

Peripartum Acute Anterior ST Segment Elevation Myocardial Infarction: An Uncommon Presentation of Acute Aortic Dissection

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Abstract

Introduction: Atherosclerotic coronary artery thrombosis is the most common cause of acute myocardial infarction. **Clinical Picture:** A 30-year-old lady presented with acute peripartum massive anterior ST segment myocardial infarction and cardiogenic shock. This was due to acute Stanford type A aortic dissection with the intimal flap occluding the left coronary ostium. The initial diagnosis was not apparent. Echocardiography confirmed the diagnosis. **Treatment and Outcome:** She underwent emergency surgical repair (Bentall procedure). Pathology confirmed underlying idiopathic cystic medial degeneration. **Conclusion:** A high index of clinical suspicion is required in acute myocardial infarction presenting without traditional cardiovascular risk factors.

Ann Acad Med Singapore 2007;36:854-6

Key words: Acute anterior myocardial infarction, Cystic medial degeneration

Introduction

Reperfusion therapy in the form of primary angioplasty or thrombolytic therapy is the cornerstone of management in patients presenting with acute ST segment elevation myocardial infarction (STEMI).¹ Thrombosis of the atherosclerotic coronary artery is by far the most common cause of acute myocardial infarction. In patients without traditional risk factors, other possible uncommon aetiologies need to be considered. In many medical centres where thrombolytic therapy is the first-line reperfusion therapy for acute STEMI, the outcome may be catastrophic in clinical situations such as acute aortic dissection. This case report stresses the importance of a high index of suspicion of non-atherosclerotic causes such as aortic dissection as a possible cause of acute STEMI. In addition, we discuss the need to be selective in performing coronary angiography in patients with acute aortic dissection, in order to improve clinical outcome.

Case Report

A 30-year-old lady, who was previously well, presented at 1 week in the postpartum period with acute severe retrosternal chest pain associated with diaphoresis. She had no significant past medical history. Clinically, she was

pale, clammy, hypotensive (blood pressure of 64/40 mm Hg) and bradycardic (42 beats per min). There was no audible murmur or palpable pulse deficit. The 12-lead electrocardiogram revealed a right bundle branch block, associated with anterolateral ST segment elevation and sinus bradycardia (40 beats per min). The chest X-ray did not show any widening of the mediastinum, pulmonary congestion or pleural effusion. The baseline complete blood count, serum creatinine and electrolytes were within normal limits. The bedside qualitative troponin T was negative. She was diagnosed to have an acute massive anterior STEMI complicated by a right bundle branch block and cardiogenic shock. She was stabilised and transferred to a tertiary care hospital. An urgent coronary angiography and subsequent aortogram was performed, which revealed a totally occluded left main coronary artery and a grossly dilated aortic root with a complex aortic dissection flap. A bedside 2-dimensional echocardiography showed a dilated aortic root with a complex aortic dissection flap associated with mild aortic regurgitation and an akinetic anteroseptal myocardial wall. She underwent a complex emergency surgical repair (Bentall procedure). Intra-operatively, a transverse dissection of the aortic root in close proximity of the left main ostium was identified with

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an incompetent aortic valve. The distal extent of the aortic dissection was not observed. The histopathology report confirmed cystic medial necrosis of the aortic wall. However, the patient died in the immediate postoperatively, predominantly due to severe myocardial dysfunction despite mechanical and inotropic cardiac support.

In summary, this is a 30-year-old lady who suffered from postpartum, Stanford Type A aortic dissection due to underlying cystic medial necrosis. It was complicated by acute massive anterior STEMI and cardiogenic shock.

Discussion

Aortic dissection is an uncommon event whose incidence is relatively unknown; in one study, the calculated incidence was 27 cases per million habitants per year.² The incidence is related to the prevalence of risk factors, in particular, uncontrolled hypertension, advanced age and disease of the aortic wall.³ A co-existing history of hypertension is found in up to 80% of the cases.⁴ The peak incidence of aortic dissection is in the sixth and seventh decades of life, with men affected twice as often as women.⁴

Aortic dissection during pregnancy and postpartum is uncommon. About 50% of all aortic dissections in women younger than 40 years occur during pregnancy, typically in the third trimester and occasionally in the early postpartum period.⁵ The increase in haemodynamic stress seen in late pregnancy may contribute to the risk, while alteration in the structure of the vascular wall may explain the predisposition to postpartum dissection.^{6,7}

Acute dissection of the ascending aorta is a highly lethal event. Without surgical intervention, the mortality is approximately 25% within the first 24 hours.⁸ Most reported cases are associated with connective tissue disease, systemic hypertension and congenital heart disease, including coarctation and bicuspid aortic valve.⁹ An inherent structural weakness of the arterial wall such as that resulting from Marfan's syndrome or mild connective-tissue abnormalities or atherosclerosis is also thought to be necessary for the initiation of dissection.⁹ In this case, the patient had cystic medial degeneration of unknown aetiology, identified only in minority.¹⁰

Aortic dissections are classified according to their location; the commonly used Stanford classification labels those involving the ascending aorta as type A and those involving the descending aorta (i.e., distal to the left subclavian artery) as type B. Type B dissections occurring during peripartum are very rare.

