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Postoperative Myocardial Infarction in Acute Type A Aortic Dissection: A Case Report

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ABSTRACT

Postoperative myocardial infarction (POMI) in acute type A aortic dissection rarely has been reported, we report a case of postoperative myocardial infarction in acute type A aortic dissection, and the clinical presentation and possible mechanisms are described. This case illustrates that postoperative myocardial infarction in acute type A aortic dissection is a rare fatal complication in patients without coronary lesions or coronary malperfusion before aortic repair. Type 2 myocardial infarction may account for POMI. Effective treatment may include anticoagulation therapy, volume adjustment, blood pressure support, administration of blood products, heart rate control, and individualized respiratory support.

INTRODUCTION

Aortic dissection, which is characterized by separation of the layers within the aortic wall with blood entering the intima-medial space, is a lethal condition requiring urgent surgical therapy. Based on anatomical location, aortic dissection is classified into Stanford A aortic dissection involving the ascending aorta or Stanford B aortic dissection occurring distal to the left subclavian artery. Severe chest pain is the most common presenting symptom, additionally, an apex-radial pulse deficit, stroke and myocardial infarction are the primary manifestations stemming from limb, cerebral, and coronary malperfusion [Hagan 2000]. Emergency surgery aims to keep the patient alive by addressing severe aortic regurgitation, cardiac tamponade, primary tear, and organ malperfusion and, if possible, by preventing late dissection-related complications in the proximal and distal aorta. With development of novel techniques and perioperative care, mortality and morbidity has been reduced. Postoperative myocardial infarction (POMI) in acute type A aortic dissection is a rare complication that rarely has been reported. In aortic dissection, POMI has a significant impact on mortality and morbidity [Khoyneahad 2014].

Here, we report a case of postoperative myocardial infarction in acute type A aortic dissection. Furthermore, the clinical presentation and possible mechanisms of postoperative myocardial infarction in acute type A aortic dissection are covered in detail. The patient provided written informed consent for publication of the study data.

CASE REPORT

A 56-year-old man with a history of hypertension and smoking was admitted to our hospital, due to the onset of chest pain radiating to his back for four hours. His blood pressure was 152/90mmHg (right arm), 133/92mmHg (left arm), 189/91mmHg (left leg), and 211/67mmHg (right leg). His heart rate was 84 beats/min. Physical examination revealed intact consciousness and a soft diastolic murmur in the aortic valve area. An electrocardiogram (ECG) showed sinus rhythm, and ultrasonic cardiography (UCG) revealed mild aortic regurgitation, normal systolic function, and pericardial effusion. A thoracic and abdominal aortic computed tomography angiogram (CTA) showed acute Stanford type A aortic dissection with a dissected flap in the ascending aorta extending to the iliac artery and a myocardial bridge in the left anterior descending artery. The patient's D2-dimer level was 80ng/mL, creatine kinase level was 137.5 U/L, creatine kinase MB level was 9.1 U/L, and cardiac troponin level was 38ng/L. He was diagnosed with acute Stanford type A aortic dissection without preoperative myocardial infarction. (Figure 1)

Subsequently, he was taken to the operating theater, and pericardial effusion was found after performing sternotomy. Intraoperatively, commissural leaflets of the aortic valve were resuspended with 2-0 prolene suture and the coronary artery bypass grafting or coronary artery repair was not performed. Eventually, ascending aorta replacement was performed, together with total arch replacement using a tetrafurcate graft with stented elephant trunk implantation. After surgery, he was taken to the intensive care unit.

