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CASE REPORT

A RARE CASE OF ACUTE STANFORD TYPE A AORTIC DISSECTION PRESENTING WITH ANTERIOR ST-ELEVATION MYOCARDIAL INFARCTION

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Abstract. Introduction. *Aortic dissections occur as a result of a tear in the intimal layer, continued longitudinal splitting within the intima and media, and formation of false lumen. This could lead to sudden death or severe aortic regurgitation and cardiogenic shock. The presented case here describes a patient with acute anterior ST-elevation myocardial infarction due to acute Stanford type A ascending aortic dissection.* ***Case presentation.*** *A 55-year-old male presented with severe chest pain, radiating into the back, jaw, and left arm, and signs of cardiogenic shock. Electrocardiography showed acute anterior ST-elevation myocardial infarction and echocardiography confirmed that there was a reduced left ventricle ejection fraction (38% calculated using the Simpson method), severe aortic regurgitation, and wall motion abnormalities. Based on these findings, we made a diagnosis of acute myocardial infarction. In accordance with the current guidelines, we opted for an interventional therapeutic approach. Angiography showed left main trunk dissection extending to the left anterior descending coronary artery caused by ascending aorta dissection. This finding altered the diagnosis and treatment plan and the patient was immediately sent to the operating room for emergency surgery.* ***Conclusions.*** *Aortic dissection should be suspected in patients presenting with acute anterior ST-elevation myocardial infarction, severe aortic regurgitation, and cardiogenic shock. Involvement of the left main trunk and left anterior descending artery occurs much more rarely than that of the right coronary artery, which causes inferior myocardial infarction.*

Key words: *ascending aorta dissection, acute myocardial infarction, intimal flap, false lumen left main trunk, left anterior descending coronary artery, cardiogenic shock, severe aortic regurgitation, angiography, surgical treatment*

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INTRODUCTION

An aortic dissection is caused by a tear in the intimal layer of the aortic wall. As a result, the blood flow provokes a continuing longitudinal splitting within the intima and media and formation of false lumen. This could lead to sudden death or development of further complications such as severe aortic regurgitation, cardiogenic shock, myocardial infarction, and arrhythmias. Aortic dissection is considered to be the most common cause of aorta-related death. These dissections are associated with

some known risk factors such as hypertension, atherosclerotic disease, male gender, connective tissue disorders, especially in younger patients (Marfan's syndrome or Ehler's-Danlos syndrome), and bicuspid aortic valve.

Aortic dissections may be categorized according to the Stanford classification into type A and type B dissections. Type A aortic dissections affect the ascending aorta and involve any part of the aorta proximal to the origin of the left subclavian artery. Type B dissections do not involve the ascending aorta and arise distal to the origin of the left subclavian artery.

A rare but life-threatening complication of type A dissection is acute myocardial infarction, which is related to the extension of the dissection flap into the ostium of the coronary artery in 1-2% of cases. Involvement of the left main trunk and left anterior descending artery is much rarer compared to that of the right coronary artery. Myocardial infarction occurs more often than aortic dissection in emergency departments [1]. Most ST-elevation myocardial infarctions are caused by a rupture of an atherosclerotic plaque, subsequent thrombosis, and coronary occlusion so patients benefit from urgent percutaneous coronary intervention. Patients presenting with type A aortic dissection need emergency surgery [2]. Therefore, when acute dissection is complicated with acute myocardial infarction, the correct diagnosis of dissection can be elusive and these patients might be treated with primary percutaneous coronary intervention (PCI) instead of surgical operation [3]. In these cases, the outcome can be catastrophic [4].

The reported case is a rare presentation of acute anterior myocardial infarction due to an acute Stanford type A aortic dissection involving the left main trunk of the coronary artery, causing cardiogenic shock, and severe aortic regurgitation.

CASE PRESENTATION

A 55-year-old male from Plovdiv, Bulgaria, was admitted for a hospital treatment due to sudden severe sharp chest pain radiating into the back, jaw, and left arm, accompanied by sweating, nausea, and vomiting. The complaints started 40 minutes before admission. The patient suffered from arterial hypertension but no current treatment was provided. He presented at the emergency department in poor general condition, pale and sweaty. The physical examination of cardiovascular system revealed sinus tachycardia (120 beats per minute) and hypotonic blood pressure (80/50 mm Hg). The diagnostic process continued with recording of an **electrocardiogram (ECG)** which revealed ST-depression in standard leads I, II, III (Fig. 1A), ST-elevation and Q wave in aVR, aVL, ST-depression in aVF (Fig. 1B), ST-elevation and Q wave in the precordial leads V1-V3 (Fig. 1C), and ST-elevation in V5-V6 (Fig. 1D).

