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# *Non-ST Elevation Myocardial Infarction Presentation in a Severe Aortic Stenotic Patient*

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## ABSTRACT

This case report demonstrates a patient who presents with typical findings for a diagnosis of NSTEMI. Upon further evaluation, this diagnosis was able to be ruled out. Presentations and labs consistent with NSTEMI masked the true diagnosis of aortic stenosis due to an oxygen supply/demand mismatch.

***Keywords:*** aortic stenosis, non-ST myocardial infarction, catheterization echocardiogram, valvular pathologies, coronary artery disease, atrial fibrillation, electrocardiogram, troponin I, cardiac myocytes, pacemaker, chest pain, oxygen supply/demand mismatch

## INTRODUCTION

Type 1 Myocardial infarctions consist of approximately 90% of cases involving damage to the coronary vessels. The other 10% is due to an oxygen supply/demand mismatch that often goes misdiagnosed. Incorrect treatment and management of coronary structure can lead to an increase in mortality, which the latter 10% already has. This is why it is crucial to keep oxygen supply/demand mismatch in mind when a case presents as that of coronary artery disease (Cohen & Visveswaran, 2020).

Aortic stenosis (AS) is a valvular stiffening that results in increased afterload of the left ventricle and hemodynamic instability (Stout & Otto, 2007). On a physical exam, AS results in a systolic ejection murmur that is described as crescendo-decrescendo. Prolonged aortic stenosis can result in increased myocardial oxygen demand and a decrease in myocardial viability due to supply/demand mismatch (Ross & Braunwald, 1968). However, there have been limited studies on how AS can be an underlying diagnosis in a clinical case that presents as a non-ST elevation myocardial infarction (NSTEMI).

Studies have shown that patients with moderate AS have increased mortality when they have a confirmed STEMI or NSTEMI (Abraham et al., 2023). However, there are only a few studies on the prevalence of patients who have symptoms presenting as MI but through diagnostic and confirmatory tests, reveal that MI is not the underlying cause of presenting symptoms; rather AS being the true diagnosis. This case brings focus on the importance of having an accurate diagnosis and treatment for patients presenting in chest pain. Our case presents with a patient who presented with symptoms of an NSTEMI but was later found to have severe aortic stenosis.

## CASE PRESENTATION

A 67-year-old, Caucasian female presented to the emergency department with a complaint of chest pain that was described as a "heaviness" radiating to the back. On admission, vitals included the following: blood pressure of 143/82, respiratory rate of 19, and temperature of 36.5C. Shortly after the initial vitals, the patient's vitals changed to a blood pressure of 76/62 and pulse of 145 with atrial fibrillation. She had a $CHA_2DS_2$-VASc score of 4 and a bled score of 2. Physical exam was notable for a grade 3/6 systolic ejection murmur. Laboratory workup demonstrated a hsTroponin I at 1-hour level elevated at 216.7ng/L (Critical), with a hsTroponin I at baseline of 16.8ng/L and an elevated BNP level of 195 pg/mL.

### Past Medical History

Her past medical history included asthma, hypothyroidism, hypertension, hyperlipidemia, and type 2 diabetes mellitus. She had an unspecified murmur since childhood. She endorsed smoking half a pack of cigarettes a day since the age of 20.

### Differential Diagnosis

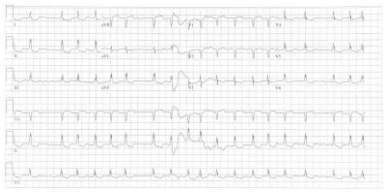
The main differential diagnosis for the patient included NSTEMI, Atrial fibrillation with RVR, Sick Sinus Syndrome, Bradycardia, and Aortic Stenosis.

