

AORTIC DISSECTION TYPE A IN COMBINATION WITH ACUTE ANTERIOR MYOCARDIAL INFARCTION. MYOCARDIAL PROTECTION

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Summary

Acute aortic dissection is an extremely serious condition with a high mortality risk. Symptoms may mimic other emergencies such as myocardial ischemia, often leading to misdiagnosis. A combination between coronary hypoperfusion and aortic dissection is relatively rare, but when present it could be fatal. Early diagnosis and correct surgical approach are essential to success in those patients and the high mortality rate requires improvement of myocardial protection. Development of acute cardiac ischemia on top of mandatory global ischemia is a serious precondition for a severe postcardiotomy syndrome, and only excellent cardiac protection can lead to successful surgical intervention.

Keywords: myocardial protection, aortic dissection, acute myocardial infarction

Introduction

Aortic dissection can present with a wide range of symptoms, and its correct diagnosis can sometimes be a challenge to emergency physicians [1]. Approximately one-quarter of patients with acute aortic syndrome have electrocardiographic abnormalities, resembling acute coronary syndrome. [2] The treatment of aortic dissection complicated by coronary hypoperfusion due to the extension of the lesion in the direction of the coronary ostium is a real challenge for surgeons and a good outcome depends on the timely diagnosis and, to a particularly large extent, on flawless myocardial protection. There are not many reported cases of this type of complication. Moreover, most of them end up fatally.

Here we present a complicated case with Stanford type A aortic dissection, which was successfully treated, despite the delay in surgical intervention and the subsequent development of acute aortic disease with STEMI. The Bentall and De Bono technique with reimplantation of the coronary ostium in the vascular prosthesis, first described in 1968, was chosen [3].

Case report

A middle-aged patient was transported by a Centre For Emergency Medical Assistance team to the emergency department (ER) of the hospital institution with a working diagnosis of unstable angina pectoris (UA). Severe, tearing chest and back pain at rest and a brief loss of consciousness were reported in the history. The patient had a five-year history of dilated ascending aorta. In the ER, he was responsive, adequate, and oriented, and complained of persistent oppression in the chest. The examination revealed a hypersthenic body habitus, pink skin and visible mucous membranes, a sonorous percutaneous sound in the chest, and bilateral rough vesicular breathing. The heart rate was rhythmic (70 bpm), the heart sounds were dull, and the blood pressure (BP) was 150/80mm Hg, with a difference between the two arms of more than 30 mm Hg. No other abnormalities were found. Echocardiography showed a dilated ascending aorta and aortic regurgitation grade II. A CT scan visualized an aortic arch with a diameter of 60 mm and an ascending aorta with a diameter of 55 mm, describing a para-aortic and mediastinal hematoma. A diagnosis of aortic dissection type A was made.

The patient refused surgical treatment and was admitted to the intensive cardiology department for conservative treatment.

Laboratory tests indicated levels of troponin I <0.20 ng/ml and D-dimer – 1672.0 ng/ml. There were no specific alterations in other parameters. Two days later, an acute anterior myocardial infarction was added to the diagnosis: Troponin I - 81.4, CPK-2035.0 U/l, CPK-MB- 140.0 U/l, and typical ECG changes were recorded. The patient changed his mind and was scheduled for surgery.

Preoperative transthoracic echocardiography provided the following data: ejection fraction (EF) 40%, end-diastolic volume (EDV) 159 ml, end-systolic volume (ESV) 96 ml, interventricular septal end (IVS) – 14 mm, left ventricular posterior wall (LVPW) – 14 mm, dissection flap in the ascending aorta, aortic arch and descending aorta, aortic regurgitation – grade III, akinesia of the left ventricular anterior wall and apex, and systolic LV dysfunction. (Fig. 1) Euro Score of 3.82% was calculated, and the patient was immediately taken to the operating theatre.

General anaesthesia was administered according to a standard protocol for surgery in acute aortic syndrome. After careful preparation of the operative field, a median sternotomy was performed. The patient was fully heparinized (activated clotting time ≥ 500 s), and cardiopulmonary bypass (CPB) was instituted via standard central cannulation. The right superior pulmonary vein was cannulated to the left ventricle venting. An aortic cross-clamp

