

AORTIC ANEURYSM IN ATHLETES – THE SERIOUS DIAGNOSTIC ET THERAPEUTIC PROBLEM

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Abstract

The aortic aneurysm is the pathology, which can lead to a sudden death caused by rupture or wall dissection during the physical effort. This disease should be excluded during the initial and periodic examinations of the sportsman. It could be done by patient's family history interview and imaging examinations. People genetically endangered should not perform any hard training. They ought to be under systematic medical control for monitoring and qualification to the surgery in the exact moment. The aortic aneurysms are treated only by open or endovascular surgery. The classical open method consist of implantation of the vascular prosthesis in place of aneurysm. It's very risk-full and can be followed by many complications. The aim of endovascular technique is an implantation of the stent-graft through the peripheral artery. It is less demanding and safer method but still requires further long-term evaluation.

Key words: aortic aneurysm, physical effort, vascular prosthesis

At present the professional sport is closely related with sub maximal and maximal physical effort. Only such training is able to provide the satisfactory results. It requires extreme efficiency of the circulatory, respiratory and motoric system. The condition of these systems should be checked during initial and periodic control. The circulatory system seems to be generally regarded only as the heart while the blood vessels are forgotten. This fact could lead to overlook of the serious pathology like the aortic aneurysm. Further, it can cause a sudden death of the athlete. The genetic and mechanical risk factors are most essential for sportsmen in terms of aortic aneurysm. Evaluation of these factors should be performed before serious training.

The term "aneurysm" comes from Latin word meaning "dilatation". The true aneurysm is a local dilatation of all layers of a vessel wall. Another kind of aneurysm is the dissecting one when layers are separated due to trauma or hypertension (1).

The aorta is an elastic vessel due to specific wall construction. It is able to change its diameter passively depending on systolic or diastolic blood pressure. This phenomenon converts pulsating blood flow into more constant. The aortic wall consists of three main layers: endothelium, media and adventitia. The aortic media is responsible for special features of this vessel because of content of collagen and elastin filaments (1, 2). It provides elasticity and pressure resistance. The durability of such construction is great but not infinite. The long - term overload leads to fatigue and loss of elasticity. It causes deformation of the filaments and - consequently - dilatation of the aorta called "true aneurysm". Such situation can be found

most frequently in abdominal aorta because of least content of filaments.

Another mentioned kind of aneurysm is a dissecting one. In some situation like trauma or sudden hypertension the internal membrane and aortic media can rupture and be separated from outer layers by the blood flow. The dissection could progress and occlude vital vessels (coronary-, carotid- or visceral arteries) or destroy aortic valve of the heart. A dissection concerns most often an aortic arch.

Known risk factors of the aortic wall pathology *Genetic factors*

The proteins of the vascular media are encoded by particular genes. If the mutation occur the improper (weaker) protein will be synthesized. The weakened wall will be prone to deformation by blood pressure. The mutation can be also heritaged so familial aneurysms can be found. According to some trials as many as 19% patients with aneurysm have at least one relative with the same disease. There are known some genetic syndromes with mutation of vascular wall proteins in a background, predisposing to the aortic aneurysm. Most popular are Marfan's and Ehlers - Danlos's syndrome. Some proteins are also elements of fascias and ligaments. This fact explains the coincidence of aortic aneurysm and hernias (especially inguinal) and motoric system malformations (1, 3, 4). Other genetic factors are congenital defects of proteases inhibitors (ex. α_1 -antitrypsine). Their dysfunction causes the degradation of elastic filaments. The proven genetic background imply the precise family history taking during the examination of an athlete.

Mechanical factors

The main influence on the aorta dilatation have the stretching forces produced by blood pressure. They are especially great in case of non-treated or mistreated hypertension. Very dangerous are also “pressure spikes” during hard effort like weight lifting and other strength disciplines. Another mechanical factor is an injury which leads to dissecting or false aneurysm.

Infectious factors

In each aortic aneurysm an inflammation can be found. It consist of interaction of immunologic cells, cytokines and enzymes. According to current theory they are directed against *Chlamydia Pneumonia* - atypical intracellular bacteria which can be found in 50% of aneurysms and not in the healthy vessel (5, 6). The inflammatory diseases of the vessels like Takayasu or Behçet's disease predispose to the aortic aneurysms occurrence (7).

The elastin and collagen degradation

These proteins creates elastic filaments. Their quantity is biggest in the aortic arch and gradually diminishes peripherally. It determines the difference of local elasticity and durability of aorta which are lowest in abdominal part. In patients genetically predisposed (deficiency of proteases inhibitors) the degradation of aortic wall proteins takes place. Such reactions lead to weakening of the aorta, it's dilatation and - finally - rupture.

Atheromatosis

Clinically the atheromatosis is most closely connected with aneurysm due to its frequent occurrence in abdominal part of aorta. Parallely, about 90% of all aneurysms are located distally from the renal arteries. The atheromatic plaques could also be found in each aneurysm. There is no scientific proof for causative role of the atheromatosis for an aneurysm formation but only coincidence (3, 4).

