Severe Aortic Stenosis presenting as a STEMI

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BACKGROUND

- A ST segment elevation myocardial infarction (STEMI) usually involves plaque rupture which is present in a coronary artery. This rupture leads to platelet aggregation and activation causing a thrombosed coronary artery resulting in a acute vessel obstruction.
- It is noted that from 2006-2011 there was a mean incidence of 258,106 STEMI's per year presenting to Emergency departments in the US(2).
- Usually STEMI's are the result of a coronary artery occlusion, however they can also occur in other conditions like myocarditis, coronary vasospasm, PE etc. In this case a STEMI was caused due to severe aortic stenosis and the lack of myocardial perfusion in the absence of a coronary obstruction.

KEY POINTS

- Aortic Stenosis affects 1.3% to 9.8% of the population above 60 years old and increases as we age.(1)
- AS causes a progressive outflow obstruction leading to compensatory Increased LV mass, increased LV systolic pressure while decreasing perfusion to the sub endocardium leading to ischemia.
- A physical exam and H&P are important during a STEMI prior to coronary angiogram.
- Any significant murmurs should be investigated with an echocardiogram
- Important differentials to consider are myocarditis, aortic dissection, pulmonary embolus and stress cardiomyopathy.

SUMMARY OF THE CASE

- A 77 year old female was brought in with neck and chest pain and anterior wall ST elevation on the EKG.
- The patient was noted to be in cardiogenic shock in the ED and was given fluids and started on Norepinephrine.
- The patient was then immediately taken to the catheter lab for primary percutaneous intervention.
- Cardiac angiography did not demonstrate any obstructive lesions
- The Angiogram did show severe calcification of the aortic valve as did the transesophageal echocardiogram done shortly after.
- The patient was hypotensive and having ventricular tachyarrhythmias; a intra aortic balloon pump(IABP) was inserted which improved her clinical status. The patient subsequently underwent a successful aortic valve(AV) replacement.
- The following is an uncommon presentation of critical Aortic Stenosis presenting as a ST segment elevation myocardial infarction(STEMI).

CLINICAL COURSE

- A 77 year old with a history of moderate AS and hyperlipidemia presented with neck,chest pain and presyncope. The patient also had an ER visit 2 days prior for palpitations and presyncope for which she was given a Holter monitor and instructed to follow up with her PCP.
- The initial EKG noted ST elevations anteriorly (figure 1). The patient was also noted to be hypotensive(cardiogenic shock) in the ER and NE was started for vasopressor support. The patient’s risk factors for CAD included poor diet, weight and a sedentary lifestyle, her non modifiable risk factors included age. Physical exam revealed a loud ejection systolic murmur.
- Given the EKG findings and the fact the patient was in cardiogenic shock she was taken to the cardiac cath lab for angiography and PCI.
- Angiogram revealed severe calcification of the aortic valve with minimal leaflet motion(figure 2). The patient’s coronary arteries were not found to have obstructive coronary disease(figure 3).
- A stat bedside echo was performed which demonstrated a severely calcified poorly mobile aortic valve with a transvalvular velocity of 5.1 m/s, a Max PG of 104.7 mmHg, a mean PG of 60mmHg and an estimated AVA of 0.5 cm2 (figure 4). A short axis view showed a calcified AV with limited mobility and narrow opening(figure 5). Other findings noted on echo were mild concentric LVH(figure 6), global hypokinesis with an EF of 35%, moderate MR and a RVSP of 36mmHg.

TREATMENT AND OUTCOME

- Prior to the angiogram the patient was given Aspirin and put on heparin. She was also put on Norepinephrine due to cardiogenic shock.
- During the angiogram it was noted that the patient’s blood pressure was unstable on norepinephrine and using the right femoral artery a arterial sheath and intra-aortic balloon was inserted and assistance began at 1:1 due to evidence of ischemia and persistent ST elevation with no evidence of an obstructing lesion. The patient’s arrhythmia and hypotension both improved post IABP placement.
- Arterial blood gas analysis was also performed which demonstrated a base deficit of -15 and 1 amp of sodium bicarbonate was given. Episodes of sustained ventricular tachycardia and supraventricular tachycardia were treated with an initial bolus of 300 mg of amiodarone followed by a subsequent 150 mg bolus because of recurrent arrhythmia.
- The patient underwent emergent Aortic Valve surgery. She has had 2 ED visits since for flank and mid thoracic pain.

DISCUSSION/CONCLUSION

- This case was a rare instance of critical AS that caused angina and a subsequent myocardial infarction due to impeded coronary blood flow in the presence of excess demand(3). This led to ischemia and hypoperfusion.
- Marcus et al(4) showed that mechanism behind this involves compensatory left ventricle hypertrophy that over time fails to overcome the outflow obstruction caused by severe AS, the hypertrophied heart has increased myocardial oxygen demand and the intramural coronary arteries may also be compressed by the hypertrophy and this in addition to decreased diastolic filling can lead to anaemia in the absence of CAD.
- Multiple case reports exist regarding this clinical scenario including one by Gue et al(5) in 2017 where the patient had a clinical picture very similar to the patient currently being discussed.
- Given the patient’s symptoms improved with placement of a IABP and the presence of symptoms despite clear coronaries a valvular pathology was determined to be the problem.
- It is important to consider differentials for a STEMI with clear coronaries. These include aortic dissection or coronary artery dissection, Coronary vasospasm, pericarditis, pheochromocytoma, Endocarditis or a Pulmonary embolus.
- This case highlights the importance of bedside echocardiography as a means to evaluate the aortic valve in a emergency setting.

REFERENCES