Zeitschrift für
Gefäßmedizin

Bildgebende Diagnostik • Gefäßbiologie • Gefäßchirurgie • Hämostaseologie • Konservative und endovaskuläre Therapie • Lymphologie • Neurologie • Phlebologie

Predicting changes in an aneurysm sac after endovascular abdominal aortic aneurysm repair // Möglichkeiten zur Prognostizierung der Dynamik des Aneurysmasacks nach Endoprothese der Bauchaorta

Furkalo SN, Khasyanova IV

Vlasenko EA

Zeitschrift für Gefäßmedizin 2020;

17 (1), 8-13

Offizielles Organ der Österreichischen Gesellschaft für Phlebologie und dermatologische Angiologie

Offizielles Organ des Österreichischen Verbandes für Gefäßmedizin

Offizielles Organ der Österreichischen Gesellschaft für Internistische Angiologie (ÖGIA)

Indexed in EMBASE/COMPENDEX/GEOBASE/SCOPUS

Krause & Pachernegg GmbH • Verlag für Medizin und Wirtschaft • A-3003 Gablitz

SITZ GUT, TUT GUT!

EINFACH SCHLUSS MIT HÄMORRHOIDALLEIDEN!

EASY-TO-USE
AKUT THERAPIE

- ✓ Einfache Einnahme
- ✓ Auf eine Akut-Therapie abgestimmt
- ✓ Wirkt gezielt von innen

*Eine Innovation von Dioscomb®, **Österreichs Nr. 1** bei Venenpräparaten**

**IQVIA Hinausverkauf aus der Apotheke in Einheiten YTD Dezember 2024*

Do_036_0325_Ins

Fachkurzinformation: Bezeichnung des Arzneimittels: Dioscomb® 1000 mg Filmtabletten; **Qualitative und quantitative Zusammensetzung:** 1 Filmtablette enthält 1000 mg mikronisierte Flavonoide, bestehend aus 900 mg Diosmin und 100 mg anderen Flavonoiden, dargestellt als Hesperidin. Sonstige Bestandteile: Tablettenkern: Magnesiumstearat, Talkum, Maisstärke, Gelatine, mikrokristalline Zellulose (Typ 102). Filmüberzug: Eisenoxid rot (E172), Eisenoxid gelb (E172), Macrogol 3350, partiell hydrolysierter Poly(vinylalkohol) (E1203), Titandioxid (E171), Talkum (E553b), Maltodextrin, Guargalactomannan (E412), Hypromellose (E464), mittelkettige Triglyzeride. **Anwendungsgebiete:** Dioscomb ist bei Erwachsenen angezeigt zur: Behandlung von chronischer Veneninsuffizienz der unteren Extremitäten bei folgenden funktionellen Symptomen: schwere Beine und Schwellungen, Schmerzen, nächtliche Krämpfe der unteren Extremitäten. Symptomatische Behandlung von akuten Hämorrhoidalbeschwerden. **Gegenanzeigen:** Überempfindlichkeit gegen den Wirkstoff oder einen der in Abschnitt 6.1 genannten sonstigen Bestandteile. **Pharmakotherapeutische Gruppe:** Kapillarstabilisierende Mittel; Bioflavonoide, Diosmin, Kombinationen. ATC-Code: C05CA53. **Inhaber der Zulassung:** ExtractumPharma zrt. H-1044 Budapest, Megyeri út 64. Ungarn. **Zulassungsnummer:** 141737 **Verschreibungspflicht/Apothekenpflicht:** Rezeptfrei, apothekenpflichtig. **Stand der Information:** 07/2024; *Weitere Angaben zu Warnhinweisen und Vorsichtsmaßnahmen für die Anwendung, Wechselwirkungen mit anderen Arzneimitteln und sonstigen Wechselwirkungen, Schwangerschaft, Stillzeit und Nebenwirkungen sind der veröffentlichten Fachinformation zu entnehmen.* Über Wirkung und mögliche unerwünschte Wirkungen dieses Arzneimittels informieren Gebrauchsinformation, Arzt oder Apotheker.

ERWO PHARMA

Predicting changes in an aneurysm sac after endovascular abdominal aortic aneurysm repair

S. N. Furkalo, I. V. Khasyanova, E. A. Vlasenko

Kurzfassung: Möglichkeiten zur Prognostizierung der Dynamik des Aneurysmasacks nach Endoprothese der Bauchaorta. *Hintergrund:* Im Rahmen von Studien wurden die Diagnostik und Stabilitätsprognose des Aneurysmasacks (AS) bei Patienten mit Bauchaorta-Aneurysma nach Aorta-Endoprothese je nach pathomorphologischen Veränderungen im Bereich des chirurgischen Zugangs durchgeführt. Eine morphologische Untersuchung der Arteria femoralis communis (AFC) wurde bei 38 Patienten (2 Frauen, 36 Männer) durchgeführt, das durchschnittliche Alter betrug 66,1 ± 2,1 Jahre.

