Case Report

Rare Occurrence of Syncope and Nodal Rhythm Caused by Left Anterior Descending Myocardial Bridge

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A 59-year-old male presented to our clinic with a one-year history of recurrent syncope, accompanied by chest pain, dizziness, and an urge to vomit. Each syncopal episode lasted less than one minute, with spontaneous recovery and no postictal confusion, distinguishing it from seizure activity. The chest pain, described as pressure-like and non-exertional, lasted approximately 20 minutes per episode. Previous neurologic evaluation, including brain computed tomography, was unremarkable, prompting a cardiac workup. The patient's medical history included hypertension and hyperlipidemia, managed with metoprolol 45.5 mg daily, acetylsalicylic acid 100 mg daily, and rosuvastatin 20 mg daily. He denied smoking, alcohol use, or allergies. On admission, vital signs were stable (blood pressure 135/85 mmHg, heart rate 45 bpm, respiratory rate 16 breaths/min, oxygen saturation 98%). An electrocardiogram (ECG) revealed a nodal rhythm at 45 bpm, characterized by narrow QRS complexes and absent P waves, consistent with a junctional escape rhythm (Figure 1A). Laboratory findings showed elevated troponin I of 911 ng/L (reference < 34.2 ng/L), indicating myocardial injury, while other tests, including electrolytes and renal function, were normal.

Coronary angiography was performed to investigate the cause of the syncope and troponin elevation. It revealed no obstructive coronary artery disease but identified a mid-left anterior descending (LAD) myocardial bridge with systolic compression, producing a characteristic "milking effect" (Figure 2).

This finding suggested that the bridge was dynamically compressing the LAD during systole, potentially reducing coronary flow. Optical coherence tomography (OCT) further confirmed a 9-mm intramural band encircling the vessel wall, providing detailed visualization of the bridge's anatomy (Figure 3).

The OCT images highlighted the extent of vessel compression and the surrounding myocardial tissue, aiding in diagnostic certainty. Echocardiography with strain analysis demonstrated normal left ventricular function (ejection fraction 60%) and no regional wall motion abnormalities, with left ventricular end-diastolic diameter of 48 mm (normal 42–58 mm), left atrial diameter of 35 mm, indexed left atrial volume of 28 mL/m² (normal < 34 mL/m²), no associated valvopathy, and global longitudinal strain of −20% (normal more negative than −18%). A 24-hour ECG Holter monitor, conducted post-angiography, recorded sinus rhythm (60–121 bpm) without nodal recurrence (Figure 1B).[1-4]

The clinical presentation—syncope, nodal rhythm, and troponin elevation—was attributed to bridge-related ischemia, possibly worsened by beta-blockade. He was switched to diltiazem 120 mg daily per guidelines[2] and discharged. At 4-week follow-up, symptoms had resolved, with normal sinus rhythm on ECG.

Our patient's presentation—chest pain, syncope, nodal rhythm (45 bpm), and troponin elevation (911 ng/L)—suggests an ischemic conduction disturbance from the 9-mm mid-LAD muscle bridge. While shorter than some symptomatic bridges (e.g., 20–30 mm),[5] its compression likely impaired perfusion to the anteroseptal myocardium or AV node. Beta-blockers, used for hypertension, may have suppressed sinus activity, unmasking a junctional rhythm.[6] Troponin elevation indicates injury beyond demand ischemia, possibly from endothelial dysfunction or microvascular effects.[7] The return to sinus rhythm on Holter monitoring post-intervention supports a transient ischemic etiology.

Literature on bridge-related bradyarrhythmias is limited, with few case reports linking myocardial bridging to syncope or conduction defects.[3,8-10] Similarities include ischemia-induced AV node involvement, as in Ishikawa et al.[3] (syncope and AV block) and den Dulk et al.[9] (paroxysmal AV block). Differences in our case are the shorter bridge length (9 mm vs. typically > 10 mm), transient nodal rhythm (vs. persistent block), and rapid resolution with diltiazem (vs. other therapies like nitrates). These findings highlight variable symptomatic thresholds influenced by depth or spasm.[7]

Guidelines from the European Society of Cardiology (ESC) on coronary anomalies note that symptomatic bridges may present atypically, advocating calcium channel blockers or nitrates over beta-blockers in such cases.[2]

Our workup — angiography, OCT, strain echocardiography, and Holter monitoring — excluded alternative causes (e.g., coronary artery disease, intrinsic conduction disease). The 9-mm length challenges thresholds for symptomaticity (typically > 10 mm),[5] suggesting that depth or dynamic factors (e.g., spasm) may contribute.[7] Switching to diltiazem, which reduces myocardial oxygen demand without risk of bradycardia, aligned with ESC recommendations and resolved symptoms.[2]

This case highlights myocardial bridging as a differential diagnosis in syncope with cardiac markers, often overshadowed

Keywords

Myocardial Bridging; Syncope; Heart Rate.

