization decreased by 31% in comparison with Taxus stent TAXUS (2,2% for XIENCE V vs.3,2% for TAXUS). The number of myocardial infarctions related to the target vessel decreased by 38% in comparison with Taxus (1,8% for Xience V vs. 2,9% for Taxus). The number of stent thromboses decreased by 80% in comparison with Taxus stent (0,17% for Xience V vs. 0,85% for Taxus); with the

use of Xience V the rate of thromboses in patients with diabetes mellitus decreased by 40% in comparison with Taxus Express stent (0.8% vs.1.33%), and in non-diabetic patients — by 94% (0.06% vs.1%). A decrease of Target Lesion Failure (TLF) was also noted in patients with small vessels — by 43% (3.9% vs. 6.8%), in patients with extended stenosis — by 35% (4.5% vs. 6.9%), and in patients with two and more involved coronary arteries — by 49% (5.1% vs. 10%) (65, 66).

6. Clinical trial Compare: on January 8, 2010 the on-line resource Lancet published the results of a large-scale clinical trial. P. C. Smits from Maasstad Ziekenhuis hospital (Rotterdam, the Netherlands) presented the results of comparison of Xience V and Taxus stents. Over 1800 complex cases have been randomized for this study in 1:1 ratio (60% — with acute coronary syndrome, 73% — with type B2/C lesions in the Taxus Liberte group and 74% — with type B2/C lesions in the Xience V group). The following data are of utmost interest: at 1 year the use of Xience V stent led to a significant reduction of MACE in comparison with Taxus Liberte (6,2 % for Xience V vs. 9,1 % for Taxus Liberte) — a decrease by 31%, as well as to the decrease of the rate of stent thrombosis (0,7 % for Xience V vs. 2,6 % for Taxus Liberte) — a decrease by 74% (67).

7. Clinical trial Lesson I, aimed at the evaluation of comparative effectiveness of Sirolimus-eluting stents Cypher (Cordis) and Everolimus-eluting stents Xience (Abbot). The authors have analyzed the data of 1532 patients who got Cypher stent during the period from 2004 to 2006, and of 1601 patients who received Xience stents from 2006 to 2009. They have identified 1342 pairs comparable by their baseline clinical parameters. Clinical outcomes were followed for up to 3 years. The results suggested a statistically reliable decrease of the number of MI and target vessel revascularization in the group of Xience (14,9% for Xience vs. 18,0% for Cypher). The number of deaths was not statistically different (6% for Xience vs. 6,5% for Cypher). The group of Xience demonstrated a decrease of the rate of definite and eventual stent thromboses (2,5% for Xience vs.4,0% for Cypher) (67) .

8. The Leaders trials have revealed a tendency towards the improvement of MACE with the implantation of Biolumus A9 -eluting stents (BES) in comparison with Sirolimus-eluting stents (SES). Major adverse cardiac events (MACE) have been noted in 15.7% patients with BES and in 19.0% with SES. In the subgroups of patients with a history of MI, there was a significant decrease of MACE among the patients with BES (9.6% vs. 20.7%). Another group of patients with BES, which comprised patients with Syntax score over 16, demonstrated a significant (by 57%) decrease of deaths in comparison with a group patients with SES (4.6% vs. 10.4%) (68).

9. Essence-Diabetes trial has shown that Xience V is more effective than Cypher for the treatment of coronary lesions in diabetic patients. The trial comprised 280 patients with diabetes mellitus and angina or confirmed ischemia and coronary arteries stenosis > 70% (with reference diameter of the vessel > 2,5 mm and lesion's length > 25 мм). Patients with the lesions of the left main coronary artery and coronary grafts, with chronic renal or hepatic failure and with bifurcation lesions requiring stenting of the side branch were excluded from the study. The patients were divided into 2 equal groups. In 8 months after the intervention control coronary angiography has been performed in all patients.

Maximal lumen loss in the Xience V group was lower than in the Cypher group (0,23 mm vs. 0,37 mm; P=0,02). Lumen loss inside the stent was 0,04 mm in the Xience V group and 0,18 mm in the Cypher group (P=0,015). Segmental lumen loss was 0,11 mm in the Xience V group and 0,20 mm in the Cypher group (P=NS). Restenosis over 50% was noted in 4,7% of patients from the Cypher group and in none patient from the Xience V group (P=0,029). Repeated intervention has been performed in 2,0% of patients from the Xience V group and in 5,3% in the Cypher group (P=0,085). During the follow-up there was 1 case of eventual stent thrombosis in each group (The results were presented by Young Hak Kim at TCT-2010).

The complications of DES include intimal hemorrhages, incomplete healing, intimal inflammation and medial necrosis (63, 69). Aneurysms, false aneurysms, stent apposition, perforations, local vasculitis, thrombosis, accelerated progressing of atherosclerosis, fibrosis and systemic disturbances are potential complications associated with DES implantation (69). The IVUS-assisted studies of Colombo and Tobis (70) have demonstrated that many stents were under-deployed, which increased their blood-contacting surface area (70). These data suggested the necessity of more aggressive stent deployment during their implantation using high pressure (71). At the same time the studies of anticoagulant therapy have revealed that the combination of antiplatelet Aspirin agents with Ticlopidine, as well as the combination of Aspirin with Clopidogrel is several times more effective than Varfarin in the prevention of stent thrombosis (72). These two new practical approaches to stenting have significantly decreased the rates of stent thrombosis and bleeding.

On July 7, 2003, the company Cordis has appealed to interventionists with an advice to strictly follow the guidelines for the implantation of Cypher stents and for subsequent patients' care for the assurance of maximal safety of the procedure. And on October 29, 2003, FDA has announced an investigation of the causes of subacute thrombosis and related complications after Cypher implantation. As for 2005 (73), FDA did not recommend the use of Sirolimus-eluting stents in cases that were not definitely studied in randomized trials, up to the discovery of true causes of thrombosis (73). Antiproliferative agents do not stop the process of neointima formation, but just delay

it, and under their influence the process of endothelization does not end by 6 months, but continues further. Nevertheless this process leads to stepwise decrease of minimal lumen of the dilated segment of the artery. The growth of neointima does not stop even after 12 months (74). Karvouni et al. (73) have noted that by 15 months after Cypher (Sirolimus-eluting) implantation, the stent was not covered by the endothelium; at the same time BX Velocity (bare metal) stent was completely endothelized. In 2005, Waters (75) has presented 40 pathological cases after DES implantation: in 24 of these cases there was a thrombosis of the stented segment. It became evident, that the delay of endothelization after DES implantation prolong the time when the development of complications is possible. These complications include thrombosis — the most dangerous adverse event after DES implantation (76).

Long-term follow-up of 746 patients in whom Clopidogrel was stopped 6 months after stenting (Basket-Late trial) have been presented in March 2006, at the Annual session of ACC (77). The study revealed that within 1 year after the cessation of Clopidogrel intake the rate of complications in patients with DES was reliably higher than in those with BMS. Herewith in the group with DES there was a tendency towards lower need in repeated PCI for restenosis (78). Wenaweser at al. (77) have shown that an angiographically confirmed stent thrombosis was noted in 1,8% of cases. Almost one half of thromboses (41%) were late (on the average — in 453 days). The majority of late thromboses (59%) occurred more than one year after stenting (77).

One can suggest, that the obtained results, if they are not accidental and are not related to some particularities of the trial, favor more prolonged (over 6 months) administration of Clopidogrel after the implantation of DES. Eisenstein et al. (79) presented the results of their study of 4666 patients followed for 6, 12, and 24 months after stenting. In patients with bare metal stents prolonged Clopidogrel therapy had no impact on the rate of death and MI at 6-12 months after the procedure. On the contrary, in patients with DES the use of Clopidogrelfor 6, 12 and 24 months was associated with the decreased rate of mortality and MI in all time intervals.

At present we assist at the introduction of new techniques of stenting, including simultaneous use of DES and BMS, the so-called “hybrid” stenting. A group of Italian researchers headed by Varani has followed 2898 patients with coronary artery disease who underwent multiple coronary stenting, from July 2003 through December 2006 (81). BMS have been implanted in 1315 of these patients, DES — in 657, and 926 patients had “hybrid” stenting. Long-term results (2 years) suggest the absence of any significant difference between the outcomes with the use of DES and “hybrid” stenting. It has been shown that isolated use of DES is more cost-effective in comparison with the use of only BMS.

Bertand et al. (82) have compared two groups of patients: the patients from group 1 (n=161) received only DES, while the patients from group 2 (n=201) — a combination of DES and BMS. Long-term results of “hybrid” stenting were found to be comparable with the results of DES implantation. The authors have confirmed the effectiveness of isolated use of DES in coronary lesions with high risk of restenosis.

Conclusion: Sirolimus-eluting stents marked the advent of a new generation of intravascular devices. They have been followed by other stents with similar and essentially different types of coating. Their apparition can be considered as a logical evolutionary step in invasive cardiology, while the decrease of the risk of restenosis can mean the beginning of a new era in revascularization. Certainly, significant decrease of the rate of restenosis and TLR with the use of DES (in comparison with BMS) has been proved by multiple trials, and constitutes the main advantages of these stents. A representative of second generation of DES, Everolimus-eluting stent Xience V, is superior to Sirolimus-eluting, as well as to Paclitaxel-eluting stents. Today we assist at the development and successfully introduction of a third-generation DES — with PLA-based biodegradable coating, that has been shown to be safe and effective in early, as well as in long-term follow-up (68). However, the true innovation in this field was the advent of new stents, implanted into the thrombotic vessels. After the implantation they dissolve within about 2 years leaving the vessel patent in the absence of a permanent metallic implant (80).

References

1. Petrosian Yu.S., Zingerman L.S. Coronarography. Moscow, Meditzina, 1974. 150 p.

2. Rudd J.H.F. , Davies J. R., Weissberg P.L. Atherosclerotic Biology and Epidemiology of Disease. In Topol E.J. Textbook of cardiovascular medicine. Lippincott Williams & Wilkins, Philadelphia, 2002.

3. Gruentzig A. Transluminal dilatation of coronary-artery stenosis. Lancet, 1978, 1, 263.

4. Sigwart U., Puel J., Mirkovitch V., et al. Intravascular stents to prevent occlusion and restenosis after transluminal angioplasty. N. Engl. J. Med., 1987, 326, 701-6.

5. Gianturco C, Wright KC, Wallace S, et al. Percutaneous endo-vascular stents: An experimental evaluation. Radiology, 1985, 156 (1), 69-72 .

6. Garas SM, Huber P, Scott NA. Overview of therapies for prevention of restenosis after coronary interventions. Pharmacol. Ther., 2001, 92(2-3), 165-78.

7. Serruys P.W., de Jaegere P., Kiemeneij F., et al. A comparison of balloon-expandable stent implantation with balloon angioplasty in patients with coronary artery disease: BENESTENT study group. N. Engl. J. Med., 1994, 331, 489–509.

8. Serruys P.W., Van Hout B., Bonnier H. et al. Effectiveness, costs and cost-effectiveness of a strategy of elective heparin-coated stenting compared with balloon angioplasty in selected patients with coronary artery disease: the BENESTENT II Study. Lancet, 1998, 352, 673.

9. Zakharova O.V., Arablinsky A.V., Iosseliani D.G. Endovascular methods of treatment of CAD patients with restenosis after primary balloon angioplasty. Klinicheskaya meditzina, 2004, 82(10), 22-6.

10. Babunashvili A.M., Ivanov V.A. Use of coronary stenting depending on clinical presentation of IHD and the type of atherosclerotic lesion of the coronary arteries. Moscow, 2000, 77 p.

11. ACC/AHA Percutaneous Coronary Intervention in United States. J. Am. Coll. Cardiol., 2002, 23, 2, 1881-2137.

12. Serruys P.W. ARTS I — the rapamycin eluting stent; ARTS II — the rosy prophecy. Eur. Heart J., 2002, 23, 757.

13. Fishman D.L., Leon M.B., Baim D.S., et al. A randomized comparison of coronary-stent placement and balloon angioplasty in treatment of coronary artery disease. Stent Restenosis Study Investigators. N. Engl. J. Med., 1995, 332 (8), 536;

14. Serruys PW, Sousa E, Belardi J et al. BENESTENT-n Trial: subgroup analysis of patients assigned either to angiographic and clinical follow-up or clinical follow-up alone. Circulation, 1997, 96, 653.

15. Rodrigues A., Ayala F., Bernardi V., et al. Optimal coronary balloon angioplasty with provisional stenting versus primary stent (OCBAS). J. Am. Coll. Cardiol., 1998, 32, 1351-7.

16. Versaci F., Gaspardone A., Tomai F., et al. A comparison of coronary artery stenting with angioplasty for isolated stenosis of the proximal left anterior descending coronary artery. N. Engl. J. Med., 1997, 12, 817.

17. Antoniucci D., Santoro G., Bolognese L., et al. A clinical trial comparing primary stenting of the infarct-related artery with optimal primary angioplasty for acute myocardial infarction (FRESCO). J. Am. Coll. Cardiol., 1998, 31,1234-9.

18. Iosseliani D.G., Arablinsky A.V., Chernysheva I.E. Immediate results of direct stenting of the coronary arteries. Kardiologia, 2002, 42 (2), 8-10.

19. Hoffmann R., Mintz G.S., Mehran R., et al. Tissue proliferation within and surrounding Palma-Schatz stents in dipendent on the aggressiveness of stent implantation technique. Am. J. Cardiol., 1999, 83, 1170-4.

20. Block P., Petch M.C., Letouzey J.P. Manpower in cardiology in Europe. Eur. Heart J., 2000, 21, 14, 1135-40.

21. Gensini GG. Coronary arteriography: role in myocardial revascularization. Postgrad Med., 1978, 63(1), 121-8, 133-8

22. Sawada Y., Nokasa H., Kimura T., et al. Initial and six months outcome of Palmaz-Schatz stent implantation: STRESS/BENESTENT equivalent vs non- equivalent lesions. J. Am. Coll. Cardiol., 1996, 27 (suppl A), 252A.

23. Varnauskas E. Twelve-year follow-up of survival in the randomized European Coronary Surgery Study. N. Engl. J. Med., 1988, 319, 332-7.

24. Grossman W., Baim D. Coronary angiography. In: Cardiac Catheterization, Angiography and Intervention. Eds Grossman W, Baim D. Lea&Febiger, 1991: 185-214.

25. Sharma SK, Karakala V, Dangas G, D. et al. In-stent restenosis after Palmaz-Schatz stent implantation. Eur. Heart J., 1998, 20, 352.

26. Lee C.H., Serruys P.W, An overview of drug-eluting stent. International Journal of interventional Cardioangiology, 2003, 1, 9

27. Hofma H.S., Whelan D.M. van Beusekom HM. et al. Increasing arterial wall injury after long-term implantation of two types of stents in a porcine coronary model. Eur. Heart J., 1998, 19, 601.

28. Topaz O., Vetrovek G. The stenotic stent: mechanism and revascularization options. Cathet. Cardiovasc. Diagn., 1996, 37,293-9.

29. Rogers C., Edelman E.R. Endovascular stent design dictates experimental restenosis and thrombosis. Circulation, 1995, 91, 2995-3001.

30. Edelman E.R.., Rogers C. Hoop Dreams. Stent without restenosis. Circulation, 1996, 94(6), 1199-202.

31. Sousa J.E., Costa M.A., Abizaid A.C., et al. Sustained suppression of neointimal proliferation by sirolimus-eluting stents: one-year angiographic and intravascular ultrasound follow-up. Circulation, 2001, 104, 2007.

32. Catala L. Is stent design affecting the biologic response? In: «Endovascular Therapy Daily», at the «Eight complex coronary angioplasty», Paris, May 23-28, 1997, p.2.

33. Dangas G., Mehran R., Lansky A.J., et al. Acute and long-term results of treatment of diffuse in-stent restenosis in aortocoronary saphenous vein grafts. Am. J. Cardiol., 2000, 86, 777.

34. Mohacsi P.J., Tuller D., Hulliger B., et al. Different inhibitory effects of immunosuppressive drugs on human and rat aortic smooth muscle and endothelial cell proliferation stimulated by platelet-derived growth factor or endothelial cell growth factor. J. Heart Lung Transplant., 1097, 16, 484-92.

35. Suzuki T., Kopia G., Hayashi S., et al. Stent-based delivery of sirolimus reduces neointimal formation in a porcine coronary model. Circulation, 2001, 104, 1188-93.

36. Van Giesen W.J., Lincoff M., Schwartz R.S., et al. Marked inflammatory sequelae to implantation of biodegradable and nonbiodegradable polymers in porcine coronary arteries. Circulation, 1996, 94, 1690–7.

37. Poon M., Marx S.O., Gallo R., et al. Rapamycin inhibitis vascular smooth muscle cell migration. J. Clin. Invest,. 1996, 98, 2277-83.

38. Heldman A.W., Cheng L., Jenkins G.M.. et al. Paclitaxel stent coating inhibitis neointimal hyperplasia at 4 weeks in a porcine model of coronary restenosis. Circulation, 2001, 103, 2289-95.

39. Beyar R., Roguin A. The sirolimus-coated stent: will the Achilles heel of interventional cardiology finally be cured? Eur. Heart J., 2001, 22, 2054-7.

40. Bruke S.E., Labbers N.L., Chen Y.W. et al. Neointimal formation after balloon-induced vascular injury in Yucatan minipigs is reduced by oral rapamycin. J. Cardiovasc. Pharm., 1999, 33, 829-35.

41. Hwang C.W., Wu D., Edelman E.R. Physiological transport forces govern drug distribution for stent-based delivery. Circulation, 2001,104, 600-5.

42. Wahl A.F., Donaldson K.L, Fairchild C, et al. Loss of normal P53 function confers sensitization to Taxol by increasing G2/m arrest and apoptosis. Nat. Med., 1996, 2, 72-9.

43. Donaldson K.L., Goolsby G.L., Kiener P.A., Wahl A.F. Activation of p34cdc2 coincident with taxol-induced apoptosis. Cell Growth Differ., 1994, 5, 1041-50.

44. Heldman A.W., Cheng L., Jenkis G.M., et al. Paclitaxel stent coating inhibits neointimal hyperplasia at 4 weeks in a porcine model of coronary restenosis. Circulation, 2001,103, 2289-95.

45. Farb A., Heller P.F., Shroff S., et al. Pathological analysis of local delivery of paclitaxel via a polimer-coated stent. Circulation, 2001,104, 473-9.

46. Whelan D.M., van Beusekom H.M., van Giessen W.J. Mechanisms of drug loading and release kinetics. Semin. Interv. Cardiol., 1998, 3, 127-31.

47. Heldman G.P. Internal testing of DES. Circulation, 2001, 103, 2289-95.

48. Grenadier E., Roguin A., Hertz I., et al. Stenting very small coronary narrowings (< 2 mm) using the biocompatible phosphoryl-choline-coated coronary stent. Catheter Cardiovasc. Interv., 2002, 55, 303-8.

49. Seldinger S.I. Catheter replacement of the needle in percutaneous arteriography: a new technique. Acta Radiol., 1953. 39, 368.

50. Volova T.G., Sevastianov V.I., Shishatskaya. Polyhydrooxyalkoanates — biodegradable polymers for medicine. Krasnoyarsk, Platina, 2006, 287 p.

51. Hofma S.H., Beusekom H.M., Serruys P.W., Giessen W.J. Recent developments in coated stents. Curr. Intervent. Cardiol. Rep., 2001, 3, 28–36.

52. Malik N., Gunn J., Shepherd L., et al. Phosphorylcholine-coated stents in porcine coronary arteries: in vivo assessment of biocompatibility. J. Invasive Cardiol., 2001, 13, 193-201.

53. Whelan D.M., Van Giessen W.J., Krabbendam S.C., et al. Biocompatibilrty of phosphorylcholine coated stents in normal porcine coronary arteries. Heart, 2000, 83, 338-45.

54. Regar E., Serruys P.W., Bode S., et al. Angiographic findings of the multicenter randomized study with the sirolimus-eluting Bx velocity balloon-expandable stent (RAVEL): sirolimus-eluting stents inhibit restenosis irrespective of the vessel size. Circulation, 2002, 90, 937-41.