Ergebnisse: Es wurden in 22 Fällen (57,8 %) direkte Anzeichen von Atherosklerose und in 14 Fällen (36,8 %) Anzeichen von degenerativen Veränderungen beobachtet. In 2 Fällen (5,26 %) dominierten im pathologischen Bild die Entzündungsreaktionen. Die statistische Analyse zeigte, dass eine stabile Beziehung zwischen der Art der morphologischen Veränderungen in der AFC-Wand und der Größe und Dynamik des AS nach Endoprothese besteht. Für atherosklerotische Veränderungen ist eine Reduktion des AS auf 77,7% typisch, während die Reduktion des AS bei degenerativen Veränderungen nur in 28,7 % beobachtet wurde. Bei 64,2 % fehlen Änderungen des Sackes oder der Sack hatte sich vergrößert.

Zusammenfassung: Die morphologischen Studien an der AFC-Wand bei Patienten schlussfolgern, dass die Wahrscheinlichkeit der Reduktion des AS nach der Endoprothese im Falle der vorherrschenden atherosklerotischen Veränderungen in den Gefäßwänden um das 16,5-Fache – OR 16,5 (2,79–97,68) steigt. Degenerative Veränderungen haben eine ungünstige Prognose. Die Wahrscheinlichkeit der AS-Reduktion wird um 94 % verringert AM–OR = 0,06 (0,01–0,36).

Schlüsselwörter: Aneurysma der infrarenalen Bauchaorta, Endoprothese, Aorta, Endoleak

Abstract: *Background:* The aim of this study is to predict the aneurysm sac condition using the analysis of morphological studies of the anterior wall of the common femoral artery.

Materials and Methods: In this study, a method for diagnosing and predicting the stability of aneurysm sac in patients with abdominal aortic aneurysms after endovascular aortic aneurysm repair, depending on the type of pathomorphological changes at the surgical access site, was used. The morphological study of the common femoral artery (CFA) was performed in 38 patients. The average age of patients was 66.1 ± 2.1, including 2 women and 36 men. The summary of the obtained results showed that in 22 cases (57.8%) there were direct signs of atherosclerosis, in 14 cases (36.8%) – manifestations of dystrophic changes and in 2 cases (5.26%) inflammatory reactions dominated among the pathologies.

Results: Statistical analysis showed that there was a stable and significant relationship between the nature of morphological changes in the CFA walls and the aneurysm sac (AS) size after endovascular repair. In atherosclerotic changes, a decrease in the AS size was observed in 77.7% of cases, while in degenerative changes, the AS size decreased in 28.7% of cases only, and in 64.2% of cases, changes were absent or the sac size was increased.

Conclusion: Therefore, the morphological studies of the CFA walls in patients showed that in prevailing atherosclerotic changes in the artery walls, the probability of the AS size reduction after endovascular repair increased by 16.5 times – OR-16.5 (2.79–97.68). In dystrophic changes, the prognosis was unfavourable – the probability of the AS size reduction decreased by 94% – OR = 0.06 (0.01–0.36). **Z Gefäßmed 2020; 17 (1): 8–13.**

Key words: infrarenal abdominal aortic aneurysm, endovascular aortic aneurysm repair, endoleak

Abbreviations

AAA = abdominal aortic aneurysm
AS = aneurysm sac
CFA = common femoral artery
EVAR = endovascular aneurysm repair
IEM = internal elastic membrane
SMC = smooth muscle cells

Introduction

Endovascular aneurism repair (EVAR) of infrarenal abdominal aortic aneurysm (AAA) is one of the high-tech, less traumatic and advanced surgical techniques. The continuous improvement of surgical equipment and development of more advanced vascular prothesis have significantly reduced perioperative mortality, injury and rehabilitation time [1, 2]. The share of endovascular aneurysm repair of infrarenal abdominal aortic aneurysm in the developed European countries and Americas is about 70% of all surgical interventions for infrarenal abdominal aortic aneurysm (AAA) [3].

Endovascular abdominal aortic aneurysm repair is a highly-technological and rapidly developing trend in the treatment of patients with abdominal aortic aneurysms (AAA) [4, 5]. With the development of endovascular equipment and clinical experience, the indications for this intervention are gradually expanding [6]. Due to this fact, a number of patients with AAA, for whom the only option was a traditional surgical correction, have an option of treatment using less traumatic mini-invasive techniques [7–9]. This is especially important for elderly patients with concomitant comorbidities and, consequently, increased risks related to surgical intervention.

At the same time, the results of long-term observations of patients after endovascular aortic repair showed the need for continuous monitoring and repeated interventions, compared to patients undergoing traditional surgical procedure [10, 11]. The results of the EVAR-1 and EVAR-2-studies directly show an increased frequency of aneurysm-related complications during a long-term follow-up (more than 10 years) [12]. Despite the fact that the above studies summed up the experience of endovascular interventions which was obtained at the initial stages of clinical use of the technique, using older and less advanced generations of endovascular equipment, these results caused a certain discussion in the medical circles, putting into question a long-term advantage of endovascular abdominal aortic aneurysm repair [13].

