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Case Report

A Case Report of Recurrent Acute Myocardial Infarction with a Peculiar Background

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ABSTRACT

Occlusion of the right coronary artery is a relative rare complication of type-A aortic dissection and an example of type-2 myocardial infarction as well, but when it occurs, it can be fatal. Misdiagnosis leads to inappropriate therapy and delays. Our case made no exception. Etiology remains unclear although the patient's background might be related to his later, life-threatening complication.

Keywords: Aortic dissection . Acute myocardial infarction . Pseudo aneurysm . Vasculitis . Hairy cell leukemia . Cardiogenic shock

Introduction

Acute chest pain is one of the most common reasons for seeking care in the emergency department and can have multiple causes: myocardial ischemia or infarction, pericardial disease, vascular disease, pulmonary conditions, gastrointestinal conditions, musculoskeletal and other conditions [1]. Myocardial infarction (MI) alone can be subdivided into Type 1 MI which is a primary coronary arterial event attributable to atherothrombotic plaque rupture or erosion and type 2 MI which occurs secondary to an acute imbalance in myocardial oxygen supply and demand without atherothrombosis. The reported prevalence of type 2 MI ranged from 2% to 58% of patients with MI [2].

Occlusion of the right coronary artery (RCA) is a relative rare complication of type A aortic dissection and an example of type 2 MI as well but when it occurs, it may have a fatal result for the patient. Aortic pseudoaneurysms are local type A dissections with restricted extent in which the majority of the aortic wall has been breached and luminal blood is held in only by a thin rim of the remaining wall, mainly purely the adventitia. They typically occur from iatrogenic trauma by interventional procedures or previous cardiac surgery but in our case the etiology remains unclear although the patient's background might be related to his later, life-threatening complication.

Case Presentation

A 56-years old male patient presented to our emergency catheterization laboratory following sudden onset of chest pain and electrocardiographic findings of inferior ST segment elevation. Upon arrival he was progressive impairment of the hemodynamic status equaling to cardiogenic shock with (heart rate of 90bpm and blood pressure of 85/50 mmHg, confirmed by non-invasive and invasive measurements, as well). He had no previous medical history other than a recently diagnosed Hairy-Cell Leukemia (HCL) and took no regular medication. Orienting echocardiography was performed prior catheterization with a hand-held pocket-size ultrasound system, as standard of care. Note, this device allows a rough diagnosis of regional wall motion abnormalities and relevant

Timeline

Date	Events
First admission to hematologist	Hairy-Cell Leukemia diagnosis (bone marrow aspiration)
Day 1, first admission	1. Ambulance Diagnosis of Inferior STEMI. Direct presentation at the Catheterization Laboratory. Complains: constrictive chest pain, mid-thorax located, mild dyspnea. Hemodynamics: BP 85/50 mmHg, HR 90 bpm, neurological status – GCS 13 pcts. 2. ED Echo (on the table) – slightly enlarged left ventricle, EF 45%, moderate aortic regurgitation. 3. Coronary angiogram – RCA stenting. Rapid relief of symptoms. 4. Chest pain and ventricular fibrillation. Successful CPR. Intubation. Second angio: stent occlusion. Second PCI. Patient stabilization. 5. Transfer to ICU. Chest CT scan – sinus Valsalva aneurysm rupture diagnosis through CT. Labs: anemia, lymphopenia, thrombocytopenia, hyperglycemia, increased serum creatinine, BNP, D-Dimer and Troponin-I. Echo – severe aortic regurgitation.
Day 2	6. Heart-Team – call for planned surgery. Patient extubated, stable, GCS 15 pcts. No complains.
Day 8	7. Heart surgery performed: aortic prosthesis and 1 RCA bypass.
Day 9	8. ECG – Paroxysmal Atrial Fibrillation 11. Treatment: amiodarone cardioversion, crystalloids, norepinephrine, midazolam adjusted and ventilation parameters, extubation.
Day 10	12. Second look angio - no complications 13. Histology interpretation: chronic aortitis with microscopic infiltrates of lymphocytes and plasmocytes.
Day 20	14. Patient discharge