55. Moses J.W., O'Schaughnessy C., Caputo R., et al. The U.S. multicenter, randomized, double-blind study of the sirolimus-eluting stent in coronary lesions: safety outcomes at 9 months. Eur. Heart J., 2002;23:264.

56. Serruys P.W., Ong A.T., Piek J.J., et al. A randomized comparison of a durable polymer Everolimus-eluting stent with a bare metal coronary stent: The SPIRIT first trial.. EuroIntervention, 2005, 1, 58-65.

57. Garg S., Serruys P., Onuma Y., et al. 3-Year Clinical Follow-Up of the XIENCE V Everolimus-Eluting Coronary Stent System in the Treatment of Patients With De Novo Coronary Artery Lesions The SPIRIT II Trial. J.Amer.Coll. Cardiol., 2009, 2, (12), 1190-8.

58. Piek J.J., Neumann F.J., Giessen W.J., et al. One-year results of a durable polymer everolimus-eluting stent in de novo coronary narrowings (The SPIRIT FIRST Trial).. EuroIntervention 2005, 1, 266-72.

59. Tsuchida K., Garcia-Garcia H.M., Ong A.T., et al. Revisiting late loss and neointimal volumetric measurements in a drug-eluting stent trial: analysis from the SPIRIT FIRST trial. Catheter Cardiovasc. Interv., 2006, 67,188-97.

60. Onuma Y., Tanimoto S., Ruygrok P., et al. Efficacy of everolimus eluting stent implantation in patients with calcified coronary culprit lesions: two-year angiographic and three-year clinical results from the SPIRIT II study. Catheter Cardiovasc. Interv., 2010, 76 (5), 634-42.

61. Applegate R.J., Hermiller J.J., Sanz M., et al. Comparison of everolimus-eluting and paclitaxel-eluting coronary stents in patients with two treated vessels: 2-year results from the SPIRIT III randomised trial. EuroIntervention, 2010, 6, 437-46.

62. Stabile E., Escolar E., Weigold G., et al. Marked malposition and aneurysm formation after sirolimus-eluting coronary stent implantation. Circulation, 2004, 110(5), 47-8.

63. Stone G.W., Midei M., Newman W . Randomized comparison of everolimus-eluting and paclitaxel-eluting stents: two-year clinical follow-up from the Clinical Evaluation of the Xience V Everolimus Eluting Coronary Stent System in the Treatment of Patients with de novo Native Coronary Artery Lesions (SPIRIT) III trial. Circulation, 2009, 119, 680-6.

64. Nikolsky E., Lansky A.J., Sudhir K., et al. SPIRIT IV trial design: a large-scale randomized comparison of everolimus-eluting stents and paclitaxel-eluting stents in patients with coronary artery disease. Am. Heart J., 2009,158, 520-6.

65. Kedhi E., Joesoef K.S., McFadden E., et al. Second-generation everolimus-eluting and paclitaxel-eluting stents in real-life practice (COMPARE): a randomised trial. Lancet, 2010, 375,201-9.

66. Stone G.W., Rizvi A., Newman W., et al. Everolimus-eluting versus paclitaxel-eluting stents

in coronary artery disease. N. Engl. J. Med., 2010, 362,1663-74.

67. Raber L., Juni P., Nuesch E., et al. Long-term comparison of everolimus-eluting and sirolimus-eluting stents for coronary revascularization. J. Am. Coll. Cardiol., 2011, 57(21), 2143-51.

68. Udupi K. et al. Next generation Endeavor Resolute Stent: role of the BioLinx polymer system. Medtronic Cardiovascular, Santa Rosa, CA 95-403, USA.

69. Vik-Mo H., Wiseth R., Hegbom K. Coronary aneurysm after implantation of a paclitaxel-eluting stent. Scand. Cardiovasc. J., 2004, 38(6), 349-52.

70. Colombo A., Tobis J. Techniques in Coronary Artery Stenting. London, Martin Dunitz Ltd. 2000, 422.

71. Waksman R., Ajani A.E., White R.L., et al. Prolonged antiplatelet therapy to prevent late thrombosis after intracoronary gamma radiation in patients with in-stent restenosis: Washington Radiation for In-Stent Restenosis Trial plus 6 months of clopidogrel (WRIST PLUS). Circulation, 2001, 103, 2332–5.

72. Waksman R., Bhargava B., Taafe M., et al. Prolonged antiplatelet therapy to reduce late thrombosis after intracoronary gamma radiation in patients with in-stent restenosis: “plavix-wrist” Circulation, 2000, 102, II, 570. (abstract).

73. Karvouni E. 17 months after SES implantation — despite ASA continuation (clopidogrel stoped at 12 months). Heart, 2005, 8, 64-71.

74. Alt E., Haehnel I., Beilharz C. et al. Inhibition of neointima formation after experimental coronary stenting. Circulation, 2000, 101, 1453–8.

75. Waters R.E. 3 cases following DES for in-stent-restenosis (at 16, 20, 43 months) — shortly after interruption of antiplatelet Tx. Catheter Cardiovasc. Interv., 2005, 4, 107-15.

76. Camenzind E., Steg P.G., Wijns W. Stent thrombosis late after implantation of first-generation drug-eluting stents: a cause for concern. Circulation, 2007, 115 (11), 1440-55;

77. Wenaweser P., Hess O. Stent thrombosis is associated with an impaired response to antiplatelet therapy. J. Am. Coll. Cardiol., 2005, 46(5), CS5-6.

78. Pfisterer M.E., Kaiser C.A., Bader F., et al. Late clinical events related to late stent thrombosis after stopping clopidogrel: prospective randomized comparison between drug-eluting versus bare-metal stenting. Program and abstracts from the American College of Cardiology 55th Annual Scientific Session, March 11-14, 2006, Atlanta, Georgia. Abstract 422-411.

79. Eisenstein E.L., Anstrom K.J., Kong D.F.,et al. Clopidogrel use and long-term clinical outcomes after drug-eluting stent implantation. JAMA, 2006, E1–E10.

80. Zilberman M., Eberhart R.C.. Expandable bioresorbable endovascular stent. I. Fabrication and properties. Annu. Rev. Biomed. Eng., 2006, 8, 153-80.

81. Varani E., Guastaroba P., Di Tanna G.L. et al. Long-term clinical outcomes and cost-effectiveness analysis in multivessel percutaneous coronary interventions: comparison of drug-eluting stents, bare-metal stents and a mixed approach in patients at high and low risk of repeat revascularisation.EuroIntervention, 2010, 5 (8), 953-61

82. Bertand O.F., Faurie B., Larose E. et al. Clinical outcomes after multilesion percutaneous coronary intervention: comparison between exclusive and selective use of drug-eluting stents. J. Invasive Cardiol., 2008, 20 (3), 99-104.

Successful Repair of a Giant Thoracic Aortic Aneurysm with De Bakey IIIB Dissection Using Hybrid Surgical Approach. A Clinical Case

R.S. Akchurin[1], S.V. Korolev, T.E. Imaev, A.E. Komlev, P.M. Lepilin, I.S. Fedotenkov, A.S. Kolegaev
Department of Cardiovascular Surgery, Russian Cardiological Scientific and Production Complex of the Ministry of Healthcare and Social Development of the Russian Federation, Moscow, Russia

Thoracic aortic aneurysms account for about 20% of all aortic aneurysms. Their incidence is 6 per 100000 of patients per year (5). Aneurysm development is connected with media degeneration resulting from the insufficiency of all layers of the aortic wall. The risk of rupture is related to the increase of aortic diameter (7). In 33-50% of cases the rupture leads to death (5,8).

Aortic dissection is a complex emergency situation, occurring twofold more frequently than abdominal aortic ruptures. The dissection of the aortic walls can cause dynamic or permanent obstruction, rupture or thrombosis of the aortic branches.

Open surgical intervention is a traditional standard treatment for this condition. The mortality associated with open interventions in the leading world centers is about 5-14% (for III type dissections) (10,11). The complications involve pulmonary, renal and cardiovascular systems and abdominal organs, thus significantly increasing the risk of death. Herewith the rate of complications is directly related to the size and the extension of the aneurysm. Concomitant diseases existing in many patients cause many problems related to open surgical interventions.

With the account of open surgery-related risks, endovascular minimally invasive approach seems attractive and advantageous. The aim of endovascular aneurysm repair (EVAR) consists in the prevention of aneurysm rupture and aortic dissection by the way of aneurysmatic sac exclusion from the circulation, decreasing of the tension of aneurysmatic sac's walls or of the pressure in the false lumen with its subsequent closure.

In order to illustrate the up-to-date possibilities of the aortic dissection repair we present a clinical case of successful repair of a giant thoracic aortic aneurysm with De Bakey IIIB dissection and a parietal thrombosis using hybrid surgical method.

Keywords: *hybrid surgery, thoracic aortic aneurysms, aortic dissection, open surgery, endovascular interventions, endoprosthesis, EVAR.*

Introduction

According to expert data, thoracic aortic disease is diagnosed yearly in over 16,000 European patients (1,6). The development of aneurysms is related to media degeneration as a result of insufficiency of all aortic wall's layers. Dissection occurs in about 50% of thoracic aortic aneurysms. In fact, these are false aneurysms, as the process of dissection does not involve all aortic wall's layers.

The incidence of thoracic aortic aneurysms rupture is 3.5-5 / 100,000 patients per year. The risk of rupture is related to the increase of aortic diameter (7). The rupture leads to death in 33-50% of cases (5, 8). Population studies have shown that 5-year survival in patients with untreated thoracic aneurysms of various genesis is only 13% (5), while 3-year survival in patients with degenerative aneurysms is 35% (8,9).

The dissection of thoracic aorta is a complex emergency situation occurring twofold as often as abdominal aortic aneurysms. Atherosclerotic degeneration of the aortic wall in hereditary diseases, such as Marfan and Ehlers-Danlos syndromes, and arterial hypertension is a predictor for dissection. Local intimal rupture or intramural hematoma leads to abrupt blood inflow into the aortic media layers which ends by aortic dissection and the development of a false lumen. The dissection can extend proximally as well as distally to the site of initial lesion, thus creating aortic branches' obstruction (dynamic or permanent), rupture or thrombosis.

Aortic dissections can be classified by their location, duration and presence of ischemic complications. According to one of the most often used Stanford classification, type A includes the dissection of the ascending aorta, type B — the dissection of the descending aorta. De Bakey's type I includes the dissection of the whole aorta, type II — the dissection of the ascending aorta, type III — the dissection of the descending thoracic aorta and of the abdominal aorta.

Pharmacological therapy and regular diagnostic studies are indicated in most cases. Surgical interventions for asymptomatic aneurysms are recommended if aneurysmatic sac attains 5.5-6 cm in diameter, while acute symptomatic aneurysms irrespective of

[1] Address for correspondence:
Dr. Renat Akchurin,
Russia, 121552, Moscow,
3rd Cherepkovskaya ul, 15a
Tel. /Fax: 007 499 140-93-36, 007 499 149-17-08
Email: dddd99999@mail.ru
Manuscript received on June 06, 2011.
Accepted for publication on August 22, 2011.

their diameter and dissecting aneurysm should be operated immediately.

Open surgical intervention is the traditional standard treatment. Mortality rate in open surgery in the leading world centers is about 5-14% (for type III dissections) (10,11), the rate of paraplegia is 5% (12, 13). Other major complications involve pulmonary, renal and cardiovascular systems and abdominal organs, which contribute to a significant rise of mortality risk. Herewith the incidence of complications correlate with the size and the extension of the aneurysm and the dissection.

Concomitant diseases present in many patients create a lot of problems related to open surgical intervention. The decision making is complicated by the following factors connected with increased risk of surgical mortality: the patients' age over 70 years, preoperative shock / hypotension, multifocal atherosclerosis (in about 1/3 of patients), partial thrombosis of the false lumen, signs of periaortic hematoma, descending aortic diameter > 6 cm, right ventricular dysfunction. One also has to keep in mind relative contraindications for open surgery: ACVA, marked LV failure, coagulopathy, pregnancy, myocardial infarction (< 6 months), marked arrhythmia, elderly age, valvular apparatus diseases.

Taking into account the risks of open interventions, the endovascular minimally invasive interventions in aortic aneurysms (EVAR — endovascular aneurysm repair) are advantageous and attractive in comparison with traditional surgery. As a result, the patients with contraindications for open surgery have gotten a chance for correction. The use of endovascular endoprosthesis allows for the closure of fenestrations, the thrombosis of the false lumen with the reconstruction of the true lumen, the decreasing of pressure in the false lumen and the increasing of distal perfusion (14, 15, 16). As a rule, short term mortality indices (30-day mortality, 3- and 6-months mortality and 1 year mortality) after endovascular interventions on aortic aneurysms and type B dissections are significantly lower than after open surgery (17,18). Endovascular repair of thoracic aortic aneurysms is associated with lower complications rate, shorter ICU and hospital stay, lower blood loss and lower percentage of paraplegia in comparison with open surgical intervention (19, 20,21). The complications associated with this technique include: incomplete exclusion of the aortic arch from the circulation due to the gaps between the endoprostheses, the endoleaks of the 1st and other types.

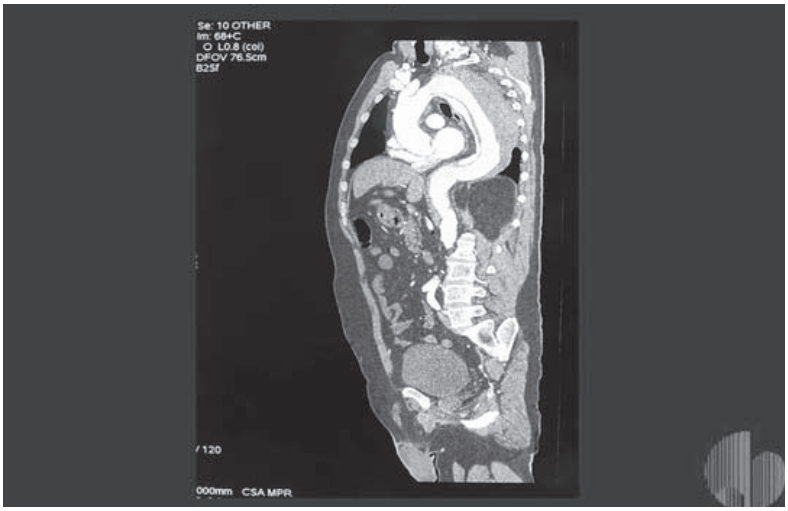


Fig. 1. MHCT-aortography of patient A

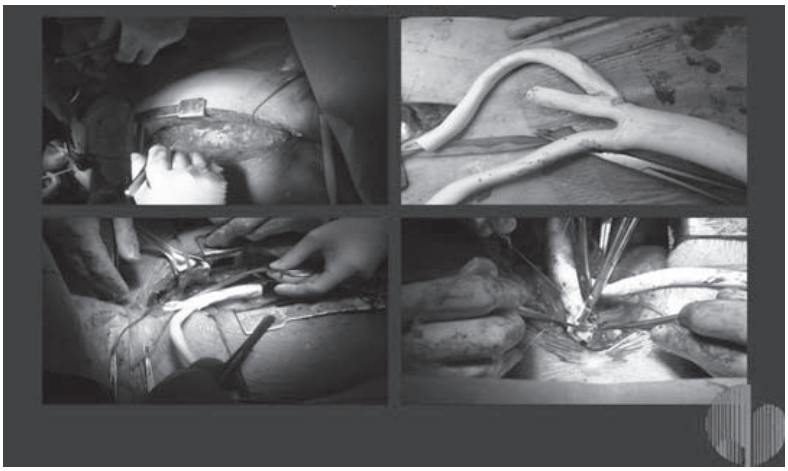


Fig. 2. The stage of open surgical intervention: the grafting of the brachiocephalic trunk, the left common carotid artery, the left subclavian artery with a trifurcating Gore prosthesis

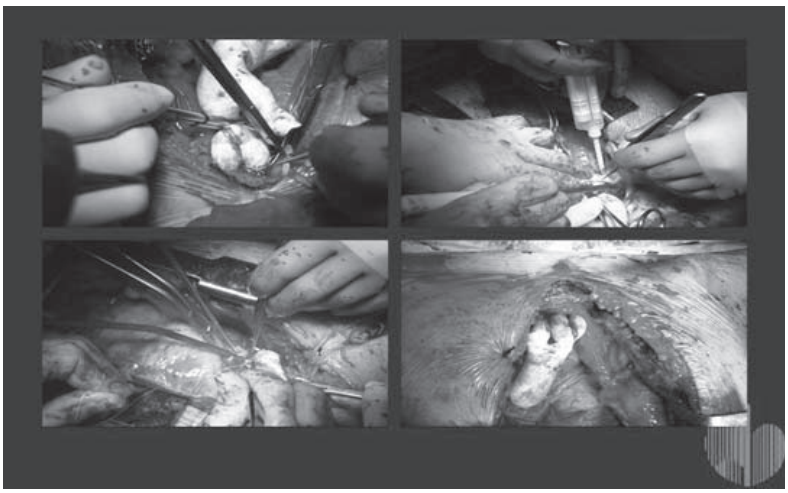


Fig. 3

A significant number of patients with acute and chronic pathology have thoracic aortic lesion involving aortic arch branches (the brachiocephalic trunk, the common carotid and the subclavian arteries). In

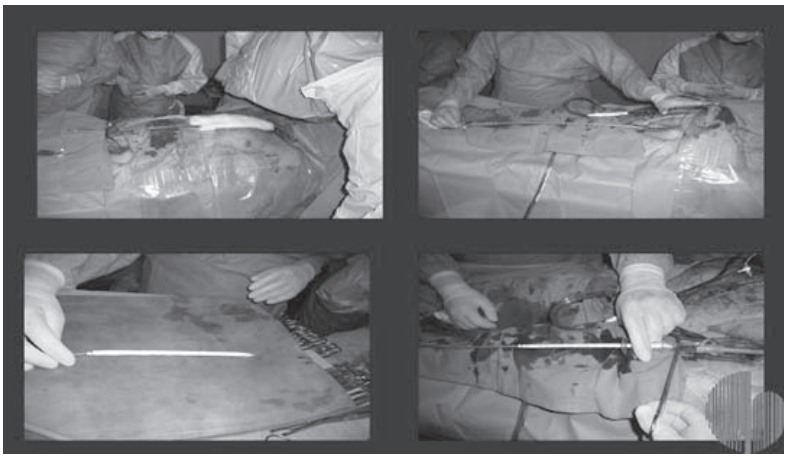


Fig. 4. Endovascular grafting of the thoracic aorta using Gore endoprosthesis

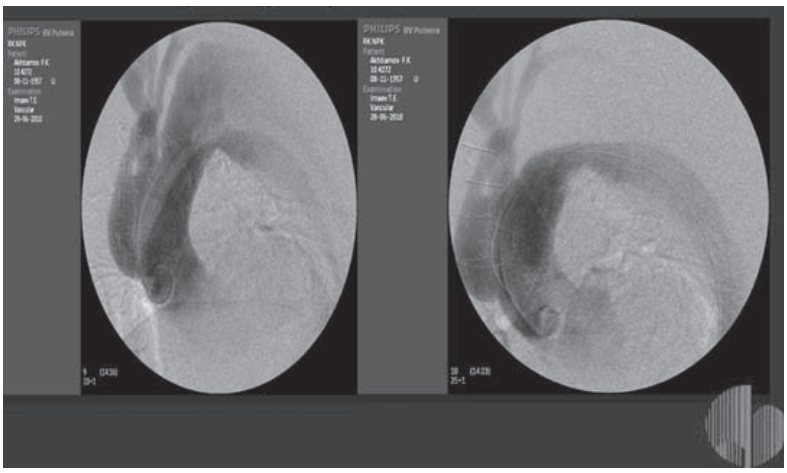


Fig. 5. Intraoperative aortography of patient A

such cases in order to enlarge the proximal area of endoprosthesis deployment during endovascular interventions it can be necessary to cover the aortic arch branches. The occlusion of supraaortic vessels by endoprosthesis can be a severe, sometimes even fatal complication of endovascular interventions on the ascending aorta and aortic arch. In such case it would be reasonable to find some balance between the "traditional open surgery" and the endovascular interventions. The so-called "hybrid" operations, combining minimal invasiveness of the open access, the providing of visceral perfusion, the minimization of ischemia duration, the creation of a safe terrain for endoprosthesis implantation and endovascular grafting for aortic aneurysm exclusion, as such, can offer this balance (22). Many innovative methods, including the reimplantation of the major aortic arch branches (rebranching), shunting operations and "elephant trunk" reconstructive surgery have been presented rather recently (23-28).