Received: May 14, 2019; accepted: May 28, 2019; Pre-Publishing Online: August 16, 2019.

From the Department of Endovascular Surgery and Interventional Radiology, Kyiv, Ukraine

Correspondence to: Sergey N. Furkalo, A.A. Shalimov National Institute of Surgery and Transplantology, 03680, 30 Heroiv Sevastopolia Str., Kyiv, Ukraine; e-mail: furkalosn@gmail.com

It is obvious that late complications, endoleaks and a tendency toward aneurysm sac enlargement do not occur in all patients and most patients have stable long-term effects, confirmed by special studies, over 10–15 years [14, 15]. Therefore, a prognostic assessment of the patients' condition and a possibility to identify patients with predicted long-term outcomes of endovascular repair, as well as identification of parameters indicating supposedly negative long-term effects of endovascular techniques, is of paramount importance in the practice of vascular surgery [16–18].

The purpose of this study is to predict the aneurysm sac condition using the analysis of morphological studies of the anterior wall of the common femoral artery.

Materials and Methods

This study is based on the analysis of the treatment results in 110 patients who underwent EVAR of AAA from November 2005 to November 2015 in the SE O.O. Shalimov National Institute of Surgery and Transplantology of the NAMSU Kyiv.

During the study, the method of life-time diagnosis of the pathological process in the aortic wall associated with aortic aneurysms, was used [19]. During endovascular repair, the CFA anterior wall is accessed using traditional surgery to insert a stent-graft through it under X-ray control. When the artery is closed with sutures, the surgeon has the opportunity to take the material for the morphological study of the vascular wall. The biopsy of the CFA anterior wall was performed before its suturing after implantation of the stent. Hypothetically, changes in the CFA wall reflect the aortic wall condition in the area of the aneurysm sac. For a morphological study, the obtained samples were treated according to the generally accepted method, stained with hematoxylin and eosin and studied under the optical microscope with a magnification of eyepiece 10, objective 20.

Morphological studies of the CFA walls were performed in 38 patients with infrarenal AAA. Mean age of the patients was 66.1 ± 2.1 years, including 2 women and 36 men. At the time of intervention, mean AS size did not show statistically significant differences (p = 0.448) and was: 55.4 ± 1.5 mm in the group of predominant atherosclerotic changes, 53.4 ± 1.9 mm in predominant dystrophic changes and 59.0 ± 2.0 mm in the group of predominant inflammatory changes. The obtained material was fixed in 10% neutral formalin solution. After routine histological treatment, 4–5 micron sections were cut from paraffin blocks and stained with hematoxylin and eosin; the collagen fibers were stained using the Van Gieson's stain and the elastic fibers using the Weigert method; the Schick test was performed.

The histological preparations were described according to our own scheme: there were changes in intima, internal elastic membrane (IEM), tunica media and tunica adventitia. In general, a degree of preservation of the CFA wall normal structures was estimated. In intima, thickness, presence of endothelium, peculiarities of the intercellular matrix and presence of dissection were studied. The appearance of the internal elastic membrane, maintaining of its structure and orientation of elastic membranes were described. In tunica media, changes in thickness, nature of degenerative changes in smooth muscle cells and elastic membranes, as well as presence of cysts, hemocapillaries and slit-like blood vessels were examined. In tunica adventitia, a degree of its sclerosis, presence of vascular reactions and severity of inflammatory infiltration were investigated.

Table 1. Distribution of patients by the nature of changes in the CFA wall

Nature of predominant morphologic changes	Number, (%)	Age, years (M ± m)	P (F)
Atherosclerotic	22 (57.9)	66.8 ± 2.2	F = 0.17
Dystrophic	14 (36.8)	65.7 ± 1.9	p = 0.846
Inflammatory	2 (5.3)	67.5 ± 2.5	
Total	38 (100)	66.1 ± 2.1	

The studied preparations were photographed. The CFA wall structures from different biopsies were compared regarding postoperative changes in the AS size and the presence or absence of endoleaks.

Results and Discussion

Based on prior histologic analysis of biopsy samples, three main types of pathological changes were identified: atherosclerotic lesions of the vessel wall, dystrophic changes without direct signs of atherosclerosis and inflammatory infiltration of the vessel wall (Table 1).

There were no significant differences in age in the groups with different pathological changes in the FCA wall. The cases of atherosclerosis were cases when biopsy revealed direct morphological signs of atherosclerosis with the presence of various types of atherosclerotic plaques. There were 22 cases. In the group with predominant dystrophic wall damage, there were no direct morphological signs of atherosclerosis. In this group, the biopsy showed mild sclerosis, as well as areas of dysplasia of the smooth muscle cells (SMC), areas of fibro-muscular dysplasia, dissection and cysts. This group included 14 cases. In 2 cases, the signs of acute and sub-acute inflammatory reaction dominated among pathological changes, indicating the presence of aortic arteritis. Such division was found feasible in the course of the study and for the assessment of changes in the AS size during a long-term follow-up.

