

MINOCA Presenting as Anterior STEMI in a 19-year-old Complicated by Complete Heart Block and Pacemaker Implantation: A Rare Case Report

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Case Report

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Abstract

Background

Myocardial Infarction with Non-Obstructive Coronary Arteries (MINOCA) accounts for 5–10% of all cases of acute myocardial infarctions. It has recently been observed in increasing numbers in younger patients without established coronary risk factors. While thought to be less severe, MINOCA may be associated with malignant arrhythmias and conduction disturbances. A post-anterior ST-elevation myocardial infarction (STEMI) complete heart block (CHB) is uncommon in this demographic and may be related to severe septal ischemia.

Case Presentation

We describe a case of a 19-year-old male who never had comorbid conditions before admission for classic ischemic chest pain with anterior STEMI. He underwent streptokinase thrombolytic therapy, for which coronary arteriography disclosed normal coronaries with suggestions of transient thrombotic occlusions with spontaneous recanalization. He returned with recurrent pain in the chest with prompt development of symptomatic CHB with hypotension for which he required dopamine infusion with transvenous pacing. His conduction disturbances progressed from CHB to bifascicular block, then to trifascicular block, with CHB eventually. Repeat coronary arteriography disclosed normal coronaries once more. The echocardiogram disclosed an ejection fraction of 45% with moderate wall motion abnormalities. Because of conduction instabilities, he underwent permanent pacing with significant improvement thereafter, to be sent home in stable conditions.

Conclusion

This case brings into focus a very rare presentation of MINOCA in a young patient that was complicated by progressing conduction system disease that needed permanent pacing. The importance of early recognition of MINOCA-related STEMI due to conduction system disease with careful monitoring, irrespective of a normal coronary cath angiogram, can once again be appreciated.

1. Introduction

Acute Myocardial Infarction (AMI) in young adults, including those under 40, is increasingly acknowledged and presents particular challenges for diagnosis and treatment. As it is unlikely that young adults would have typical atherosclerotic coronary artery disease, alternative mechanisms, including coronary thrombosis with recanalization, coronary spasm, embolisms, myocarditis, and hypercoagulable states, either congenital or acquired, also warrant consideration [1, 2]. This disease is presently generally referred to using the designation 'myocardial infarction with non-obstructive coronary arteries' (MINOCA). This is now known to be more common in younger people who lack conventional

coronary disease risk factors and is reported to constitute 5–10% of MI cases [10]. Notably, MINOCA is not an innocent disease, even if it does not show significant coronary disease obstruction, and is associated with significant morbidity, potential arrhythmic disturbances, and also detrimental long-term outcomes.

There may be varied rhythm disturbances like supraventricular tachycardia (SVT), atrial fibrillation, ventricular rhythm disturbances, as well as intraventricular conduction disturbances in the type of bifascicular or trifascicular block that might be more frequent in MINOCA patients [3]. Moreover, the development of conduction disorders requiring permanent pacing has been noticed in MINOCA patients, that point towards the severity of electrical complications [4].

We will highlight an unusual case of a 19-year-old patient with an anterior wall myocardial infarction, complete heart block, and episodes of arrhythmias with progressive disturbance of the conduction system function with normal coronary arteries at angiography. It will illustrate the complexity of AMI in a young patient and the role of transient coronary thrombosis with recanalization.

2. Case Presentation

The patient, a 19-year-old male with no previously identified medical conditions, initially presented to the ED with acute chest pain of typical character. The pain was central, associated with diaphoresis, generalized weakness of the whole body, shortness of breath, and nausea, and referred to the left arm and back. The patient never had smoking, drug use, preceding viral illnesses, or myopericarditis symptoms. His family history identified only hypertension with no history of sudden deaths in relatives or hyperlipidemia. He had electrocardiographic evidence of an anterior wall myocardial infarction. He also took streptokinase thrombolytic medication on admission to the ED in an effort to open the blocked coronary arteries. However, he had a heart biomarker with a highly sensitive troponin-I level of 5.4 mg/mL. He also had coronary angiographic studies at this point, with normal coronary arteries with a presumed recanalized thrombus involving the left anterior coronary vein.

