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Case Report

A Case Report of Non-Atherosclerotic Driven Myocardial Infarction in a Patient Presenting with Coronary Artery Spasm

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Abstract: Non-atherosclerotic processes are regarded as equally important contributors to a substantial number of coronary problems mainly myocardial infarction. This includes coronary spasm which has been considered as one of the coronary syndromes leading to myocardial infarction. These non-atherosclerotic events ensuing in Major Adverse Cardiac Events (MACE) not only require various diagnostic and therapeutic strategies but also there is a need to delineate the underlying etiology for their effective treatment and management. **Case Summary:** We report a case of Anterior Wall Myocardial Infarction (AWMI) driven by a non-atherosclerotic event i.e. coronary spasm. Concomitant marked ST-segment elevation recorded on ECG revealed a diffuse mid distal disease in our patient. We report here the initial presentation, coronary care & intervention and throughout the clinical course of our patient. **Conclusions:** Myocardial infarctions involving non-atherosclerotic causes in young individuals as in our study should be reported by medical practitioners and given equal importance as they might indicate the underlying root cause of such events. Effective treatment of such future cases can be done by taking management strategies, diagnostic findings and prognostic data into consideration.

Introduction:

Myocardial infarction (MI) is one of the leading causes of mortality and morbidity worldwide. Although today, the survival chances have fairly increased, however, the likelihood of re-infarction among survivors is very common and poses a life-threatening risk [1]. Though driven by atherosclerotic and non-atherosclerotic events, the latter one is often not highlighted and reported in the differential diagnosis [2]. Coronary Artery Spasm (CAS) or Coronary Vasospasm is a critical non-atherosclerotic event that if not treated promptly can progress to a deadly condition like MI or even sudden death. It is characterized as the sudden, intense vasoconstriction of an epicardial coronary artery that may result in partial or complete vessel occlusion [3]. The pathological mechanisms that contribute to CAS include endothelial dysfunction, vascular smooth muscle cells and other microvascular dysfunction [4]. It is reportedly said to occur between 40 and 70 years and is not a much common event among individuals lesser than the reported age [5]. In this case report we highlight and discuss CAS as a non-atherosclerotic event in an early thirty male patient presented with ST-elevation myocardial infarction (STEMI) or AWMI on diagnosis.

Case Presentation:

A 34-year male patient (resident Pakpattan City) was presented in the Emergency Room (ER) at Mayo Hospital on 31st December, 2020 with an episode of chest pain for the past two hours. On admission to ER, he had established severe central chest pain along with profuse sweating.

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He had no prior experience of chest pain. The patient had no established conventional risk factors like diabetes mellitus, hypertension, ischemic heart disease and active smoking. Electro-cardiography (ECG) revealed a typical pattern of changes with ST-segment elevation in anterior precordial leads. He was shifted directly from ER to Cath. Lab for immediate primary percutaneous coronary intervention (PCI). His vitals at presentation were: BP=100/60mmHg, Pulse=100/min, SaO_2=98% and Respiratory Rate (RR) =20/min. Upon intervening with coronary angiography, performed through right femoral artery using 6F femoral sheath, revealed constriction of the left anterior descending (LAD) artery of the heart indicating mild mid disease with diffuse mid distal disease. Coronary angiography identified coronary vasospasm as the primary cause of AWMI. The condition was relieved by intracoronary nitroglycerine.