Morphological results obtained during the study of the CFA wall were extrapolated to the aortic walls and aneurysm. We consider this reasonable, as many studies confirm systemic nature of large arteries involvement. It should be noted that the division into atherosclerotic and dystrophic changes is rather conventional and is necessary for the analysis of the material obtained during biopsy. The absence of direct evidence of atherosclerosis in biopsy samples does not exclude its presence in neighbouring, non-examined sites [20, 21]. In addition, the above mentioned severe degenerative changes in the aortic wall may be observed in combination with atherosclerosis.

In primary histological studies of biopsy samples, all layers of the CFA wall were expected without significant structural damage, as the macroscopic analysis showed intact artery walls in the area of biopsy. However, the first results showed significant

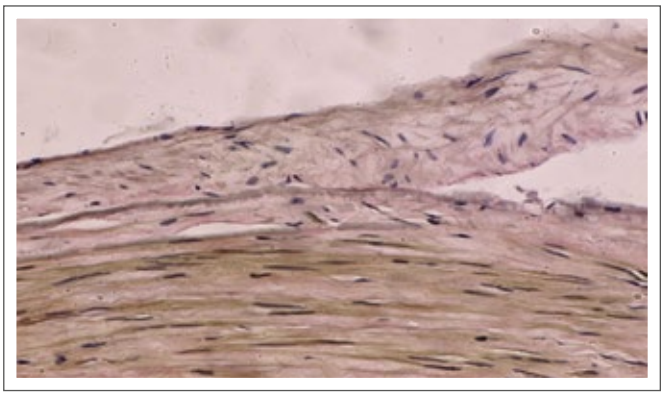

Figure 1. CFA wall. Moderate intimal fibroplasia, individual SMCs, slight connective tissue swelling and sclerosis. Cell separation and exfoliation. Van Gieson staining (x200).

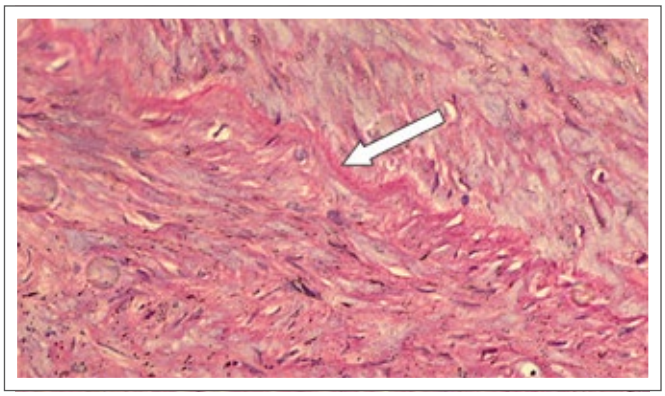

Figure 3. CFA wall. Severe IEM (indicated by the arrow). Intima and media sclerosis. Van Gieson staining (x200).

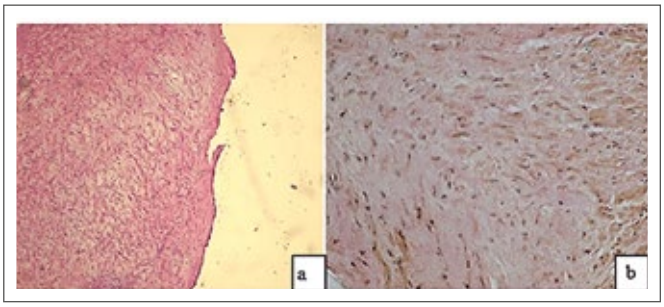

Figure 2. CFA wall. **(a)** Intimal hyperplasia, SMC proliferation, focal myxomatosis. Hematoxylin and eosin staining (x100); **(b)** edema, moderate fibroplasia, intimal sclerosis, SMC dystrophy. Van Gieson staining (x200).

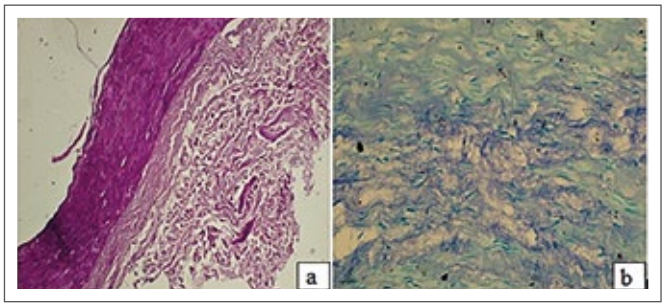

Figure 4. CFA wall. **(a)** Thinning and sclerosis of the vessel wall, adventitious sclerosis. Schiff (PAS) reaction (x40); **(b)** lysis of SMCs and elastic membranes with cysts. Azure II and eosin staining (x400).

degenerative changes in the CFA wall, involving all layers of the vessel walls (intima, IEM, tunica media, tunica adventitia and surrounding cellular tissue) in most cases.

