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CASE REPORT

Coexistence of Coronary Artery Ectasia and Myocardial Bridging: A Case Report

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Abstract: Ectasia is the term used to describe the dilation of a coronary artery when its diameter is 1.5 times that of a normal artery. Myocardial bridge (MB) is a congenital coronary abnormality where a coronary artery segment tunnels across myocardial tissue, with coronary angiography (CAG) is the gold standard method for diagnosis. Here, we describe a case of an Afghan man who had both coronary artery ectasia and MB in the same segment of his left anterior descending artery (LAD).

Keywords: coronary artery ectasia, myocardial bridge, coronary angiography, case report

Introduction

The hallmark of coronary artery ectasia (CAE) is a coronary artery diameter greater than 1.5 times the greatest diameter of a nearby normal artery. According to several researches, the prevalence of CAE varies from 1.2% to 7.4%, making it an uncommon illness. It primarily affects men, with about 90% of them being affected. Although not in every patient, risk factors are comparable to atherosclerosis.[1–3]

Myocardial bridging (MB) is a cardiac abnormality in which a section of the coronary artery crosses the myocardium. To determine which patients benefit from treatment, functional imaging and hemodynamic characterization are required.[4–6] The clinical implications of myocardial bridges and slow coronary flow remain uncertain.[7] The gold standard for diagnosing MB can be coronary angiography (CAG),[8] and the left anterior descending artery's proximal region is the area where it is mostly found.[9]

Research has indicated that symptoms including dyspnea and chest discomfort are worsened in people with MB who have other cardiovascular risk factors (such as smoking, age, sex, occupation, and ethnicity).[10] Isolated MB had no effect on mortality, myocardial infarction (MI), effort-induced angina, or positive stress tests for ischemia in 99% of patients. Seldom seen in MB, acute MI and resting angina with electrocardiographic (ECG) abnormalities are primarily caused by persistent spastic constriction at the site, which may be exacerbated by luminal secondary clotting.[11] Though, asymptomatic patients with MB are common.[12] On the other hand, it has been linked to myocardial infarction, syncope, arrhythmias, angina, coronary spasm, sudden cardiac arrest, and mortality.[13] We describe a rare instance of circumflex coronary artery ectasia and slow-flow left anterior descending artery ectasia that is marked by the presence of anterior descending artery (LAD) myocardial bridging.

Case Presentation

A 27-years-old male dentist presented with palpitation for 3 years, the patient reported a sudden onset of palpitations, which were not accompanied by dizziness and primarily occurred during physical activity or emotional events. These episodes lasted for about 10 to 15 minutes and resolved spontaneously. Although the primary complaint appeared to be cardiac in nature, neurological causes such as seizures, autonomic disorders, or anxiety-related conditions were also considered in the differential diagnosis to accurately identify the underlying cause of the palpitations. On physical examination, heart sounds

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(S1, S2) were normal, and no murmurs were detected. Also he had exertional dyspnea for more than 2 years. It is not at ordinary activity and at rest, stress angina for 2 years is increased by physical activity and emotional events and weakness from one year. The clinical examinations found a conscious patient, heart rate is 63, regular beats per minute at rest during exam, respiratory rate is an 18 cycle per minute at rest, temperature is 37 degrees of Celsius, blood pressure is 115/70 mmHg at sitting position and the oxygen saturation was 97% in room temperature. Additional examinations at fasting time had the following results: renal function test creatinine: 0.9 mg/dl, blood sugar: 93 mg/dl, triglyceride: 140 mg/dl and cholesterol: 110 mg/dl. All the 12 leads resting electrocardiography show the following views: T inversion in V1, mild ST elevation in V2-V3 of chest leads but other leads are normal. The ST elevation in V2-V3 leads are shown in Figure 1. Transthoracic echocardiography report was performed, showing normal global and segmental biventricular systolic function (ejection fraction at 68%), atrial and ventricular chambers are in normal size and shape, and there is no hypertrophy, all four valves are in normal structure there are no stenosis and regurgitation, pulmonary artery systolic pressure is normal (25 mmHg), there is no evidence of hypokinesia, akinesia and dyskinesia at left and right ventricular and there is no sign of congenital anomaly. Coronary angiography was preformed showing coronary arteries are without stenosis with a myocardial bridge of left anterior descending and ectasia of left anterior descending and circumflex arteries with slow flow. The myocardial bridge and ectasia during systole is shown in Figure 2, also they are shown during diastole in Figure 3. The patient was monitored over a two-