The initial diagnosis based on the clinical presentation and ECG findings was acute anterior myocardial infarction and the patient was transported to the cardiology department. During monitoring at the intensive care unit, a ventricular ectopic beats R-on-T phenomenon (V Lown class) was registered. To assess cardiac function, urgent echocardiography was performed. It showed left ventricular hypertrophy (septum and left ventricle posterior wall 1.2 cm) as a result of untreated arterial hypertension. Left ventricle systolic function was assessed by measuring the left ventricle ejection fraction (LVEF) using two separate methods. It was found to be significantly reduced – LVEF 40% measured by the Teichholz method and 38% measured by the Simpson method (normal range 55 to 70%). Valve assessment performed using color Doppler showed moderate mitral regurgitation (II grade), severe aortic regurgitation (IV grade), severe tricuspid regurgitation (III grade), and elevated tricuspid valve gradient (52 mHg) (normal range 12.6 to 29. 3 mm Hg). Elevated systolic pulmonary artery pressure of 62 mm Hg (normal range 18 to 25 mm Hg) was indicative for pulmonary hypertension and was registered by using pulse wave Doppler in the parasternal short axis view. Wall motion abnormality such as apical dyskinesia was observed in apical four-chamber view and was taken as a result of regional myocardial ischemia due to coronary artery occlusion.

The laboratory blood tests revealed leukocytosis (13.29×10^9/l) (normal range 3.5 to 10.5×109/l) and elevated blood glucose (8.76 mmol/l) due to severe stress reaction. Myocardial injury was suspected on the basis of elevated creatine phosphokinase (CPK, 178 U/l) (normal range 0 to 171 U/l) and elevated CPK-MB (25.4 U/l) (normal range 0 to 25 U/l). The lipid profile revealed an additional risk factor for atherosclerotic disease, a concomitant dyslipidemia: elevated total cholesterol (6.14 mmol/l) (normal range 3.5 to 5.17 mmol/l) and triglycerides (5.38 mmol/l) (normal range up to 1.69 mmol/l). The initial diagnosis based on clinical presentation, ECG changes, echocardiography data, and laboratory results was acute anterior myocardial infarction. In accordance with the European guidelines for the treatment of acute coronary syndrome, interventional strategy was undertaken. The percutaneous coronary intervention (PCI) using radial access showed left main trunk dissection involving proximal left anterior descending artery (LAD) (Fig. 2) with partial perfusion and thrombolysis in myocardial infarction (TIMI) flow grade II.