Diagnostics

Most of the aneurysms are diagnosed incidentally during the examination for different reason (8).

History

The patient should be asked directly about any case of aneurysm in their family. In case of negative answer the question about sudden deaths and “internal hemorrhages” should be asked. Confirmation should induce further imaging examination.

Physical examination

In a skinny person the big aneurysm can be palpated during two-handed abdomen examination as the pulsative tumor over the navel or “wide” pulse in

that localization. In case of ruptured aneurysm these symptoms will be intensified. The tenderness of the abdomen and symptoms of hemorrhagic shock can be easily noticed.

Ultrasonography

It is the simplest and cheapest imaging examination (9). It is possible to diagnose an aneurysm, describe its localization among adjacent organs and presence of inflammation. If the colored or double-colored Doppler (Color Doppler, Power Doppler) is used it is possible to confirm the blood extravasations or approximate indication of a rupture.

Computed tomography

The more sophisticated method, more expensive, requires special equipment and service but offers precise imaging. The precise measurement of the aneurysm and evaluation of its surroundings can be done. If the contrast is injected to the circulation (angiography of the computed tomography, angio-CT) it is possible to confirm or exclude of the rupture. The spiral tomography provides enough data to render three-dimensional reconstruction of aorta and its branches. Because of its accuracy this method allows to work up the conception of surgery and estimate the effects. Computed tomography is regarded as the golden standard for qualification to endovascular procedures and evaluation of their effects.

Magnetic resonance

This method is more precise than CT and gives more detail data. It is not in a common use because of the costs and sufficiency of CT.

Arteriography

Consist in taking the series of radiologic photos immediately after the contrast agent is injected. The popularity of non-invasive imaging examinations dedicates this method for pre-endovascular measurements or control of the endovascular prosthesis location.

Clinically the aneurysms are divided into three groups: asymptomatic, symptomatic and ruptured. This systematization concerns the moment of examination because the aneurysm evolves into next state.

Asymptomatic aneurysms

About 20% of aneurysms are asymptomatic. Small ones do not give any characteristic syndromes and their growth can be missed. During the history taking the patient could complain about cough, chest pain, pulsation in the abdomen and abdomen pain.

Symptomatic aneurysms

The symptoms come from compression on the adjacent organs. They can be noticed when the aneu-

rysm is significantly big. The back and lumbal pain are most common. The pain usually radiates to the scapulas, groins, thighs or scrotum. If such symptoms appears and the growth of an aneurysm is observed it is very probable that the aneurysm will rupture immediately.

Ruptured aneurysms

Rupture is the final of the aneurysm evolution. It is the state of extremely serious life risk because of rapidly growing hemorrhagic shock. The death rate is approximately 80%. The patient feel the hit of pain then the shock symptoms appears - hypotension, tachycardia and anemisation. Often such patient has no chance to reach the operation theater. The rupture could be caused by the physical effort like weight lifting for example.

The aneurysm can be treated only by surgery. The indications for it are urgently or scheduled. The urgent cases include symptomatic and ruptured aneurysms. Scheduled can be patients with relatively small aneurysms about 50-60 millimeters in an abdomen or 60-70 millimeters in a thorax and patients with proven growth of an aneurysm. In each case the risk of the surgery should be significantly lower than the risk of eventual rupture (10-14). The coincidence of the internal diseases like ischemic heart disease, hypertension, cerebral ischemia or insufficiency of respiratory system, chronic kidney and liver insufficiency or ulcer disease should be always taken into consideration.

The radical aneurysm surgery consist in implantation of the vascular prosthesis into the aneurysm. It could be performed by open or endovascular surgery. The classic open surgery includes the implantation of the vascular prosthesis through the wide surgical access sometimes with opening the thorax. The endovascular surgery consists of the placing of the stent-graft in the lumen of an aneurysm through the small incision of the peripheral vessel by use of the set of guide wires. Present vascular prosthesis are usually made of dacron or politetraethylene. Due to shape the vascular prosthesis are divided into straight and divided. They are also available in different sizes and diameters (15-18). The stent-graft is the combination of classic vascular prosthesis and elastic metal chassis. Such construction allows for implantation through the peripheral artery (like a femoral one) and its spontan expansion in the aneurysm. The indications for endovascular procedure are similar to classic surgery. In respect for limited knowledge about further results this method is regarded to be used in patients with severe internal diseases who can not be qualified to the open surgery.

There are known some complications of the classic surgery. The most common are the hemorrhages during the implantation and the anesthesiologic complications like hypovolemia, acidosis or heart ischemia.

Next group includes the early complications - local and general ones - which occur within 30 days from operation. The local complications are the bleedings from the anastomosis, prosthesis thrombosis, peripheral embolisation, necrosis of sigmoid colon and infections. The general early complications includes respiratory and circulatory insufficiency, acute renal failure, bleeding to a digestive track, neurological deficits and coagulopathy (as DIC). The late postoperative complications are prosthesis occlusion and false aneurysms of anastomosis usually with concomitant infection. Very dangerous are fistulas between anastomosis and adjacent tubular organs (19-21).