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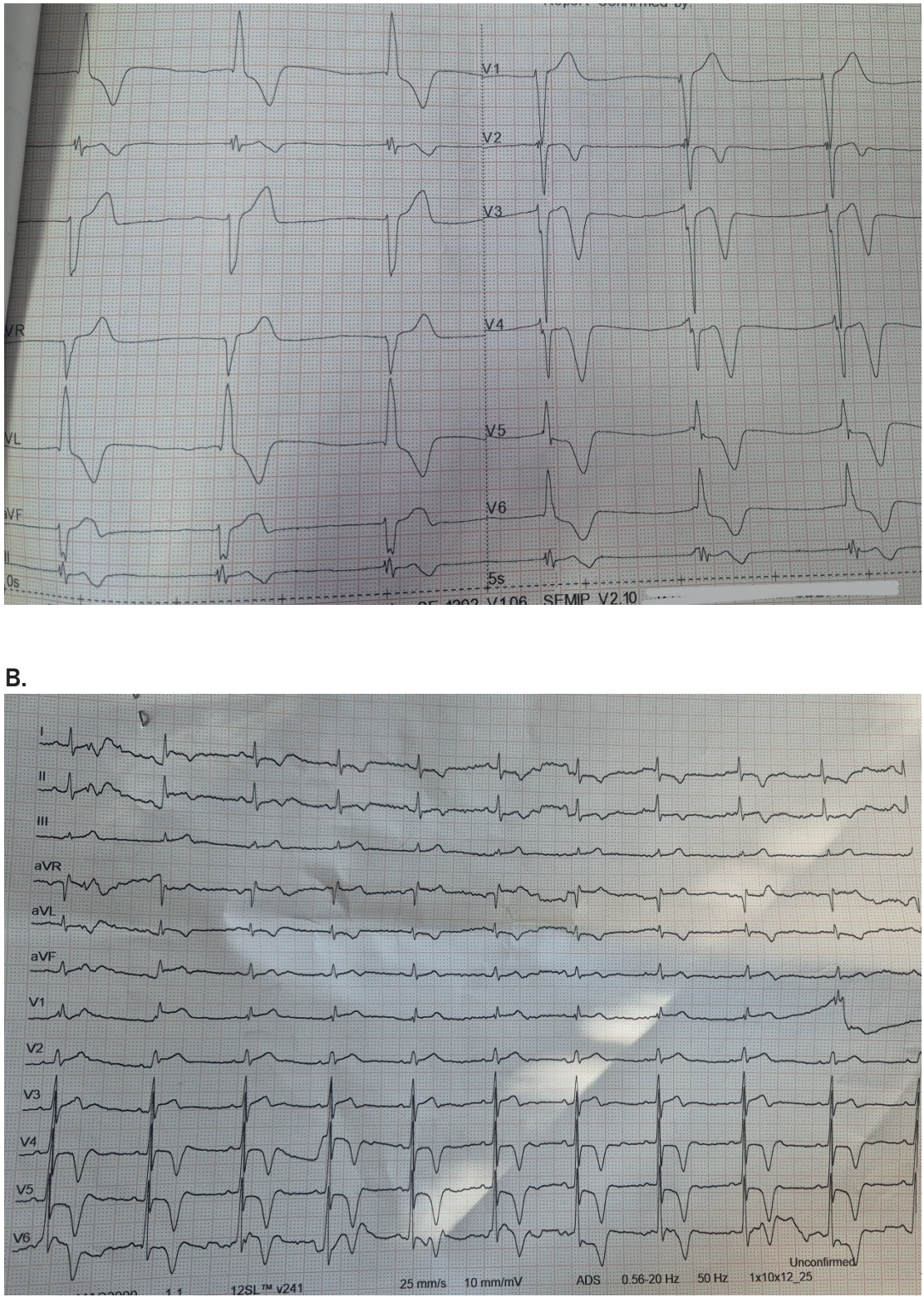


Figure 1 – *(A) ECG on admission showing nodal rhythm at 45 bpm; (B) ECG before discharge showing sinus rhythm.*