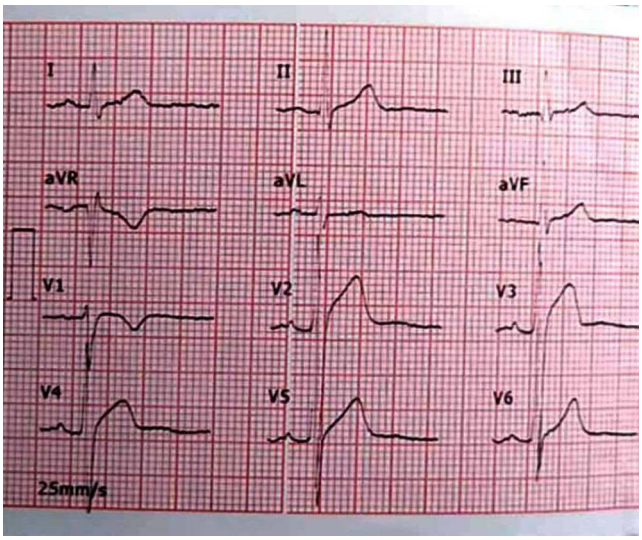

Figure 1 Electrocardiogram (ECG) demonstrating mild ST elevation in leads V2-V3.

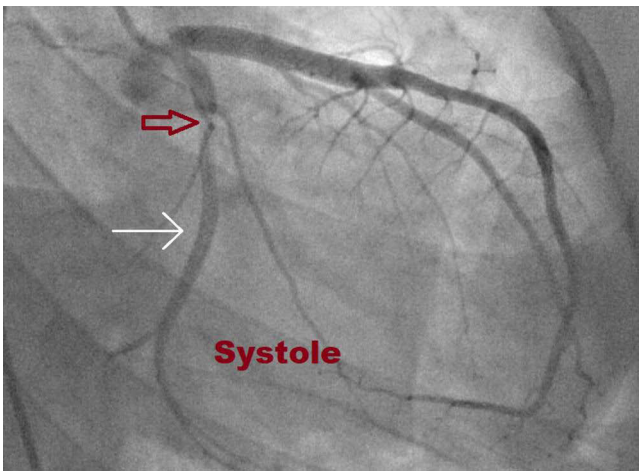

Figure 2 Shows myocardial bridge (red arrow) and coronary artery ectasia (white arrow) at the left anterior descending coronary artery during systole on coronary angiography and the angle of angle of the angiographic image is left anterior oblique with cranial tilt.

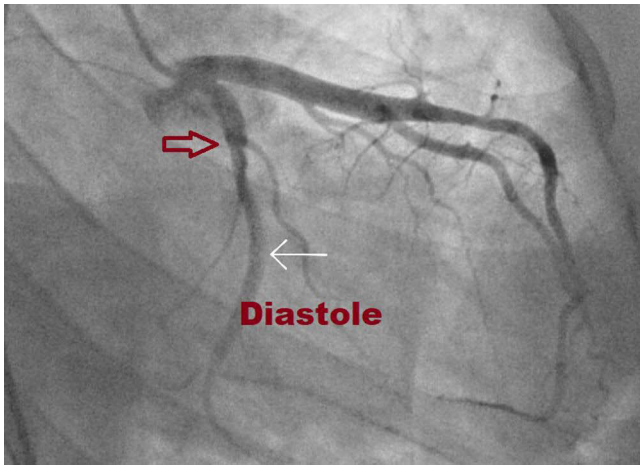

Figure 3 Shows myocardial bridge (red arrow) and coronary artery ectasia (white arrow) at the left anterior descending coronary artery during diastole on coronary angiography and the angle of angle of the angiographic image is left anterior oblique with cranial tilt.

month period following the initial diagnosis, during which no recurrence of symptoms or evidence of ischemia was observed. Regrettably, extended follow-up beyond this period was not feasible due to limited patient accessibility and broader healthcare system constraints. Despite this limitation, the short-term clinical course remained stable, providing valuable insight into the early management of this rare coexistence. As the patient received optimal non-dihydropyridine calcium channel blockers (CCBs), antiplatelet agents and high intensity statin, the patient's clinical sign and symptoms were improved.