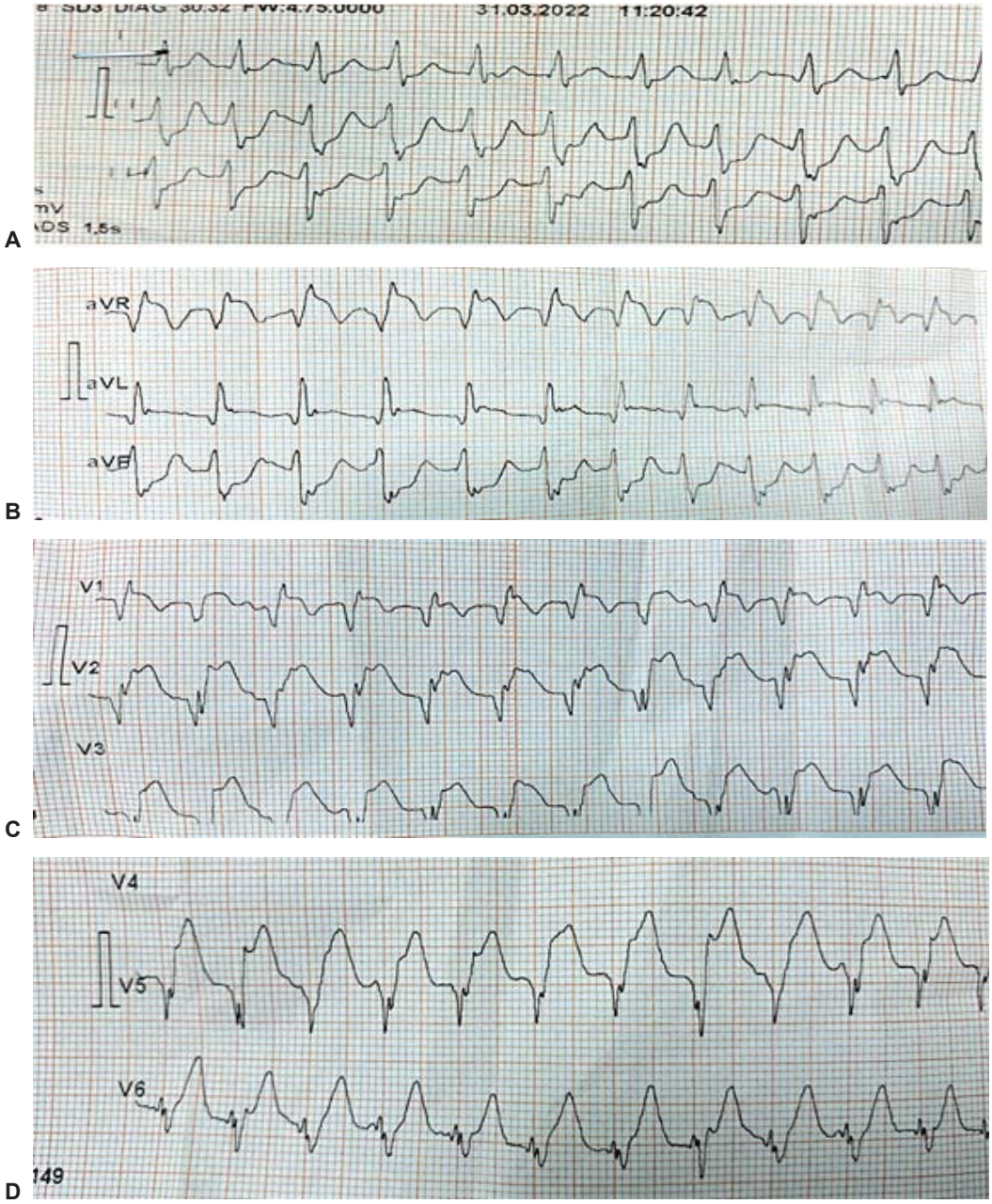

Fig. 1. Electrocardiogram. **A)** ST-depression in standard leads I, II, III; **B)** ST-elevation and Q wave in standard leads aVR, aVL, ST-depression in aVF; **C)** ST-elevation and Q wave in precordial leads V1-V3; **D)** ST-elevation in precordial leads V5-V6

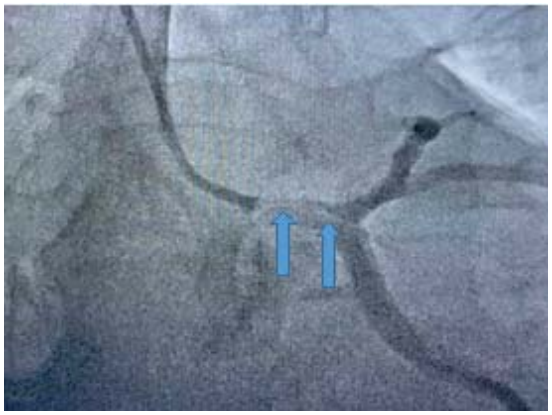

Fig. 2. Percutaneous coronary intervention. Left main trunk dissection

The left circumflex and right coronary arteries were intact with normal blood flow.

A decision to undertake an aortography investigation was made based on the echo findings of severe aortic regurgitation. The investigation revealed dilated aortic root and ascending aorta (Fig. 3A) and presence of intimal flap (Fig. 3B). The pigtail catheter was placed into the true lumen (Fig. 3C).

All these findings confirmed the accuracy of the diagnosis of aortic dissection previously misdiagnosed with acute myocardial infarction due to coronary atherothrombosis. Treatment was based on the initial diagnosis. As the patient presented with typical

complains, ECG changes, echo data of wall motion abnormalities, and reduced left ventricle ejection fraction, laboratory data indicating acute anterior STEMI, the initial treatment included anticoagulant (unfractionated heparin 5000 E loading dose followed by an infusion of 1000 E/hour) and antiplatelet agent (acetylsalicylic acid 250 mg loading dose). Due to hemodynamic instability that led to the developed cardiogenic shock, inotropic drugs were administered (dopamine and dobutamine at an infusion rate of 2.5 μg/kg/min). The complex ventricular arrhythmias were treated with amiodarone infusion and magnesium. Urgent transportation to cardiac surgery clinic was arranged right after confirmation of severe ascending aorta dissection of Stanford type A. Unfortunately, the patient died in the ambulance during the transportation.