In addition, pathological signs and their rates in different patients were different, which created a polymorphic and rather individualized picture in each individual case. Therefore, it was considered appropriate to determine the morphological semiotics of lesions in each structural element of the CFA wall while describing and evaluating the material.

Pathological changes in the intima were present in several variants. In the first variant, the intima had an ordinary thickness, was well differentiated with uniformly arranged SMCs, the intercellular matrix had moderate volume, homogeneous, stained pink, and had a relatively dense layer of flattened endothelial cells. However, this condition of the intima was observed only in one of the observations. In the second variant, the intima is thinned, unevenly scleroid (moderate intimal fibroplasia), exfoliated from the inner elastic membrane, edema of the extracellular matrix is observed, the SMC is disorganized, the endothelium is differentiated as individual flattened cells (Fig. 1).

In the following variant (Fig. 2), the intima is uneven, focally thickened, with edema, fibrosis and myxomatosis areas. In some cases, collagen fibers form foci with SMCs with small nuclei and pronounced dystrophic changes. In some cases, the intima is not differentiated at all, as it merges with the sclerotic medium sheath. In each case, changes of various severity and their various combinations can be observed. These are the main signs of intimal structure disorders.

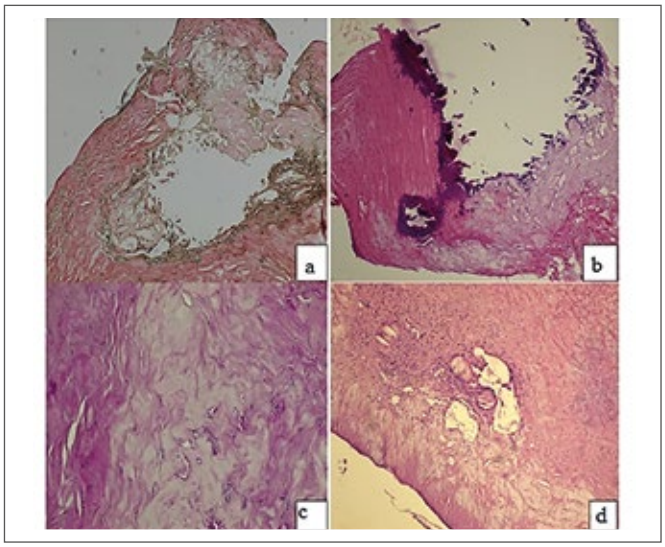

Figure 5. CFA wall. **(a)** Atheromatous plaque. Van Gieson staining (x100); **(b)** atheromatosis and calcification associated with sclerosis of the media. Schiff (PAS) reaction (x100); **(c)** lipid plaque in the wall of the CFA, neoangiogenesis. Schiff (PAS) reaction (x200); **(d)** atherosclerotic plaque with inflammatory infiltration. Hematoxylin and eosin staining (x100).

In some cases, the internal elastic membrane with a relatively clear structure was preserved (Fig. 3). In several cases, its full or partial fragmentation, swelling, uneven thickness, splitting of fibers, areas of intima detachment or its complete absence were observed.

The most severe pathological changes were observed in the medium sheath. The most common changes include sclerosis

Table 2. Dependence of AS volume changes on the type of morphological changes in the wall of the common femoral artery

Wall changes	Aneurysm sac shrinkage (in %)	P (F)
Atherosclerotic	– 20.38 ± 2.79	F = 10.32 p = 0.0003
Inflammatory	– 10.9 ± 1.6	
Dystrophic	– 0.17 ± 3.3	
Total	– 13.16 ± 2.52	–

and thinning of the medium sheath, dystrophic changes of SMCs with edema and cell lysis and cysts, sclerosis, disorganization and disorientation of SMCs and elastic membranes. Different sclerosis areas (diffuse, linear, reticular, with vortex and nodules) were observed. Focal proliferation of small capillaries located separately, as chains or clusters; areas of stratification of various sizes, foci of fragmentation and lysis of elastic membranes with the formation of cysts could be observed in the vessel wall at different levels. As a result of the SMC dystrophy and diffuse sclerosis, the artery wall resembled the structure of cartilage in density and structure in some areas (Fig. 4, 5). Some of these signs were typical of fibromuscular dysplasia, and some of them were characteristic of stratification and pathological deformations.

Atherosclerotic lesions had a significant frequency and were observed in more than half of the studied biopsy specimens. These include lipid, atheromatous, fibrous plaques, areas of mucoid degeneration and calcification, sometimes with perifocal minor lymphohistiocytosis infiltration (Fig. 5). In some cases, thrombotic layers were observed.