Case report

In order to illustrate the current possibilities in the treatment of aortic dissection we would like to present a clinical case of successful correction of a giant thoracic aortic aneurysm with De Bakey IIIB dissection using hybrid surgical approach.

The 52-years old patient A, male, was admitted to the Department of Cardiovascular Surgery in June 04, 2010, with chest pain syndrome, complaints of arterial hypertension, neurological disturbances and impossibility to urinate. The analysis of his history revealed 3rd degree arterial hypertension of many years duration (maximal BP 220/160 mm HG) despite regular hypotensive therapy. MHCT performed in September 2009 revealed aortic arch aneurysm with III type dissection. After acute development of lower paraplegia on May 25, 2010, the patient was diagnosed with acute impairment of spinal circulation and paradoxical ischuria ((neurogenic bladder)

EchoCG: the aorta is indurated, with descending segment diameter of 3,7 cm, significantly dilated (up to 9 cm) in the descending thoracic segment, where intimal dissection can be seen. An area filled with up to 3 cm thick thrombus is visualized at the external contour of the descending aorta. The cardiac cavities are not enlarged. The LV EF > 55%, there are no areas of local contractility impairment. There are insignificant valvular regurgitations.

Coronary angiography: the left main coronary artery without changes. Irregular contours of the LAD, the CxB and the RCA.

MHCT-angiography (Fig.1): the walls of the ascending aorta are not thickened, there are no calcification. Maximal diameter of the aortic root -– 3,9 cm, the aortic arch and the descending segment are enlarged up to 9 cm, a massive parietal thrombosis can be seen at the external contour. The intimal dissection starts from the left subclavian artery ostium, the true lumen is several times smaller than the false one. The aortic dissection can be found up to the celiac axis (arising from the true lumen), below the celiac axis there is no dissection.

Consultation of a neurologist: the episode of acute impairment of the spinal circulation was caused by intima dissection in the ostium of an intercostal artery at the level of Th X-XI (proximal to the end of aortic dissection) with subsequent restoration of the collateral circulation.

The patient was diagnosed with a giant thoracic aortic aneurysm with De Bakey IIIB dissection, with parietal thrombosis; the impairment of the spinal circulation with the development of transient lower paraplegia and urination disturbances; arterial hypertension.

On June 28, 2010, the patient underwent hybrid surgical intervention: endovascular repair of the thoracic aorta with the grafting of the brachiocephalic trunk, the left common carotid artery and the left

subclavian artery using a trifurcation Gore prosthesis.

The access to the heart and aortic arch branches was achieved through median sternotomy. Simultaneously the common femoral artery was approached through the left thigh. Three distal prosthetic anastomses were performed — to the left subclavian artery (LSA), the left common carotid artery (CCA) and an "end-to-side" anastomosis to the brachiocephalic trunk (BCT) using lateral dislocation of the aorta (Fig. 2). After that a proximal "end-to-side" Gore anastomosis to the aorta was performed (Figs. 2, 3). The CCA, LSA and BCT were ligated. The anastomoses were patent and non-leaking. An introducer was inserted through a puncture to the CFA, and a diagnostic catheter was advanced into the ascending aorta via the stiff guide. Diagnostic aortography was performed. Simultaneously a delivery system was inserted through the left CFA. A Gore Tag 45x20 endoprosthesis (Fig. 4) was delivered through this system into the ascending aorta and deployed from the level of distal edge of the trifurcating Gore prosthesis anastomosis with the aorta (Fig. 5). Control opacification of the aorta demonstrated effective grafting of the proximal aortic dissection, no endoleak signs were revealed.

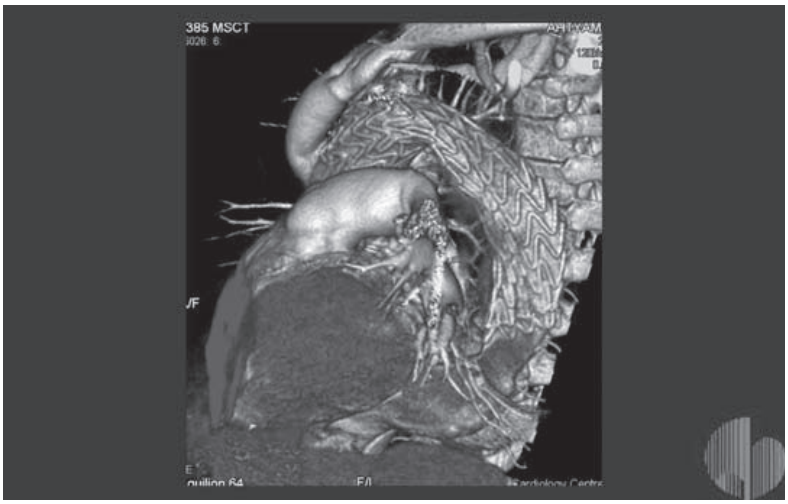


Fig. 6. Postoperative MHCT-aortography of patient A

Results of study

The patient's rehabilitation was achieved within the shortest term. The patient was discharged at day 5. Postoperative course was uneventful. The patient has been followed in the long-term (1 month — 1 year), no complications were revealed. According to control MHCT aortography (Fig. 6), the false lumen did not opacify, there were no signs of endoleak. The aortic arch arteries opacified through the trifurcating prosthesis from the ascending aorta.

Discusson

In view of latter achievements in endovascular therapy, endovascular aortic repair (EVAR) is being used more frequently for the treatment of aneurysms and aortic dissection with satisfactory initial results.

In accordance with the existing indications for EVAR, the necessary zone of endoprosthesis fixation should be at least 20 mm and the diameter of the aorta less than 40 mm. For this reason, from the viewpoint of surgical indications, the anatomical factors, the character and the length of arterial injury, as well as aneurysm morphology play an important role in the choice of the tactics of treatment, and EVAR should not become the method of choice if the aneurysm / dissection is located in the close proximity of the main aortic branches. Fenestrated and multi-branched endoprosthesis have been developed just for such cases — in order to provide the blood flow to the branches in aortic aneurysms / dissections, but at present they are submitted to detailed clinical testing in single clinical centers, and the accumulated information will be carefully analyzed by the experts. These endoprostheses are made by hand, adjusted for the anatomy of each patient and necessitate a precise sizing as well as 6 to 8 weeks to be manufactured.

Hybrid procedures combining traditional surgical approach and EVAR, are being developed and introduced with the aim of enlarging the indications for endovascular interventions in high-risk patients. This approach is minimally invasive, associated with low rate of complications and mortality. Hybrid approaches for the treatment of aortic arch pathology will continue to develop, the methods and the techniques will be elaborated and brought to perfection. The future role of these procedures will be determined by their long-term results.

Hybrid surgery is a particular transition to eventual primacy of endovascular multi-branch grafting in the future and represents a real alternative for patients with contraindications to endovascular or open surgical intervention.

References

1. Advanced endovascular therapy of aortic disease. Ed. by Alan B. Lumsden et al. Blackwell Futura, Malden, 2007.

2. Endovascular and hybrid management of the thoracic aorta. Ed. by Edward B. Diethrich et al. Wiley-Blackwell, Hoboken, 2008.

3. Aortic surgery. New developments and perspectives. Ed. by C. Setacci et al. Edizioni Minerva Medica, Turin, 2009.

4. Comprehensive vascular and endovascular surgery. 2nd ed. Ed. by John W. Hallett et al. Mosby, Philadelphia, USA, 2009.

5. Bickerstaff L.K., Pairolero P.C., Hollier L.H., et al. Thoracic aortic aneurysms: A population-based study. Surgery, 1982, 92, 1103–8.

6. Joyce J.W., Fairbairn J.F., Kincaid O.W., Juergens J.L. Aneurysms of the thoracic aorta. A clinical study with special reference to prognosis. Circulation, 1964, 29, 176–81.

7. Juvonen T., Ergin A., Galla J.D., et al. Prospective study of the natural history of thoracic aortic aneurysms. Ann. Thorac. Surg., 1997, 63, 1533–45.

8. McNamara J.J, Pressler V.M. Natural history of arteriosclerotic thoracic aortic aneurysms. Ann. Thorac. Surg., 1978, 26, 468–73.

9. Clouse W.D., Hallett J.W., Schaff H.V., et al. Improved prognosis of thoracic aortic aneurysms: A population based study. JAMA, 1998, 280,1926–9.

10. Miller D.C., Mitchell R.S., Oyer P.E., et al. Independent determinants of operative mortality for patients with aortic dissections. Circulation, 1984, 70 (Suppl. 1), 1153–64.

11. Svensson L.G., Crawford E.S., Hess K.R., et al. Dissection of the aorta and dissecting aortic aneurysms. Circulation, 1990, 82 (Suppl. IV), IV24–IV 28.

12. Coselli J.S., LeMaire S.A., Miller C.C., et al. Mortality and paraplegia after thoracoabdominal aortic aneurysm repair: A risk factor analysis. Ann. Thorac. Surg., 2000, 69, 409–14.

13. LeMaire S.A., Miller C.C., Conklin L.D., et al. Estimating group mortality and paraplegia rates after thoracoabdominal aortic aneurysm repair. Ann. Thorac. Surg., 2003, 75, 508–13.

14. Chavan A., Hausmann D., Dresler C., et al. Intravascular ultrasound-guided percutaneous fenestration of the intimal gap in the dissected aorta. Circulation, 1997, 96, 2124–7.

15. Slonim S.M., Nyman U., Semba C.P., et al. Aortic dissection: Percutaneous management of ischemic complications with endovascular stents and balloon fenestration. J. Vasc. Surg., 1996, 23, 241–53.

16. Williams D.M., Lee D.Y., Hamilton B.H., et al. The dissected aorta: Percutaneous treatment of ischemic complications— Principles and results. J .Vasc. Interv. Radiol., 1997, 8 (4), 605–25.

17. Lepore V. Lann L., Delle M., et al. Endograft therapy for aneurysms and dissections of the thoracic aorta in 43 consecutive patients. J. Endovasc. Ther., 2002, 9, 829–37.

18. Lann L., Delle M., Falkenberg M., et al. Endovascular treatment of type B thoracic aortic dissections. J. Card. Surg., 2003, 18, 539–44.

19. Makaroun M.S., Dillavou E.D., Kes S.T. et al. Endovascular treatment of thoracic aortic aneurysms: results of the phase II multicenter trial of the Gore TAG thoracic endoprosthesis. J. Vasc. Surg., 2005, 41, 1–9.

20. Wheatley G.H., III, Gurbuz A.T., Rodriguez-Lopez J.A. et al. Midterm outcome in 158 consecutive Gore TAG thoracic endoprostheses: single center experience. Ann. Thorac. Surg., 2006, 81 (5), 1570–7.

21. Bavaria J.E., Appoo J.J., Makaroun M.S., et al. Endovascular stent grafting versus open surgical repair of descending thoracic aortic aneurysms in low-risk patients: a multicenter comparative trial. J. Thorac. Cardiovasc. Surg., 2007, 133, 369–77.

22. Diethrich E.B., Ghazoul M., Wheatley G.H., et al. Surgical correction of ascending type a thoracic aortic dissection: Simultaneous endoluminal exclusion of the arch and distal aorta. J. Endovasc. Ther., 2005, 12, 6660–6.

23. Inoue K., Hosokawa H., Iwase T., et al. Aortic arch reconstruction by transluminally placed endovascular branched stent graft. Circulation, 1999, 100 (Suppl.), 316–21.

24. Ischimaru S. Endografting of the aortic arch. J. Endovasc. Ther., 2004, 11 (Suppl. 2), 62–71.

25. Greenberg R.K., Haddad F., Svensson L., et al. Hybrid approaches to thoracic aortic aneurysms: The role of endovascular elephant trunk completion. Circulation, 2005, 112, 2619–26.

26. Bergeron P., Coulon P., De Chaumaray T., et al. Great vessel transposition and aortic arch exclusion. J. Cardiovasc. Surg. (Torino), 2005, 46, 141–7.27. DeSanctis R.W., Dorgoghazi R.M., Austen W.G., Buckley M.J. Medical progress (aortic dissection). N. Engl. J. Med., 1987, 317, 1060–67.

28. Duhaylongsod F.G., Glower D.D., Wolfe W.G. Acute traumatic aortic aneurysm: the Duke experience from 1970 to1990. J. Vasc. Surg., 1992, 15, 331–43.

Endovascular Correction of a Post-Traumatic False Aneurysm of the Right Subclavian Artery. A Case Report

A.I. Kvashin, S.A. Atamanov, A.V. Melnik[1], A.O. Bykov, A.A. Pomkin, M.G. Shirkin, S.A. Kyshtymov.

Research Center of Reconstructive Surgery, East Siberian Scientific Center of Siberian Branch of Russian Academy of Medical Sciences, Irkutsk Regional Clinical Hospital, Irkutsk, Russia

False aneurysm of the subclavian artery is an uncommon complication. Most frequently this pathology is caused by fracture of clavicle, however other causes also can exist. We present a case of diagnosis and endovascular treatment of a false aneurysm of the right subclavian artery in a 31-years-old man, heroin addict, who performed multiple injections into the right subclavian artery. At admission he had paresis and decreased sensitivity in the right arm due to compression neuritis of the brachial plexus. The diagnosis was made on the basis of clinical examination, duplex scanning, multihelix computed tomography (MHCT) and angiograpnic study of the subclavian vessels. With the account of large false aneurysm and difficulties of surgical approach we decided to abstain from open intervention.

A successful endovascular correction of the blood flow in the right subclavian artery using a stent-graft was performed.

This clinical example demonstrates that endovascular approach to the treatment of false aneurysms located in this area is associated with high clinical effectiveness, minimal traumatism and absence of blood loss.

Keywords: *false aneurysm, stent-graft, subclavian artery, brachial plexus.*

Abbreviations

RSA — right subclavian artery.

MHCT — multihelix computed tomography

Introduction

According to the definition, the false aneurysm is a cavity filled with blood and communicating with the true arterial lumen. The walls of the false aneurysm are formed by the aneurysm-limiting tissues that do not contain the elements of the arterial wall (4,8).

Most commonly the false aneurysms of the subclavian artery develop after traumatic lesions of the clavicular area (4, 8, 11). As a rule, this pathology is diagnosed late, which is mostly related to the gradual increase of the volume in the clavicular area and the appearance of the signs of brachial plexus compression (6,9,12).

Clinical case

A 31-years-old man was admitted to the Department of Surgery of the Irkutsk Regional Clinical Hospital in January 2010 with the diagnosis of a right-sided post-traumatic aneurysm of the subclavian artery. The patient was heroin addict. He attributed his disease to drug injection into the right subclavian area about 2 months ago. Palpation revealed a mass of about 7 cm in diameter in the right supraclavicular area, at auscultation a systolic murmur was heard. There was paresis and decreased sensitivity in the right arm.

Duplex scanning of the right subclavian and axillary vessels was performed.

The signs of phlebothrombosis of the right subclavian and axillary veins were revealed. A heterogenous, roundish mass measuring 60x40 mm, with an intraluminal parietal thrombus and turbulent blood flow was found at the anterolateral wall in the projection of the 2nd segment of the right subclavian artery (RSA). This cavity was connected with the lumen of the right subclavian artery through a 4 mm channel.

MHCT also confirmed the presence of a thrombosed aneurysm of the 2nd segment of the RSA.

Taking into account the big size of this aneurysm as well as the difficulties of surgical intervention, we decided to abstain from open surgery.

The patient was transferred into the cathlab for elective angiographic study and solution of the problem of endovascular correction of his false aneurysm. The right common femoral artery was punctured in accordance with Seldinger technique. A catheter was advanced into the RSA ostium through a 5F sheath and angiographic study was performed. In the 2nd segment of the RSA we found a post-traumatic multi-chambered false aneurysm (53x64 mm) with turbulent blood flow and protracted retention of the opacified blood (fig.1). The RSA was 7 mm in diameter. The length of the damaged segment was 50 mm. After the evaluation of x-ray semiotics we decided to perform endovascular separation of the RSA from the false aneurysm cavity. After the puncture of the left common femoral artery a 9F sheath was inserted. A peripheral stent-graft Fluency plus (BARD) 9.0 x 60,0 mm was advanced by the cord guide and implanted in the plane of the aneurysm's neck. At control an-

[1] Address for correspondence:
Dr. Alexey Melnik.
Russia, 664000, Irkutsk, microdistrict Yubileyny, 100,
Irkutsk Regional Clinical Hospital, department of Angiography.
Tel. +7 964 212 25 45
e-mail: am78@bk.ru
Manuscript received on June 2, 2011.
Accepted for publication on July 27, 2011.

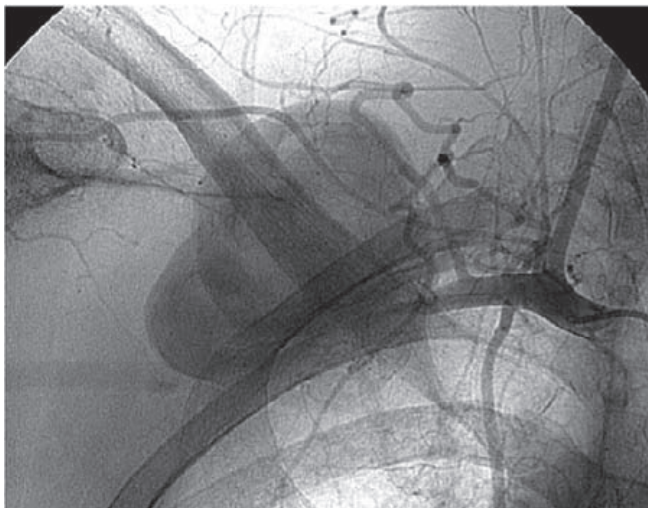


Fig. 1

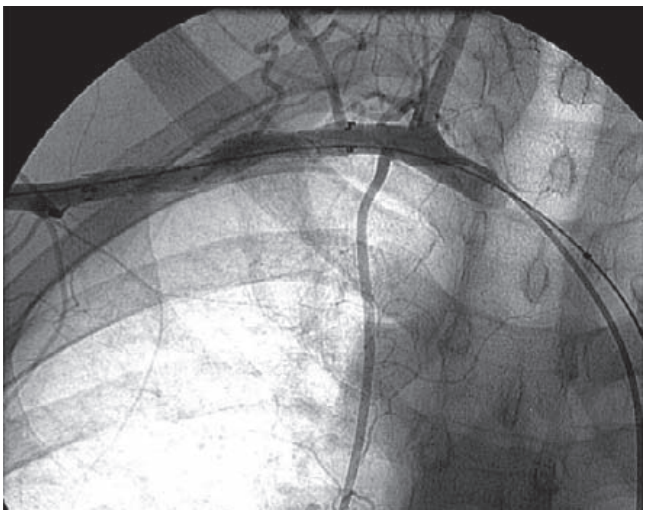


Fig. 2

giography the false aneurysm did not opacify, the blood flow in the stented segment was preserved, there was an over 50% stenosis in the initial segment of the implanted stent. Balloon dilatation of this segment was performed with the use of AgilTrac balloon (Guidant) 6.0x40.0 mm.

Control angjography revealed a stented segment with regular contours, without residual stenosis. The aneurysm was not opacified (fig2).

Control duplex scanning in the projection of the 2nd segment of the RSA revealed a heterogenous roundish mass measuring 60 x 40 mm, with intraluminal parietal thrombosis, located at the anterolateral wall. Turbulent blood flow was not seen. The stent-graft was patent, the magistral blood flow was preserved.

Postoperative condition of the patient was satisfactory. He had no complaints. The symptoms of compression neuritis of the brachial plexus regressed. In 7 days after endovascular intervention the patient was discharged under the care of a local physician.