valvular dysfunction, or mechanical complications. However it is not reliable for further fine assessment of structural disorders. Emergency diagnostic coronary angiography showed a patent left coronary system and the RCA could not be found. Aortic angiography was performed, showing an aberrant orifice of the RCA and also a weird contour of the right sinus (video 1), however, being focused on the RCA occlusion, the latter was not carefully evaluated at this moment. No intimal flap or double-lumen was detected. With the support of non-selective visualization, RCA was finally found, showing sharp-contoured obstruction in the proximal segment (Figure 1). PCI was performed, ST segment elevation resolved and TIMI 3 flow achieved (Figure 2), with rapid relief of symptoms. Operators considered the angiographic appearance to matched to 'typical occlusion of an RCA with atypical orifice' with no clinical or angiographic hint for embolic background. Even though the unique expansion of the right sinus Valsalva is visible on one of the cine loops (video 1), the operator missed to take it in serious consideration during the procedure. This can be explained partially by personal experience, however also by human nature of focusing on what one is looking for (i.e. a culprit stenoses) and not necessarily the bystander issues.

Shortly after, the patient presents cardiac arrest (ventricular fibrillation), CPR and intubation was performed. Second-look coronary angiogram reveals reocclusion of the RCA. Two more stents were implanted in the proximal part, with difficulty in maintaining the vessel open, with significant stent-protrusion in the aorta. (Figure 3 and Figure 4). Patient was stabilized and transferred in the CCU where echocardiogram showed a slightly enlarged left ventricle, with mild

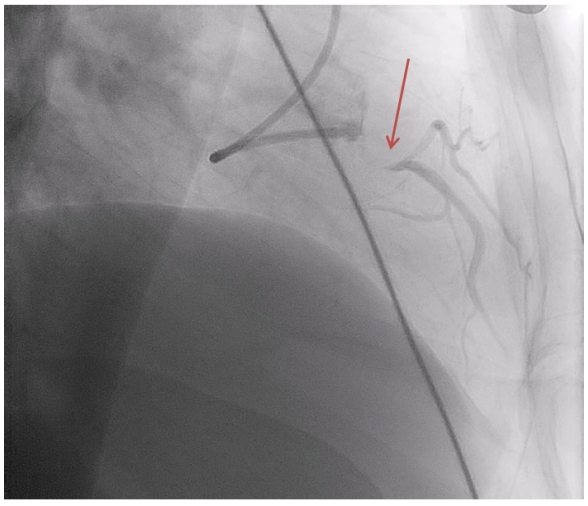

Figure 1: Non-selective injection of the RCA. Ostial stenosis.

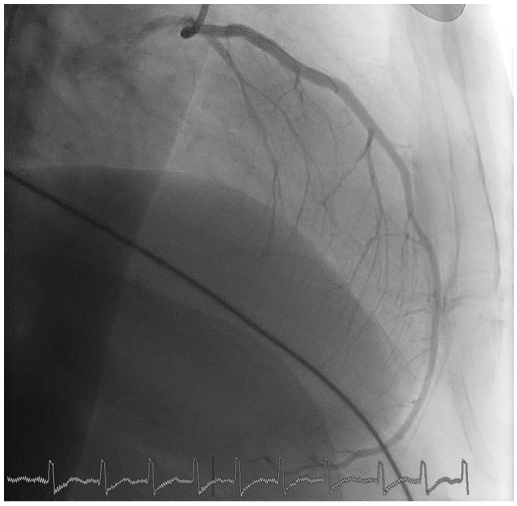

Figure 2: First RCA stenting.

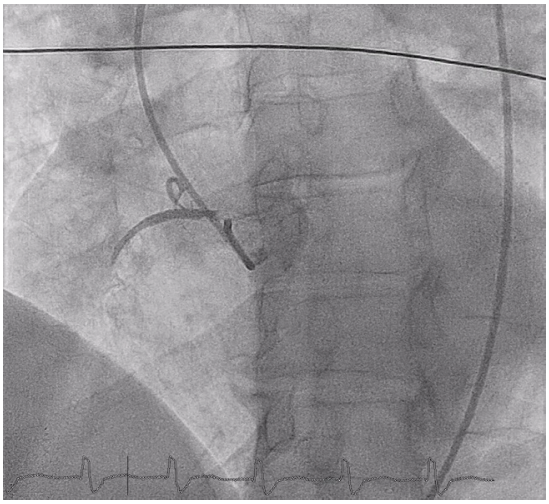

Figure 3: RCA re-occlusion shortly after the first PCI.