In adventitia and in the surrounding tissue, the pathological changes were fairly uniform. Severe sclerosis in combination with numerous sclerosed capillaries, dilation and congestion of the veins, expansion of lymphatic collectors and lymphostasis dominated. In most cases, diffuse, diffuse and focal, pericapillary lymphoid infiltration, lymphoid nodules were observed in adventitia. In two cases, areas of acute inflammation were found in the biopsy specimens of arterial walls with fibrosis (Fig. 6), which was regarded as a manifestation of aortoarteritis.

A generalization of the results showed that a complex of fairly uniform morphological changes was found in biopsy specimens, but direct signs of atherosclerosis were confirmed in 22 cases (57.8%). In 14 cases (36.8%), there were no direct signs of atherosclerosis, but lamination areas, fibromuscular dysplasia areas and other manifestations of dystrophic changes were observed. In one case, dystrophic changes were observed in combination with leiomyoma. In 2 cases (5.26%), acute inflammatory reactions dominated the pathological pattern.

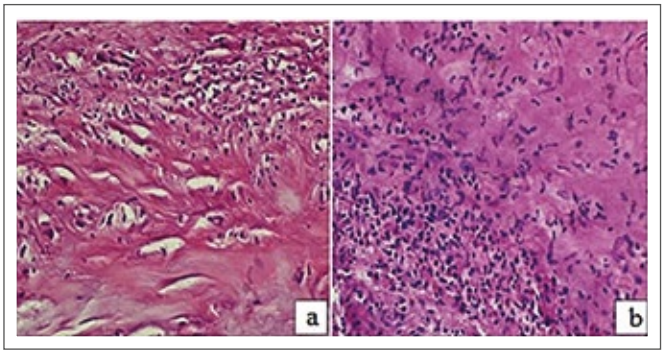

Figure 6. CFA wall. **(a, b)** Areas of acute inflammation in the vessel walls, vessel wall sclerosis. Schiff (PAS) reaction (x40).

Based on the results of histopathological studies, the links between changes in the CFA wall and the dynamics of AS changes in the preoperative, early and late postoperative period were analysed. The dependence of the changes in AS size, when morphological characteristics of the CFA walls were preoperatively monitored in patients, is presented in Table 2.

The statistical analysis showed that there is a stable reliable correlation between the nature of the morphological changes in the CFA wall and the changes in the AS size after the endoprosthesis. Atherosclerotic changes are characterized by a decrease in AS volume of 77.7%, while a decrease in AS volume is observed with dystrophic changes without signs of atherosclerotic lesion only in 28.7%, and changes in the sac size are absent or the sac increases in 64.2%.

It can be considered a proven fact that in cases with severe atherosclerotic changes in biopsy, the frequency of leakage is less compared to dystrophic changes [22, 23]. This gives reasons for a more detailed study of the correlation of these patterns to explain and create a therapeutic prognostic model in order to predict the possible unsatisfactory results of endoprosthetics in the form of blood leakage in AS based on the study of morphological changes in the CFA wall biopsies.

In the absence of the dynamics of AS changes, the morphological characteristics of the CFA wall are also not significantly different, except for individual cases with intimal edema and hyperplasia areas, an increase in the number of cystic formations and media stratifications, and a slight increase in the inflammatory response in adventitia.

Increased AS size cases had slightly different morphological characteristics according to their list and severity. Increased size was associated with increased size and number of cysts and stratification sites, signs of fibromuscular dysplasia and increased inflammatory response in adventitia. These signs can be arranged in the following order by their significance: 1) increased inflammatory reaction in adventitia with edema, severe microangiomatosis and lymphocytic focal and diffuse infiltration; 2) signs of fibromuscular dysplasia; 3) intimal edema and hyperplasia; 4) cystic formations and stratification in media.

Each of the above symptoms has a specific clinical significance. Thus, an increase in the inflammatory reaction in adventitia with edema, severe microangiomatosis, and lymphocytic focal and diffuse infiltration indicates that adventitia, which plays a significant form-reducing role in pathology of the vessels, loses rigidity and can be relatively easily deformed under the influence of lateral pressure.

Fibromuscular dysplasia is an independent pathology with systemic lesions of large vessels or can be observed as an element of a local dystrophic process [24]. In the presence of

fibromuscular dysplasia, the vessel wall also loses its elastic properties and can be deformed under pressure. Intimal edema and hyperplasia are most likely the evidence of tissue reaction and reorganization of the vessel wall under the conditions of abnormal blood pressures in the great vessels.