On the second day after surgery, the patient became dynamically unstable and unresponsive to vasoactive agents (that were administered to maintain hemodynamic stability), manifesting as low blood pressure, high level of lactic

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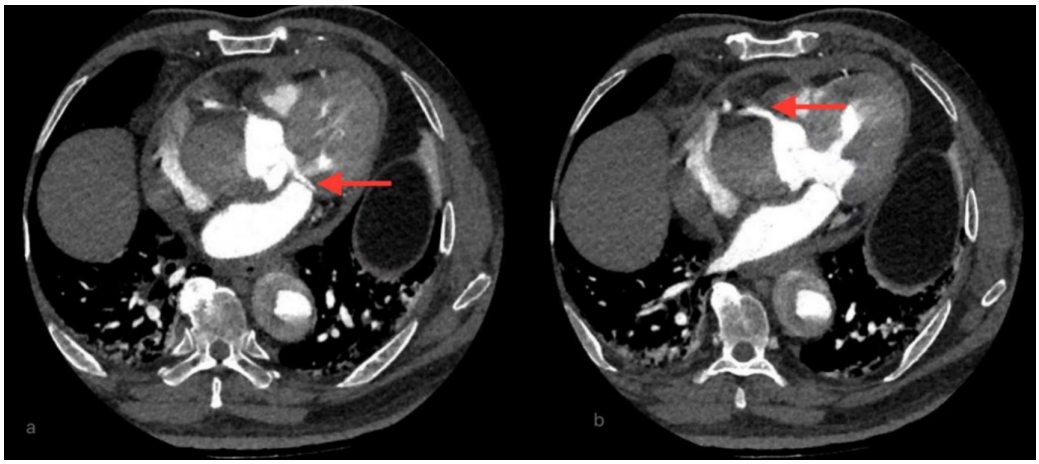

Figure 1. CTA demonstrates proximal segment of right and left coronary artery. At the level of ostia of left coronary artery (A). At the level of ostia of right coronary artery (B).

