Case report

SURGICAL TREATMENT OF PATIENT WITH RUPTURED DISSECTION OF THE THORACIC AORTA – STANFORD A, DE BAKKEY I – CASE REPORT OF THE PATIENT SURGICALLY TREATED AT UC OF STATE CARDIOSURGERY – SKOPJE

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Abstract

Introduction. A 59-year-old male patient with dissection of the thoracic aorta, DeBakey I, Stanford A is presented in this case study. We present his symptoms, his preoperative condition, diagnostic procedures, the surgical procedure and his postoperative treatment at the UC of State Cardiosurgery-Skopje.

Case report. The surgery was performed two days after establishment of the diagnosis and more than 3 days (72 hours) after the symptoms occurred, due to absence of patient's consent for the surgery. This resulted in more difficult preoperative condition of the patient, surgical procedure harder to perform, and reduced survival expectations. Preoperative risk of predicted mortality from the cardiovascular surgery calculated according to EUROSCORE was 28.6%.

Results. In this case study we also present classification, etiology, pathophysiology, and some statistics about the incidence of thoracic aorta dissection and survival rates emphasizing the increased mortality rate in delayed surgical interventions.

Keywords: thoracic aorta, dissection, rupture, surgical treatment

Introduction

Definition

An aortic dissection is a very severe medical condition where the inner layer of the aorta (intima) tears and then the blood surges through the tear between the inner and the middle layer separating (dissecting) them. The acute form of the dissection is often rapidly lethal while patients that survive the initial event

usually develop chronic dissection with more variable symptoms.

Classification

Depending on the timing, the dissection can be acute, when the patient is admitted to the clinic within the first two weeks, or chronic when the patient comes to the clinic more than two months after the initial event. Subacute dissection is added recently to this classification to describe patients in the period between two weeks and two months from the initial event.

There are two classification systems most frequently used in the clinical practice, and they describe different types of dissection depending on the location, and the extent of the dissection-De Bakey, and Stanford classification. In De Bakey classification there are four groups of aortic dissections depending on the location and the extent (Figure 1).

Stanford classification is more functional one, and it differs two types of dissection, type A-where all dissections that include the ascendant part of the aorta are classified, and type B when the ascendant part of the aorta is not included in the dissection.

The tear in the inner layer occurs in the ascendant part of the aorta in 70% of all dissections, 10% in the arch, and the rest is in the descendent part.

In Stanford A in 90% the tear occurs in the ascendant part of the aorta with distal propagation, and in 10% the tear is in the arch.

In Stanford B in 85% the tear is in the descendent part of the aorta, and in 15% in the arch.

Stanford A dissections are twice more often than Stanford B.

Epidemiology

Aortic dissection is the most frequently diagnosed lethal condition of the aorta. There is an estimated worldwide prevalence of 0.5-2.95 per 100.000 per year. In Macedonia it will be 10-30 cases per year. It is an estimated number, and the real number is probably quite larger due to autopsies that show that many of the dissections have fatal results without being diagnosed [1,2].

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Fig. 1. Types of dissection of the thoracic aorta according to De Bakey

Etiology

Around 40% of patients with acute aortic dissection die. If not treated, Stanford A, 25% of the patients will die in the first 24 hours, 50% in the first week and 90% in the first month. Type 2 has 70% survival with medical therapy [5,6]. There is not a single factor that directly causes the dissection. Usually we speak about risk factors. The most common risk factor is hypertension. It weakens the media and gives the opportunity to other forces to result in dissection [7,8]. Other risk factors are shown in Table 1.