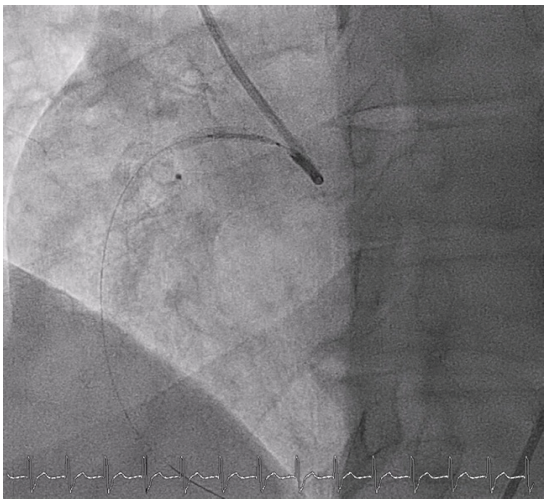

Figure 4: Second RCA stenting, with significant stent protrusion into aorta.

systolic dysfunction, severe aortic regurgitation but normal sized ascending aorta, with no signs of dissection. Careful review of the coronary angiogram and especially the aortogram raised the suspicion for an actual aortic root pathology. CT was performed which revealed formation of pseudoaneurysms in the right and non-coronary sinuses, measuring 1,5 cm each (Figure 5). Interdisciplinary heart team decided for semi-elective surgical sanitation of the pseudoaneurysm. Full replacement of the ascending aorta, aortic valve, relocation of left coronary system and graft over the RCA, distal of the stents, with ostial ligature of the native vessel were performed (Bentall procedure). Intraoperative findings nicely illustrate the protruding stent covered with dissected intima (Figure 6) but also wall tissue ruptures forming "pouches" in the non-coronary sinus, due to destruction of the layers and loss of intima (Figure 6).

Patient made a good recovery and was extubated the next day, control angiogram was performed: both left native coronaries and right vein-bypass were

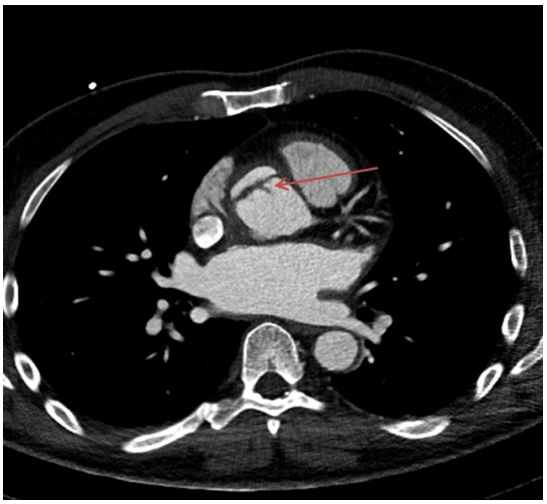

Figure 5: Chest CT Scan, the intimal flap marked by the arrow, marking the pseudoaneurysm.

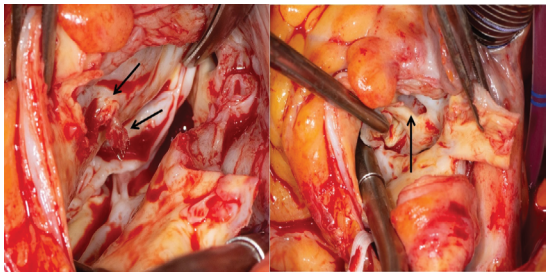

Figure 6: Intraoperative pictures of the aortic sinus, showing the exaggerated stent protrusion with the intimal flap coversing the stent. Pouch orifice is marked by the arrow from the second picture.

found normal, with TIMI 3 flow, aortic bioprosthesis in physiological position. He was discharged home after 20 days, without complications.

Discussion

Risk factors for aortic dissection are hypertension, heritable or genetic thoracic aortic disease and syndrome, congenital diseases/syndromes, atherosclerosis, trauma, blunt or iatrogenic and inflammatory/infectious diseases. Patients with acute aortic dissection have very high early mortality, with up to 1% per hour reported in the first 24 hours before surgery for type A dissection [3].