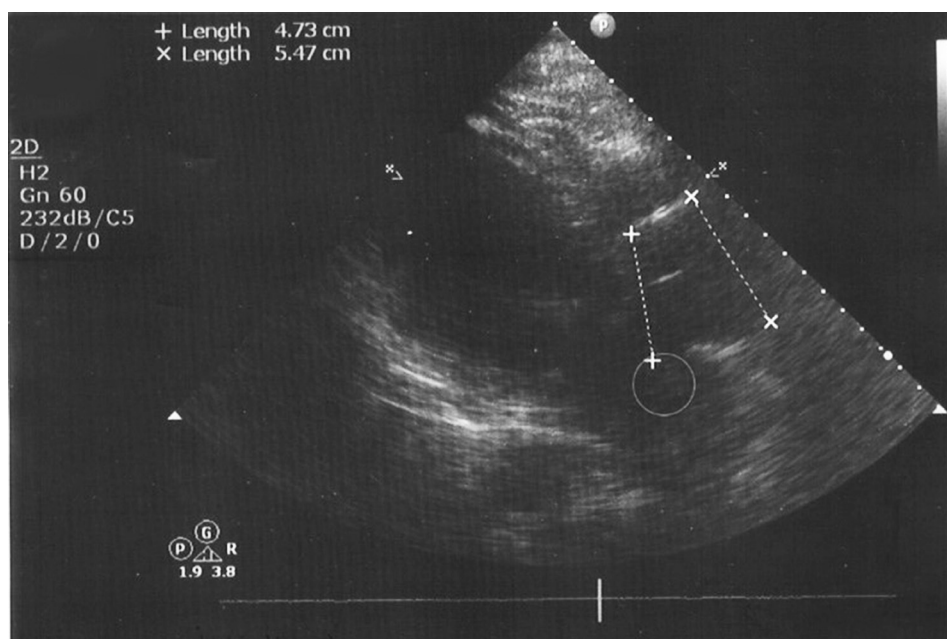


Figure 1. Preoperative transthoracic echocardiography

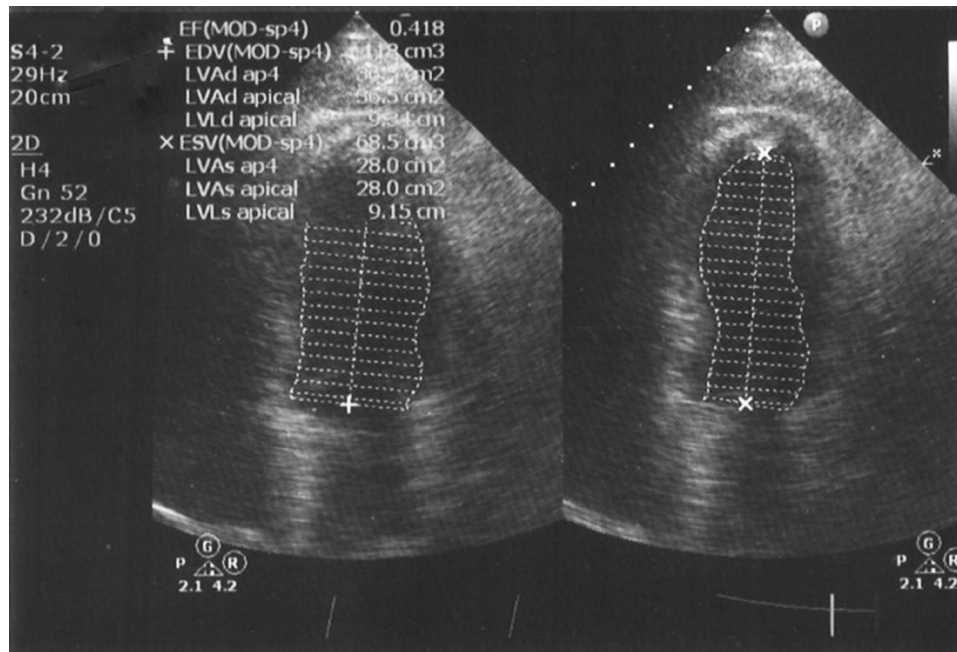


Figure 2. Control transthoracic echocardiography

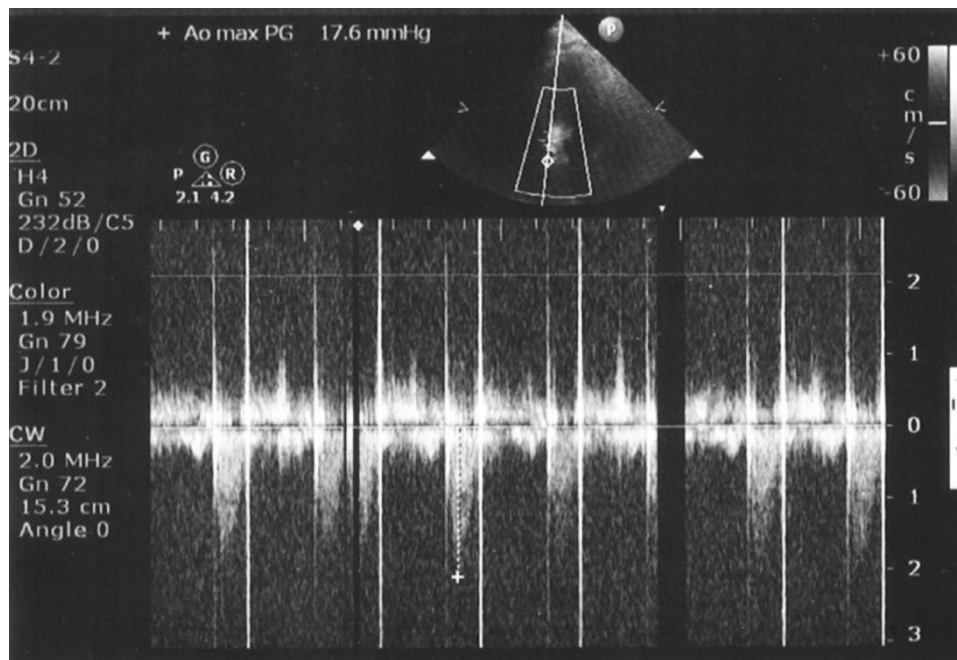


Figure 3. Control transthoracic echocardiography

at 32°C was performed. The ascending aorta was transected on its anterior surface. An infusion of 2000 ml cold-modified del Nido cardioplegic solution was delivered through the coronary ostia. An aortic flap was found that reached the left coronary ostium. Bentall de Bono surgery was performed under the conditions of CPB with a flow rate of 5.0-5.50 l/min. The valve and the aneurysmal portion of the aorta were excised. A Carbomedics Conduit 23/26 mm was implanted using 19 single U-shaped etibond 2/0 sutures with

felt, and an adequate function of the prosthesis was achieved. A second dose of 1000 ml cold-modified del Nido cardioplegic solution was given. The coronary arteries were re-implanted through button incisions in the vascular prosthesis using a continuous 5/0 prolene suture. The distal anastomosis between the vascular prosthesis and the native aorta was constructed using a continuous 3/0 prolene suture with felt tape. The cross-clamp was removed. After the required reperfusion time, CPB was discontinued using

catecholamine support (Dopamine, Dobutamine, and Adrenaline), additionally implanting an intra-aortic balloon pump (I.A.B.P.) Because of the acute myocardial infarction. Protaminization was administered to neutralize the heparin. According to the protocol, a decision was made to extend the reperfusion time to restore cardiac function after the longer clamping time: CPB time - 307 minutes, aortic cross-clamp time 150 minutes, reperfusion time 131 minutes.