Discussion

The lesions of the subclavian artery can be caused by penetrating injury by the fragments of fractured clavicle, the displacement of metal elements of constructions used for ostheosynthesis (6, 7, 9, 10, 12). Paresthesia and paresis of the arm are typical symptoms of brachial plexus compression (6, 9, 12). As a rule, the time interval between the primary trauma and the diagnosis of a false aneurysm is long. Skeletal and muscular lesions as well as the gradual growth of the aneurismal cavity hamper timely diagnostics and treatment of this condition. Severe neurological abnormalities, related to the compression of brachial plexus, compression embolism of the subclavian vessels, as well as the rupture of false aneurysm with the development of arterial bleeding, are severe, life-threatening complications.

Open surgical approach in various segments of the subclavian artery is associated with sternotomy or the resection of a fragment of clavicle with subsequent ostheosynthesis, is rather traumatic and elongates the rehabilitation period. The risk of wound infection and massive blood loss is always higher with open surgery (3, 5, 7, 10, 12).

Endovascular treatment is minimally traumatic, minimizes the blood loss and the probability of infection. The rehabilitation period after intravascular intervention is enormously shorter in comparison with open surgery.

There are few reports of such cases in world literature (1, 2). However we did not find any description of endovascular correction of a post-injection false aneurysm with the use of a stent-graft.

Conclusion

Due to currently available materials and technologies, the method of endovascular separation of the false aneurismal cavity from the true arterial lumen allows to obtain good results in the correction of vascular wall lesions.

Minimal invasiveness, high effectiveness, absence of blood loss allow to consider the endovascular method as one of the leading modalities of treatment of false aneurysms of different genesis.

References

1. Babatasi G., Massetti M., Le Page O. Endovascular treatment of a traumatic subclavian artery aneurysm. J. Trauma, 1998, 44, 545-7.

2. Becker G., Benenati J., Zemel G. Percutaneous placement of a balloon-expandable intraluminal graft for life-threatening subclavian arterial haemorrhage. JVIR, 1991, 2, 225-9.

3. Jeganathan R., Harkin D., Lowry P. Iatrogenic subclavian artery pseudo-aneurysm causing airway compromise : treatment with percutaneous thrombin injection. J. Vasc. Surg., 2004, 40, 371-4.

4. Johnston K.W., Rutherford R.B., Tilson M.D., et al. Suggested standards for reporting on arterial aneurysms. J. Vasc. Surg., 1991,13, 452–8.

5. Mcconnell P., Rehm J., Oltman D. Thrombin injection for treating a subclavian artery pseudo-aneurysm. Surg., 2000, 127, 716-8.

6. Pfammatter T., Kunzli A., Hilfiker P. Relief of subclavian venous and brachial plexus compression syndrome caused by traumatic subclavian artery aneurysm by means of transluminal stent-grafting. J. Trauma, 1998, 45, 972-4.

7. Renger R., De Bruijin A., Aarts H. Endovascular treatment of a pseudo-aneurysm of the subclavian artery. J. Trauma, 2003, 55, 969-71.

8. Rizzoli H.M., Caboclo J.L.F., Godoy J.M.P., et al. Resolucao de pseudoaneurisma de arteria subscapular, por molas de Gianturco. HB Cientifica 1996,3,28–30.

9. Schellhammer F., Muller H. J. Endovascular treatment of subclavian pseudo-aneurysm causing delayed brachial plexus paralysis. VASA, 2004, 33, 239-41.

10. Serrano J., Rodriguez P., Castro L. Acute subclavian artery pseudo-aneurysm after closed fracture of the clavicle. Acta Orth, Belg,, 2003, 69, 555-7.

11. Stockinger Z., Townsend M., Mcswain N. Acute endovascular management of a subclavian artery injury. J. La State Med. Soc., 2004, 156, 262-4.

12. Watanabe K., Matsumara T. Late-onset brachial plexus paresis caused by subclavian pseudo-aneurysm formation after clavicular fracture. J. Trauma, 2005, 58, 1073-4.

Significance of Anatomical Variability of Tibial and Foot Arteries for Peripheral Angioplasty in Patients with Critical Ischemia

D.V. Ovcharenko[1], M.Yu. Kapytin, A.A. Voronkov, S.A. Platonov.
Department of endovascular diagnostics and treatment, Djanelidze Research Institute of Emergency Medicine, St. Petersburg, Russia

The authors present a retrospective evaluation of the incidence of atypical anatomical variants of tibial arteries origin and foot blood supply associated with critical ischemia, treated by peripheral angioplasty. Intraprocedural complications caused by atypical anatomy have been taken into consideration.
Keywords: *peripheral angioplasty, critical lower limb ischemia, popliteal artery, anterior tibial artery, posterior tibial artery.*

Purpose of study. This study was aimed at the evaluation of the significance of atypical anatomical variants of popliteal artery branching and foot arteries origin for the performance of peripheral angioplasty in patients with critical lower limb ischemia (CLLI), in whom angioplasty has been used as a primary revascularization method.

Material and methods. In total, 248 peripheral angioplasties (PAP) have been performed in 240 patients with CLLI. The incidence of atypical anatomical variants of popliteal artery branching and foot arteries origin has been studied retrospectively. Clinical results and complications of peripheral angioplasty in patients with typical and atypical branching of the popliteal artery and foot blood supply have been studied.

Results. Atypical branching of the popliteal artery and foot blood supply have been noted in 31 (12,5%) cases (out of 248 legs, on which PAP was performed). Aplasia — hypoplasia of the tibial branches with dorsalis pedis artery (DPA) and/or plantar artery (PlA) arising from the peroneal artery was seen in 17 cases (6,8% of 248 legs, in which PAP has been performed). Herewith only 14 (45%) cases (out of 31 with atypical anatomy) were revealed during diagnostics. In seven (41%) of the remaining 17 cases (non-recognizable before PAP) the peculiarities were suspected only after the occurrence of complications. There were no significant differences between legs preservation rate in the groups with typical and atypical anatomy.

Conclusion. Atypical variants of lower leg and foot blood supply are rather frequent and if not timely recognized can cause specific complications.

[1] Address for correspondence:
Dr. Dmitry Ovcharenko
Russia, 192242, St. Petersburg, ul. Budapeshskaya, 3
Tel. 007 812 313-46-38
Cell. 007 911-915-93-88
факс. (812) 313-46-46
E-mail: dovcharenko@rambler.ru
Manuscript received on May 13, 2011.
Accepted for publication on July 14, 2011.

List of abbreviations
CLLI — critical lower limb ischemia
PAP — peripheral angioplasty
PA — popliteal artery
ATA — anterior tibial artery
PTA — posterior tibial artery
PeA — peroneal artery
DPA — dorsalis pedis artery
PlA — plantar artery
TPT — tibioperoneal trunc

Introduction

Endovascular interventions are playing an increasing role in the treatment of critical lower ilmb ischemia (CLLI), expecially in diabetics. In order to achieve clinical success it is necessary to restore antegrade blood flow into the arterial arch of the foot. It often requires recanalization of extended occlusions of the tibial and foot arteries, being the predominant form of arterial lesions in such patients (1, 2).

Besides typical branching of the popliteal artery (PA) below the cleft of the knee into the anterior tibial artery (ATA) and tibioperoneal trunk (TBT) prolonged by the posterior tibial artery (PTA), there are other anatomical variants. They are caused by embryological development and their incidence varies from 7,4% to 17,6% (3, 4). Basing on the results of 1000 legs' angiography, Kim et al. (3) suggested a practical classification of anatomical variants of branching, being a modification of Lippert и Pabst classification (5). The authors suggested three types of anatomical variants that account for the level and the character of branching of the popliteal artery, as well as the origin of the foot arteries. Type I includes the variants with normal level of PA branching, where typical anatomical variant is denominated as IA. Type II comprises the cases with high level of popliteal artery branching above the cleft of the knee, and type III — the cases with aplasia or hypoplasia of the tibial branches with dorsalis pedis artery and/or plantar artery arising from the peroneal artery (Fig.1).

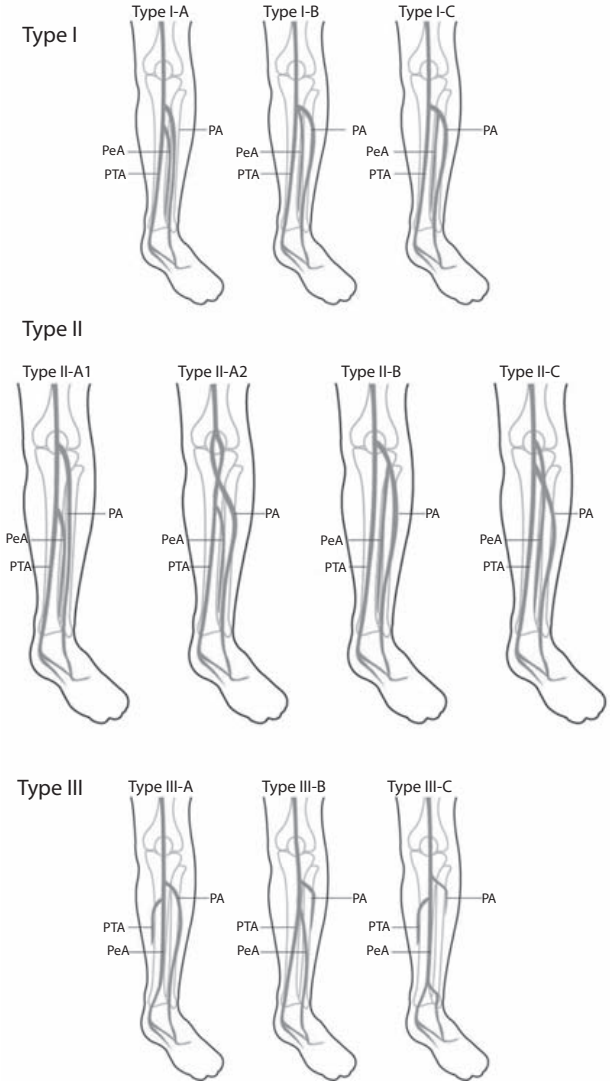


Fig. 1. Scheme of anatomical variants of the PA branching and foot blood supply, suggested by Kim et al.

Type I — normal level of branching: I-A — typical anatomical variant, I-B — trifurcation of the PA, I-C — anterior tibioperoneal trunk, PeA arises from the ATA;

Type II — high branching of the PA: II-A1 and II-A2 — the ATA arises on the level or above of the cleft of the knee, II-B — high origin of the ATA, anterior tibioperoneal trunk, II-C — high origin of the PeA from the PA;

Type III — hypoplasia of the PTA and/or ATA with plantar and/or dorsalis pedis artery arising from the PeA: III-A — hypoplasia of the PTA, the plantar artery arises from the PeA, III-B — hypoplasia of the ATA, the dorsalis pedis artery arises from the PeA, III-C — hypoplasia of the PTA and the ATA, the plantar and the dorsalis pedis arteries arise from the PeA

Abbreviations: PA — popliteal artery; ATA — anterior tibial artery, PTA — posterior tibial artery, PeA — peroneal artery.

The occlusions of the arterial segments in the area of popliteal artery branching, lower leg and foot arteries often complicate the recognition of atypical anatomical variants, which can lead to technical problems and specific complications during endovascular interventions.

The significance of anatomical variability of the PA for various open surgical interventions has been described in the literature (3), however we could not find the works assessing its significance for endovascular interventions. Kawarada et al. (6) reported three clinical cases of successful PAP in type III anatomical variants, with all these variants being evident at diagnostic stage of the intervention (6).

The aim of our study consisted in the evaluation of the significance of anatomical variants of popliteal artery branching and foot arteries origin for PAP performance in patients with CLLI, in whom angioplasty was applied as a primary revascularization method.

Material and methods

From December 2004 through June 2009 in total 248 PAP have been performed in 240 patients with CLLI caused by arterial lesions in femoropopliteal segments, as well as in tibial and foot arteries. Diabetes mellitus was present in 189 (76%) patients. The procedures were performed under local anesthesia, antegrade transfemoral approach was used in most cases. Intraluminal angioplasty was applied for stenoses and non-extended occlusions. In cases of extended (over 5 cm) occlusions in femoropopliteal segment, as well as in crural arteries planned subintimal recanalization and angioplasty according to Bolia technique were used (7, 8).

PAP was considered as technically successful if a continuous antegrade blood flow to the foot was restored in at least one crural artery without >50% residual stenoses. The patients with trophic lesions received necessary local surgical treatment and therapy with antiobiotics. Leg preservation after PAO was determined in accordance with Rutherford recommendations (9).

During retrospective analysis of pre- and post-procedural angiograms, as well as of protocols of procedures we evaluated the incidence of atypical anatomical variants of the origin of tibial arteries and of foot blood supply. Only the cases where the anatomy was univocally interpreted by two experienced interventional radiologists were taken into account. The cases of atypical anatomy were assigned to one of the two groups depending on time of their identification during endovascular intervention. The first group comprised the cases of atypical anatomy that were evident at the stage of preoperative selective angiography. The second group comprised the legs in which atypical variant of PA branching or foot blood supply was identified only during PAP. The intra-PAP complications caused by atypical anatomy and their clinical consequences were taken into consideration.

The preservation of the legs in the groups of patients with typical and atypical anatomy of the PA and foot arteries was determined using Kaplan-Meier method. Parametric and non-parametric statistical techniques were used for the identification of differ-

Table.

Distribution of atypical variants of the PA branching and of the foot blood supply in 248 cases of CLLI when PAP has been performed

TYPE	Cases in total n (%)	Evident before PAP n	Recognized during PAP n	Complications n
Type I				
I-B	3 (1,2%)	2	1	0
I-C	5 (2%)	3	2	1
Type II				
II-A	2 (0,8%)	1	1	0
II-B	4 (1,6%)	2	2	1
Type III				
III-A	8 (2,85%)	3	5	2
III-B	7 (2,82%)	2	5	2
III-C	2 (0,8%)	1	1	1
Total n (%)	31 (12,5%)	14 (45%)	17 (55%)	7 (23%)

ences during group comparison. P value of ≤0,05 was considered as statistically significant.

Results

The incidence of atypical variants of the PA branching and the foot blood supply in the group of study is presented in the Table.

Distribution of atypical variants of the PA branching and of the foot blood supply in 248 cases of CLLI when PAP has been performed

In total, atypical anatomy was noted in 31 cases (12,5%). Aplasia/hypoplasia of the tibial branches with DPA and/or PIA arising from the PeA were noted in 17 (6,8%) cases. Only 14 (45%) cases of atypical anatomy were evident at the stage of preoperative selective angiography. In seven (41%) of the remaining 17 cases (which could not be recognized before PAP) atypical anatomy has been suspected only after the occurrence of specific PAP-related complications. Atypical variants of PA branching were associated with 2 complications related to the search of the ostium of the posterior tibial artery (PTA) in its "typical" location (Fig. 2). In 5 cases with foot blood supply from the PeA system the attempt of recanalization of hypoplastic tibial arteries resulted in their perforation. Clinical consequences have been noted in 1 (20%) of these cases — the perforation of the anterior TA led to hematoma development in the anterior crural muscular bed, which necessitated surgical decompression.

In the group with atypical anatomy technical success was achieved in 28 out of 31 cases (90,3%), while the rate of success in the group of 217 cases without anatomical variants was 93,5% (203 cases). The comparison of these results with the use of exact Fisher's test gave P=0,8. Herewith both groups were similar in age, sex and concomitant pathology. One-year rate of leg preservation was 83,7% for the cases with atypical and 85,9% — for the cases with typical anatomy. The comparison of Kaplan-Meier curves using LogRank Test did not reveal significant difference (P=0.65).

Discussion

According to the data of two large angiographic studies, atypical variants of PA branching and foot blood supply were seen in 7,4% and 17,6% of cases (3, 4). Our results (12,5%) are within these limits. In our opinion, the problem of specific complications caused by atypical anatomy still represents a pressing challenge, despite the absence of significant differences in the rate of technical success and clinical indices in the groups with typical and atypical anathomy.

The incidence of interventions on distal arterial segments during PAP for CLLI is steadily increasing. Most patients in our study had ischemic diabetic foot (76%), and extended occlusions of lower leg arteries is the main type of lesion in such patients. The attempts of intraluminal or subintimal recanalization of a hypoplastic ATA or PTA, at best, will be an exercise in futility, and in the worst will end by perforation and/or occlusion of the artery, with eventual clinical consequences for the patient. The differentiation between an occluded "normal" and hypoplastic tibial artery is not always feasible even in the presence of selective intraoperative angiographic data. In our study only 45% atypical anatomical variants have been recognized before the start of PAP, while in 23% of cases they were revealed only after the development of complications. Most complications (5 out of 7) occurred in the group of patients with type III atypical anatomical variants. In 32% of cases an atypical variant has been suspected on the base of indirect signs, which allowed to change the tactics of intervention and avoid complications. One such case is presented in Fig. 3. If possible, it is necessary to carefully evaluate the blood supply in the other leg, as according to the literature, atypical variants of blood supply in both legs occur in 27,5% — 50% of cases (10, 11).

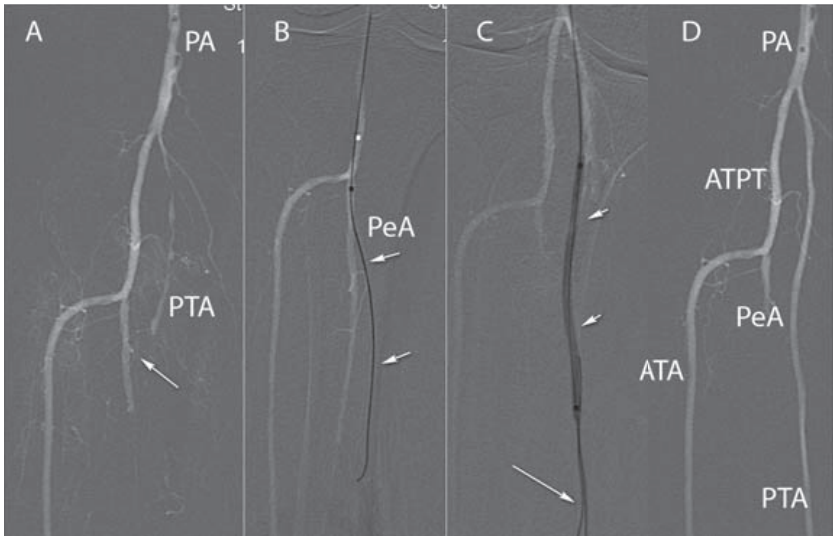


Fig. 2. Angioplasty in high PA branching and anterior tibioperoneal trunk (ATPT).
A *Angiogram of the PA after successful subintimal recanalization of the anterior tibial artery (ATA). The variant of PA branching was erroneously interpreted as typical. Diffusely stenotic and occluded branch of the PA, arising at the level of the cleft of the knee was not recognized as the posterior tibial artery (PTA). The arrow indicates the assumed ostium of the PTA.*
B *The attempts to find the ostium of the posterior tibial artery in its "typical" location led to the perforation with guide (arrows) of a small branch of the peroneal artery (PeA), that had a course similar to this one of a typical PTA;*
C *Subintimal angioplasty of the true PTA was performed. Short arrows indicate the balloon inflated in its lumen in order to facilitate the advancement of a hydrophilic guide' loop (long arrow)*
D *Final result: both tibial arteries are reconstructed. The II-B type is evident.*

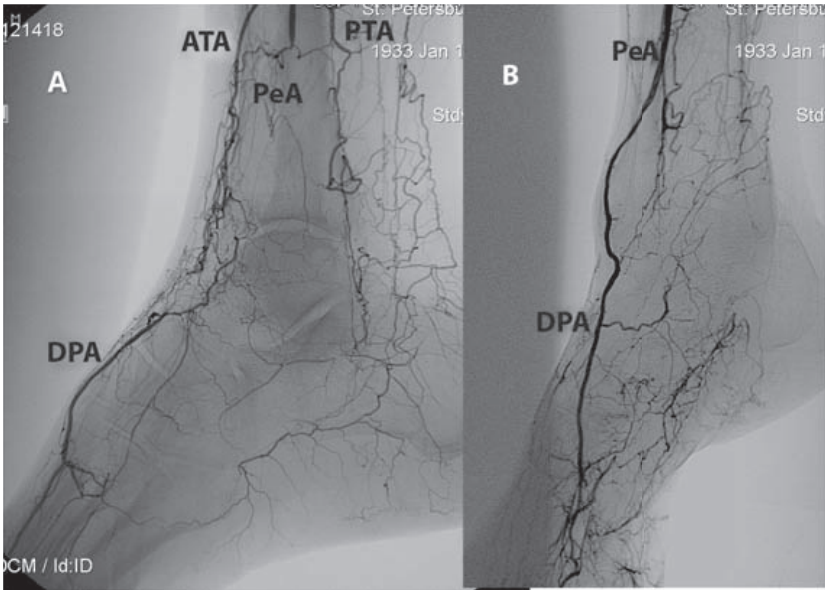


Fig. 3. Dorsalis pedis artery (DPA) arises from the peroneal artery (PeA).
A *Diagnostic stage of intervention: supposed hypoplasia of the anterior tibial artery (ATA), occlusion of the peritoneal artery (PeA) and of the posterior tibial artery (PTA). Large diameter and direct course of the PeA in association with the small tortuous ATA allowed to suppose the DPA origin from the PeA.*
B *Angiograms of final results of PAP: the DPA is a branch of the PeA.*

Maybe, the use of x-ray technique, allowing, unlike contrast angiography, to visualize not only the lumen, but also the course of occluded arteries, for preoperative evaluation of the vascular status will be useful for the recognition of atypical anatomical variants before the procedure. However this approach requires task-oriented studies in patients with CLLI.

