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ACUTE AORTIC DISSECTION MANIFESTING WITH ACUTE MYOCARDIAL INFARCTION: CASE REPORT

DISSECÇÃO AGUDA DE AORTA MANIFESTANDO-SE COM INFARTO AGUDO DO MIOCÁRDIO: RELATO DE CASO

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ABSTRACT

Acute Aortic Dissection (AAD) is a cardiovascular emergency that entails high mortality - 50-68% in 48 hours and up to 85% in one month. This case involves a 65-year-old male ex-smoker who had onset of precordial pain with a burning sensation, radiating into the upper limbs, in combination with nausea. Electrocardiogram showed inferolateral wall ST elevation. He received treatment for acute myocardial infarction with acetylsalicylic acid, clopidogrel, enoxaparin and tenecteplase. Cardiac catheterization revealed Stanford type A AAD and unobstructed coronary arteries. Transthoracic echocardiogram showed moderate aortic regurgitation and aortic dissection extending from the aortic root to its proximal descending portion. The patient underwent a Bentall-De Bono procedure and right coronary artery bypass grafting using the saphenous vein due to obstruction during surgery, with good postoperative progress. AAD remains a diagnostic challenge in the emergency room. According to the International Registry of Acute Aortic Dissection, clinical findings in type A dissections include: sudden severe chest pain (86%), dorsal irradiation (47%), aortic regurgitation murmur (44%), asymmetric blood pressure (50%) or pulse (30%), mediastinal widening on chest radiograph (63%) and ST-segment elevation (4%), mainly due to right coronary ostium occlusion. The case is distinctive because of favorable progress in spite of the potentially catastrophic treatment initially targeting acute coronary atherothrombotic disease.

Keyword: Dissection; Aorta; Myocardial Infarction.

RESUMO

A dissecção aguda da aorta (DAA) é uma emergência cardiovascular que acarreta mortalidade alta, 50% a 68% em 48 horas e até 85% em um mês. Este caso refere-se a um homem com 65 anos, ex-tabagista, que teve precordialgia com queimação irradiada para membros superiores, associada a náuseas. O eletrocardiograma mostrou supradesnivelamento ST em parede inferolateral. Recebeu tratamento para infarto agudo do miocárdio com AAS, clopidogrel, enoxaparina e tenecteplase. O cateterismo cardíaco evidenciou DAA tipo A de Stanford e coronárias sem obstruções. O ecocardiograma transtorácico mostrou insuficiência aórtica moderada e dissecção estendendo-se da raiz da aorta até a porção descendente proximal. O paciente foi submetido à cirurgia de Bentall de Bono e enxerto safeno-coronário direito devido à obstrução durante a cirurgia, com boa evolução pós-operatória. A DAA continua a ser um desafio diagnóstico na sala de emergência. De acordo com International Registry of Acute Aortic Dissection, os achados clínicos nas dissecções tipo A incluem dor torácica súbita e intensa (86%), irradiação dorsal (47%), sopro de insuficiência aórtica (44%), assimetria de pressão arterial (50%) e pulsos (30%), alargamento de mediastino à radiografia de tórax (63%) e supradesnivelamento de ST (4%), majoritariamente por oclusão de óstio da coronária direita. O caso destaca-se pela evolução favorável a despeito do tratamento com potencial catastrófico inicialmente direcionado para doença coronariana aguda aterotrombótica.

Descritores: *Dissecção; Aorta; Infarto do Miocárdio.*

INTRODUCTION

Acute aortic dissection (AAD) is a cardiovascular emergency that often goes undiagnosed until an acute and often catastrophic complication occurs.[1-3] It is associated with high mortality, reaching 50-68% in 48 hours, 70% in 1 week and up to 85% in 1 month.[4-6]

The formation of a false lumen caused by delamination of the intimal layer or by hemorrhage of the medial layer

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due to rupture of the vasa vasorum leads to variable clinical conditions of chest pain, bleeding, and poor perfusion of vital organs.[5,6] The triggering event is still unknown, but most patients have a structural abnormality of the arterial wall, most commonly due to cystic medial degeneration, associated with risk factors such as uncontrolled chronic systemic arterial hypertension, atherosclerosis, smoking, drug use (crack, cocaine), and connective tissue disorders (Marfan's syndrome), among others.[1,6]

When Stanford type A AAD affects the coronary ostium (due to vessel wall dissection or compressive hematoma), complete acute coronary occlusion can occur, leading to acute ST-elevation myocardial infarction (STEMI).[6-8] The incidence of STEMI secondary to AAD is approximately 2.5-4%.[1,2]

In STEMI patients, AAD is often diagnosed late.[8-10] Incorrect therapy with antiplatelet, anticoagulant, and fibrinolytic agents leads to a significant increase in the mortality rate.[9,10] The case reported here shows the challenge of diagnosing AAD and the differences in treatment of STEMI according to the pathophysiological mechanisms of classical atherothrombosis or coronary ostium dissection secondary to AAD.