### Investigations

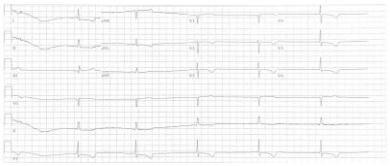
Due to the chief complaint of persistent chest discomfort, troponin levels and ECG were taken. Troponin-I levels were elevated (baseline 16.8 Gn/L to 1 hour 216.7ng/L) and initial ECG showed atrial fibrillation as well as some ST segment abnormalities. Several repeat ECGs were done the same day and the next day which demonstrated T-wave inversions from V2 to V6, II and AvF with sinus bradycardia. (Figure 1).

Figure 1: ECG findings – (a) patient's initial ECG showed an irregular rhythm and tachycardia, consistent with atrial fibrillation, (b) a secondary ECG of the patient showed bradycardia

(a)



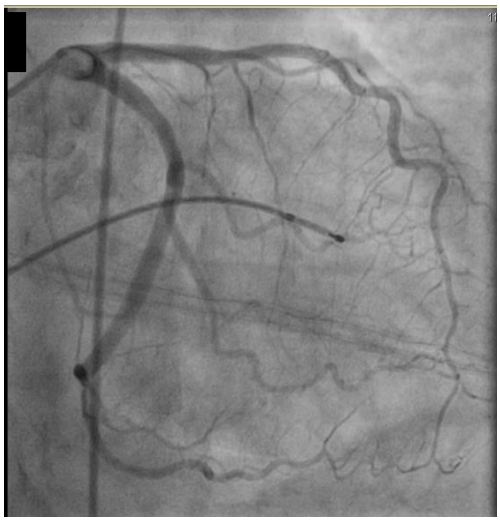
(b)

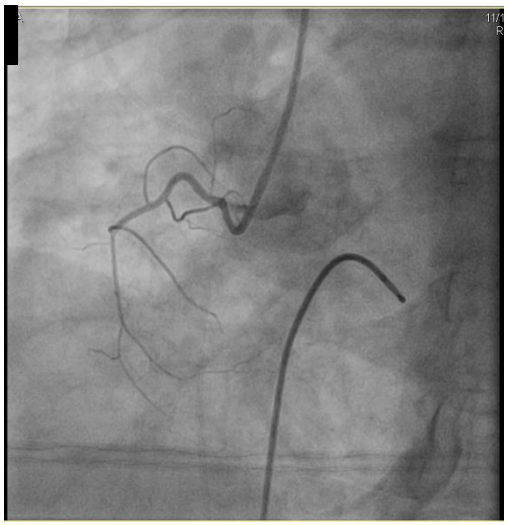


Due to severe bradycardia, ECG findings of atrial fibrillation, greatly elevated troponin levels, and symptom of chest pain, a cardiac catheterization was completed instead of performing an echocardiogram first. Angiogram results showed normal left main coronary artery, left anterior descending artery, and right coronary artery. In the left circumflex artery, there was mild luminal irregularity in the proximal vessel, but otherwise, no significant stenosis was noted (Figure 2).

Figure 2: Catheterization of coronary vessels – patient's coronary vasculature shows adequate perfusion and no signs of coronary artery disease in the left anterior descending, left circumflex artery (a) and the right coronary artery (b).

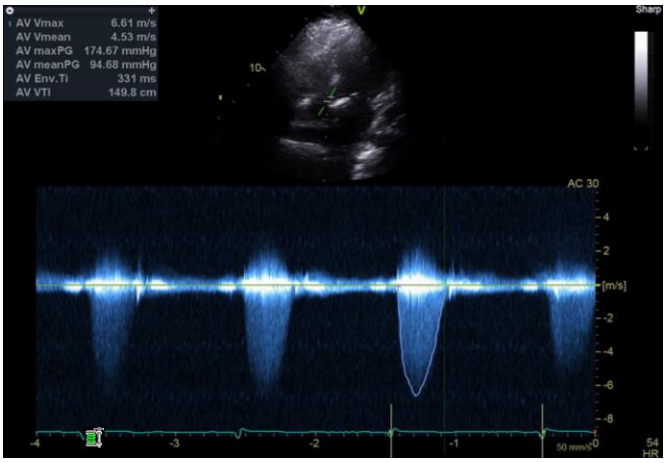
(a)



(b) 

During catheterization, a peak 100mmHg gradient was noted across the aortic valve, suggesting severe aortic stenosis. Further investigation with an echocardiogram was completed, and it showed findings of an aortic valve consistent with critical stenosis. Aortic valve area of .46 cm2 and mean gradient of 94mmHg was recorded (Figure 3).

