### Title of case report or series

**Critical Aortic Stenosis Presenting as STEMI**

### Authors

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### Summary

A 73-year-old male was brought into hospital with chest pain and inferior ST elevation on ECG. He immediately proceeded to the catheter lab for primary percutaneous coronary intervention. Angiography did not identify any culprit lesions to account for the patient’s electrocardiographic changes and ongoing symptoms of chest pain. Bedside echocardiography revealed critical aortic stenosis. Intra-aortic balloon pump was inserted resulting in resolution of chest pain and ST-segment changes. The patient underwent successful aortic valve replacement without the need for coronary intervention. This is a rare presentation of critical aortic stenosis presenting as STEMI.

### Learning points

1. Aortic Stenosis affects 2-9% of population above 65 years old and increases with age.  
2. AS induces ischemia via abnormal cardiac coronary coupling  
3. Focused clinical examination in patients with STEMI is vital prior to cardiac catheterisation  
4. Detection of murmurs should be followed on by an echocardiography examination  
5. Other differentials of STEMI includes acute aortopathy, endocarditis with embolus, myopericarditis and intracranial haemorrhage.

### Background

ST-segment elevation myocardial infarction (STEMI) commonly occurs when a coronary artery becomes totally occluded by a blood clot disrupting blood flow to the myocardium. The UK national registry has shown that out of 80724 admissions in 2013/14 with ACS, 39% had STEMI. [1] However, not all cases of STEMI are due to coronary artery occlusion: in this case, critical aortic stenosis led to inadequate myocardial perfusion in the absence of demonstrable epicardial coronary stenosis or occlusion. AS is present in 2-9% of general population over the age of 65 and its incidence increases with age. [2] The risk factors for AS is similar to that of atherosclerosis (age, male sex, smoking, hypertension and raised lipoprotein and LDL). [3]

### Case presentation

A 73-year-old male presented with typical ischaemic chest pain at rest and inferior ST-segment elevation on a background of exertional chest tightness over a six-month period. Paramedic ECG
(Figure 1) showed inferior STEMI with voltage criteria for left ventricular hypertrophy (LVH). Past medical history included hypertension and BPH. His risk factors for CAD included a previous history of smoking and a positive family history of premature cardiovascular disease. Examination revealed an ejection systolic murmur with an absent second heart sound. Given his ECG findings, he was brought directly into the cardiac catheterisation lab for coronary angiography and primary PCI.

**Investigation**

Coronary angiography revealed minimal atheroma within the right coronary artery (RCA) (Figure 2) and a mild stenosis of the mid left anterior descending (LAD) artery (Figure 3). Incidentally, heavy calcification was noted on the aortic valve on fluoroscopy (Figure 2). In view of the ongoing chest pain and persistent ST elevation a pressure wire study with intravenous adenosine was performed across the LAD lesion which showed a fractional flow reserve (FFR) of 0.90, indicating that the lesion was not significantly flow limiting.

Bedside echocardiography was performed which showed critical aortic stenosis (Vmax 5.5m/s, Max PG 90mmHg, Mean PG 72.2mmHg, AVA 0.8cm$^2$) (Figure 4) and evidence of LVH with no regional wall motion abnormalities (Figure 5). Short axis view (Figure 6) of the aortic valve revealed a calcified aortic valve with limited mobility and narrow opening.

**Treatment and outcome**

Prior to coronary angiography the patient was loaded with Aspirin and Ticagrelor. On identification of critical AS an IABP was inserted in view of ongoing ischemic chest pain, with persistent ST-segment elevation and no evidence of coronary occlusion. The case was discussed with the cardiac surgeons and he was accepted for urgent aortic valve replacement.

Following insertion of the IABP there was rapid resolution of chest pain and resolution of ST-segment elevation (Figure 7). The patient underwent emergency aortic valve surgery without bypass grafting. He made a good recovery and was symptom-free at 8-week follow-up.

**Discussion**

This is a rare case of critical aortic stenosis resulting in significant disruption of normal laminar haemodynamic across the valve, [4] which can lead to significant myocardial hypo-perfusion in the face of left ventricular hypertrophy despite unobstructed coronary arteries.

Lumley et al. [5] showed that the underlying mechanism for ischemia in patients with severe aortic stenosis is due to an abnormal cardiac coronary coupling – the inability to increase blood flow in proportion to cardiac workload resulting in ischemia. A similar case was published in 2010 by Wayangankar et al. [6] However, the patient in this case did exhibit ST-segment elevation.

While plaque rupture may result in STEMI despite the absence of a severe arterial stenosis, the persistence of ECG changes without arterial occlusion and the response to IABP is highly suggestive of a valvular cause for this gentleman’s presentation. Other differentials of STEMI with non-obstructing coronaries include acute aortopathy, endocarditis with embolus, myo-pericarditis and intracranial haemorrhage.

This case highlights the importance of the correlating clinical examination with focused bedside investigation like echocardiography in the management of patients presenting with chest pain. The European Association of Cardiovascular Imaging and the Acute Cardiovascular Care Association has recommended the use of echocardiography in patients presenting with acute chest pain, however, a level of competency is required for accurate interpretation of results. [7]
Funding statement

N/A

Declaration of interest

The authors declare that there is no conflict of interest.

Patient consent

Patient consent was obtained and consent form was signed

Author contributions and acknowledgements

YXG was involved with the patient’s care and wrote the case presentation, investigation and treatment outcome
SSB wrote the discussion and learning points of the case
DJK was the consultant in charge of the case and did the final draft.

References


Legends to tables/figures/videos

Figure 1. ECG on arrival
Figure 2. RCA (arrowhead showing diseased segment and arrow showing calcified AV)
Figure 3. Left sided coronary angiogram (arrowhead showing diseased segment in LAD)
Figure 4. Echocardiogram showing Doppler measurements
Figure 5. PSAX and 4-chamber view showing concentric LVH
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Figure 6. PLAX view showing calcified aortic valve (orange arrow)
Figure 7. ECG 2 hours post IABP
ECG on arrival

90x33mm (300 x 300 DPI)
RCA (arrowhead showing diseased segment and arrow showing calcified AV)

151x66mm (300 x 300 DPI)
Left sided coronary angiogram (arrowhead showing diseased segment in LAD)
Echocardiogram showing Doppler measurements

171x61mm (300 x 300 DPI)
PSAX and 4-chamber view showing concentric LVH

191x73mm (300 x 300 DPI)
PLAX view showing calcified aortic valve (orange arrow)

159x119mm (300 x 300 DPI)
ECG 2 hours post IABP

90x67mm (300 x 300 DPI)