CASE REPORT

A 65-year-old male presented with burning precordial pain of 10/10 intensity, radiating to both upper limbs and associated with nausea and dizziness. He had a 10 pack-year smoking history, but stopped 28 years prior. He also had a 20-year history of chronic hepatitis B.

He presented to a municipal hospital in São Paulo as an emergency, where an electrocardiogram (ECG) showed inferior-lateral-dorsal ST-segment elevation (Figure 1). He was treated with 300 mg of acetylsalicylic acid, 300 mg of clopidogrel, and 60 mg of subcutaneous enoxaparin; fibrinolytic therapy with tenecteplase was performed 1 h after the onset of symptoms, with a decrease in pain and improvement of ST-segment elevation (Figure 1A and B). However, mild pain recurred 1 h after thrombolytic therapy, without ECG changes.

He was transferred to a tertiary care hospital on dobutamine 8 μg/kg/min, with chest pain of 3/10 intensity and progression to hypotension (blood pressure 90/60 mmHg) and tachycardia (heart rate 120 bpm); however, the cardiac and pulmonary examination was stable, and the blood pressure and peripheral pulses were bilaterally symmetrical. Emergency angiography and aortography showed non-occluded coronary arteries and Stanford type A AAD (Figure 2A and B). A transthoracic echocardiogram showed moderate aortic regurgitation, moderate pericardial effusion, and dissection extending from the aortic root to the proximal descending aorta.

Dobutamine was discontinued, beta-blocker treatment was started, and intravenous saline was administered. The hemodynamics improved, the heart rate decreased to 90 bpm, and the blood pressure stabilized. The patient was referred for emergency cardiac surgery 25 h after onset of pain. The aortic valve and a portion of the ascending aorta were replaced using a bovine pericardial tube graft, the coronary ostia were reimplanted (Bentall-De Bono surgery), and a saphenous vein graft was performed on the right coronary artery due to obstruction during surgery, with no other intraoperative complications. The patient remained hemodynamically stable postoperatively and was discharged after 17 days of hospitalization.

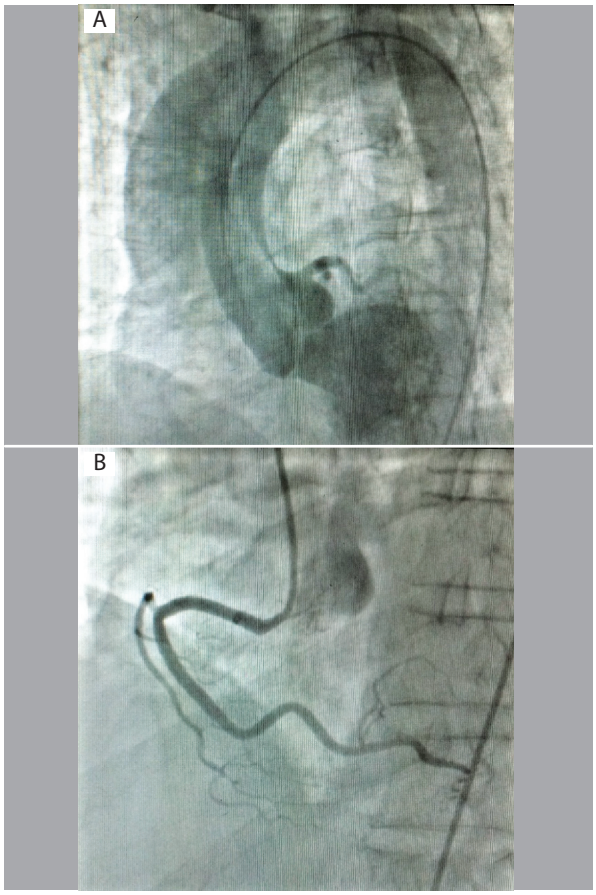

Figure 2. A) Aortography showing aortic dissection and moderate aortic regurgitation. B) Cardiac catheterization with non-occluded right coronary artery.