The patient was re-presented for the complaints of recurrent chest pain and palpitations. The patient was given a dose of 1 mg intravenous atropine, following which electrocardiography showed a complete heart block. At the time of presentation, the patient was hypotensive with a systolic blood pressure of 80 mm of Hg and bradycardic with a pulse rate of 40 per minute. The patient commenced on a dopamine infusion. The patient complained of palpitations, dizziness, lightheadedness, and mild chest pain a few hours after being admitted to the hospital.

2.1 Investigations

Electrocardiography was suggestive of complete heart block with ST elevations in the anterior leads. Blood tests showed CK-MB levels of 42 U/L. Thyroid profile revealed TSH of 5.03 μ IU/mL with normal thyroxine of 5.62 μ g/dL, and Vitamin D3 level of 0.8 ng/mL. Electrolytes and kidney functions were found to be within normal limits. Liver functions showed mildly elevated ALT of 65.4 U/L, Alkaline phosphatase

of 71.3 U/L, and normal Total bilirubin of 0.61 mg/dL. The virology profile was negative for HBs Ag, Anti-HCV, and Anti-HIV.

Glycemic profile showed an HbA1c of 5.28%. Complete Blood Counts showed WBC of $8.44 \times 10^3 /\mu\text{L}$, Hemoglobin of 14.8 g/dL, Hematocrit of 42.7%, Mean corpuscular volume of 82.3 fL, and Platelets of 215,000/ μL . The coagulation study was normal with PT of 12 seconds, INR of 1.0 units, and aPTT of 28 seconds. Lipid profile, including triglycerides, LDL, and HDL, was also normal. Transthoracic echocardiography showed a left ventricular ejection fraction of 45% with mild left ventricular systolic dysfunction, regional wall motion abnormalities, a normal pericardium, and a small pericardial effusion.

2.2 Management

A temporary transvenous pacemaker was employed because of symptomatic bradycardia and hemodynamic instability. During the second day of hospital admission under a temporary pacemaker, the patient developed Supraventricular tachycardia with aberrancy. The following day, the off TPM-ECG showed a bifascicular block, which eventually transformed into a trifascicular block. However, repeat coronary angiography once again showed normal coronaries. The patient remained paced on temporary pacemakers for a total of five days. After which, a permanent pacemaker was implanted.

During hospital stay, the patient was maintained on a variety of medications, namely, dapagliflozin, rosuvastatin, enoxaparin, spironolactone with furosemide, aspirin, clopidogrel, cefoperazone-sulbactam, and IV omeprazole. The sacubitril-valsartan was started; however, because the patient remained persistently hypotensive with a blood pressure range of 80/50 mmH

2.3 Diagnostic Assessment

The final clinical diagnosis was acute myocardial infarction complicated by complete heart block and probably caused by a recanalized thrombus in a young patient with normal coronary arteries.

2.4 Follow-up and outcomes

The patient's health had shown improvement, had remained vitally stable, and had been discharged with instructions for follow-up with the cardiology clinic after a month.

3. Discussion

This case expands on some atypical, highly relevant aspects, namely, acute anterior ST-elevation myocardial infarction (STEMI) occurring in a 19-year-old individual despite having normal coronaries, the progression through complete heart block, bifascicular, and eventually advancing to trifascicular conduction delay, besides arrhythmias, being dependent on temporary pacing, and eventually requiring permanent pacemaker therapy. It represents the spectrum of the general condition of myocardial infarction with non-obstructive coronaries (MINOCA)

Many young individuals with acute myocardial infarction have non-traditional risk factors, such as hypercoagulability, substance use, or coronary anomalies [6, 7, 17]. Our young patient did not have the traditional risk factors (no smoking, no hyperlipidemia, and so on), which are consistent with the literature, which showed that MINOCA patients are comparatively young, predominantly female, with fewer traditional risk factors for atherosclerosis [6, 8]. Previous case series among young individuals with normally appearing coronaries included hypotheses such as coronary vasospasm, thrombus, or spontaneous coronary artery dissection without overt plaque [17]. In contrast to older MI patients with diffuse atherosclerosis, our patient's only notable finding was a remote history of hypertension in the family; no hyperlipidemia, diabetes, or illicit drug use were present. European cohorts even report MINOCA in ~ 10% of acute coronary syndromes [9]. Despite having "normal" coronaries, MINOCA is not benign, and its diverse etiologies demand thorough evaluation [10]. Contemporary expert statements likewise stress the need for multimodality imaging and tailored diagnostic approaches to uncover the underlying etiology [16].