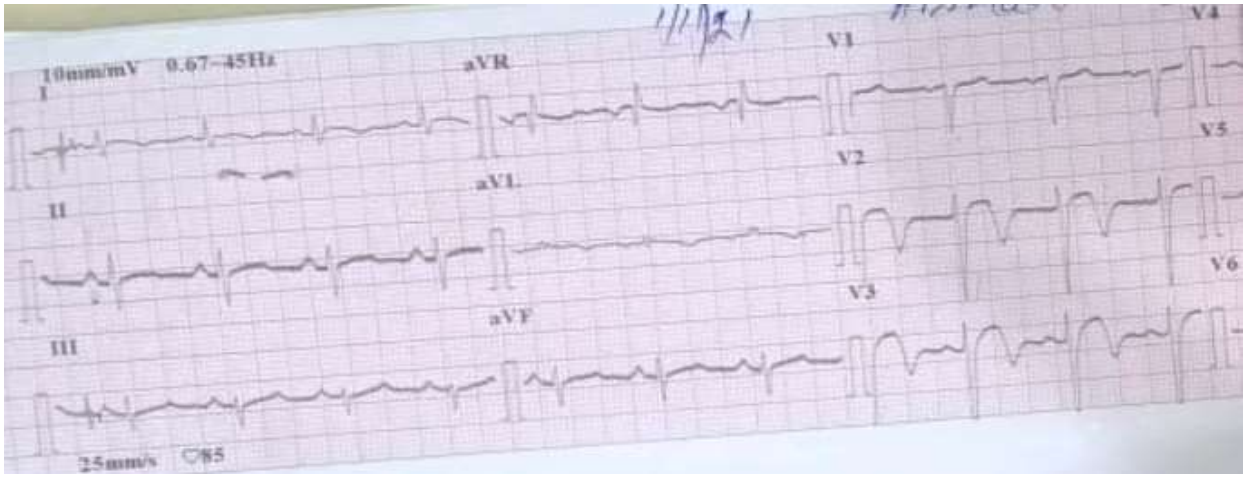

Figure 1

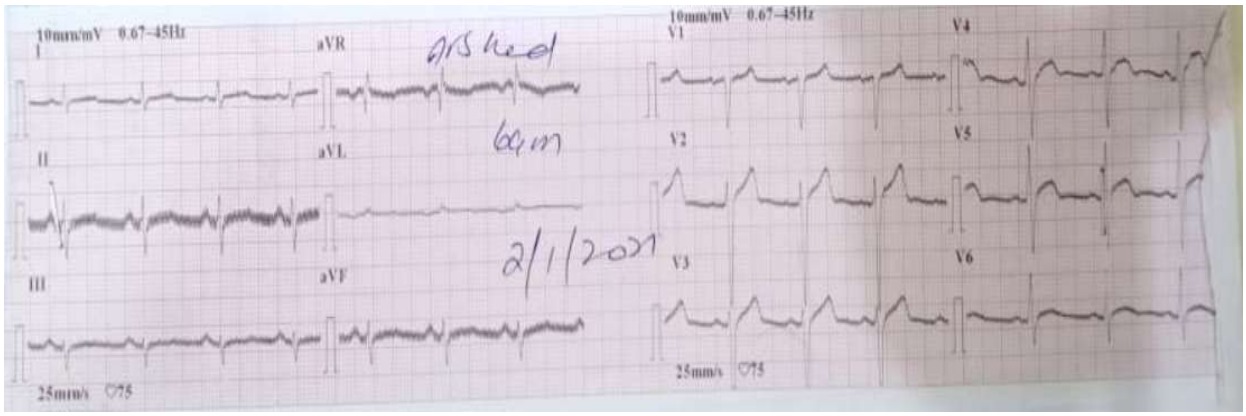

Figure 2

The above Figures1 & 2 represents the electrocardiogram of the patient under study. Significant ST-segment changes in the anterior leads were recorded.

The next day the patient underwent echocardiography to assess LVEF (Left Ventricular Ejection Fraction). Echocardiography revealed an LVEF value of 60% which falls under a normal range. Furthermore, no significant wall motion abnormalities were observed. ECG performed the next day to look for any changes were also settled. After 48 hours of being under surveillance which was completely uneventful, he was discharged from the hospital. He was treated and discharged with calcium channel blockers, dual anti-platelets, statins and nitrates. He came as an outpatient after 18 days for follow-up.