Figure 3: Echocardiogram showing aortic stenosis – a mean pressure gradient of 94.68 mmHg is seen across the aortic valve in the echocardiogram




### Management

Due to echocardiogram findings showing severe stenosis of the aortic valve, the patient was scheduled for aortic valve replacement surgery and permanent pacemaker implantation for her severe bradycardia.

## DISCUSSION

This case highlights a patient with a true diagnosis of AS who had the common presentation of NSTEMI. Our patient demonstrates the importance of considering AS as a differential diagnosis even when disease presentation and labs point to NSTEMI. The common presentation of NSTEMI includes nausea, vomiting, and chest pain that can radiate down the left arm, back, and/or neck, and dyspnea either at rest or exertion. When suspicion of NSTEMI exists, it is further evaluated with ECG and cardiac biomarkers. ECG findings can include ST elevation/depression or T wave inversions while troponin at baseline and after 1 hour is used to further confirm the suspicion of NSTEMI. A 1-hour troponin level five times higher than baseline troponin is a positive finding for an MI (Gilutz et al., 2019). This patient had a baseline troponin I level of 16.8 ng/L which jumped to 216.7 ng/L within an hour. She also presented with complaints of nausea and chest pain radiating to the back which supports a diagnosis of MI. Her dyspnea during her walks was described by her as being her baseline. Furthermore, she has several risk factors for coronary artery disease including a history of hypertension, hyperlipidemia, diabetes, and smoking half a pack a day. Despite several factors pointing to an NSTEMI, echocardiogram and catheterization revealed an underlying pathology of AS.

AS is a valvular stiffening that results in increased afterload of the left ventricle and hemodynamic instability (Stout & Otto, 2007). Pathogenesis of AS involves calcification from lipid deposition and oxidation, which triggers an immune response that continues the calcification of the valve (Olsson et al., 1999). On presentation, patients who are found to have AS will display angina similarly to that of our patient. However, other symptoms typically seen in AS including exertional dyspnea, syncope, and ultimately, heart failure was not present in this patient (Lindman et al., 2013). Research studies have also supported that patients found to have AS had an elevated troponin level not related to NSTEMI (Patel et al., 2022). In this patient, the initial presentation showed an elevated troponin at 1 hour in addition to their history would suggest the diagnosis of NSTEMI. Studies on the masking effect NSTEMI diagnosis have on valvular disorder pathologies have been limited, so

we strive to provide additional support that shows an underlying diagnosis of AS can be mistaken for NSTEMI.

An oxygen supply/demand mismatch offers a possible explanation for why this patient with severe AS presented so similarly to NSTEMI. The mismatch causes damage in the cardiac myocytes, which can contribute to the elevated troponin levels seen on the lab values (Cohen & Visveswaran, 2020). Some secondary causes of the mismatch include arrhythmias, sepsis, anemia, and hypotension (Pillai et al., 2020). Our patient had uncontrolled atrial fibrillation and hypotension that may have contributed to the mismatch in oxygen supply/demand through the coronary vessels.

NSTEMI and AS are managed very differently, contributing to the importance of keeping both differentials in mind when a patient presents with symptoms similar to those seen in the case. AS management depends on cause and severity. Severe cases, like that seen in our patient, are treated with valve replacement (Patel et al., 2022). On the contrary, coronary blockages seen in NSTEMI are managed with stent placement and blood thinners (Gilutz et al., 2019).