Multiple pathophysiological processes underlie MINOCA. An occlusive thrombus (often transient) can cause MI and then lyse, leaving a "normal" angiogram. In fact, our angiogram described an apparent "recanalized thrombus" in the LAD, fitting this mechanism. Intense vasospasm can induce transmural infarction despite patent arteries [5, 8]. Spasm is implicated in ~ 27% of MINOCA cases when provoked. Hypercoagulable states and paradoxical emboli can produce MI in unobstructed arteries. For example, a 19-year-old with an undiagnosed atrial septal defect suffered MI from a paradoxical coronary embolus. [8] (No embolic source was found in our patient.) In a MINOCA series, 14% had detectable thrombophilia [11]. Our patient had no prodromal viral illness, normal inflammatory markers, and persistent ECG changes, making myocarditis unlikely. In our case, the leading hypothesis is a brief LAD thrombotic occlusion with spontaneous lysis, possibly augmented by subtle endothelial dysfunction, given the normal coronaries on two angiograms.

High Atrio Ventricular block (HAVB), including Complete Heart Block (CHB), complicating ST elevation MI is known, but the incidence and significance vary depending on the MI location. In anterior MIs, CHB typically indicates severe septal and His-Purkinje system damage. Kawamura et al. demonstrated that CHB in acute anterior STEMI protected cent-percent patients, although 82% died compared to 20% without CHB in follow-up. [12]. In inferior MIs, CHB is usually nodal and transient. In AWTMI, CHB usually becomes infranodal, associated mainly with wide-complex escape rhythms, and frequently, clinically, precipitates cardiovascular collapse.[12, 13] In the patient, CHB, associated with bilateral bifascicular, trifascicular block, indicated extensive septal ischemia. Moreover, CHB occurred after atropine and associated chest pain, suggesting transient ischemia, and not sustained conduction system injury.

Similar cases like ours are very rare. There was a case that was older, where a 45-year-old patient had an anterior MI that progressed into transient trifascicular delay and CHB, but eventually resolved without the need for a permanent pacemaker [14]. This case was somewhat different, as our patient eventually needed a permanent pacemaker. Another patient, aged 19, experienced MI because of pseudoephedrine

ingestion caused by vasospasm [5], but he had normal stable conduction. In contrast, our case is unique for combining a young age, anterior STEMI, MINOCA picture, and life-threatening

Quality of life can be severely affected following a MINOCA, with reports of decreased exercise performance and mental health or vitality issues compared to healthy controls.[19] A current systematic review indicates that individuals with MINOCA or Takotsubo suffer from poor mental health status or quality of life compared to non-cardiac diagnoses, with recovery not always occurring.[20]

As per guidelines, in our patient, we have resorted to temporary pacing, which was associated with improvement of conductivity in days, but later replaced with a PPM in order to prevent recurrence of complete AV block. While there is less mortality in comparison to obstructive MI, there are adverse events, including arrhythmias and heart failure, which vary according to the basic cause[15]. Our patient's course (an EF of 45% and conduction defects) reminds us that extensive myocardial injury can occur even without visible atherosclerosis

4. Conclusion

Firstly, in children, chest pain requires complete investigation for MI, even in the absence of risk factors. Secondly, in patients suspected of having true MI, initial negative coronary arteriography does not preclude later arteriography or intravascular imaging in the event of persistent ischemia. Thirdly, in cases of high AV block complicating MI, according to guidelines, temporary pacing is recommended for hemodynamic stability, with consideration of implantation of a permanent pacemaker in cases of prolonged block.

Declarations

Ethics Approval

Ethical approval is not required for case reports and case series in our institution

Consent

Written informed consent was obtained from the patient for publication of this case series and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Conflict of Interests

The authors declare that they have no conflicts of interest

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Not commissioned, externally peer-reviewed

Contribution And Acknowledgment

Abbas Khan, Okasha Tahir, and Alina Batool: Formal analysis and data curation. Jaweria Farman is writing the 1st draft. Askar Nisar is reviewing the methodology. Muhammad Hassaan Javaid, Raghabendra Kumar Mahato and Hakim Ullah Wazir: References and editing, writing the final draft, and final review. The author(s) read and approved the final manuscript

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Author Contribution

Abbas Khan, Okasha Tahir, and Alina Batool: Formal analysis and data curation. Jaweria Farman is writing the 1st draft. Askar Nisar is reviewing the methodology. Muhammad Hassaan Javaid, Raghabendra Kumar Mahato and Hakim Ullah Wazir: References and editing, writing the final draft, and final review. The author(s) read and approved the final manuscript

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Not applicable.