Table 1. Risk factors for Type A and B Thoracic Aortic Dissection

Hypertension
Connective tissue disorders:
<i>Ehlers-Danlos Syndrome</i>
<i>Marfan disease</i>
<i>Turner Syndrome</i>
Cystic medial disease of aorta
Aortitis
Iatrogenic
Atherosclerosis
Thoracic Aortic Aneurysm
Bicuspid Aortic Valve
Trauma
Pharmacologic
Coarctation of the aorta
Hypervolemia (pregnancy)
Congenital aortic stenosis
Polycystic kidney disease
Pheochromocytoma
Sheehan syndrome
Cushing syndrome

Clinical presentation – Symptoms

Clinical presentation of thoracic aorta dissection depends on the type of the aorta. Usually the symptoms begin with strong interscapular pain, followed by retrosternal pain, together with dyspnea, and later symptoms depending on the compromitiation of the blood vessels exits from the aorta (neurological symptoms, anuria, cold ex-

tremities, lack of sensibility and motion). Type A may rupture intrapericardially resulting in tamponade. Type B ruptures in the left pleural space. If the rupture doesn't occur, then false lumen usually progresses in saccular aneurysm. In patients with chronic dissections, 56% of the lethal results are due to a rupture of the saccular aneurysm [3,4].

Type A dissection may be presented with aortic regurgitation (due to the loss of the commissural function), myocardial infarction (because of the compression of the coronary ostia), cardiac tamponade, vena cava superior syndrome, neurological symptoms, shock.

Around 40% of the patients suffering from acute dissection of the thoracic aorta die. Mortality of the Stanford type A dissections if left untreated is 25% in the first 24 hours, 50% in the first week, and 90% in the first month after the initial event. Type B has 60-80% survival rate with medicamentous treatment.

Case report

Preoperative condition

A 59-year-old patient with symptoms of chest pain and pain in the right leg was admitted in the ICU at the University Cardiology Clinic-Skopje. The symptoms began on the night, one day before admission, starting with chest pain and dyspnea, and afterwards the pain in the right leg started in the hip and later spreading to the foot with inability to move the leg.

The patient had history of sinus bradycardia and obstructive syndrome. He said he was receiving cardiac and pulmonary drug therapy, but without documentation on the condition and treatment (Tab. Aspirin, Tab. Enap, Tab. HHTZ, Tab. Aminophylline).

The patient is a heavy smoker, and has no food or drug allergies.

On admission to the Cardiology Clinic, the patient had high-pitched auscultatory sounds bilaterally. His blood pressure was 120/80 mmHg and heart rate 45.

His laboratory results were normal.

A cardiac surgeon was called for consultation and the patient underwent diagnostic procedures to assess his condition.

Echocardiography

The dimensions of the left ventricle were normal, with good systolic function. Kinetics of the basal third of the lower wall was reduced.

Valves: mitral valve had fibrosis and light mitral regurgitation.

Aortic valve had three leaflets, no stenosis and fibrosis on the leaflets.

Tricuspid valve – light regurgitation.

Aorta: Atheromatous wall. Dimensions of the aorta in the visualized part were within the reference range. The arch and the descending part could not be visualized.

No signs of PAH, and pericard without effusion.

Doppler of lower extremities showed pathologic signals on the right AFD and AP.

CT angiography showed dissection of the aorta beginning right after the exit from the heart, continuing through the ascending aorta and aortic arch where double lumen exists. Dissection continued through the whole thoracic aorta and abdominal aorta. Dissection of both iliacas is shown, the right in the upper part, and the whole left (Figure 2, 3, 4 and 5).

With these investigations the patient was diagnosed with dissection of the aorta, Stanford A, De Bakey I, and our cardiosurgery team indicated an urgent surgery of the thoracic aorta.

The situation was explained to the patient, and the urgent need for surgery was emphasized, but still, he did not want to sign the consent for the surgery. Thus, at that moment he did not undergo the surgery.

Fig. 2. Dissection of the left carotid artery and the left subclavia and brachiocephalic trunk without dissection

Fig. 3. Dissection of the aortic arch

He stayed at the Cardiology Clinic, where he was treated with analgesic and antihypertensive drugs.

Two days later the patient's condition worsened and he finally signed the consent for surgery, and he was transferred to UC for State Cardiosurgery.

He was admitted in an extremely bad general condition, tension of the left arm was 190/102 mmHg, and of the right arm 180/93 mmHg, with heart rate 96/min, with border blood gas values. He had obvious venous distention of the chest wall and his neck and cyanosis, as part of well developed vena cava superior syndrome.