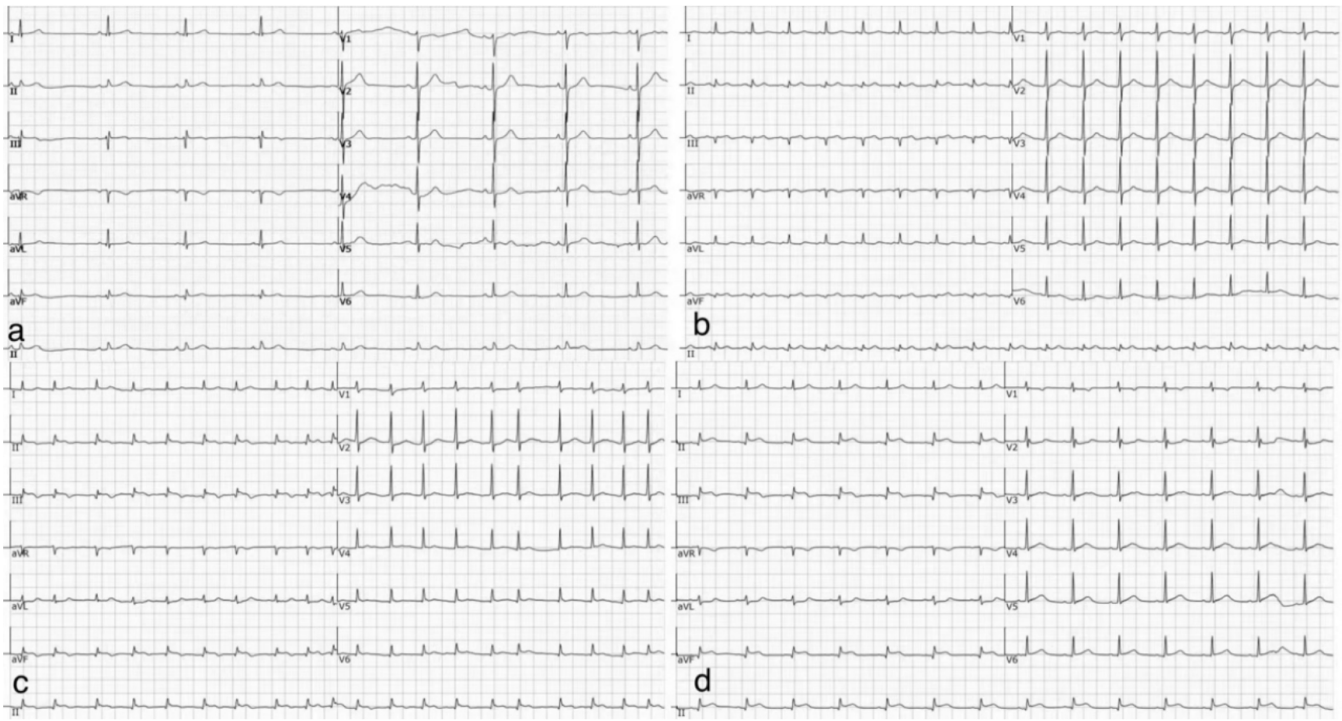

Figure 2. Representative electrocardiogram (ECG) of the patient. Panel A: A 12-lead ECG at initial admission without ST-segment changes. Panels B, C, and D: ECG on 1st, 2nd, and 3rd day after admission shows ST segment elevation and new Q waves in leads II, III, and aVF.

acid, and peripheral hypoperfusion. Based on the evidence in leads II, III, and aVF of the 12-lead ECG (Figure 2), poor systolic function of the right ventricle in transthoracic echocardiogram, and elevated levels of myocardial enzymes and troponin (Figure 3), right ventricular myocardial infarction was highly suspected. (Figure 2) (Figure 3) Immediately, coronary angiography was scheduled on the fourth day, and the result did not show the presence of significant stenosis in the three main coronary arteries. (Figure 4) Gradually, the patient became hemodynamically stable with gangrene of his toes and fingers (Figure 5) and increased systolic ventricular function but had multiple organ dysfunction, including pneumonia, hepatic and renal dysfunction, and coagulopathy. (Figure 5) On the 11th postoperative day, tracheostomy was performed to facilitate airway management, by sputum aspiration using a fiber bronchoscope. With comprehensive treatment such as vasoactive agents, volume adjustment, conservative interrupted anticoagulation titrated with D-dimer

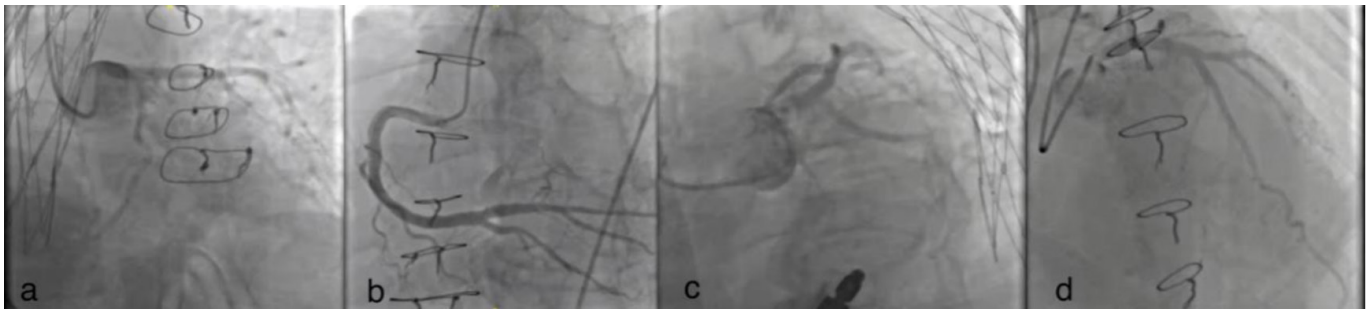

Figure 3. CAG (A-D) demonstrates no significant stenosis in the left main artery, left coronary artery, left anterior descending artery, and right coronary artery.

Figure 4. Gangrene of the limbs because of multisite embolism.

changes, and anti-infective therapy, the patient survived the postoperative myocardial infarction. However, he died on the 28th day after surgery, due to MODS while receiving continuous renal replacement therapy.