Table 3. Changes in aortic diameter at the intervisceral level in patients with different patterns of histopathological changes in the anterior CFA wall

Pattern of predominant morphological changes	Quantity, (%)	Changes in diameter, after 3–5 years, (M ± m)	P (F)
Atherosclerotic	22 (57.9)	-1.7 ± 0.2	F = 52.62 p = 0.0001
Dystrophic	14 (36.8)	+3.2 ± 0.7	
Inflammatory	2 (5.3)	+0.6 ± 0.3	

Cystic formations and media stratification also suggest the loss of elastic properties of the vessel wall and, accordingly, opportunities for the appearance of pathological deformities. It also leads to a decrease in the vessel wall resistance under the influence of pathogenic factors (pressure, endoprosthetics), to the possibility of the formation of various protrusions and microaneurysms in the endoprosthetics area.

It is known from the literature that the above-mentioned morphological characteristics of the vessel wall in the conditions of development of aneurysms of the abdominal aorta are systemic [25]. The prognostic assessment of the probability of finding favourable dynamics (decreased bag) for various types of dominant changes in the blood vessel walls is shown in Figure 7.

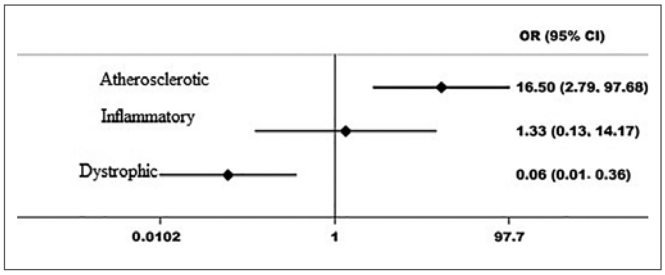

Figure 7. Prognostic assessment of the probability of finding favourable changes (decreased AS) with various types of dominant changes in the vessel walls (estimation of the odds ratio – OR). Source: Compiled by the authors.

Taking into account the systemic nature of changes in the arterial walls of the elastic type, the correlation between the morphology of the anterior CFA wall and AS dynamics after operations was clearly proved, regardless of other adverse factors (outflow endoleaks were detected during control angiography or ultrasound scan before discharge). In addition, we analysed the dynamics of the aorta diameter at the intervisceral level and its relationship with the pattern of histopathological changes in the arterial CFA walls (Tab. 3).

The analysis of the obtained data confirmed the significance of the differences with a tendency toward an increased diameter of the aorta, proximal to the place of prosthesis fixation in patients with predominant dystrophic changes in the arterial walls. This observation confirms the etiology of concomitant aneurysms of the thoracic aorta, popliteal, coronary and other arteries found in approximately 10% of patients with infrarenal AAA.

Conclusions

This study demonstrated that, in predominant atherosclerotic changes in the artery walls, the probability of a successful result, i.e. AS size reduction, increased 16.5 times – OR = 16.5 (2.79–97.68). In inflammatory processes, this assessment was uncertain and statistically insignificant. In dystrophic changes, the prognostic assessment was unfavourable – the probability of favourable changes (AS size reduction) decreased by 94% – OR = 0.06 (0.01–0.36).

In addition, depending on the nature of changes in the walls of large arteries of the elastic type, we observed different changes in the intervisceral aortic diameter, which might also be a predictor of both most prognostically significant endoleaks and the possibility of aneurysmal changes in the thoracic aorta and large arteries of elastic type.

The severity of aortic calcinosis, provided that its prevalence does not exceed 50% of the circumference in the sites of prosthesis implantation, mainly depends on the age of the patient and has no statistically significant differences in patients with different histopathological changes in the CFA.

Conflict of interest

Authors have no conflict of interest.

References:

1. Ali MM, Flahive J, Schanzer A, et al. In patients stratified by preoperative risk, endovascular repair of ruptured abdominal aortic aneurysms has a lower in-hospital mortality and morbidity than open repair. J Vasc Surg 2015; 61: 1399–407.

2. Gillum RF. Epidemiology of aortic aneurysm in the United States. J Clin Epidemiol 1995; 48: 1289–98.

3. Nikulnikov PI, Liksunov AV, Ratushnyuk AV, et al. Surgical treatment of abdominal aortic aneurysm rupture. Actual problems of medical medicine. Bull Ukr Med Stomat Acad 2009; 9: 318–20.

4. Arko FR. Considerations beyond anatomy to best manage aortic disease: the benefits of endoanchors. Endovascular Today 2016. https://evtoday.com/2016/06/supplement2/considerations-beyond-anatomy-to-best-manage-aortic-disease-the-benefits-of-endoanchors/ (last seen 06.06.2019).

5. Schanzer A, Messina L. Two decades of endovascular abdominal aortic aneurysm repair: enormous progress with serious lessons learned. J Am Heart Assoc 2012; 1: e000075.

6. De Martino RR, Goodney PP, Nolan BW, et al. Optimal selection of patients for elective abdominal aortic aneurysm repair based on life expectancy. J Vasc Surg 2013; 58: 589–95.

7. Al-Jubouri M, Comerota AJ, Thakur S, et al. Reintervention after EVAR and Open Surgical Repair of AAA. A 15-Year Experience. Ann Surg 2013; 258: 652–8.