There are also some postoperative complications characteristic for endovascular aneurysm surgery. Most common are the endoleaks to the lumen of an aneurysm. It could be intraoperative as well as late complication. The endoleaks can occur between the wall of prosthesis and aortic wall, recurrently from aorta branches, between the modules of the stent-graft or from damaged prosthesis wall. The reason of endoleak can be the prosthesis migration. It is the uncontrollable movement of the stent-graft downstream. The prosthesis thrombosis can also occur so the patients should receive some chronic anticoagulation or anti-agregation drugs. Extremely dangerous complication is the prosthesis infection due to nearly impossible explanation of the infected material. Another serious complication is aneurysm rupture because of growth induced by endoleak (22).

Conclusions

The qualification to serious training and supervising the athlete is very responsible task for a doctor. He ought to detect any pathologies which endanger the sportsman life during the hard effort also these concerning the vascular system. Basing on the precise history taking and imaging examinations is possible to diagnose the aortic aneurysm. Sick person should be banned from strength sports disciplines and stay under strict periodic control. Early diagnose and appropriate qualification to surgery can prevent from sudden death.

References

1. Noszczyk W. Tętniak aorty brzusznej. *Chirurgia tętnic i żył obwodowych*. PZWL 1998; 463-80.
2. Welsh U. *Histologia*. U&P 1998; 126-7.
3. Grange J, Davis V. Pathogenesis of abdominal aortic aneurysm: an update and look toward the future. *Cardiovasc Surg* 1997; 5: 256-65.
4. van Keulen C, Pals G. Familial abdominal aortic aneurysm: a systematic review of a genetic background. *EJVES* 2002; 24: 105-16.
5. Petersen E, Boman J. In vitro degradation of aortic elastin by chlamydia pneumoniae. *Eur J Vasc Surg* 2001; 22: 443-7.
6. Pupka A, Skóra J. The detection of Chlamydia pneumoniae in aneurysm of abdominal aorta and in normal aortic wall of organ donors. *Folia Microbiol* 2004; 49: 79-82.
7. Sakalihan N, Limet R. Abdominal aortic aneurysm. *Lancet* 2005; 1577-89.

8. Kakost C, Mukhopadhyay U. Abdominal aortic aneurysm: The role of clinical examination and opportunistic detection. *EJVES* 2000; 19: 299-303.
9. Lindholt J. Screening for abdominal aortic aneurysms. *EJVES* 2003; 25: 377-9.
10. Girardi LN, Krieger KH, Altorki NK. Ruptured descending and thoracoabdominal aortic aneurysms. *Ann Thorac Surg* 2002; 74: 1066-70.
11. Coselli JS, LeMaire SA. Surgical techniques. Thoracoabdominal aorta. *Cardiol Clin* 1999; 17: 751-65.
12. Sternbergh WC, Gonze MD, Garrard CL. et al. Abdominal and thoracoabdominal aortic aneurysms. *Surg Clin North Am* 1998; 78: 827-43.
13. Lobato AC, Puech-Leao P. Predictive factors for rupture of thoracoabdominal aortic aneurysm. *J Vasc Surg* 1998; 27: 446-53.
14. Zarins Ch, Gewertz B. Atlas of Vascular Surgery. Churchill, Livingstone, New York 1989; 74-81.
15. Skóra J, Janczak D, Jeleń M. et al. Wczesna odpowiedź komórkowa i humoralna po wszczepieniu protezy naczyniowej w układ tętniczy. *Polim Med* 1997; 27: 27-31.
16. Zimoch L. Porównanie procesów wgajania się protez naczyniowych Dallon standard i Dalton dwustronny welur. *Polim Med* 1992; 22: 4-13.
17. Głowiński S. Wpływ tworzyw różnych typów protez naczyniowych na płytki krwi, układ krzepnięcia i fibrynolizę. *Polim Med* 1992; 22: 4-9.
18. Maruszyński M, Osiecki M. Leczenie chorych z tętniakami aorty brzusznej przy użyciu rozwidlonych stent-graftów aortalnych. *Merk Lek* 2005; 18: 253-6.
19. Szyber P, Janczak D, Skóra J. et al. Czynniki wpływające na wyniki leczenia operacyjnego tętniaków aorty brzusznej. *Terapia* 2000; 8: 3-5.
20. Skóra K, Szydlowski Z, Szyber P. et al. Ocena wpływu wybranych czynników na śmiertelność po operacjach tętniaków aorty brzusznej. *Pol Przegl Chir* 1991; 63: 1119-25.
21. Gattuso R, Gossetti B. Aorto-enteric fistula following abdominal aortic aneurysm repair by endograft. *EJVES Extra* 2002; 4: 48-51.
22. Szmidt J, Rowiński O, Gałązka Z. et al. Simultaneous endovascular exclusion of thoracic aortic aneurysm with open abdominal aortic aneurysm repair. *Eur J Vasc Endovasc Surg* 2004; 28: 442-8.

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C – Statistical Analysis

D – Data Interpretation

E – Manuscript Preparation

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