Case report

Cardiac compression causing fatal acute ST-segment elevation myocardial infarction

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A 71-year-old woman presented 2 weeks after thoracic endovascular repair of a mycotic descending aortic aneurysm. One week after discharge she presented with chest pain and transient lateral ST-segment elevation on electrocardiogram (Figure 1a). Urgent computerized tomography (CT) scanning (Figure 1B) demonstrated no evidence of endovascular leak or aortic dissection. Her symptoms recurred with dramatic lateral ST-segment elevation and there was akinesis of the lateral wall of the left ventricle (LV) on echocardiography. A diagnosis of ST-segment elevation myocardial infarction was made, but emergency coronary angiography showed no flow-limiting stenoses. Cardiac compression secondary to probable haemorrhagic expansion of the aortic aneurysm was diagnosed. She was not considered fit for re-operation and died shortly afterwards.

On several occasions, we have seen patients present with an apparent syndrome comprising of (i) severe cardiac chest pain, (ii) dramatic ST-segment elevation, (iii) unobstructed and brisk epicardial coronary artery blood flow, (iv) large LV regional wall motion abnormality and (v) cardiac compression.

In a 79-year-old woman, this constellation of features was precipitated by a large hiatus hernia with intrathoracic strangulation and compression of the lateral LV wall. After emergency cardiac investigation, oesophagoduodenoscopy and laparotomy revealed irreversible oesophageal and stomach necrosis, and she rapidly died. Subsequently, a 41-year old presented with chest pain with this apparent syndrome 3 weeks after thoracic surgery for resection of a malignant mediastinal mass. Echocardiography revealed an akinetic lateral LV wall and coronary angiography demonstrated normal coronary arteries with brisk blood flow. CT scanning confirmed encasement of the heart. Sudden chest pain and marked ST-segment elevation recurring led to circulatory collapse. Emergency thoracotomy, dissection of fibrotic tissue and internal cardiac massage was unsuccessful.

There are rare reports¹–⁴ of myocardial infarction secondary to direct external coronary arterial compression, but this was not the case here. There were no features of cardiac tamponade and the clinical course was rapidly fatal in this index and previous cases. What is the explanation of this clinical course?

ST-segment elevation myocardial infarction is caused by acute obstruction of coronary arterial blood flow, usually caused by thrombotic occlusion at the site of atherosclerotic disease. Here, there was no evidence of obstructive atherosclerotic disease, intracoronary thrombosis or reduced coronary blood flow at the time of coronary angiography. Clearly, cardiac compression played a major role although the epicardial arterial vessels were not compressed even when angiography was performed during the dramatic ST-segment elevation. The presentation may reflect microvascular obstruction due to local compression, mechanical deformation of the ventricle and cardiac tissues or local accumulation...
of toxic or ionic metabolites. Thebesian veins drain directly into the cardiac chambers and, to some extent, mitigate the effects of cardiac venous obstruction. However, experimental studies of abrupt coronary sinus occlusion have demonstrated clinical and electrocardiographic features very similar to those of acute ST-segment (‘arterial’) myocardial infarction and post-mortem features reveal predominant intra-myocardial haemorrhage. We believe it highly conceivable, and anatomically plausible, that external compression of these low pressure vessels might have precipitated coronary venous infarction in these patients.

The cause of ST-segment elevation presented here is uncommon, but may be observed more frequently as referral for primary percutaneous coronary reperfusion continues to increase. A high index of suspicion for unusual causes of regional ST-segment elevation needs to be maintained. The hazard of missing a true acute ST-elevation myocardial infarction needs to be weighed against potential harm from delay in diagnosis of a more obscure and potentially life-threatening ‘compressive’ cause. The precise pathophysiology of these cases remains obscure, but rapid diagnosis by CT and immediate surgical decompression of the heart would appear to be the most appropriate and urgent approach in such cases.

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**Figure 1.** (A) Electrocardiogram demonstrating marked anterolateral ST-segment elevation in leads I, aVL and V2–V6. (B) Left: descending thoracic aortic stent (white arrow) with retrocardiac aneurysm expansion and compression of left atrium and lateral left ventricle (*). Right: coronary angiogram demonstrating normal coronary arteries (the right coronary artery was non-dominant and small).
References