8. Burkitbaiev JK, Abdrakhmanova SA, Savchuk TN, Zhiburt EB. The implementation of nat-screening of infections in blood donors of the republic of Kazakhstan. Klinicheschеskaya Laboratornaya Diagnostika 2017; 3: 154–6-

9. Baimbetov AK, Rzaev FG, Nardaya ShG, et al. New trends in interventional arrhythmology: experience of two centers in cryoablation of atrial fibrillation. J Arrhythm 2018; 92: 5–10.

10. Homoliak IV, Nikulikov PI, Liksunov OV, et al. State of the system of neutrophil granulocytes as a risk factor for postoperative complications in abdominal aortic aneurysms. Heart Blood Vessels 2015; 3: 80–5.

11. Robinson WP, Schanzer A, Aiello FA, et al. Endovascular repair of ruptured abdominal aortic aneurysms does not reduce later mortality compared with open repair. J Vasc Surg 2016; 63: 617–24.

12. Nordon IM, Karthikesalingam A, Hinchliffe RJ, et al. Secondary interventions following endovascular aneurysm repair (EVAR) and the enduring value of graft surveillance. Eur J Vasc Endovasc Surg 2010; 39: 547–54.

13. Greenhalgh RM, Brown LC, Powell JT. Endovascular versus open repair of abdominal aortic aneurysm. N Engl J Med 2010; 362: 1863–71.

14. De Basso R, Sandgren T, Ahlgren ÅR, et al. Increased cardiovascular risk without generalized arterial dilating diathesis

in persons who do not have abdominal aortic aneurysm but who are first-degree relatives of abdominal aortic aneurysm patients. Clin Exp Pharmacol Physiol 2015; 42: 576–81.

15. De la Motte L, Falkenberg M, Koelemay MJ, et al. Is EVAR a durable solution: indications for re-interventions. J Cardiovasc Surg (Torino) 2018; 59: 201–12.

16. Dangas G, O'Connor D, Firwana B, Brar S, Ellozy S. Open versus endovascular stent graft repair of abdominal aortic aneurysms: a meta-analysis of randomized trials. JACC. Cardiovasc Intervent 2012; 5: 1071–80.

17. Baimbetov AK, Bizhanov KA, Yergeshov KA, et al. Immediate results of pulmonary veins entries cryoablation of patients with atrial fibrillation. Z Gefäßmed 2018; 15: 11–6.

18. Herrmann F, Pesenatto GG, Goldani E, Silva JB. The use of alternative experimental models on microsurgery technique's training for medicine undergradute students. Periodico Tche Quimica 2017; 14: 117–22.

19. Lederle FA, Johnson GR, Wilson SE, et al. The Aneurysm Detection and Management Study Screening ProgramValidation Cohort and Final Results. Arch Intern Med 2000; 160: 1425–30.

20. Erbel R, Aboyans V, Boileau C, et al. 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases. Eur Heart J 2014; 35: 2873–926.

21. Tuveson V, Löfdahl HE, Hultgren R. Patients with abdominal aortic aneurysm have a high prevalence of popliteal artery aneurysms. Vasc Med 2016; 21: 369–75.

22. Maras D, Lioupis C, Moulakakis KG. Giant abdominal aortic aneurysms: clinical and technical considerations. Acta Chir Belg 2009; 109: 376–80.

23. Stather PW, Sidloff D, Dattani N, et al. Systematic review and meta-analysis of the early and late outcomes of open and endovascular repair of abdominal aortic aneurysm. Br J Surg 2013; 100: 863–72.

24. Goyal A, Keramati AR, Czarny MJ, et al. The genetics of aortopathies in clinical cardiology. Clinical Medicine Insights. Cardiology 2017; 11: 1179546817709787.

25. Abdul-Hussein H, Hanemaaijer R, Kleemann R, et al. The pathophysiology of abdominal aortic aneurysm growth: corresponding and discordant inflammatory and proteolytic processes in abdominal aortic and popliteal artery aneurysms. J Vasc Surg 2010; 51: 1479–87.

Mitteilungen aus der Redaktion

Besuchen Sie unsere Rubrik

☑ Medizintechnik-Produkte

Neues CRT-D Implantat
Intica 7 HF-T QP von Biotronik

Artis pheno
Siemens Healthcare Diagnostics GmbH

Philips Azurion:
Innovative Bildgebungslösung

Aspirator 3
Labotect GmbH

InControl 1050
Labotect GmbH

e-Journal-Abo

Beziehen Sie die elektronischen Ausgaben dieser Zeitschrift hier.

Die Lieferung umfasst 4–5 Ausgaben pro Jahr zzgl. allfälliger Sonderhefte.

Unsere e-Journale stehen als PDF-Datei zur Verfügung und sind auf den meisten der marktüblichen e-Book-Readern, Tablets sowie auf iPad funktionsfähig.

☑ Bestellung e-Journal-Abo