Fig. 4. Dissection of the aorta and aorta descendens at the level of the branching

Prior to the surgery his laboratory results were: WBC-13.7; RBC-4.12; HGB-140; Hct-0.417; Plt-62; Glucose-5.86; Urea-10.2; Creatinine-88; Total proteins-66; Albumins-

Fig. 5. Dissection of the abdominal aorta at the level of the branching of renal arteries of pulmonary arteries

39; Globulins-27; ALT-40; AST-63; LDH-364; Na-137; K-3.8; Cl-99; Ca-2.23; Mg-0.77; CRP-305.9; Fe-2.9; Lact-2.82. Heart enzymes were: CK-1817; CK-MB-49; Troponin-27.43. INR-1.0, D-dimers-24500.

Preoperative risk of cardiac surgical intervention as estimated mortality according to EUROSCORE calculation was 28.6%.

Operation

Resectio aortae ascendens, arcus aortae, pars proximalis aortae thoracalis, pars proximalis arteriae subclaviae sinister et pars proximalis a carotis communis sinister. Reconstructio aortae ascendens cum Dacron graft No. 24. Reconstructio pars proximalis aortae thoracalis et arcus aortae cum Dacron graft No. 22. Sutura arteriae subclaviae. Reconstructio a. carotis communis sinister cum interpositionem Dacron grafti No. 8.

The patient with acute dissection of the aorta (type I De Bekey) (type A Stanford), and interpericardial rupture of the aorta, with chronic obstructive pulmonary disease, occlusion of the right iliac artery, ischemia of the right leg, without neurological disorder.

He was operated on under general endotracheal anesthesia. First we found and isolated the right axillary artery, and then we looped it. Then we gave 5000 IU of heparin and sutured 8 mm graft "end to side" on the artery, placing an arterial cannula through the graft (Figure 6).

Median sternotomy followed, then pericardiotomy, venous cannulation. Then we put the patient on the EKG/ ECG machine. Then we clamped the aorta just beneath the brachiocephalic trunk, we started with antegrade perfusion through the axillary artery, we resected the aneurysm and gave cardioplegia direct into the coronary ostia.

Then we cooled the patient to 18°C, started with total circulatory arrest, declamped the aorta, and we excised

Fig. 6. Arterial cannula placed through the graft in the right axillary artery

the whole diseased aorta (the arch and part of the descending aorta). Then we reimplanted the brachiocephalic trunk and the left carotid to the new arch. We put a new clamp on the prosthesis, made the proximal anastomosis, rewarmed the patient, and when the parameters including heart work were good, we stopped the machine. After that we did hemostasis and closed the patient in usual manner (Figure 7, 8 and 9).

Postoperative treatment

Postoperatively the patient was transferred and treated in the ICU at the UC of State Cardio surgery. After transferring the patient from the operating theater he was given adrenaline and noradrenaline support, which was reduced in the following days and fully stopped at the third postoperative day. The therapy included solutions, laxatives, cardio tonics, antihypertensive, antiulcerogenics, antiemetics, antibiotics and blood derivatives.

Fig. 7. False lumen obliterated with tissue adhesive glue inserted between the flap and the adventitia, and Dacron graft size 24 sutured afterwards

Fig. 8. Suturing the proximal to distal graft and forming a new arch with consequent sutured dacron graft 8 mm

Fig. 9. Final view of the newly formed arch with dacron graft size 8 mm from the left carotid artery and truncus brachiocephalicus sutured on it

The patient was hemodynamically stable in regular sinus rhythm. Regarding postoperative respiratory condition, the patient was still with marginal levels of blood gases, because of the bad pulmonary condition preoperatively. He had normal hour portions of diuresis, with normal laboratory results. He was extubated on the second postoperative day, but still was respiratory limited, so in few times backed on the invasive ventilation. Drains were taken out on the sixth postoperative day, after the drainage stopped. On X-ray we saw atelectasis, and

performed bronchial aspiration and bronchial lavage, which resulted in improvement of the patient condition. At the fourteenth postoperative day, the patient was in improved general condition and transferred to the cardiology ward.

Conflict of interest statement. None declared.

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