This patient had an unusual presentation of severe AS. Her chest pain and elevated troponin levels can be explained by the oxygen supply/demand mismatch seen as a result of AS. AS due to oxygen supply/demand mismatch should be kept on the differential in patients with elevated troponin levels and a disease presentation more commonly associated with an NSTEMI as both are managed differently.

## CONCLUSION

The patient's symptoms of nausea, chest pain, and elevated troponin levels are consistent with a diagnosis of NSTEMI. However, this patient did not have NSTEMI, but rather AS discovered during catheterization. AS would not commonly present in this fashion, which makes this an unusual presentation of this diagnosis. It is important to keep AS on the list of differential when a patient presents with symptoms that would commonly indicate NSTEMI. AS would also be managed differently than NSTEMI. The conclusion of the patient case included a prosthetic valve replacement of the aortic valve followed by a dual chamber (LBBAP) permanent pacemaker implantation.

## REFERENCES

Abraham, B., Farina, J.M., Fath, A., Abdou, M., Elbanna, M., Suppah, M., Sleem, M., Eldaly, A., Aly, M., Megaly, M., Agasthi, P., Chao, C. J., Fortuin, D., Alsidawi, S., Ayoub, C., Alkhouli, M., El Sabbagh, A., Holmes, D., Brilakis, E.S., & Arsanjani, R. (2023). The impact of moderate aortic stenosis in acute myocardial infarction: A multicenter retrospective study. *Catheterization and cardiovascular interventions: official journal of the Society for Cardiac Angiography & Interventions*, *102*(1), 159–165. https://doi.org/10.1002/ccd.30676

Cohen M. & Visveswaran G. (2020). Defining and managing patients with non-ST-elevation myocardial infarction: Sorting through type 1 vs other types. *Clinical cardiology*, *43*(3), 242–250. https://doi.org/10.1002/clc.23308

Gilutz, H., Shindel, S., & Shoham-Vardi, I. (2019). Adherence to NSTEMI Guidelines in the Emergency Department: Regression to Reality. *Critical pathways in cardiology*, *18*(1), 40–46. https://doi.org/10.1097/HPC.0000000000000165

Lindman, B.R., Bonow, R.O., & Otto, C.M. (2013). Current management of calcific aortic stenosis. *Circulation research*, *113*(2), 223–237. https://doi.org/10.1161/CIRCRESAHA.111.300084

Olsson, M., Thyberg, J., & Nilsson, J. (1999). Presence of oxidized low density lipoprotein in nonrheumatic stenotic aortic valves. *Arteriosclerosis, thrombosis, and vascular biology*, *19*(5), 1218–1222. https://doi.org/10.1161/01.atv.19.5.1218

Patel, K.P., Rathod, K., Akhtar, M., Jones, D.A., Ozkor, M., Kennon, S., Mathur, A., Pugliese, F., Mullen, M. J., & Baumbach, A. (2022). Differentiating Between Acute Decompensated Aortic Stenosis and Myocardial Infarction. *Cardiovascular revascularization medicine:*

*including molecular interventions*, *43*, 13–17. https://doi.org/10.1016/j.carrev.2022.04.003

Pillai, B., Trikkur, S., Farooque, U., Ramakrishnan, D., Kakkra, J. J., Kashyap, G., Lalwani, C., Mani, A. B., & Vishwanath, J. (2020). Type II myocardial infarction: predisposing factors, precipitating elements, and outcomes. *Cureus, 12(*7), e9254. https://doi.org/10.7759/cureus.9254

Ross Jr. J. & Braunwald E. (1968). Aortic stenosis. *Circulation.* 38(1 Suppl):61-7. doi:10.1161/01.cir.38.1s5.v-61. PMID: 4894151.

Stout K.K. & Otto C.M. (2007). Indications for aortic valve replacement in aortic stenosis. *J Intensive Care Med.*;22(1):14-25. doi:10.1177/0885066606295298. PMID: 17259565.