After the end of the operation, the patient was taken to the intensive care unit with satisfactory hemodynamics, HR - 90/min, BP - 100/60. CVP at discharge was + 5 mmHg, pH 7.35 (7.35-7.45), pO_2 74 mm Hg (80–100 mm Hg), pCO_2 44 mmHg (35-45 mm Hg), Lac 5.9 mmol/l (0.5 - 2.2 mmol/l), BE 1.3 mmol/l (-2 to +2 mmol/l), Hct 35% (38-48%), CPK 5948U/l (55-170 U/L), MB 515U/l (5-25 U/L), creatinine 160 mmol/l (65-120 mmol/l) and urea 7.7 mmol/l (2.5-7.1 mmol/l). After 24 hours, CPK 2301U/l, MB 56U/l, creatinine 150 mmol/l and urea 7.9 mmol/l were reported. The patient was extubated on the 36th postoperative hour and was discharged from intensive care on the fifth postoperative day. On the 2nd postoperative day, control echocardiography was performed and revealed EF 41%, EDV 118 ml, ESV 69 ml, AVR - mechanical prosthesis with a gradient of 18/9 mm Hg, without pericardial effusion. (Fig. 2,3)

The patient was discharged on the 10th postoperative day. At follow-up, within a month later he reported he had resumed his usual daily physical activity.

Discussion

Aortic dissection is often misdiagnosed as an acute coronary syndrome, in which there is a well-established diagnostic approach based on ECG changes in combination with elevated cardiac enzymes. However, there are no readily available equivalent diagnostic tests for aortic dissection. An investigation on biomarkers in Acute Aortic Dissection (IRAD-Bio) evaluated the diagnostic performance of D-dimer testing in a study population of patients with suspected aortic dissection [4]. At a cut-off level of 1600 ng/mL, D-dimer can be a useful tool in the differential diagnosis between