The thoracic aortic disease guidelines suggest a management pathway for patients with acute aortic dissection. Initial medical management includes stabilizing the patient, controlling pain, and lowering BP with beta blockers to reduce the rate of rise in the force of left ventricular contraction. These measures should commence immediately while the patient is undergoing diagnostic evaluation. Lowering BP may help prevent further propagation of the dissection and lessen the risk for aortic rupture. Emergency surgery leads to improved survival in patients with acute type A dissection, with an 18% in-hospital mortality for surgically treated type A dissection and 50% mortality in the first 48 hours, if not treated [3].

Acute MI related to the false lumen compressing the coronary ostium or the dissection flap involving the coronary artery complicates 10-15% of patients with acute type A aortic dissection. [6] It most frequently involves the right coronary artery and leads to acute inferior MI. [4] Troponin elevations and electrocardiographic changes may occur in acute dissection.

Aortic dissection may not be suspected as a cause of coronary ischemia, and misdiagnosis may lead to inappropriate therapy and delays in treatment. Our case made no exception. Emergency echocardiogram showed normal size aorta with no signs of dissection. Encountering difficulty in cannulating RCA and subsequent aortography aspect were indirect signs of aortic dissection but the patient's hemodynamic instability and malignant arrhythmia forced us to focus on the myocardial infarction and proceed with PCI. Planned surgery resolved both dissection and valvulopathy. While surgical repair of aortic dissection is normally a highly acute intervention, but in the present case the following aspects were considered when defining the best timing for the procedure: (1) recent transmural myocardial infarction with massive rise of necroenzymes, making the patients conditions unfavorable for an acute cardiac surgery with extracorporeal circulation; (2) dissection did not show the typical radiologic signs of an acute dissection on the CT images, suggesting that rapid progression as natural history is not to be expected; (3) however newly introduced double-antiplatelet therapy might destabilize the dissection, if waiting too long. The acute event occurred from one of its complications, which was resolved and patient was stabilized, therefore affording to schedule the surgical intervention in a semi-elective fashion.

The histopathological report described chronic IgG4 periaortitis with findings of lymphocytic and plasmocytic infiltrates in the aortic adventitia (Figure 7). There are few but similar cases of vasculitis reported in patients with HCL. We suspect a similar substratum, considering it was not a typical type A aortic dissection but more like multiple focal lesions, with subacute evolution, otherwise associated with the incriminated hematological disorder. Although rarely occurring, it is important to recognize this condition in patients with HCL. Forty-two cases of vasculitis coincident with HCL have been reported, of which 17 had panarteritis nodosa, 21 had cutaneous leukocytoclastic vasculitis, and 4 had vessel wall infiltration by hairy cells [5]. Therapy is controlling the underlying leukemia.

MI resulted from external coronary artery occlusion due to aortic dissection, where the dissection flap temporarily occluded the ostium of

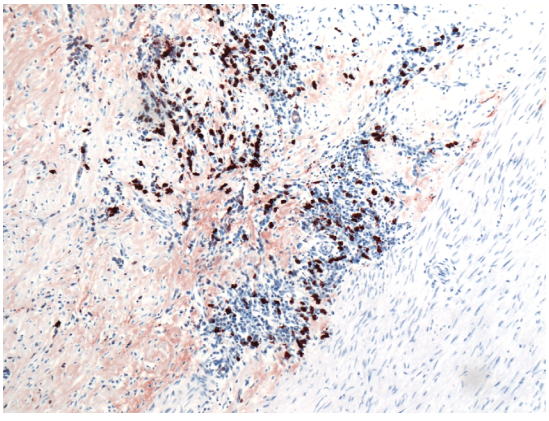

Figure 7: Immunohistochemical-staining (IgG4) showing diffuse loss of elastic fibers with lymphocytic and plasmocytic infiltrates.

the right coronary artery and affected the blood flow to the vessel. The significant aortic regurgitation in a young patient with acute chest pain should make every physician suspicious of aortic dissection. Prompt diagnosis and treatment can be lifesaving. Later understanding through multidisciplinary approach can offer the bigger picture.

Learning Objectives

Aortic dissection need to be considered as a cause of acute coronary syndrome. Carefully clinical examination should be performed before rushing into invasive treatments.

- Screening for other diseases that may affect body vasculature should be assessed; Hairy –Cell Leukemia can cause vasculitis.
- Considering medical decisions throughout larger, multidisciplinary medical teams unmistakably provides both, a better understanding of the disease and a better outcome for the patient.

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COI Statement

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Patient Informed Consent Statement

Informed consent for patient information and images to be published was provided by the patient.

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