Data Availability

Data available on request from the authors

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Figures

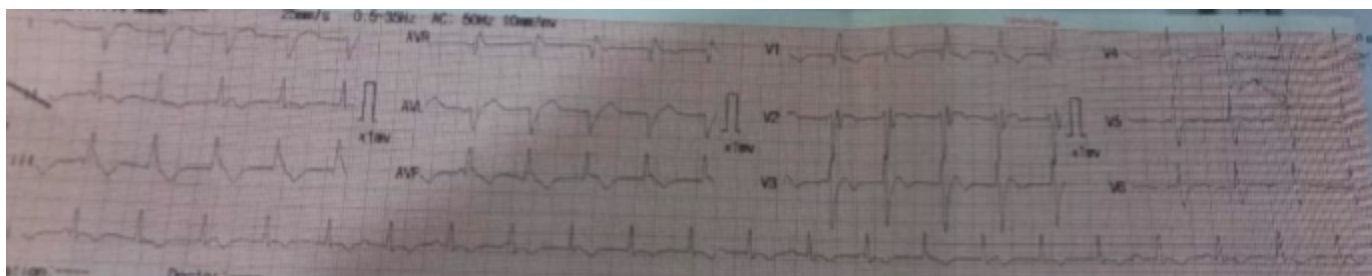


Figure 1

Initial 12-lead ECG at presentation showing marked ST-segment elevation in anterior leads (V1–V4) consistent with acute anterior wall STEMI.

