CASE REPORT

Aortic arch variant presenting as an acute ST elevation myocardial infarction

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SUMMARY

A woman in her 40s presented to our institution with a first episode of sudden-onset right-sided chest pain. Initially, her ECG revealed ST segment elevation in leads V1–V3 and cardiac biomarkers were elevated with a troponin I (TnI) 5 µg/l. Her cardiac risk factors included type II diabetes mellitus and a history of hypertension. Intravenous heparin was initiated and emergent cardiac catheterisation revealed non-obstructive coronary artery disease. A two-dimensional echocardiogram confirmed hypokinesis of the anterior wall. A CT thorax demonstrated a rare anatomical variant of the aortic arch with an aberrant right subclavian artery. This is a rare case of an aortic arch anomaly presenting as a myocardial infarction.

BACKGROUND

This case report describes the initial assessment and management of a patient presenting with acute coronary syndromes. It highlights the diagnostic and therapeutic role of cardiac catheterisation in the management of these syndromes. Following resuscitation and appropriate anticoagulation, treatment should be directed towards identifying and correcting the underlying cause. Failure to identify an immediate cause for 'typical' chest pain should prompt a more thorough evaluation. By far, acute thrombosis of a coronary vessel remains the most common underlying aetiological mechanism, particularly in the presence of multiple risk factors and should be the initial consideration. For lack of a better explanation, investigation of other causes should be performed. This case serves to highlight the unique presentation of an aortic arch anomaly as an acute myocardial infarction. For the clinician faced with an acute ST-elevation myocardial infarction and the absence of clinically obstructive disease on angiography, the presence of an aortic arch anomaly on angiography/imaging should prompt re-evaluation.

CASE PRESENTATION

A woman in her 40s presented to our emergency department with a 2 h history of severe right-sided chest pain radiating to the back, and only partially relieved by sublingual nitroglycerin tablets. Her medical history was only significant for hypertension and diabetes mellitus. She was noted to be on metformin, metoprolol and a multivitamin. On admission, she was hypertensive with a blood pressure of 220/120 mm Hg. Clinical examination was unremarkable. Her 12-lead ECG is shown in figure 1. It demonstrates sinus rhythm of 75 bpm and new ST

segment elevation in the anterior precordial leads. The initial troponin I (TnI) level was 5 µg/l and a chest x-ray on admission was unremarkable. The working diagnosis was acute myocardial infarction. She was given aspirin, intravenous nitroglycerin and heparin. Metformin was discontinued. She was taken to the interventional suite for emergent cardiac catheterisation. Angiography was performed and this revealed non-obstructive coronary artery disease. She was transferred to the coronary care unit for close monitoring. Serial TnI levels continued to rise to a peak of 22 µg/l. The nitroglycerin infusion was titrated to achieve a mean arterial blood pressure of 80 mm Hg over the next 36 h. A two-dimensional echocardiogram performed after an improvement of blood pressure demonstrated anterior wall hypokinesis with an ejection fraction (EF) of 40%. Serial TnI levels began to trend downwards. CT thorax was performed which showed her to have an anatomic variant of the aortic arch (figure 2). The particular aortic arch variant seen was type V, in which the common carotid arteries share a trunk and the aberrant right subclavian (RS) artery arises as the last branch of the aortic arch (figure 3). The patient remained chest pain free and the remainder of her stay was uneventful. A follow-up ECG post-discharge showed resolution of the ST elevation.

DIFFERENTIAL DIAGNOSIS

Myocarditis was high in our differential; however, there was no history of any autoimmune disorder or viral or enteric infection prior to the onset of symptoms. No biopsy was done.

OUTCOME AND FOLLOW-UP

Twelve months after the acute onset, a repeat echocardiogram demonstrated no focal areas of hypokinesis and a normal EF of 55%. At her most recent follow-up visit, the patient was chest pain free and continued to lead her usual lifestyle.

Figure 1 ECG suggestive of ST segment elevation in anterior leads.

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Figure 2 (A) CT scan showed an aortic arch variation; a common origin of both the common carotid arteries with an aberrant right subclavian (RS) artery. (B) CT scan showed an aortic arch variation; a common origin of both the common carotid arteries with an aberrant RS artery.

Figure 3 CT angiogram showing type-V variation of the aortic arch.

DISCUSSION

To our knowledge, this is the first reported case of acute myocardial infarction resulting from an aortic arch variant. Typically, aortic arch variants are asymptomatic.¹ When they do cause symptoms, they are usually related to iatrogenic complications resulting from confusion of anatomic structures during angiography,²⁻³ aortic instrumentation and supra-aortic head and neck surgery. In the particular variant in this woman, type V variant, previously reported complications have also included dysphagia⁴ (if the aberrant RS vessel runs behind the oesophagus) and dyspnoea⁵⁻⁶ (if the aberrant RS contacts the trachea).

Learning points

- ▶ Recognise aortic arch anomalies as a cause of acute myocardial infarction.
- ▶ Review the importance of angiography in the diagnosis of aortic arch anomalies.
- ▶ Recognise that knowledge of aortic arch anomalies can help prevent complications resulting from interventions to the aortic arch.

Patient consent Obtained.

Competing interests None.

Provenance and peer review Not commissioned; externally peer reviewed.

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