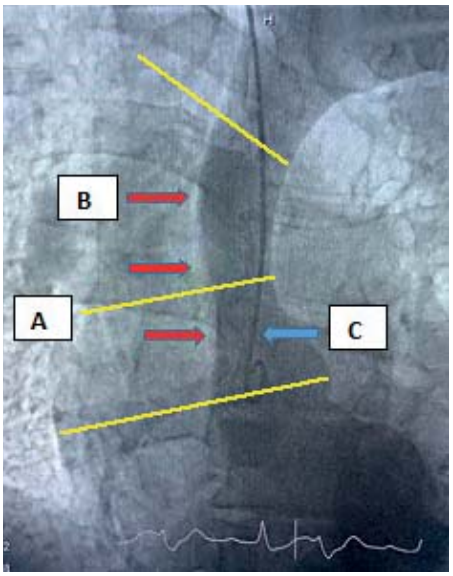

Fig. 3. Aortography. **A)** Dilated aortic root and ascending aorta; **B)** Presence of intimal flap; **C)** Catheter placed into true lumen

DISCUSSION

The acute aortic syndrome includes aortic dissection, intramural hematoma, and a penetrating aortic ulcer. Although dissection is one of the most common disorders associated with aortic disease, it is also the most fatal one, for a variety of reasons, including the patient presenting with unusual symptoms or severe acute heart disease. Therefore, healthcare professionals are faced with a serious challenge leading to difficulties in making an accurate diagnosis and, as a result, delaying the commencement of the correct surgical therapy, resulting in an increase in mortality rate. Several case reports of a Stanford type A aortic dissection in combination with a myocardial infarction have been published [5-12]. When a type A aortic dissection extends to the left main trunk of the coronary artery, life-threatening hemodynamic instability may occur, including cardiogenic shock, severe systolic LV dysfunction, frequently ending with sudden death [15]. Acute myocardial infarction due to extension of an acute Stanford type A aortic dissection is very rare. When combined with myocardial infarction due to left main trunk involvement, it is one of the most lethal clinical situations. This requires a prompt and accurate diagnosis and urgent surgical treatment to save the patient's life. However, it is difficult to make a differential diagnosis between acute myocardial infarction and myocardial infarction due to the extension of aortic dissection into the coronary artery. Ischemia could be a result of dynamic hemodynamic changes as the intimal flap causes an occasional obstruction of the coronary artery. Another possible mechanism is static, in which the hematoma narrows the lumen of the vessel [2]. Evaluation of the ascending aorta using either transthoracic or transesophageal echography and a computed tomography (CT) scan, when possible, is extremely important for accurate diagnosis. Interventional study using aortography with manual injection of the contrast through a pigtail catheter is effective for detecting the aortic dissection. The presence of an intimal flap and dilation of the aorta are crucial for exact diagnosis of aortic dissection. A resistance noted while advancing the diagnostic catheter during PCI is suspicious for aortic dissection. If the catheter is placed in the true aortic lumen, the movement of the catheter is free and smooth but if placed in the false lumen, the advancement is difficult [1].

The presented clinical case is significant for clinical practice because it is a rare demonstration of acute type A dissection involving the trunk of the left coronary artery, resulting in acute blood flow disruption and the development of another life-threatening condition, acute anterior myocardial infarction with ST-elevation. In comparison, involvement of the right coronary artery in acute aortic dissection is far more common. Aortic dissection must be addressed as an underlying etiological cause when patients present to the emergency room with ST-elevation acute coronary syndrome. Because of its rarity, the current clinical case demonstrates the challenges in reaching an accurate diagnosis, which requires experience and modern technical equipment, including imaging equipment. Severe aortic dissection was not suspected by emergency department physicians even during the performance of transthoracic echocardiography. Percutaneous coronary intervention and aortography provided complete vi-

sualization of the aorta and in this case served as invasive diagnostic imaging modalities for the establishment of the correct diagnosis.

CONCLUSIONS

Patients with acute anterior STEMI, significant aortic regurgitation, and cardiogenic shock should be specifically evaluated for aortic dissection. Although acute myocardial infarction has a characteristic appearance, it is crucial to actively look for symptoms of aortic dissection during physical examination, imaging (echocardiography and CT scan), and interventional tests to obtain an accurate diagnosis. Any delay could result in the patient's death. The only treatment for acute Stanford type A aortic dissection is immediate surgical repair.

Disclosure Summary: *The authors have nothing to disclose.*

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