We are aware of certain limitations of our study, as it does not take into consideration the cases where even after PAP we could not clearly determine the anatomical variant. It is also probable, that some cases interpreted as foot arteries origin from the peroneal artery, in fact could represent hypertrophied collaterals. However we are sure that our experienced with PAP in patients with atypical anatomy of tibial and foot arteries will be useful for specialists dealing with endovascular treatment of patients with CLLI.

References

1. Menzoian J.O., LaMorte W.W., Paniszyn C.C. et al. Symptomatology and anatomic patterns of peripheral vascular disease: differing impact of smoking and diabetes. Ann. Vasc. Surg., 1989, 3, 224-8.

2. Graziani L., Silvestro A., Bertone V. et al. Vascular involvement in diabetic subjects with ischemic foot ulcer: a new morphologic categorization of disease severity. Eur. J. Vasc. Endovasc. Surg., 2007, 33, 453-60.

3. Kim D., Orron D.E., Skillman J.J. Surgical significance of popliteal arterial variants. A unified angiographic classification. Ann. Surg., 1989, 210, 776–81.

4. Mavili E., Donmez H., Kahriman G. et al Popliteal artery branching patterns detected by digital subtraction angiography. Diagn. Interv. Radiol., 2011, 17 (1), 80-3

5. Lippert H., Pabst R. Arterial variations in man: classification and frequency. Bergman Verlag, Munich, 1985, 60–4.

6. Kawarada O., Yokoi Y., Honda Y., Fitzgerald P.J. Awareness of anatomical Variations for infrapopliteal interventions. Cathet. Cardiovasc. Interv., 2010, 76, 888-94.

7. Bolia A., Miles K.A., Brennan J., Bell P.R. Percutaneous transluminal an-

gioplasty of occlusions of the femoral and popliteal arteries by subintimal dissection. Cardiovasc. Intervent. Radiol., 1990, 13, 357–63.

8. Bolia A, Sayer RD, Thompson MM, et al. Subintimal and intraluminal recanalization of occluded crural arteries by percutaneous balloon angioplasty. Eur. J. Vasc. Surg., 1994, 8, 214 –9.

9. Rutherford R.B., Baker J.D., Ernst C., et al. Recommended standards for reports dealing with lower extremity ischemia. Revised version. J. Vasc. Surg., 1997, 26, 517-38.

10. Day C.P., Orme R. Popliteal artery branching patterns - an angiographic study. Clin. Radiol., 2006, 61, 696-9.

11. Keen J.A. A study of the arterial variation in the limb with special reference to symmetry of vascular pattern. Am. J. Anat., 1960, 108, 245-61.

Clincial Case of Successful Two-Stage Correction of type 1 Aortic Dissection Using Hybrid Surgical Approach

R.S. Akchurin[1], T.E. Imaev, A.E. Komlev, M.R. Osmanov, T.N. Veselova, M.E. Nikonova,I.A. Pokidkin Fedotenkov, A.S. Kolegaev

Department of Cardiovascular Surgery, Russian Cardiological Scientific and Production Complex of the Ministry of Healthcare and Social Development of the Russian Federation, Moscow, Russia.

Aortic dissection is an exclusively emergency situation with the incidence twofold higher than the incidence of abdominal aortic rupture, that is 10-20 cases per 1 mln. of population per year. About 20% of patients die before admission, 30% die in hospital and up to 20% — within the next 10 years.

Concomitant insufficiency of the aortic valve, ring and aortic root dilatation occurring in 50-60% of cases usually requires surgical replacement of the aortic valve, or aortic valve replacement with ascending aortic grafting following Bentall-De Bono technique, or some valve-preserving operations in David and Yacoub modifications.

Innovative hybrid surgical approach as a method of effective revascularization, combines the effectiveness of traditional open surgery and of percutaneous endovascular intervention with balloon valvuloplasty and endograft implantation. Due to its minimal invasiveness, the hybrid approach allows to minimize the complications and to decrease the mortality in high-risk patients.

The purpose of this paper is to present a case of successful use of hybrid approach for the correction of a chronic dissecting aortic aneurysm of type 1, extending from the level of Valsalva sinus and associated with aortic valve insufficiency.

The configuration of the thoracic aortic arch and the design of endografts often require more proximal zone for endograft fixation. Isolated endovascular intervention becomes risky because of eventual closure of aortic arch branches with the endograft. Hybrid surgical interventions possess all advantages of endovascular and traditional open surgery, thus allowing to avoid dangerous complications and minimize the duration of rehabilitation.

Keywords: *: hybrid surgery, thoracic aortic aneurysms, open surgery, endovascular interventions, endograft.*

Introduction

Aortic dissection is an exclusively emergency situation with the incidence twofold higher than the incidence of abdominal aortic rupture, that is 10-20 cases per 1 mln. of population per year. About 20% of patients die before admission, 30% die in hospital and up to 20% — within the next 10 years (1-4).

Aortic dissections can be classified by their location, length and presence of ischemic complications. According to one of the most often used Stanford classification, type A dissection involves the ascending, and type B — the descending aorta. De Bakey's type I involves the whole aorta, type II — the ascending aorta, type III –the descending aorta.

Atherosclerotic degeneration of the aortic wall in hereditary diseases, eg. Marfan and Ehlers-Danlos syndromes, and in arterial hypertension, are facrots predisposing for dissection. The fenestrations in the intima permit the blood flow to enter into the aortic media, thus peeling the intima away from the aortic wall and forming two lumen — the true and the false ones. The exfoliation of the aortic wall can produce an obstruction, rupture or thrombosis of the aortic branches. The obstruction can be dynamical or static, leading to malperfusion. Repeated blood flow into the true lumen decrease dynamical load on the aortic wall, that causes spontaneous fenestrations. The formation of a false aortic lumen leads to a dangerous sequence of events, that constitute the indications for emergency intervention: rapid formation of aortic aneurysm, ongoing organs' ischemia caused by the obstruction of the major aortic branches, unstable hemodynamics, cardiac tamponade, aortic rupture.

As a rule, the symptoms are very sharp, with marked chest pain irradiating into the neck or arms. Backache is usually related to type B dissection. Other symptoms depend on the degree of dissection and other organs' involvement. Abdominal pain, renal failure, paraplegia, lower limb ischemia can be present.

Type A dissections are particularly dangerous and without treatment or in case of inappropriate diagnosis are associated with high mortality. Pharmacological therapy is not a basis for the management of type A dissections. Mortality rate at early stages is 1-2% per hour (5). Mortality can be as high as 40% per week, which is significant in comparison with about 10% mortality associated with open surgical interventions.

In type B dissections hypotensive therapy usually is applied. The purpose of treatment consists in the

[1] Address for correspondence:
Dr. Renat Akchurin,
Russia, 121552, Moscow,
3rd Cherepkovskaya ul, 15a
Tel. /Fax: 007 499 140-93-36, 007 499 149-17-08
Email: dddd99999@mail.ru
Manuscript received on June 06, 2011.
Accepted for publication on August 22, 2011.

limitation of dissection, minimization of the impact on the aortic wall, prevention of dilatation and creation of conditions for false lumen thrombosis. Surgical mortality can be as high as 30% or even more (5).

Concomitant aortic valve insufficiency, ring and aortic root dilatation present in 50-60% of cases of aortic dissection usually require surgical aortic valve replacement, or aortic valve replacement with ascending aorta grafting using Bentall-De Bono technique, or valve-preserving surgery using David and Yacoub technique.

The fenestrations in the aortic arch occur in 30% of dissection cases. In case of fenestrations in the aortic arch, distal anastomosis between the graft and the aorta is performed to bypass a part of aortic arch, not involved in dissection. In any case, if prolonged multiple fenestrations extend beyond the site of aortic arch passage into the descending part of the aorta, it is necessary to perform subtotal or total arch replacement with rebranching of several or all aortic branches into the graft. This is performed in the settings of hypothermic circulatory arrest and antegrade cerebral perfusion. If the dissection involves also the descending aorta, the "elephant trunk" aortic arch reconstruction is indicated.

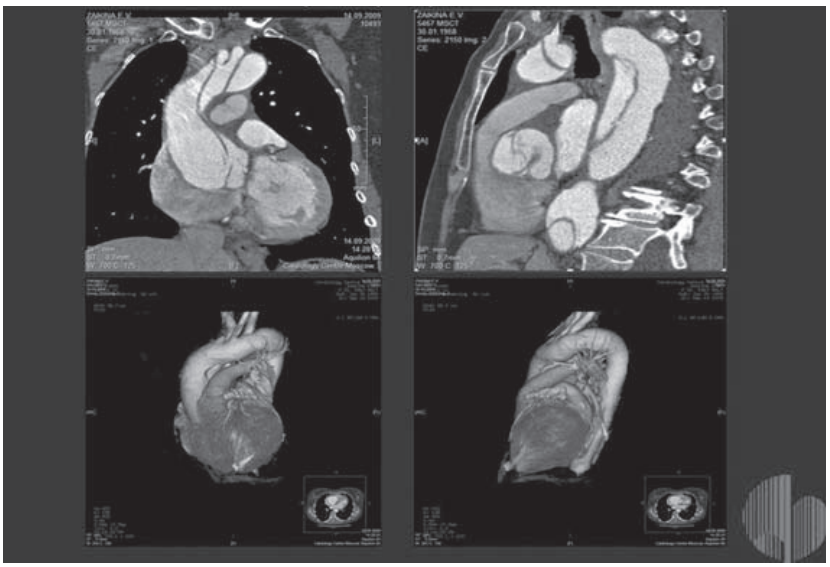


Fig. 1. MHCT of patient Z

Most patients with aortic dissection are severely ill and unlikely to survive traditional open surgical intervention with deep circulatory arrest, aortic arch reconstruction, etc. Such high-risk patients, as well as the complications occurring during open heart surgery and pharmacological therapy, have stimulated the search of new technologies for the treatment of aortic dissections — minimally invasive endovascular interventions.

In general endovascular treatment is being used for both types of aortic dissection — A and B, — both in acute and in chronic forms. Recent results of meta-analyses have shown technical feasibility of endovascular intervention in 98% of cases, as well as encouraging immediate and mid-term results (1-2 years) (6, 7). The advantages of endovascular interventions and hybrid surgery for aortic dissections in comparison with traditional open surgery are evident. In particular, endovascular approach allows to avoid thoracotomy with subsequent formation of pleural adhesions (8-11). Usually the procedure is performed through the femoral approach. Unlike open operation, primary or repeated endovascular intervention can be performed under spinal or even local anesthesia. It is evident that in high-risk patients endovascular interventions have significant advantages over surgical treatment and offer hope for recovery for patients with contraindications for open surgery. These advantages comprise the decreased rate of mortality and complications, the minimization of time spent in the ICU and in hospital, as well as of postoperative rehabilitation.

Innovative hybrid surgical approach as a method of effective revascularization, combines the effectiveness of traditional open surgery and of percutaneous endovascular intervention with balloon valvuloplasty and endograft implantation. Due to its minimal invasiveness, the hybrid approach allows to minimize the complications and to decrease the mortality in high-risk patients. In case of lower limb ischemia this approach is preferable for legs perfusion using extracorporeal circulation.

Clinical case

The purpose of our paper is to present a clinical case of successful two-stage correction of type I aortic dissection using hybrid surgical approach.

A 52-years-old woman was admitted to the Department of Cardiovascular Surgery on September 10, 2009 with complaints of restrictive retrosternal pain associated with minimal physical load, palpitations and pulsation of the neck vessels. History analysis revealed that the patient had poorly controlled arterial hypertension for over 20 years (max. BP 190/110 mm Hg). She has considered herself sick from May 20, 2009, when in the night she suddenly felt an intensive retrosternal pain irradiating into the neck and the mandible. After 40 minutes the pain has been stopped with Nitroglycerin. In-hospital examination excluded myocardial infarction, EchoCG revealed severe aortic valve insufficiency. Within 2 weeks after the pain attack her body temperature remained high — 38^{0}C. Her condition at admission was severe, with respiration rate at rest — 25/min. and visible pulsation of the cervical vessels. A loud diastolic murmur heard over the whole heart, radiating to the carotid arteries, interscapular space, as well as a systolic murmur with maximal

loudness in the 2nd right intercostal space were revealed. HR — 50 b/min.. BP in the right arm — 180/70 mm Hg, in the left arm — 160/65 mm Hg.

ECG: sinus bradycardia with HR 50b/min., transient I degree AV block (PQ 180-480 msec.), signs of LV overload and hypertrophy.

EchoCG: the diameter of ascending aorta is enlarged up to 6.0 cm, the aortic root — 5.0 cm. Left atrium — 3.5 cm, LVEDS — 6.5 cm, LVESS — 3.9 cm, LV EF — 55%. 1st degree mitral and tricuspid regurgitation. 3rd degree aortic regurgitation. The exfoliation of the aortic intima is seen immediately after the aortic valve cusps and extends up to the brachiocephalic trunk. The false lumen is significantly larger than the true one, without signs of thrombosis.

Coronary angiography, aortography: the left main coronary artery, the LAD and the CxB with irregular contours. The angiographically intact RCA arises from the false channel. At the level of the right coronary sinus there is a large communication between the aorta and the false channel, with its lumen extending in the ascending aorta up to the brachiocephalic trunk ostium.

MHCT of the aorta (Fig.1): dissecting aneurysm of the ascending aorta starting at the root, with multiple intimal fenestrations at the root level. Maximal dilatation of the aorta at the level of the right coronary sinus — 6.0 cm, the diameter of the ascending aorta — 4.0 cm, of the aortic arch — 2.7 cm, of the descending segment — 2.2 cm. The false lumen starts from the right sinus of Valsalva, and has a helix-like course, the RCA arises from the false lumen. A proximal fenestration is located near the isthmus, aortic dissection extends to the level of the common iliac artery on the left and to the external iliac artery on the right. The brachiocephalic trunk and the left common carotid artery arise at the border of the false and true lumens, the dissection does not spread into the proximal segments of brachiocephalic arteries.

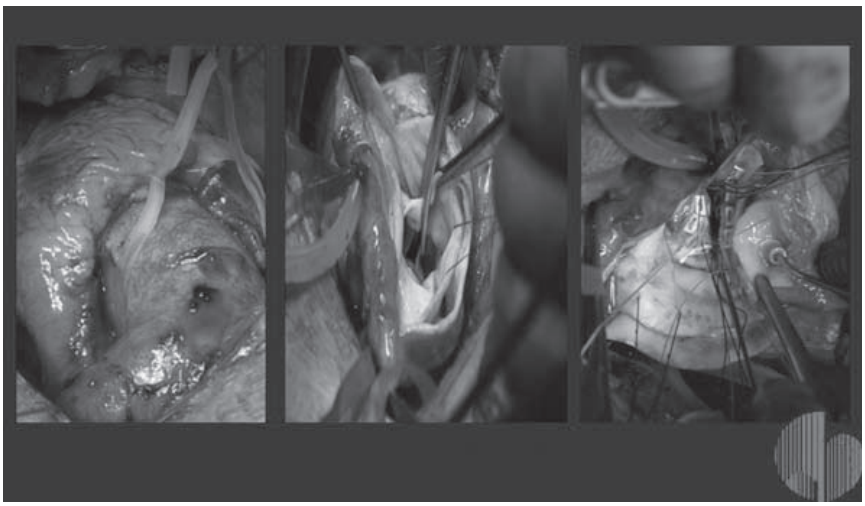


Fig. 2. First stage — surgery. Replacement of the ascending aorta, reimplantation of aortic arch branches

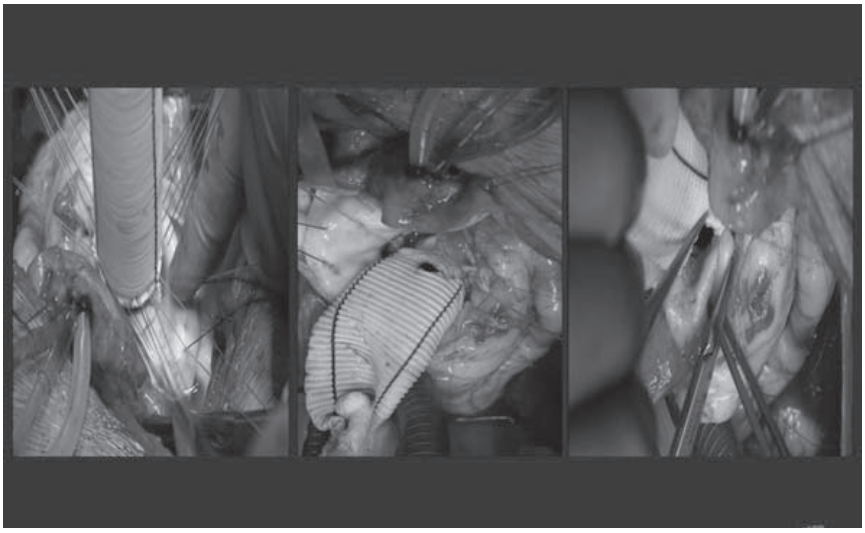


Fig 3.

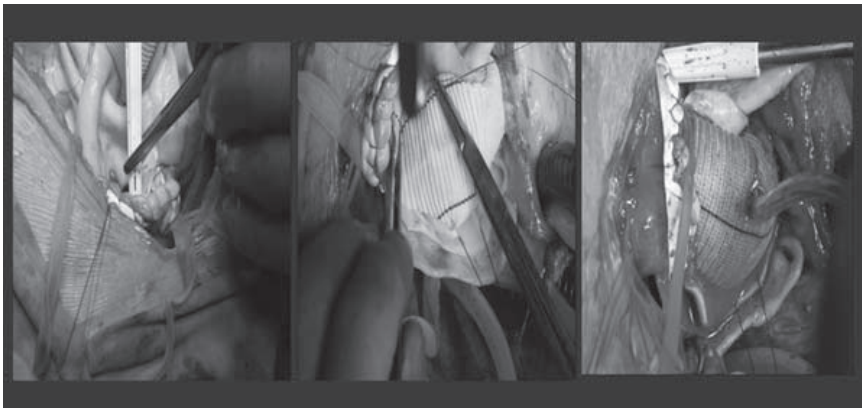


Fig. 4. Duration of extracorporeal circulation — 184 min.; of aortic cross-clamping — 125 min.; of circulatory arrest — 31 min.