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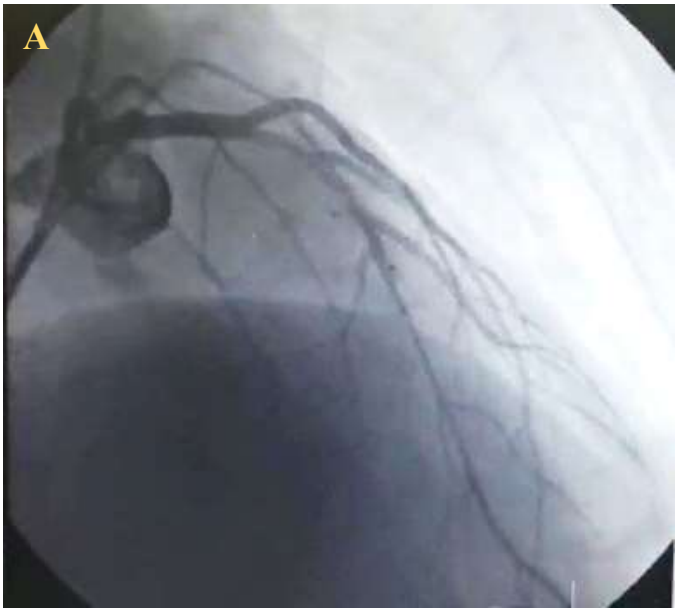
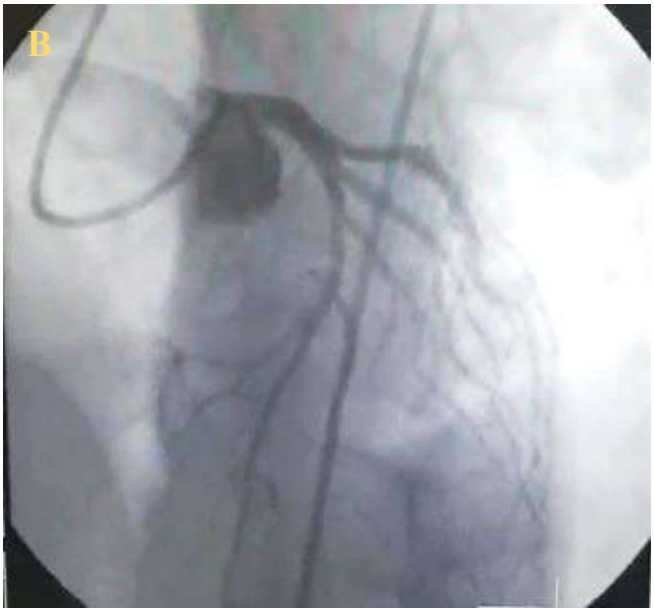
His vitals were: Pulse= 102/min, BP=110/70mmHg, S2O2=99% and RR= 16/min. He had no active complaints, did not require any hospital admission and his condition was controlled and stabilized by medical management.

<table>
<tr><th colspan="6">M-MODE DIMENSIONS</th><th>DOPPLER</th></tr>
<tr><th>Parameter</th><th>Normal Range</th><th>Result</th><th>Parameter</th><th>Normal Range</th><th>Result</th><td rowspan="2">DT=257ms EA Ratio=1.4, ETD1=10m/s E/E=11</td></tr>
<tr><td>Aortic Root</td><td>20-38mm</td><td>33</td><td>IV Septum</td><td>6-11mm</td><td>11</td></tr>
<tr><td>AOV Annulus</td><td></td><td></td><td>Posterior LV Wall</td><td>6-11mm</td><td>8</td><td>AVPG=9mmHg</td></tr>
<tr><td>Valve Opening</td><td>15-27mm</td><td></td><td>LV Dimension EV</td><td>36-56mm</td><td>50</td><td>RVSTD1=16cm/s</td></tr>
<tr><td>Left Atrium</td><td>19-39mm</td><td>39</td><td>LV Dimension (ES)</td><td>25-41mm</td><td>34</td><td>TVPG=mmHg</td></tr>
<tr><td>RV</td><td>7-25mm</td><td>25</td><td>E/F</td><td>50-70%</td><td>60%</td><td rowspan="3">Color Flow<table><tr><td>AR=</td><td>MR=</td><td>TR=</td></tr></table></td></tr>
<tr><td>RA</td><td></td><td></td><td>F/S</td><td>29-37%</td><td></td></tr>
<tr><td>IVC</td><td></td><td></td><td>EPSS</td><td>6mm</td><td></td></tr>
</table>

Table 1: Post-angiography Echocardiography Report

The above Table 1 represent a normal sized aortic root, normal sized LV cavity with good LV systolic function along with no segmental wall abnormalities. Intact IAS & IVS activity and normal pericardium. Hence a good biventricular systolic function was concluded.