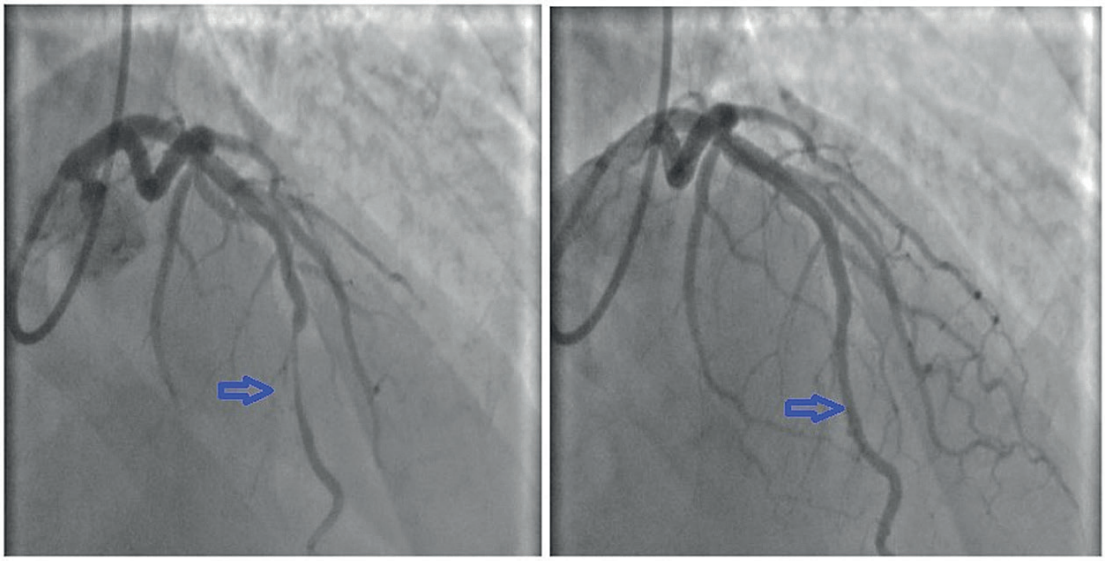


Figure 2 – *Coronary angiography demonstrating mid-LAD myocardial bridge with systolic compression and "milking effect".*

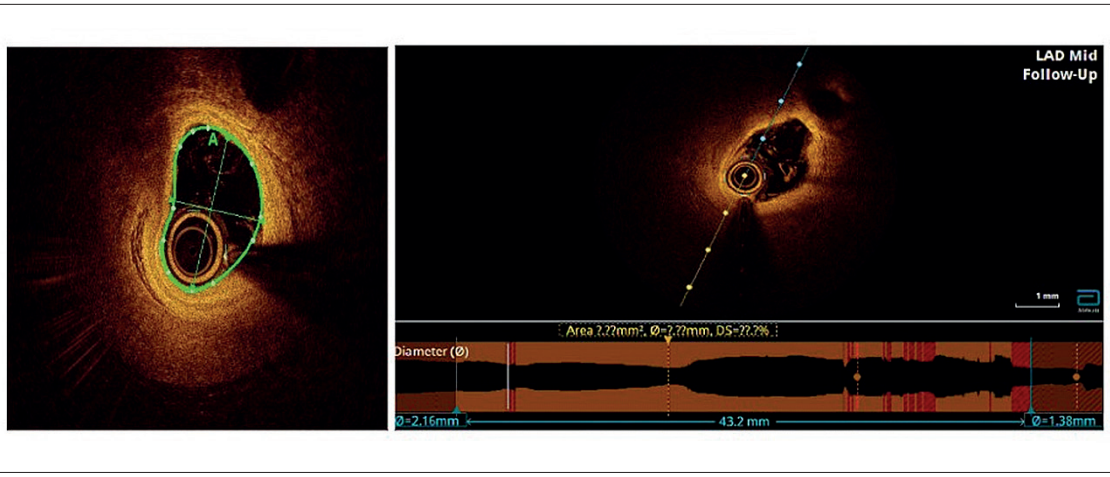


Figure 3 – *OCT confirming a 9-mm intramural band in the LAD.*

by neurologic or arrhythmic causes.[10] Limitations include the absence of stress testing to quantify ischemia and the bridge's modest length. Future studies could assess the prevalence of bridge-related bradyarrhythmias and refine risk stratification with tools like intravascular ultrasound (IVUS) or fractional flow reserve.[8]

Conclusion

We describe a 59-year-old man with syncope and nodal rhythm due to a 9-mm mid-LAD myocardial bridge, marked by chest pain and troponin elevation. This rare presentation underscores the ischemic potential of myocardial bridging beyond angina, urging its consideration in unexplained syncope and arrhythmias. Tailored therapy with calcium channel blockers proved effective, supporting guideline-directed management. OCT and IVUS can help in evaluation of coronary muscle bridges and determine the correct treatment strategy.

Author Contributions

Conception and design of the research, statistical analysis, writing of the manuscript and preparation of the figures: Kandic E; acquisition of data: Kandic E, Vrajnko E; analysis and interpretation of the data: Shopov B; critical revision of the manuscript for intellectual content: Kandic E, Biljana Zafirovska Taleska; literature review: Vrajnko E; final approval of the manuscript: Kandic E, Taleska BZ.

Potential conflict of interest

No potential conflict of interest relevant to this article was reported.

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Study association

This study is not associated with any thesis or dissertation work.

Ethics approval and consent to participate

This article does not contain any studies with human participants or animals performed by any of the authors.

Use of Artificial Intelligence

The authors did not use any artificial intelligence tools in the development of this work.

Data Availability Statement

The data cannot be made publicly available.

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