The diagnosis comprised: chronic large dissecting type I aortic aneurysm, starting at the level of the sinuses of Valsalva, severe aortic valve insufficiency, arrhythmia (transient 1st degree AV block), 3rd degree arterial hypertension. The patient was in NYHA class III.

The first stage of correction consisted in hybrid operation for aortic valve replacement with valved conduit (Figs. 2,3,4), during the second stage left-sided carotid-subclavian bypass and partial aortic

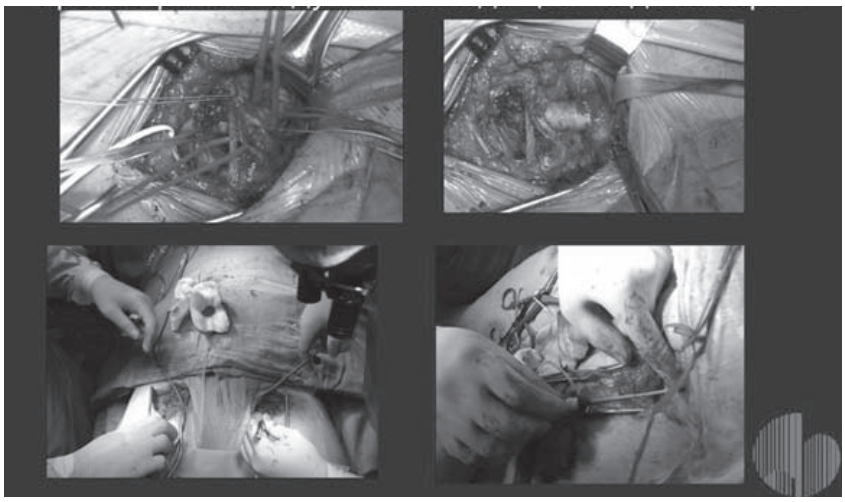


Fig. 5. Second stage — left-sided carotid-subclavian bypass grafting, endovascular grafting of the aortic arch and the descending aorta

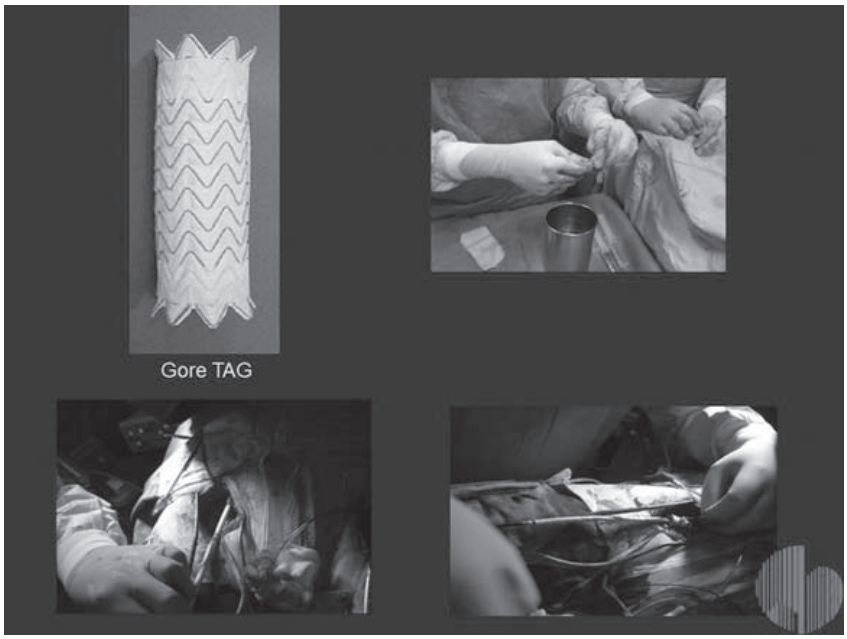


Fig. 6. Postoperative MHCT of patient A

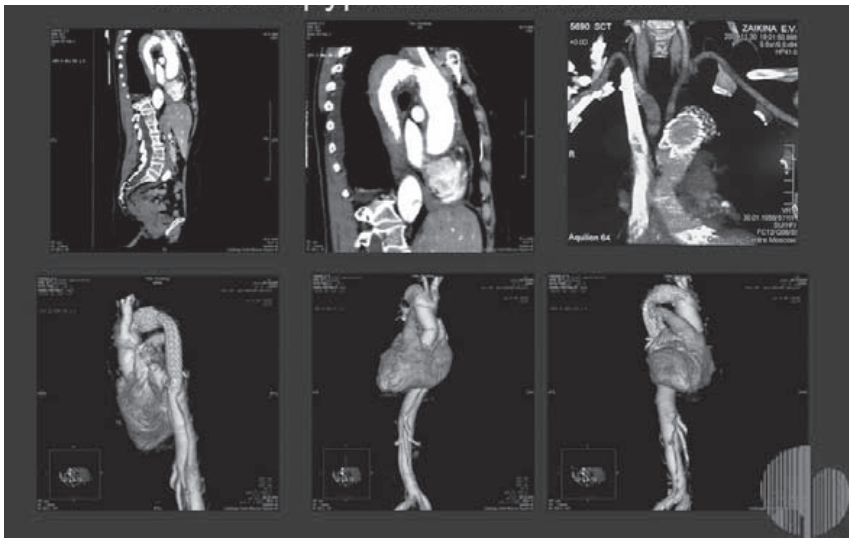


Fig. 7. MHCT-aortography of patient Z. after the 2nd stage of intervention

arch replacement with an endograft were performed (Fig. 5).

The first stage (October 4, 2010): the heart was approached through median sternotomy. A T-shaped aortotomy was performed above the sinoaortic area. The revision showed the dissection starting from the level of aortic valve's commissures, spreads into the anterior, lateral and posterior aortic walls involving the right coronary artery ostium, and then spreads into the aortic arch in the shape of a two-barreled gun. The button-like isolation of the coronary ostia was performed for subsequent implantation. The valve's cusps were sectioned. The aortic valve and the ascending aorta were replaced by a valved conduit Carbomedics Inc. (aortic valve prosthesis 25, graft diameter 28). The right and the left coronary arteries were reimplanted onto the conduit. The revision of the aortic arch revealed that the dissection spreading in the upper wall of the aorta, ends by a fenestration at the level of brachiocephalic trunk; the dissection involving the posterior and the lateral walls spreads into the arch and ends in the area of the left subclavian artery ostium. The aortic arch branches arise from the true lumen. The aortic arch was sectioned in oblique way, with the preservation of the upper wall and a part of lateral walls. The false lumen was eliminated and the distal anastomosis line was reinforced with internal and external padded sutures (the "layered cake"). The distal anastomosis between the conduit and the aorta was performed using the open technique.

The second stage (October 7, 2010): the procedure was performed under endotracheal anesthesis. The approach to the left common carotid artery and the left subclavian artery was achieved through the supraclavicular incision. An "end-to-side" anastomosis was performed between the subclavian artery and the common carotid artery. Simultaneously an introducer was inserted into the left common femoral artery; on the right the introducer was inserted percutaneously. The diagnostic catheters were advanced by

the guidewires inserted through the introducers into the thoracic aorta. The position of diagnostic catheters in the true aortic lumen was confirmed. A super-stiff guidewire was inserted through the left diagnostic catheter and positioned in the thoracic aorta, and a Gore Tag 34x20 endograft was advanced by this guidewire. The endograft was implanted from the ostium of the left common carotid artery up to the diaphragm. Neither leakage nor false lumen filling were seen.

Results of study

The patient's rehabilitation was achieved within the shortest term. The patient was discharged at day 5. Postoperative course was uneventful. The patient has been followed in the long-term (1 month — 1 year), no complications were revealed. Control MHCT-aortography (Fig. 6) revealed Gore endograft positioned from the level of the left common carotid artery to the celiac trunk. The segment of false aortic lumen, with an entry fenestration, immediately under the ostium of the left subclavian artery, is filled by partially thrombotic masses, there is no blood flow in this segment.

Discussion

Traditional open surgical treatment of type I aortic dissection can leave uncorrected the false lumen of the arch and the descending thoracic aorta, thus causing aneurysm progressing up to its rupture, or distal malperfusion, which is accompanied by high morbidity and mortality.

The configuration of the thoracic aortic arch and the design of endografts often require more proximal zone for endograft fixation. Isolated endovascular intervention becomes risky due to the danger of eventual covering of the aortic arch branches with endograft.

The replacement of the ascending aorta and the aortic arch with a conduit with supraaortic vessels' debranching is a safe and effective method for the treatment of acute as well as of chronic aortic pathology, provides the creation of an optimal and safe zone for endograft fixation during endovascular stage of arch and descending aorta replacement, allows for partial solution of the problem of "oversized" endografts; optimizes the technique of surgery, significantly decreases the duration of circulatory arrest and extracorporeal circulation, allows to avoid deep hypothermia, prevents potential development of proximal endoleak, thus contributing to low rate of morbidity and mortality.

Hybrid surgical interventions possess all advantages of endovascular and traditional open surgery, thus allowing to avoid severe complications and minimizing the duration of rehabilitation period. This technique deserves to be included into the basic arsenal of surgical options used for revascularization.

In our opinion, hybrid surgery is a transition stage to the eventual primacy of endovascular intervetions. In the future, the use of fenestrated and multi-branch endografts could allow to completely exclude traditional open interventions on the aorta and its branches.

References

1. Advanced endovascular therapy of aortic disease. Ed. by Alan B. Lumsden et al. Blackwell Futura, Malden, 2007.

2. Endovascular and hybrid management of the thoracic aorta. Ed. by Edward B. Diethrich et al. Wiley-Blackwell, Hoboken, 2008.

3. Aortic surgery. New developments and perspectives. Ed. by C. Setacci et al. Edizioni Minerva Medica, Turin, 2009.

4. Comprehensive vascular and endovascular surgery. 2nd ed. Ed. by John W. Hallett et al. Mosby, Philadelphia, USA, 2009.

5. Hagan P.G., Nienaber C.A., Isselbacher E.M., et al. The international registry of acute aortic dissection (IRAD): New insights into and old disease. JAMA, 2000, 283, 897–903.

6. Lann L., Delle M., Falkenberg M., et al. Endovascular treatment of type B thoracic aortic dissections. J. Card. Surg., 2003, 18, 539–44.

7. Eggebrecht H., Nienaber C.A., Neuhauser M., et al. Endovascular stent-graft placement in aortic dissection: A meta-analysis. Eur. Heart J., 2006, 27 (4), 489–98

8. Appoo J.J., Moser W.G., Fairman R.M., et al. Thoracic aortic stent grafting: Improving results with newer generation investigational devices. J. Thorac. Cardiovasc. Surg., 2006, 131, 1087–94

9. Greenberg R.K., O'Neill S., Walker E., et al. Endovascular repair of thoracic aortic lesions with the Zenith TX1 and TX2 thoracic grafts: Intermediate-term results. J. Vasc. Surg., 2005, 41, 589–96.

10. Dake M.D., Wang D.S. Will stent-graft repair emerge as treatment of choice for acute type B dissection? Semin. Vasc. Surg., 2006, 19, 40–7.

11. Peterson B.G., Eskandari M.K., Gleason T.G., Morasch M.D. Utility of left subclavian artery revascularization in association with endoluminal repair of acute and chronic thoracic aortic pathology. J. Vasc. Surg., 2006, 43, 433–9.

Morphogenetic Particularities of Acute Myocardial Infarction with and without Early Thrombolytic Therapy

G.A. Nefedova (1)[1], I.E. Galankina (1), I.E. Chernysheva (2).
(1) Sklifosovsky Research Institute of Emergency Medicine,
(2) Moscow City Center of Interventional Cardioangiology, Moscow, Russia

Despite continuous improvement of therapeutic tactics, current mortality from acute myocardial infarction (AMI) remains high (3, 4). The extent of AMI being of significant importance for the outcome depends on the time of blood flow restoration in the ischemic myocardium and the formation of the collateral flow. This imposes the necessity to achieve an early and consistent reperfusion in the occluded vessel, resulting in the decrease of the necrotic area expansion, the improvement of the blood flow in the peri-infarction area, the decrease of electrical instability of the myocardium and the improvement of the residual left ventricular (LV) function. The results of different clinical trials suggest that the use of thrombolytic therapy (TLT) after AMI allows for a significant decrease of the rate of morbidity and mortality (1, 2, 5, 6). The information concerning the TLT-related complications — the increased rate of external heart rupture, intracerebral hemorrhage, bleeding from the puncture site, allergic reaction to thrombolytic agent — is rather abundant (1, 2, 7, 8). It is worth noting that in any of the analyzed cases we did not reveal severe TLT-related complications, caused by the disturbances of rheological properties of the blood (hemostasis) and playing an important role in the thanatogenesis. Pathoanatomical particularities of AMI development and mechanisms of thanatogenesis in early reperfusion (RP) for AMI are virtually not discussed in the literature.

The purpose of our study consisted in the determination of the influence of early (within 6 hours after the onset of pain syndrome) TLT on the particularities of the course of AMI and the development of its lethal complications.

Material and methods. In order to achieve this purpose we have studied the results of 230 specimens of hearts of patients who died after AMI with early TLT (50 cases, TLT group) and without early TLT (180 cases — non-TLT group) during the period from 2003 through 2010 (145 males, mean age 63,7 years, 85 females, mean age 67,65 years). The groups did not significantly differ by sex and age, one third of patients from each group were under 60 years old (Tables 1, 2). The material of the study was collected in the joint prosectorium of the Sklifosovsky Research Institute of Emergency Medicine, and comprises the lethal cases from the Institute and from other high-skilled cardiological departments.

We determined macroscopic localization, surface area and depth of the myocardial infarction (in planar and transverse sections) and evaluated the presence of hemorrhagic component (hemorrhages) in the necrotic area. In each case the extent and the localization of the AMI was analyzed in relation to the extent and the degree of atherosclerotic coronary stenosis, the presence of thrombus in the lumen of the infart-related artery (IRA) and the type of coronary circulation.

Comparative analysis of lethal cases occurring after AMI with and without early TLT revealed the following features.

One of the important pathoanatomical macroscopic criteria of the TLT effectiveness is the absence of thrombus in the IRA. Our study has shown a clear tendency to the decrease of the rate of IRA thrombosis in the TLT group ($p<0,05$). Average index of the number of thrombi in the non-TLT group was 65% (117 of 180), while in the TLT group is was twofold lower — 34% (17 of 50).

The rate of IRA thrombosis was significantly decreased ($p<0,05$) with the increase of the number of the involved branches, as well as the involvement of the middle and distal segments of the coronary arteries (CA) into the atherosclerotic process in both groups (Fig. 1). In combined atherosclerotic stenosis of two or three coronary branches the rate of thrombosis of the IRA in the non-TLT group (68% of cases, 122 of 80) was 57% (69 of 122). In the TLT group the rate of AMI developed against the background of multivessel coronary disease was 64% (32 of 50), the occluding thrombus in the lumen of one CA was found only in 38% of cases (12 of 32).

The difference is mostly evident in the presence of AMI developed in the settings of isolated atherosclerosis of one CA. In the non-TLT group such AMI was revealed in 32% of cases (58 of 180). The rate of thrombosis of a single involved CA in this group was 83% (48 of 58). In the TLT group the single-vessel coronary disease was seen somewhat more frequently — in 36% of cases (18 of 50), and the rate of thrombosis of a single involved CA was significantly lower — 28% (5 of 18).

The second pathological macroscopic criterion of TLT impact is the presence of a hemorrhagic compo-

[1] Address for correspondence:
Нефедова Галина Александровна,
НИИ им. Склифосовского,
129090, Москва, Большая Сухаревская пл., 3
Тел.
e-mail: nefe_ga@mail.ru
Статья получена 3 августа 2011 г.
Принята в печать 10 октября 2011 г.

Table 1.

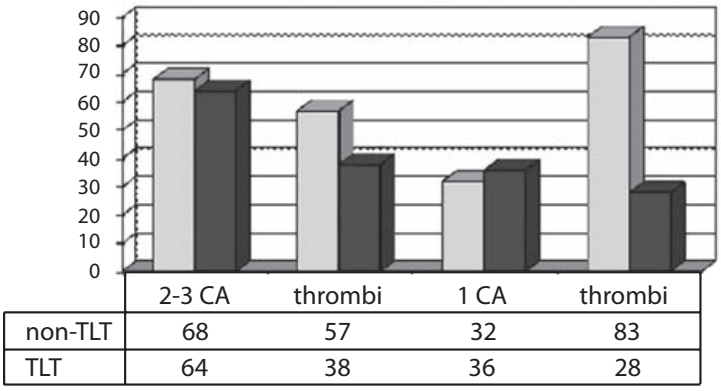
Distribution of cases by sex and age in TLT group

Sex	41 – 50		51 – 60		61 – 70		> 71		Total	
	Абс	%	Абс	%	Абс	%	Абс	%	Абс	%
Males	2	4%	8	16%	9	18%	9	18%	28	56%
Females	0	0	6	12%	7	14%	9	18%	22	44%
Total	2	4%	14	28%	16	32%	18	36%	50	100%

Table 2.

Distribution of cases by sex and age in non-TLT group

Sex	41 – 50		51 – 60		61 – 70		> 71		Total	
	Абс	%	Абс	%	Абс	%	Абс	%	Абс	%
Males	13	7,2%	35	19,4%	38	21,1%	31	17,3%	117	65%
Females	0	0	18	10%	25	13,8%	20	11,2%	63	35%
Total	13	7,2%	53	29,4%	63	35%	51	28,4%	180	100%



	2-3 CA	thrombi	1 CA	thrombi
non-TLT	68	57	32	83
TLT	64	38	36	28

Fig. 1. The rate of stenotic atherosclerosis in thrombosis in the coronary branches

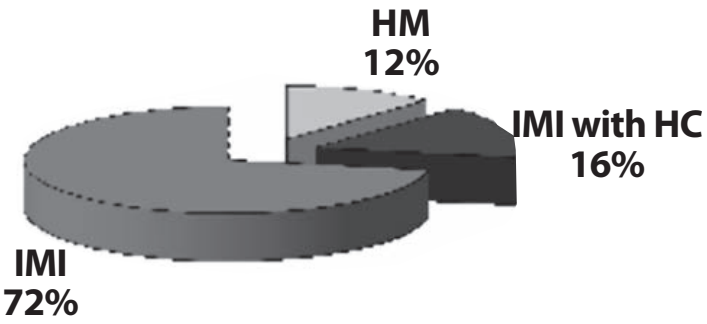


Fig. 2. The rate of various forms of myocardial infarction in the TLT group.

nent (HC) in the myocardial infarction area (within the central sites of a formed necrotic focus).

The intensity of HC in the myocardial necrotic focus was different.

The cases with diffusely dark-red coloration of the necrotic zone appearing within the first hours after the onset of the disease (min. 2 hours) were considered as hemorrhagic myocardial infarction (HMI) — such cases made 12% (6 of 50). Commonly HMI was located transmurally, somewhat less frequently — subendocardially and subepicardially (Fig. 3). As a rule, the HMI had clear borders, a faint pale-yellow peri-infarction area of varying width was seen at the periphery of the focus; this zone had rather distinct boundaries with the preserved myocardium.

In 16% of the other cases (8 of 50) the infarction was represented by a faint pale-yellow focus with distinct borders and multiple merging dark-red hemorrhages — an ischemic myocardial infarction with hemorrhagic component (IMI with HC). Most commonly the hemmorhages were revealed in the peripheral segments of the focus, somewhat less frequently — in its central area (Fig. 4).

Ischemic myocardial infarction (IMI) was found in 72% of cases (36 of 50) in the TLT group (Fig. 5).

Unlike the TLT group, ischemic infarction (without hemorrhagic component) was found in all 180 cases in the non-TLT group. Within the first hours the IMI either could not be revealed macroscopically, or its area seemed somewhat paler than the surrounding myocardium. Only by the end of the first day the infarction's borders became relatively distinct, and its color changed to faint pale-yellow. No hemorrhages were seen neither in the center, nor at the periphery of the focus.

We analyzed the association of hemorrhagic component in the myocardial infarction area with the absence on intracoronary thrombus in the IRA.