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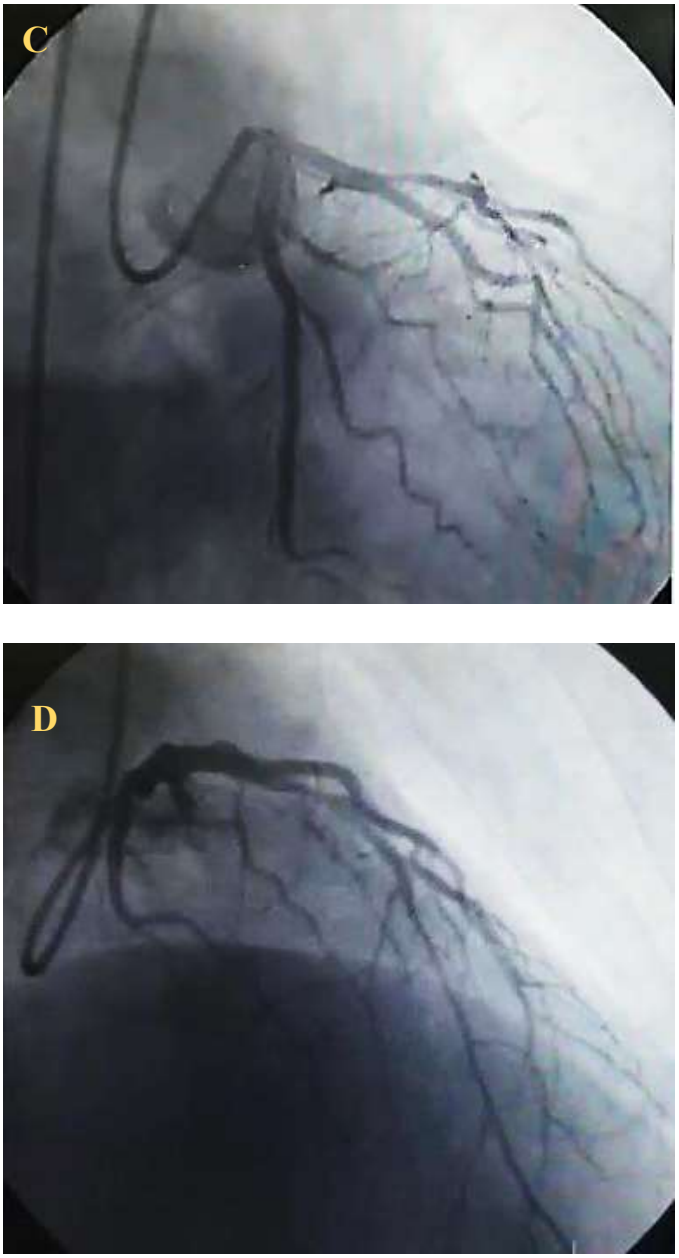

Figure 3: Coronary Angiogram of patient**:** The above fluoroscopy indicates the LAD disease.

Discussion:

Coronary artery spasm is a vasoconstrictive cardiac disorder that leads to transient myocardial ischemia, accompanied by chest pain and visible ECG changes. In worst scenarios, prolonged conditions can also lead to myocardial infarction. It occurs predominantly due to endothelial dysfunction or hyper-reactive vascular smooth muscle cells [6]. However, in this case report, we present a patient of CAS leading to complication of myocardial infarction with transient ST-segment changes.

Due to challenges in diagnosis, as stated by Matta, A et al., (2020) [4] and Slavich, M et al., (2016) [5] the actual prevalence of condition i.e. CAS remains largely unknown however, in contrast to traditional angina pectoris, it occurs less frequently, afflicting males more in comparison to females. Our case also indicated CAS affecting the young male individual (early thirties) as stated by Matta (2020) & Salvich (2016). A complex interplay of factors are reported to be responsible for the underlying pathology of coronary spasm, the most established and accepted ones apart from above mentioned also include atherosclerotic stenosis. Although atherosclerosis and CAS are two distinct clinical entities however, both the entities may co-exist. Plaque may tend to induce spasm whereas coronary spasm itself can induce the rupture of atherosclerotic plaque, subsequently leading to MI. This means that the progression of one can influence the onset of other [7]. However, in our case, the coronary spasm was driven by a non-atherosclerotic event which means that the underlying factor that contributed to this event needs to be investigated further.

According to Muhammed, I et al., (2014) [3] and Ertan C et al., (2017) [8] CAS do not always present with ECG changes however, this was not similar with the findings of our case as ECG changes were recorded in our patient. ST-segment changes were recorded in leads aVL, V1 V2 and V3 in our patient before treatment. In addition to this, our patient was treated with intracoronary nitroglycerin intervention and many studies highlight the importance of administration of intracoronary nitrates during diagnostic coronary angiography before PCI. Vishnevsky et al., (2017) [9]. Documented a series of patients referred for PCI with >70% stenosis on angiography that resolved after treatment with intracoronary nitroglycerin. Hence, routine injection of intracoronary

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nitroglycerin before proceeding to angioplasty could help diagnose some cases of CAS.

Conclusion:

To sum it all, CAS is a complex multifactorial disorder that can lead to serious complications like MI. Hence, early recognition and an appropriate management approach is primordial. It has important symptomatic and diagnostic implications, it still remains difficult to diagnose. While medications like calcium channel blockers and statins are available to effectively treat and control symptoms however, suppression of spasm still remains a problem. Therefore, non-atherosclerotic coronary diseases such as CAS should not be overlooked during differential diagnosis of MI to avoid future complications.

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