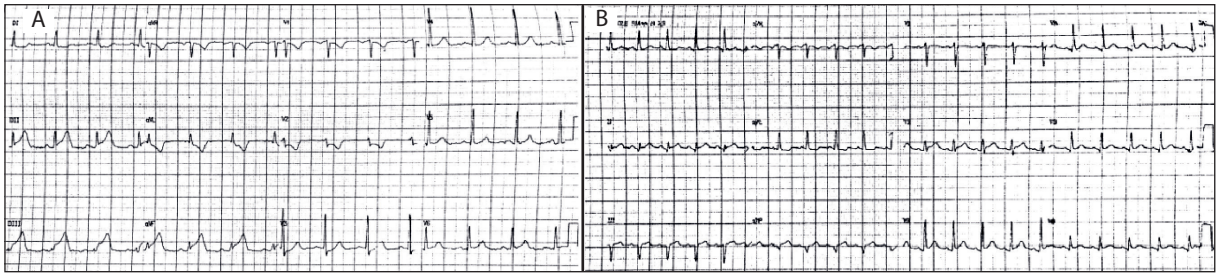

Figure 1. A) Electrocardiogram with ST-segment elevation in leads II, III, aVF, V5, and V6, and ST-segment depression in V1 and V2. B) Electrocardiogram after thrombolysis, with improvement in the electrocardiogram, but appearance of a Q wave in the inferior leads.

DISCUSSION

AAD is the most common acute aortic condition requiring urgent surgical therapy, with an estimated incidence of 5-30/million/y.[1] With a male:female ratio ranging from 2:1 to 5:1, AAD is more common in individuals aged 45-70 y, with a peak at 50-55 y for cases of proximal dissection and 60-70 y for cases of distal dissection.[5]

Systemic arterial hypertension is the most common risk factor and is present in more than 70% of patients.[6] The clinical findings in Stanford type A dissections include sudden and intense chest pain in 86%, dorsal irradiation in 47%, aortic regurgitation murmur in 44%, asymmetric blood pressure in 50%, asymmetric pulse pressure in 30%, mediastinal widening on the chest X-ray in 63%, and ST-segment elevation in 4%.[1]

STEMI chest pain is more commonly associated with Stanford type A AAD.[2] When dissection involves the descending aorta, pain can radiate to the abdomen, lumbar region, or lower limbs.[1] The present case was difficult to diagnose; the patient had severe chest pain that did not radiate to the upper back, and showed no asymmetry of pulses or blood pressure. The aortic regurgitation murmur was obscured by tachycardia at the time of admission to our service. A chest X-ray was not performed, since the patient was promptly referred to interventional cardiology after arriving at our hospital.

Dissection and myocardial infarction with ST-segment elevation can occur concomitantly; the incidence is low, with involvement of the right coronary artery and inferior wall infarction more common than left coronary artery involvement.[2,4] Myocardial ischemia can be explained by associated dissection of the coronary ostium or by reduced coronary perfusion resulting from compression of the coronary artery due to expansion of the false lumen; the ischemia can occur in the absence of obstructive coronary lesions, as in our case.[3]

Differentiation between AAD and inferior STEMI is important, but becomes difficult with concurrent presentation. A thorough history (to identify risk factors such as poorly controlled arterial hypertension and a bicuspid aortic valve), physical examination (including cardiac auscultation and assessment of bilateral peripheral pulses and blood pressure), and correct use of imaging studies (such as chest radiography, echocardiography, and computed tomography) are essential.[2,3] When diagnosis is difficult, echocardiography can reveal 3 important clues to the presence of AAD, including aortic root dilatation, eccentric aortic regurgitation, and pericardial effusion.[1,2]

The moderate aortic regurgitation found in this case was due to dilation of the proximal aorta and enlargement of the aortic ring, preventing coaptation of the leaflets, leaflet pressure down to the coaptation point, hematoma, and failure of annular support or prolapse of intimal delamination towards the left ventricle during diastole.[5]

AAD remains a diagnostic challenge in the emergency room. It is surprising to note that up to 85% of patients with dissection may not receive appropriate medical treatment in the first few hours due to diagnostic failure.[5] Imaging methods, such as echocardiography and computed tomography, can aid in rapid diagnosis.[9]

Accurate diagnosis is necessary for correct therapy, since treatment for coronary syndrome with the use of thrombolytics and anticoagulant agents can increase the mortality rate by more than 70%, mainly due to the risk of cardiac tamponade.[3]

CONCLUSION

STEMI caused by AAD is rare, difficult to diagnose, and potentially life-threatening. Inappropriate treatment with thrombolytics increases mortality. Adequate knowledge of this condition is necessary for early and accurate diagnosis.

CONFLICTS OF INTEREST

The author declares that he has no conflicts of interest in this work.

AUTHORS ´CONTRIBUTIONS: Each author contributed individually and significantly to the development of the manuscript. MCB, PIMM: design, writing, and interpretation of the data; ACCC, PIMM: critical review of intellectual content.

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