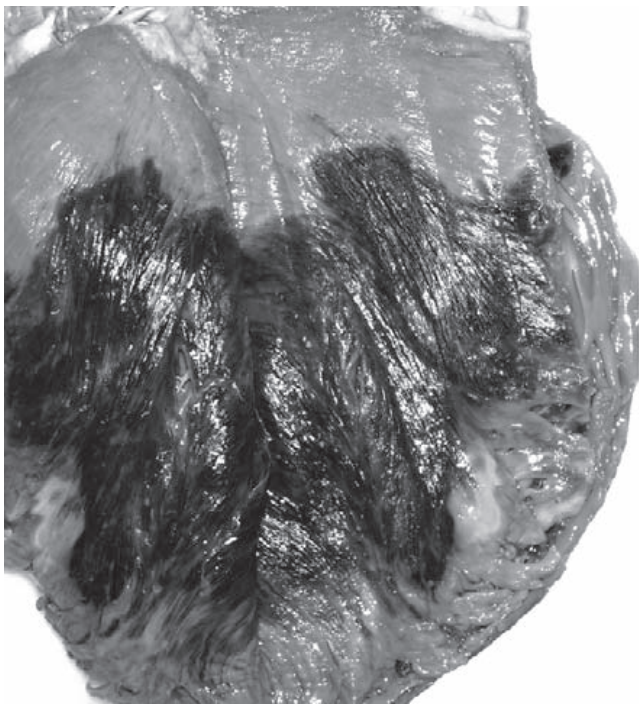


Fig. 3. Hemorrhagic myocardial infarction

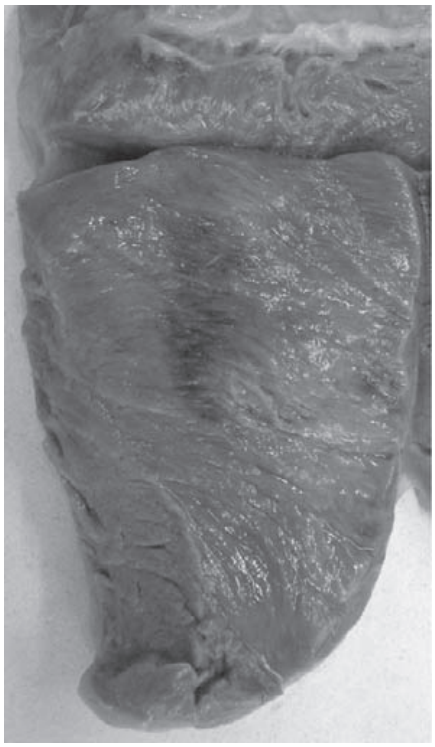


Fig. 4. Ischemic myocardial infarction with hemorrhagic component

In the TLT group of our series the presence of a HC in the area of AMI was not always associated with the absence of thrombus in the IRA. In 12 cases of AMI with the signs of accomplished RP after TLT (i.e., with HC of various intensity in the infarction area) the intracoronary thrombus was absent. Unlike this observation, in 2 cases of HMI a thrombus was present in the lumen of the left coronary artery.

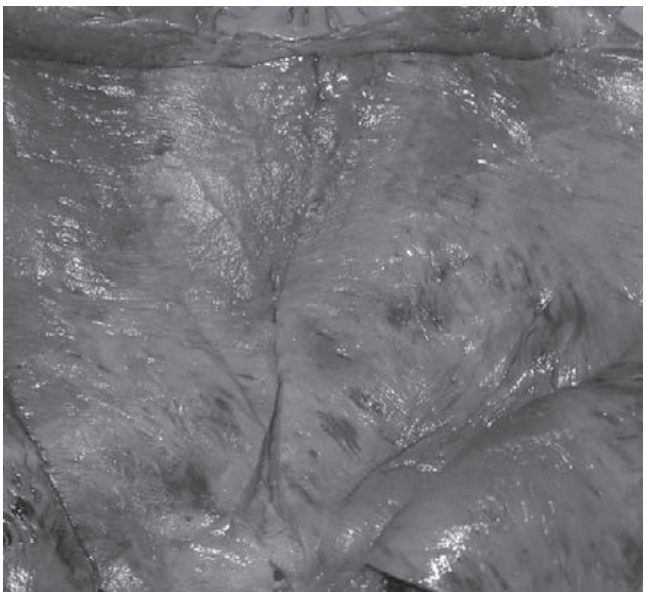


Fig. 5. Ischemic myocardial infarction

Comparative analysis of AMI-related lethal cases in both groups says for the tendency to decreasing area of contractile myocardial lesion after TLT ($p<0,05$).

Non-extensive (< 30% of the working surface of the LV) AMI was seen only in 22% of cases (40 of 180) in the non-TLT group (Fig 6), and in 34% of cases (17 of 50) in the TLT group.

In the non-TLT group, non-extensive AMI was more commonly associated with atherosclerotic stenosis of one CA — 67% of cases (27 of 40) with a lumen-occluding thrombus in 78% (21 of 27). Rarely severe atherosclerosis of all coronary branches was revealed in 33% of cases (13 of 40), the frequency of IRA thrombosis was 59%.

In the TLT group, non-extensive AMI was more commonly associated with severe atherosclerosis of all coronary branches — 59% (10 of 17), with a lumen-occluding thrombus in one half of cases (5 of 10). Atherosclerotic stenosis of one CA in this group was revealed in 41% of cases (7 of 17), and the rate of thrombosis was only 29% (2 of 7). The LAD or the RCA were the most common sites of isolated lesions.

Fatal complications of a non-extensive AMI in the groups of study were different (Fig.7 A, B).

In the non-TLT group the death was most commonly caused by external heart rupture (EHR, 55%, 22 of 40), by decompensation of an important concomitant disease, or resulted from thromboembolic complications (25%, 10 of 40).

Unlike these findings, in the TLT group a non-extensive AMI most commonly — in 53% of cases — led to complex heart rhythm and conduction disturbances (9 cases out of 17), and only in 41% (7 of 17) the death was caused by EHR or the decompensation of an important concomitant disease.

A certain tendency towards the increase of life duration was noted in the TLT group.

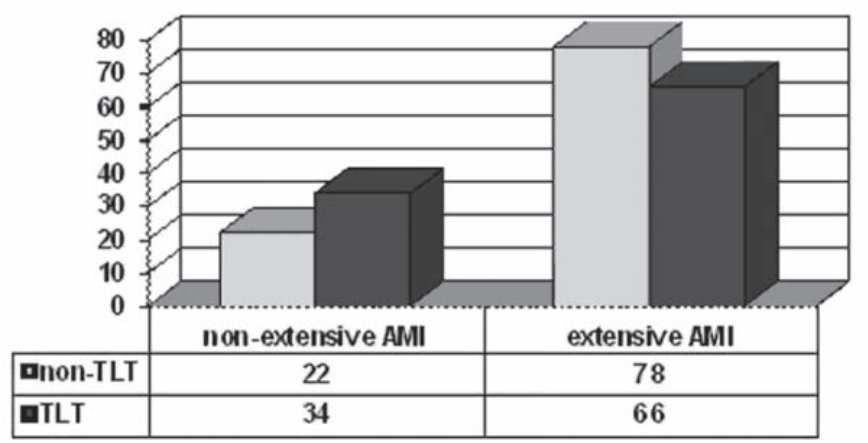


Fig. 6. The rate of extensive and non-extensive myocardial infarction in the groups of study

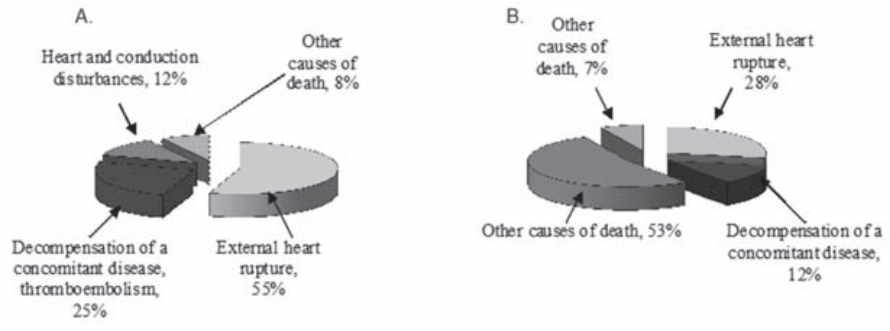


Fig. 7. Fatal complications of non-extensive AMI in the non-TLT (A) and the TLT (B) groups

TLT gave clinically positive results in 30% of cases with non-extensive AMI (5). In 3 cases the section revealed a hemorrhagic component of different intensity in the area of AMI without coronary thrombosis. The death occurred at day 2-9 from the onset of the disease and the administration of TLT, and was due to complex rhythm and conduction disturbances or EHR.

However in 70% of cases on non-extensive AMI, the TLT did not result in the improvement of the patients' condition and positive changes of the ECG (12). An occluding thrombus in the coronary artery was found in 7 of these cases, and HMI — in 3. The patients died within the first hours after the onset of the disease from EHR and complex rhythm and conduction disturbances.

An extensive AMI was found in the majority of the analyzed cases (Fig. 6). Its surface area varied from 40 to 80% of the working surface of the LV, in cases of re-infarction the cumulative surface of myocardial damage was 85-90%. Rather frequently the AMI involved the papillary muscles, less often — the right ventricular myocardium. This was seen in 78% of cases in the non-TLT group (140 of 180), and in 66% (33 of 50) in the TLT group.

The extensiveness of the AMI commonly was determined by severe atherosclerotic stenosis of all coronary branches: 78% of cases in the non-TLT group (109 of 140) and 67% of cases in the TLT group (22 из 33). Fibrotic and calcified atherosclerotic plaques narrowing the arterial lumen by 75-90%, were found in all segments of the involved CA, in the second-order branches. A lumen-obturating thrombus was found in 59% of cases in the non-TLT group (64 of 109), and more rarely– in 32% of cases — in the TLT group (7 of 22).

Sometimes an extensive AMI developed in atherosclerotic stenosis of one CA: 22% of cases in the non-TLT group (31 of 140) and 33% of cases in the TLT group (11 of 33). Alike the non-extensive AMI, isolated lesions were found most commonly in the LAD or the RCA. A single atherosclerotic plaque, narrowing the lumen by 75-90%, usually was located in the proximal segment of the involved CA. Up to 50% stenosis was found in the distal segments as well as in other branches of the CA. It is worth noting that an occluding thrombus was revealed in the vast majority of cases in the non-TLT group — 87% (27 of 31), and only in 27% of cases in the TLT group (3 of 11).

The character of lethal complications was similar in the groups with extensive AMI (Fig. 8 A, B). One half of patnets in both groups died from true cardiogenic shock (TCS) and acute left ventricular failure (ALVF), and in the one third of cases — from complex rhythm and conduction disturbances.

As well as in non-extensive AMI, a certain tendency towards the increased life time was noted. TLT was clinically effective in 18% of cases (6) of extensive AMI, the patients' condition improved, pain syndrome did not recur, positive ECG changes were noted. In two of these cases the section revealed a hemorrhagic component of different intensity in the infarction area, which is suggestive of the presence of reperfusion syndrome. In 5 of these cases no intracoronary thrombi were revealed. The patients died at day 3-12 after TLT, with the signs of rhythm and conduction disturbances and ALVF caused by the extensive lesion of the contractile myocardium. Along with AMI many patients had other, not less severe diseases — cancer, decompensated diabetes mellitus, chronic pyelonephritis with renal failure — that have played an important role in thanatogenesis.

In 82% of other post-TLT lethal cases due to extensive AMI (27) the patients' condition did not improve and there were no positive ECG changes. The patients died from ALVF, TCS, complex rhythm and

conduction disturbances at day 1-2 (22 cases) and at day 3-5 (5 cases) after the onset of the disease. However one cannot completely exclude the fact of accomplished reperfusion, as in 11 of these cases the section has revealed either the HMI or the hemorrhagic component of different intensity within the necrotic focus; in 9 cases there were no intracoronary thrombi. In another 11 cases the AMI was ischemic, with an occlusive thrombus present in 4 of these cases.

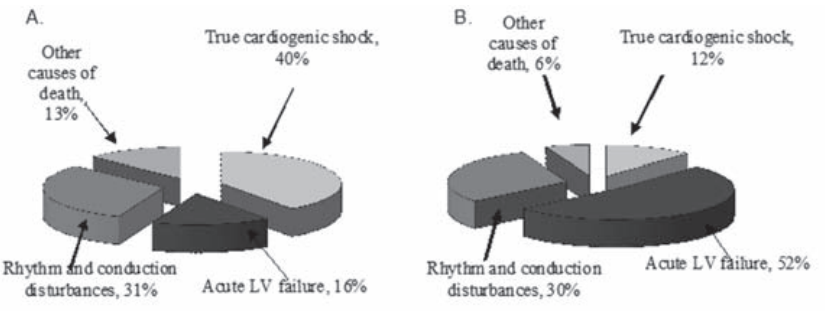


Fig. 8. Lethal complications of extensive AMI in the non-TLT (A) and TLT (B) groups

Therefore, comparative analysis of the deaths form AMI with and without early TLT reveals the following particularities. Firstly, they concern the frequency of IRA thrombosis with a clear tendency towards the decrease of the number of thrombi in the TLT group ($p<0,05$). Thus, the average rate of thrombosis in the non-TLT group was 65%, while in the TLT group — only 34%. This difference is mostly evident in the cases of AMI developed in the settings of isolated atherosclerosis of one CA. The rate of thrombosis in the non-TLT group was 83%, while the TLT group demonstrated an abrupt decrease of this value — 28%.

Another important distinguishing feature of the accomplished reperfusion (RP) is the presence of hemorrhagic component in the infarction area. Its intensity can be different. A bright earmark of reperfusion after TLT is diffuse hemorrhagic imbibition of the necrotic focus, it becomes dark-red and acquires the character of a hemorrhagic MI. We did not find HMI in the non-TLT group. In the TLT group HMI was found in 12% of cases. In 16% of cases the hemorrhages in the central parts of necrosis were focal and of different intensity. However in 72% of cases there were no focal hemorrhages within the IMI.

The presence of hemorrhagic component is not always associated with the absence of a thrombus in the IRA. Thus, in 3 cases the HMI developed in the presence of a thrombus in the left main coronary artery, which, probably, is suggestive of a temporal antegrade reperfusion and (or) is a result of collateral blood flow. Evidently, the opinion on blood flow restoration in the area of IMI after TLT should be based on the analysis of clinical parameters, such as positive dynamics of the course of AMI and regular deterioration of patients' condition as a result of reperfusion syndrome.

An extensive AMI was revealed after TLT in 66% of cases (with all CA involved — 67%, with a single-vessel disease — 33%), which is consistent with the data in the non TLT group (with all CA involved — 78%, with a single-vessel disease — 22%). The mechanisms of thenatogenesis were not significantly different. In both groups the extensive AMI was most commonly complicated by the true cardiogenic shock and acute left ventricular failure, and in one third of cases by complex rhythm and conduction disturbances.

Some data are suggestive of a tendency towards the increase of cases of AMI with a small surface area (up to 30% of the working surface of the LV). Thus, in the TLT group there were 34% of small AMI, and in the non-TLT — 22%; this could occur in the presence of three-vessel disease as well as in isolated atherosclerotic lesion of one CA. In such cases the mechanisms of thanatogenesis are significantly different. Thus, in the non-TLT group the most common cause of death was external heart rupture (55%) followed by decompensation of a concomitant disease (25%), while in the TLT group the most important cause of death was severe rhythm and conduction disturbances (53%, 9 out of 17 non-extensive AMI), and external heart rupture occurred only in 28% of cases (5 of 17).

References

1. Bokarev I.N.. Current achievements and problems of anti-thrombolytic therapy. Ter. Arkhiv, 1992, 10, 101 — 5.

2. International guidelines on myocardial infarction. Ronald V.F. Campbell, ed. Moscow, Informpoligraf 1997, 87 pp.

3. Machitidze E.Ts., Iosseliani D.G. Does Endovascular Reperfusion Affect In-Hospital Prognosis in Q-Wave AMI Patients Within the First Hours of the Disease? International Journal of Interventional Cardioangiology, 2009, 17, 10-3.

4. Nefedova G.A., Gazarian G.A., Galankina I.E. Particularities of thanatogenesis in myocardial infarction in the settings of thrombolytic therapy. «Current problems of pathological anatomy ». Proceedings of the 3rd Congress of Russian Society of pathological anatomists. Samara, 2009, 2, 367 — 9.

5. Ruda M.Ya. Acute circulatory insufficiency in myocardial infarction and its treatment. Clin, Farmakologia i Terapia, 1993, 4, 40 –46.

6. Di Mario C., Dudek D., Piscione F. et al. Immediate angioplasty versus standard therapy with rescue angioplasty after thrombolysis in the Combined Abciximab REteplase Stent Study in Acute Myocardial

Infarction (CARESS-in-AMI): an open, prospective, randomised, multicentre trial. Lancet, 2008, 371, 559–68.

7. Kober L., Torp-Pedersen C.T., Carlsen J.E. The significance ol trandelapril for mortality after AMI in patients with reduced lefl ventricular function. TRACE Study Group Trandolaprils betydnins for overlevels after AMI hos patienter med nedsat funktion afvenstre ventrikel. Ugeskr. Laeger, 1997, 159 (11), 10, 1616 — 22.

8. White H. Thrombolytic therapy in the elderly. Lancet, 2000, 356, 2028 — 30.

Clinical Case of a Successful Endovascular Management of Pulmonary Bleeding

E.R. Khayrutdinov[1], A.V. Arablinsky, V.N. Yakovlev.
Botkin City Clinical Hospital of Moscow Healthcare Department, Chair of therapy and adolescent medicine of Russian Medical Academy of post-graduate education, Moscow, Russia

The treatment of pulmonary bleedings is a complex and thrilling problem of modern surgery. The development of endovascular surgery allowed for the introduction of new methods for minimally invasive management of this pathology. The right choice of embolization technique, embolizing material, as well as the knowledge of anatomical particularities of pulmonary blood supply form the basis of successful management of these severely ill patients. This review gives an example of successful endovascular embolization of the left bronchial artery with polyvinyl alcohol particles in a patient with massive pulmonary bleeding.
Keywords: *pulmonary bleeding, bronchial artery, polyvinyl alcohol particles.*

Massive pulmonary bleeding is a serious medical problem associated with high mortality. Depending on the etiological cause, pulmonary bleeding -related mortality varies from 35 to 85%. The death from pulmonary bleeding is rarely caused by blood loss, in most cases it is related to blood asphyxia. Pulmonary bleeding is considered massive if 24-hours blood loss exceeds 300 ml (1-3). In the absence of etiotropic therapy pulmonary bleeding has recurrent character and is associated by 50% mortality within 6 months (4). The results of surgical treatment of this pathology remain unsatisfactory, in case of urgent surgery the mortality is as high as 40%. We present a case of successful endovascular management of pulmonary bleeding.

Patient B., male, aged 25, was admitted to Botkin City Clinical Hospital on October 18, 2008, with hemoptysis. Chest X-ray examination revealed foci-like shadows in the lower lobe of the left lung. Clinical picture and chest X-ray data gave the basis for differential diagnosis between tuberculosis and pneumonia of unclear etiology. Fiberoptic bronchoscopy (FBS) performed on the same day because of repeated pulmonary bleeding revealed a hemorrhage from the lumen of the left lower lobe bronchus. The bleeding was stopped by bronchus irrigation with aminocaproic acid and Etamsylate solution. However, in several hours the patient had recurrent pulmonary bleeding. Control FBS showed recidivating bleeding from the left lower lobe bronchus; repeated attempts of bronchus irrigation with hemostatic solutions were unsuccessful, and for this reason bronchus tamponade has been performed. The patient was transferred to the intensive care unit and mechanical lung ventilation (MLV), antibacterial and infusion therapies were initiated. General blood count revealed a decrease of hemoglobin concentration to 79 g/l, and in this connection erythrocyte mass transfusion was started. During the next three days the patient had several episodes of recurrent pulmonary bleeding, which required FBS and endoscopic hemostasis. Control chest X-ray revealed single foci-like shadows in both lungs. Computed tomography (CT) of thoracic organs revealed homogenous infiltration of both lungs' lower lobes. The diagnosis of bilateral aspiration pneumonia was made and antibiotic therapy with Tienam and Ciprofloxacin was started. Taking into account recurrent character and unclear etiology of pulmonary bleeding, as well as short-time effect of endoscopic hemostasis during FBS, it was decided to perform angiopulmonography and angiography of the bronchial arteries in order to find the source of bleeding and decide on the method of its embolization.