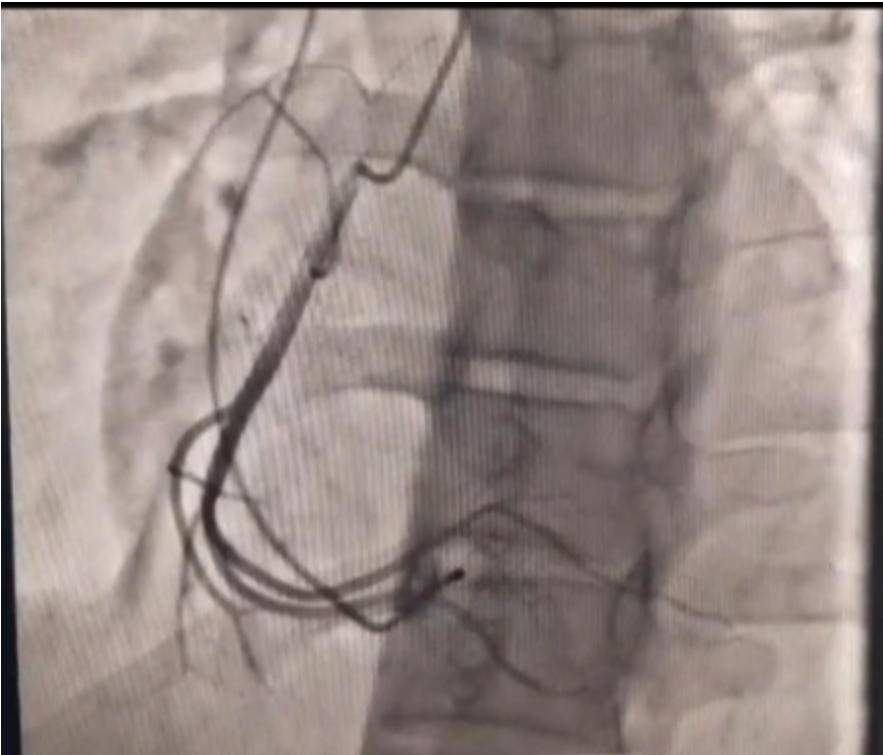


Figure 2

ECG prior to PPM showing sinus bradycardia with conduction disturbance

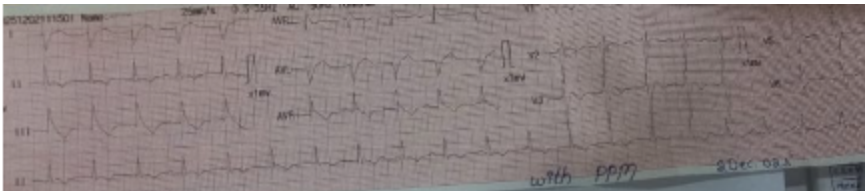


Figure 3

ECG after Permanent Pacemaker Implantation showing ventricular pacing spikes preceding widened paced QRS complexes, confirming pacemaker capture and restoration of hemodynamically effective rhythm.

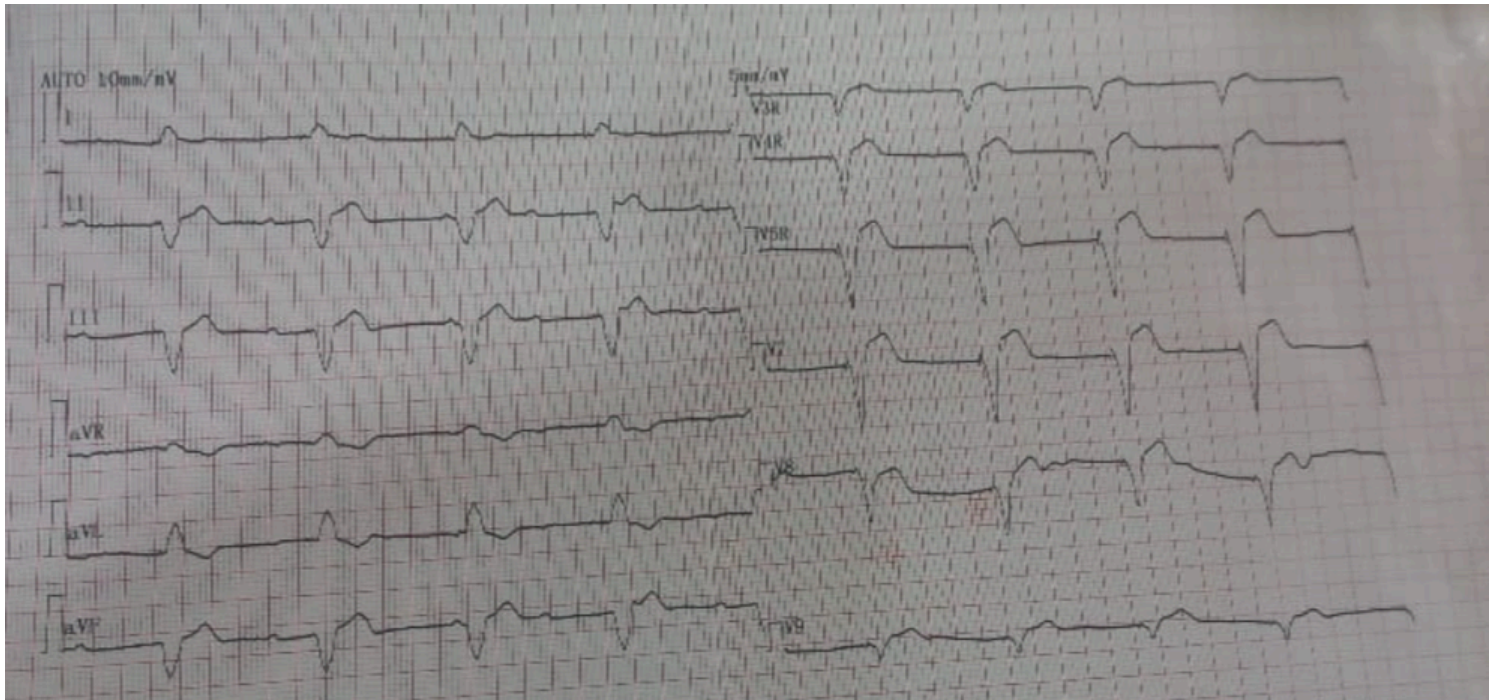


Figure 4

Coronary Angiography demonstrating normal LAD with no obstructive stenosis and smooth vessel contours consistent with MINOCA



Figure 5

Coronary Angiography showing normal non-obstructive coronaries