aortic dissection and myocardial infarction [2]. The main diagnostic methods which help to localize the affected segment are instrumental, including transthoracic and transesophageal echocardiography, computed tomography, magnetic resonance, and aortography. D-dimer testing and transthoracic echocardiography (TTE) are useful in the initial screening of patients with chest pain, and contrast-enhanced CT provides further details about the affected segment of the aorta and its outflow vessels. Due to its simplicity, the most commonly used classification of aortic dissections is that of Stanford University. It includes two types of dissection: type A (involving the ascending aorta, including or not the descending aorta) and type B (only the descending aorta). The classification of the disease is of particular importance, due to the specificity of the surgical approach in different cases. Acute myocardial infarction due to coronary ostium extension of Stanford type A aortic dissection is rare but life-threatening. It occurs in approximately 3% of patients with acute aortic syndrome, most commonly affecting the right coronary artery orifice [5]. Despite the advances in diagnostics and surgical techniques, recent reports have revealed a surgical mortality rate of over 20%. In 2001, Neri published a study conducted between 1985 and 2000. He reported that 24 patients out of 211 (11.3%) had an acute aortic dissection type A in combination with dissection of at least one of the coronary ostia. In eleven cases, the ostium of the left coronary artery was affected, in four cases the ostium of the right coronary artery was affected, and in the remaining nine cases both coronary arteries were affected. In his study, he prefers to repair the dissected coronary arteries instead of revascularizing the affected vessel by coronary artery bypass grafting (CABG). He describes various techniques for local repair, depending on the type and extent of the lesion, as well as the coronary vessel involved [6]. The reported in-hospital mortality rate was 20%. From this study, he concluded that direct repair of the affected coronary arteries is a safe alternative to coronary artery bypass grafting. In contrast, Kawahito in his report presented 12 patients out of 196, with a preference to perform coronary artery bypass simultaneously with aortic reconstruction [7]. This author recorded 4 mortality cases or an

intra-operative mortality rate of 33.3%. Different surgical approaches largely solve the problems posed by modern cardiac surgery. The persistent high mortality in patients with combined acute syndrome requires improvement of myocardial protection, which will subsequently improve outcomes. Kazui emphasizes the importance of myocardial protection during this type of surgery. He proposed using blood cardioplegia delivered both retrogradely through the coronary sinus and anterogradely through the undissected coronary ostium [8]. The Bentall de Bono procedure is the primary treatment choice for patients with dilatation of the sinuses of Valsalva and the ascending aorta. Regardless of its initial application in aneurysms of the aorta, it quickly found its place in the surgical palette of methods used in acute aortic syndrome. In the considered case, the aortic root was 50 mm, which determined the choice of operative approach to the particular case. For myocardial protection, we used antegrade cardioplegic protection modified del Nido, combined with moderate hypothermia and longer reperfusion. We chose to implant the I.A.B.P. to improve coronary perfusion, which was started in the reperfusion period and continued for the following 72 hours. Longer clamping of the aorta in combination with myocardial ischemia predisposes to an increased risk of postcardiotomy syndrome, but postoperative laboratory results confirmed the efficiency and reliability of the protection used. The patient was successfully extubated on the 36th postoperative hour and discharged from the hospital on the 10th postoperative day.

Conclusions

We presented a Bentall de Bono procedure in a patient with type A dissection in combination with an acute anterior infarction. This surgical approach is a safe alternative to other more conservative options. A longer period of global cardiac ischemia can be effectively managed by choosing appropriate myocardial protection. Carrying out the intervention should not pose any additional difficulties for a team with experience in a classical approach.

References:

1. Hsieh TH, Tsai LM, Tsai MZ. Characteristics of and atypical presentations in patients with acute aortic dissection - A single center experience. *Acta Cardiol Sin.* 2011;27:238-43.
2. Wu BT, Li CY, Chen YT. Type A Aortic Dissection Presenting with Inferior ST-Elevation Myocardial Infarction. *Acta Cardiol Sin.* 2014;30(3):248-52.
3. Bentall H, De Bono A. A technique for complete replacement of the ascending aorta. *Thorax*1968;23:338-9
4. Suzuki T, Distanto A, Zizza A, Trimarchi S, Villani M, Salerno Uriarte JA et al. Diagnosis of acute aortic dissection by D-dimer: the International Registry of Acute Aortic Dissection Substudy on Biomarkers (IRAD-Bio) experience. *Circulation.* 2009;119(20):2702-07.
5. Hagan PG, Nienaber CA, Isselbacher EM, Bruckman D, Karavite D J, Russman P L et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. *JAMA.* 2000;283(7):897-903.
6. Neri E, Toscano T, Papalia U, Frati G, Massetti M, Capannini G, et al. Proximal aortic dissection with coronary malperfusion: presentation, management, and outcome. *J Thorac Cardiovasc Surg.* 2001;121(3):552-60.
7. Kawahito K, Adachi H, Murata S, Yamaguchi A, Ino T. Coronary malperfusion due to type A aortic dissection: mechanism and surgical management. *Ann Thorac Surg.* 2003;76(5):1471-6
8. Kazui T. Invited commentary. *Ann Thorac Surg.* 2006;82(5):1677-8