During angiopulmonography there were no evidences of contrast medium extravasation into the lung parenchyma (Fig. 1). Angiography of the descending thoracic aorta revealed a pathological arterial network from the left bronchial artery (Fig. 2).

Selective angiography of the left bronchial artery showed the increase of its diameter, with hypervascularized area and contrast medium extravasation into the parenchyma of the left lung's lower lobe (Fig. 3).

The left bronchial artery was embolized with polyvinyl alcohol (PVA) particles (500 μc). Control angiogram did not reveal signs of contrast medium extravasation, total occlusion of the left bronchial artery was achieved (Fig. 4).

After the procedure the patient remained severely ill because of respiratory failure against the background of bilateral pneumonia, but pulmonary bleeding did not recur. FBS did not reveal signs of continuing bleeding. The results of laboratory examinations allowed to exclude pulmonary tuberculosis, however, the discovery of worms in patient's feces did not allow to exclude parasitic etiology of his pneumonia. Scatology revealed the presence of Ascaris lubricoides eggs. Antibiotic therapy with Tienam and Ciprofloxacin was suggested. Taking into account the revealed ascariasis, the therapy was supplemented

[1] Address for correspondence:
Dr. Evgueny Khayrutdinov,
Russia, 117574, Moscow, proezd Odoevskogo, 7, bld.1, apt.12.
Tel. +7 916 830 49 64
e-mail: eugkh@yandex.ru
Manuscript received on August 9, 2011.
Accepted for publication on September 5, 2011.

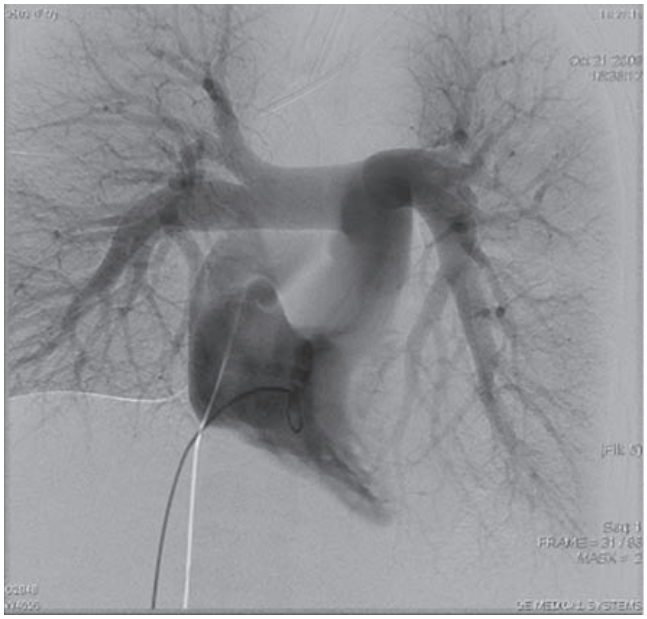


Fig. 1. Angiopulmonography

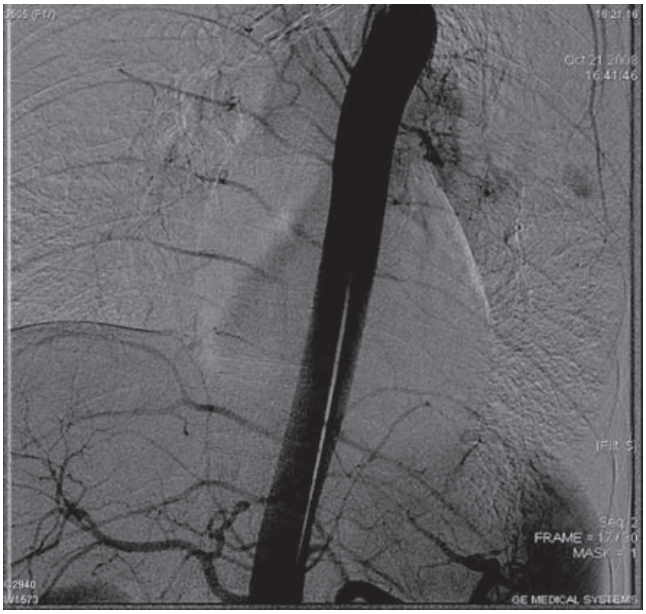


Fig. 2. Angiography of the descending thoracic aorta

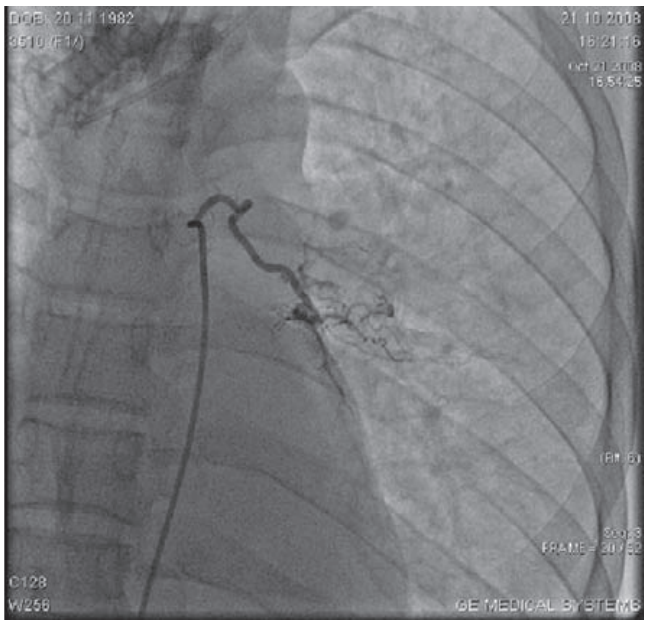


Fig. 3. Angiography of the left bronchial artery

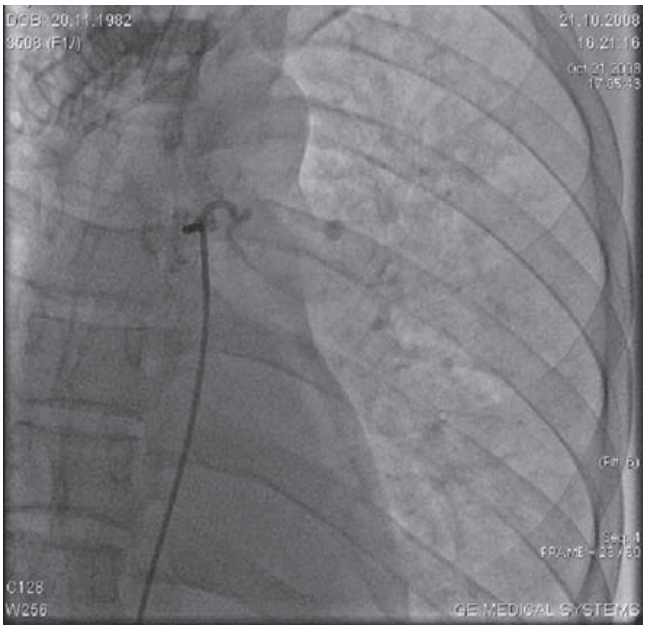


Fig. 4. Total embolization of the left bronchial artery with PVA particles.

by anti-worm agent Mebendazol. This therapy led to the stabilization of patient's condition. In 5 days after the embolization of the left bronchial artery he was extubated, pulmonary bleeding did not recur. Chest X-ray examination performed in 2 weeks after the embolization revealed the resolution of bilateral pneumonia. The patient was discharged in a satisfactory condition.

We present a case of successful endovascular treatment of pulmonary bleeding. Bronchial arteries are the source of pulmonary bleeding in more than 90% of cases. The bleeding from the pulmonary arterial system (e.g., pulmonary arteriovenous malformation, pulmonary endometriosis, pulmonary arterial aneurysm, etc.) is rare and occurs in approximately 5%. In the remaining 5% of cases the source of bleeding is not found in bronchial systemic collateral arteries. It can be located in the internal thoracic artery, the thyrocervical trunk of the subclavian artery, the lateral branches of the subclavian and axillary arteries, the intercostal arteries and the inferior diaphragmatic artery (5, 6). In most cases pulmonary bleeding is caused by acute or chronic inflammatory lung disease. Etiological causes of pulmonary bleedings are presented in Table 1.

Pulmonary blood flow in patients with acute or chronic pulmonary diseases is decreased due to hypoxia-caused vasoconstriction, thrombosis and

vasculitis at the level of pulmonary arterioles. This leads to the growth and the enlargement of bronchial arteries aimed at the compensation of decreased pulmonary blood flow. Bronchial arterial blood flow in patients with bronchoectatic pulmonary disease can reach up to 30% of the cardiac output (7). The impact of increased blood pressure and bacterial agents on the bronchial arteries contributes to their rupture and subsequent pulmonary bleeding (2, 8).

Chest X-ray, FBS and CT are the main methods for the diagnostics of pulmonary bleeding. In most cases chest X-ray examination allows to reveal some pulmonary disease, but does not detect the source of bleeding. FBS is the main diagnostic procedure in patients with pulmonary bleeding. It allows to find the source of bleeding in 93% of cases. However, the diagnostic value of FBS in patients without any pathology revealed by chest X-ray is significantly reduced (0-30%). The advantage of bronchoscopy consists in the feasibility of local application of hemostatic agents to stop the bleeding. The examination with CT allows to reveal simultaneously bronchopulmonary and vascular pathology, thus CT is the method of choice for the diagnostics of pulmonary bleeding. It allows to detect the source of bleeding in 63-100% of cases (1, 9).

Angiography of the bronchial arteries is the final step in the diagnostics of the source of pulmonary bleeding prior to embolization. The main distinctive angiographic features of bronchial arterial pathology in pulmonary bleeding are: the enlargement of the main trunk of the bronchial artery (>3 mm), the hypervascularization of the pathological focus area, the impregnation of parenchyma with contrast medium and the presence of bronchopulmonary shunts.

Virtually in all cases bronchial arteries arise from the thoracic aorta at the level of T4-T7 vertebrae (10, 11), in 90% of cases their origin is located at the level of the intervebral disc between the T5 and T6 vertebrae. Bronchial arteries supply the trachea, the bronchi, regional lymphatic nodes, the pleura and the esophagus.

There are 4 main types of bronchial arteries origin (2):

• Type 1: two bronchial arteries arise to the left, and one — to the right of the aorta (intercostal bronchial trunk) — is seen in 40% of cases.

Table 1.

Etiological causes of pulmonary bleeding

Infection: • Bronchoectatic disease • Pneumonia • Chronic bronchitis • Lung abscess • Aspergillosis, mycetoma • Ascariasis • Tuberculosis • Cystic fibrosis
Tumors: • Adenocarcinoma • Bronchus adenoma • Bronchus carcinoid • Endometriosis • Metastases in lungs
Cardiovascular diseases: • Severe left ventricular failure • Mitral stenosis • Pulmonary arterial thromboembolism • Aortic aneurysm • Pulmonary arterial aneurysm • Arteriovenous malformation • Yatrogenic causes (e.g., damage by Swan-Ganz catheter)
Vasculites: • Wegener's disease • Systemic lupus erythematosis • Goodpasture's syndrome
Idiopathic pulmonary hemosiderosis
Aspiration of a foreign body
Lung trauma (contusion)
Use of anticoagulant agents, thrombolythic therapy

• Type 2: one bronchial artery arises to the left, and one intercostal bronchial artery — to the right of the aorta — in 20% of cases.

• Type 3: two bronchial arteries arise to the left, and one bronchial and one intercostal bronchial artery — to the right of the aorta — in 20% of cases..

• Type 4: one bronchial artery arises to the left, and и one bronchial and one intercostal bronchial artery — to the right of the aorta — in 10% of cases.

However there are many variants of bronchial arteries origin. They can arise from the aortic arch, the internal thoracic artery, the thyrocervical trunk, the left subclavian artery, the inferior thyroid artery and the abdominal aorta (11). Bronchial arteries have a lot of collaterals with other anatomic areas, which has to be taken into consideration while performing the embolization. The most important are the collaterals to the anterior cerebral artery supplying the spinal cord, the right and the left subclavian arteries and the right coronary artery.

The embolization of a bronchial artery was first described by Remy (12) in 1970, and with the course of time became the generally adopted method for the treatment of massive pulmonary bleeding (13-16). Technical improvements in catheters' design and production methods, as well as in embolizing materials have contributed to the enhancement of safety and availability of this procedure. The search of the source of pulmonary bleeding during angiographic examination should be started with thoracic angiography. At the next stage selective catheterization of bronchial arteries is performed. It can be done with the use of catheters like Cobra, Simmons, Mammary, Multi-purpose, as well as of special catheters for bronchial arteries. If lateral branches arising from the bronchial artery supply other anatomical areas, superselective embolization using microcatheter is performed, because the embolization of these branches can lead to complications.

Hemostatic sponge, microspheres and coils can be used as embolization material. The main advantage of a hemostatic sponge consists in its availability and low cost. However with the course of time the sponge resolves which can lead to recanalization of the embolized vessel and recurrent bleeding. Thus, hemostatic sponge can be used only for temporary bleeding control. The most frequently used embolizing material are PVA particles. Being biocompatible and non-biodegradable they provide long-term hemostasis (17). Recently it was suggested to use for this purpose the embospheres produced on the basis of triacryl gelatine, however the experience with the use of this material for bronchial arteries embolization is still limited. The main advantage of embospheres over the PVA particles consists in their better penetrating capacity and the lack of propensity for clots formation due to particles' glueing, which is especially important with the use of microcatheters. As a rule, bronchial embolization is performed using particles of 350 — 700 μc; the use of smaller particles is undesirable as it can lead to the embolization of branches supplying healthy tissues. Metallic and plastic coils are seldom used as primary material for bronchial arteries embolization. It is related to the fact that they cause proximal occlusion of the vessel with subsequent development of collaterals to the damaged area and recurrent bleeding. The presence of bronchial arterial aneurysms is main indication for the use of coils (18, 19).

Embolization of bronchial arteries is a high-effective procedure for the treatment of acute pulmonary bleeding. Its one-month effectiveness is 73 to 98% (12, 20, 21). The rate of late pulmonary bleeding recurrence can reach 52%, however, with the use of repeated embolizations and the treatment of the underlying disease the success rate is close to 100%. The most frequent causes of pulmonary bleeding recurrence are recanalization of the embolized vessel, incomplete primary embolization of the sick vessel, the development of new collaterals and the progressing of the underlying disease (22). The frequency of recurrent pulmonary bleeding also depends on its etiological cause, most frequently they occur in patients with cancer and chronic tuberculosis.

The most frequent (24-91%) complication of bronchial arteries embolization is chest pain related to the ischemia of the embolized branches of the bronchial artery. However, it can be more pronounced after embolization of the intercostal arteries' branches. The second most frequent complication, occurring in 0,7 — 18,2% of cases, is dysphagia related to the embolization of esophagus-supplying branches (23). As a rule, the symptoms resolve spontaneously within several days. Spinal cord ischemia occurring in 1,4 — 6,5% of cases is the most severe complication of embolization of the bronchial arteries. There are reports of rare complications, such as bronchus necrosis, unilateral phrenoplegia, bronchus stenosis, infarction lung pneumonia. Eventual ways for the decrease of the risk such complications consist in the use of superselective embolization technique with microcatheters and the due choice of embolization materials.

Thus, embolization of the bronchial arteries is the method of choice for the treatment of patients with acute pulmonary bleeding. The knowledge of anatomical particularities of the bronchial arteries and their collaterals, the due choice of the technique and the material for embolization form the basis for successful embolization in patients with pulmonary bleeding.

References

1. Hirshberg B., Biran I., Glazer M., Kramer M.R. Hemoptysis: etiology, evaluation, and outcome in a tertiary referral hospital. Chest, 1997, 112, 440-4.

2. Yoon W., Kim J.K., KimY.H. et al. Bronchial and nonbronchial systemic artery embolization for life-threatening hemoptysis: a comprehensive review. Radiographics, 2002, 22, 1395-409.

3. Wong M.L., Szkup P., Hopley M.J. Percutaneous embolotherapy for life-threatening hemoptysis. Chest, 2002, 121, 95-102.

4. Knott-Craig C.J., Oostuizen J.G., Rossouw G. et al. Management and prognosis of massive hemoptysis. Recent experience with 120 patients. J. Thorac. Cardiovasc. Surg., 1993, 105, 394-7.

5. Foster D.C., Stern J.L., Buscema J. et al. Pleural and parenchymal pulmonary endometriosis. Obstet. Gynecol., 1981, 58, 552-6.

6. DeLima L.G., Wynands J.E., Bourke M.E. et al. Catheter-induced pulmonary artery false aneurysm and rupture: case report and review. J. Cardiothorac. Vasc. Anesth., 1994, 8, 70-5.

7. Do K.H., Goo J.M., Im J.G. et al. Systemic arterial supply to the lungs in adults: spiral CT findings. Radiographics, 2001, 21, 387-402.

8. Chun H.J., Byun J.Y., Yoo S.S. et al. Added benefit of thoracic aortography after transarterial embolization in patients with hemoptysis. Am. J. Roentgenol., 2003, 180, 1577-81.

9. Hsiao E.I., Kirsch C.M., Kagawa F.T. et al. Utility of fiberoptic bronchoscopy before bronchial artery embolization for massive hemoptysis. Am. J. Roentgenol., 2001, 177, 861-7.

10. Won J.H., Park S.I., Park K.J. et al. Microcatheter placement through a side hole created in a 5-F catheter into proximal subclavian arterial branches causing hemoptysis. J. Vasc. Interv. Radiol., 2004, 15, 881-4.

11. Sancho C., Escalante E., Dominguez J. et al. Embolization of bronchial arteries of anomalous origin. Cardiovasc. Intervent. Radiol., 1998, 21, 300-4.

12. Remy J., Arnaud A., Fardou H. et al. Treatment of hemoptysis by embolization of bronchial arteries. Radiology, 1977, 122, 33-7.

13. Haponik E.F., Fein A., Chin R. Managing life-threatening hemoptysis: has anything really changed? Chest, 2000, 118, 1431-5.

14. Johnson J.L. Manifestations of hemoptysis. How to manage minor, moderate, and massive bleeding? Postgrad. Med., 2002, 112, 101-9.

15. Jougon J., Ballester M., Delcambre F. et al. Massive hemoptysis: what place for medical and surgical treatment. Eur. J. Cardiothorac. Surg., 2002, 22, 345-51.

16. Marshall T.J., Flower C.D., Jackson J.E. The role of radiology in the investigation and management of patients with haemoptysis. Clin. Radiol., 1996, 51, 391-400.

17. White R.I. Bronchial artery embolotherapy for control of acute hemoptysis: analysis of outcome. Chest, 1999, 115, 912-5.

18. Barben J., Robertson D., Olinsky A. et al. Bronchial artery embolization for hemoptysis in young patients with cystic fibrosis. Radiology, 2002, 224, 124-30.

19. Saluja S., Henderson K.J., White R.I. Embolotherapy in the bronchial and pulmonary circulations. Radiol. Clin. North Am., 2000, 38, 425-48.

20. Uflacker R., Kaemmerer A., Picon P.D. et al. Bronchial artery embolization in the management of hemoptysis: technical aspects and long-term results. Radiology, 1985, 157, 637-44.

21. Lampmann L.E., Tjan T.G. Embolization therapy in haemoptysis. Eur. J. Radiol., 1994, 18, 15-9.

22. Marshall T.J., Jackson J.E. Vascular intervention in the thorax: bronchial artery embolization for haemoptysis. Eur. Radiol., 1997, 7, 1221-7.

23. Ramakantan R., Bandekar V.G., Gandhi M.S. et al. Massive hemoptysis due to pulmonary tuberculosis: control with bronchial artery embolization. Radiology, 1996, 200, 691-94.