DISCUSSION

POMI is defined as a new-onset myocardial infarction (MI) that occurs after aortic repair for type A aortic dissection. Electrocardiogram, echocardiography, and laboratory analysis are the commonly used investigations for its diagnosis. The diagnostic criteria are as follows: ECG demonstrating is new Q waves that are 0.03 seconds in width (one third or more of the QRS complex) in two or more contiguous leads, or ST elevation more than 1.0 mm in the early phase, or new Q waves or new left bundle branch block; patients with POMI who are found to have any new persistent wall motion abnormality or other evidence of MI such as free wall rupture, acute ventricular septal defect, and mitral regurgitation due to papillary muscle rupture or ischemia; laboratory diagnosis was made with creatine kinase-MB level five times or more than the upper limit of normal; and cardiac troponin values 10 times higher than the normal upper limit of.

Waterford et al. analyzed data from the International Registry of Acute Aortic Dissection and concluded that the bicuspid aortic valve, root involvement, pericardial effusion, and greater extent of surgical repair are strongly associated with POMI [Balsam 2017; Waterford 2017]. In our case, the patient who did not have preoperative coronary artery disease had hemorrhagic pericardial effusion and underwent aortic vavuloplasty, and total arch replacement using a tetrafurcate graft with stented elephant trunk implantation. The patient had POMI, despite a negative result from the coronary angiogram. It is difficult to determine the exact underlying mechanism of POMI; however, some theories may explain this complication.

Myocardial infarction, which is caused by an imbalance between myocardial oxygen supply and demand, can be divided into two types [Sandoval 2019; Montone 2018]. Type 1 MI is secondary to acute atherosclerotic plaque disruption. Type 2 MI is the result of a direct mismatch between myocardial oxygen supply and demand in the absence of acute atherothrombosis. There are several different mechanisms that can lead to the supply-demand imbalance occurs, and some cause a direct narrowing leading to the restriction of coronary blood flow, thus causing spontaneous coronary artery dissection, coronary intramural hematoma, coronary spasm, coronary embolism, and aortic dissection causing coronary obstruction. It is essential but challenging to distinguish myocardial injury from Type 1 and Type 2 MI. The treatment regimen varies, depending on the different etiologies of the two types of myocardial ischemia.

We believe that type 2 MI might be the mechanism of POMI in our case because the coronary angiogram was negative. As for the exact mechanism of type 2 MI, we consider

that coronary artery embolism (CE) might have caused myocardial infarction in our patient [Popovic 2018]. The diagnosis of CE is a formidable challenge, and its exact aetiopathogenesis remains uncertain. Aortic and mitral prosthetic heart valves, atrial fibrillation (AF), atrial septal defect, dilated cardiomyopathy, neoplasia, infective endocarditis, and intracardiac tumor are linked to coronary embolism. Contemporary data showed that AF was the most frequent cause of CE was AF. Additionally, an underlying hypercoagulable state is a potential predisposing prothrombogenic factor. In patients with such predisposition to thromboembolism, an inherited or acquired disorder and autoimmune diseases such as antiphospholipid syndrome and malignancy should be suspected. During ICU therapy, our patient had AF and thrombus may have formed and been dislodged from the left atrium. Our patient had: (1) ST-segment elevation in the inferiorly directed leads and paroxysmal atrial fibrillation on ECG, (2) elevated levels of creatine kinase-MB and cardiac troponin, (3) coronary embolism and multisite systemic emboli leading to gangrene in his hands and legs (Figure 4), (4) abnormal wall motion on echocardiogram, and (5) coexistence of a hypercoagulable state, but without any severe atherosclerotic stenosis on angiography. Based on the aforementioned evidence, the patient was suspected to have suffered a type 2 myocardial infarction secondary to coronary embolism.

For Type 2 MI, personalized treatment tailored to each etiological factor should be made for the rectification of the supply-demand imbalance. Additionally, comprehensive treatment including volume adjustment, blood pressure support, administration of blood products, heart rate control, and respiratory support should be included in the basic treatment.

CONCLUSIONS

Postoperative myocardial infarction in acute type A aortic dissection is a rare fatal complication in patients without coronary lesions or coronary malperfusion before aortic repair. Type 2 myocardial infarction may account for POMI. Effective treatment may include anticoagulation therapy, volume adjustment, blood pressure support, administration of blood products, heart rate control, and individualized respiratory support.

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