The clinical presentation is highly variable. The most common presenting symptom is severe, sharp pain in the chest and upper back that is characteristically stabbing, tearing or ripping.⁴ The pain is frequently migratory, generally following the path of propagation of the dissection.

Classic findings on physical examination such as pulse deficits and aortic regurgitation murmur are often unrecognised or absent, as in our patient. Thus in this case, where a young lady presented with acute massive anterior STEMI, a high index of clinical suspicion was needed. Not surprisingly, a number of case reports showed that the diagnosis was initially missed in the peripartum period.¹¹⁻¹³ Rapid diagnosis of a suspected aortic dissection is critical and appropriate treatment, including surgical repair, must be instituted immediately. In-hospital mortality is approximately 25%, with most deaths occurring soon after the onset of symptoms.⁴

A normal ECG is present in one-third of patients with coronary involvement; most of these patients have non-specific ST-T segment changes.¹⁴ About 20% of patients with type A dissection have ECG evidence of acute ischaemia or acute myocardial infarction.¹⁴ In 1% to 2% of cases, a proximal dissection flap may involve the ostium of a coronary artery and cause acute myocardial infarction. The right coronary artery is more often involved than the left.¹⁰ An uncommon involvement of the left main coronary ostium occurred in our patient. In the case of older patients with ECG evidence of acute STEMI, underlying aortic dissection may go unsuspected or recognised. In centres where thrombolysis is the first-line reperfusion therapy, the outcome could be fatal. A high index of suspicion, especially in younger patients or those without established major coronary risk factors, is essential to avoid a potential catastrophe. The presence of inappropriate sinus bradycardia in the acute phase despite significant hypotension, as in this patient, deserves special comment. This patient was seen very early during the course of myocardial infarction, a complication of the ascending aorta dissection. The bradycardia is almost invariably an exhibition of increased autonomic (vagal) activity, a manifestation of the Bezold-Jarisch reflex. Sinus bradycardia is also often secondary to vasovagal response, which may be intensified by severe pain as well as morphine.¹⁵ Sinus bradycardia occurring more than 6 hours after the onset of acute myocardial infarction is more often caused by sinus node dysfunction or atrial infarction rather than vagal hyperactivity.

Chest radiography is often insufficient to exclude the diagnosis of aortic dissection.¹⁶ A classic chest radiography feature such as widened mediastinum or abnormal aortic contour is uncommon and often unrecognisable, as in this case. Initial evaluation with rapid, non-invasive diagnostic modalities such as transoesophageal echocardiography, rapid sequence spiral computed tomography with intravenous contrast, or magnetic resonance imaging have a sensitivity and specificity approaching 100% in the evaluation of acute dissection of the ascending aorta.¹⁷

Nevertheless, the 2-dimensional transthoracic echocardiography remains a helpful tool in the initial screening test, particularly in patients with type A dissections, for which its sensitivity ranges from 78% to 100%.¹⁸ In addition, the test can uncover important complications such as valvular regurgitation and pericardial effusions. A typical echocardiographic criterion for aortic dissection – an undulating intimal flap – was evident in this patient, with associated mild aortic regurgitation.

The role of coronary angiography in aortic dissection remains controversial. Cardiac catheterisation may be technically difficult and time-consuming. In addition, there are potential risks of extending the dissection, perforation of the aorta and displacement of thrombotic material from the dissected aorta. Routine angiography may result in the delay of surgical intervention and impose a greater risk than benefit.¹⁹ Coronary angiography should only be performed on a stable patient with a strong history or clinical suspicion of coronary artery disease, such as a history of angina or myocardial infarction, known coronary artery disease, recent abnormal radionuclide imaging, or on patients who have undergone previous coronary artery bypass grafting and acute electrographic changes suggestive of myocardial ischaemia on presentation.²⁰ In aortic dissection, coronary angiography should not be performed on patients with haemodynamic instability, pericardial effusion, cardiac tamponade or aortic rupture.²¹ Similar to previously reported cases,¹¹⁻¹³ the diagnosis of aortic dissection in this case was not apparent in the initial assessment. The initial diagnosis made in this atypical clinical presentation was acute anterior STEMI with cardiogenic shock, where coronary angiography with an aim to perform primary coronary angioplasty is the preferred therapy.¹ If cardiac catheterisation is needed, several technical issues should be considered. A careful choice of the arterial access site and the usage of at least size 6 French catheters will permit better visualisation and torque control. The initial usage of a pigtail catheter to enter the left ventricle to confirm proper positioning of true lumen is advocated by some.²⁰ We will generally use an exchange length guide wire for catheter exchange to ensure that the catheters stay in the true lumen.

Conclusion

The above case scenario highlighted a few important clinical points. A high index of clinical suspicion is required in diagnosing aortic dissection, particularly in younger patients without the traditional cardiovascular risk factors presenting with acute myocardial infarction. Diagnosis is often clinched by non-invasive imaging tests; the selection will have to be based on the availability of the test, and the medical and logistics constraints of the centre. The role of coronary angiography in aortic dissection is controversial.

It is generally needed only for patients with established coronary artery disease or suggestion of it. In this era of primary reperfusion therapy for acute STEMI, physicians should pause and reassess the issue.

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