Discussion

A coronary artery segment that dilates to more than 1.5 times the diameter of nearby normal coronary segments is known as coronary artery ectasia.[6] Morgagni first described CAE in 1761. The frequency of CAE, a very uncommon coronary angiographic finding, varies by area and ranges from 1.5% to 5%.[1]

The majority of patients with myocardial bridging (MB) are young men, and the condition is often limited to the middle LAD, with a length of 1 to 5 cm,[9] and systolic compression of the affected epicardial coronary artery is the primary angiographic finding. A distinctive diastolic flow disruption has been reported by intravascular ultrasonography, intracoronary Doppler investigations, and quantitative coronary angiography. The location, thickness, length, and degree of cardiac contractility of the muscle bridge all affect how much the myocardial bridge blocks the coronary arteries. According to coronary angiography, the stated estimated frequency ranges from 1.5 to 16%; nevertheless, in certain autopsy series, it can reach 80%.[14]

Since actual diagnostic accuracy metrics have not been fully investigated, diagnostic procedures present a difficulty because the majority of patients remain asymptomatic throughout their lives. Noninvasive and invasive techniques are included in the two diagnostic criteria. Stress echocardiography and cardiac computer tomography angiography are the noninvasive techniques. Coronary angiography, the most invasive and widely used diagnostic technique, shows the systolic compression of artery segments as well as the distinctive milking effect, or the decompression of vessels during diastole. The pathogenic consequences of bridging on hemodynamics and flow may be better understood with the use of more recent techniques such as intravascular ultrasonography, intracoronary Doppler, and fractional flow reserve evaluation.[15]

Beta-blockers and non-dihydropyridine calcium-channel blockers are the first-line treatments for patients believed to be exhibiting symptoms due to myocardial bridging.[16] By increasing systolic compression of the bridged segment and vasodilating segments proximal to the bridge, nitrates can exacerbate symptoms by exacerbating retrograde flow in the proximal segment and lowering the myocardial ischemic threshold, even though they have antispasmodic qualities and can lower preload. Therefore, unless there is a severe concomitant coronary vasospasm, vasodilators should be avoided.[12]

Both medicinal and surgical management are used to treat MB. For patients with MB symptoms, pharmacological treatment with beta blockers or calcium channel antagonists is advised as first-line therapy.[17] If maximal medical therapy

fails to alleviate symptoms, revascularization should be considered. Both surgical (CABG and myotomy) and percutaneous coronary intervention (PCI) with stent implantation are options for revascularization. The length and depth of myocardial bridging also influence the choice of revascularization technique. To determine the most effective treatment strategy for patients with myocardial bridging, more study is needed.[5]

Surgical myotomy and coronary artery bypass graft surgery are two surgical procedures for myocardial bridging. Patients with refractory symptoms despite medical therapy should be the only ones who undergo surgical myotomy, which involves resection of the overlying muscle fibers. This is especially true for those who have demonstrated inducible ischemia and are at high risk for myocardial infarction, ventricular tachycardia, or resuscitated cardiac arrest.[16]

Previous reports describing the coexistence of CAE and MB within the same coronary segment are exceedingly limited. The first documented case was reported by Çağlar et al, in which an elderly male patient presenting with acute coronary syndrome was found to have ectasia and a severe myocardial bridge involving the mid-left anterior descending artery on invasive coronary angiography. In that case, the authors postulated that superimposed atherosclerotic changes on a congenital myocardial bridge might have contributed to the development of ectasia. More recently, Czepe et al described a similar coexistence detected incidentally by coronary computed tomography angiography (CCTA) in a female patient without significant atherosclerotic disease, highlighting the diagnostic value of advanced non-invasive imaging in characterizing both anatomical abnormalities simultaneously. Compared with these reports, the present case further supports the notion that CAE and MB may coexist independent of obstructive coronary artery disease and that their pathophysiological interaction may vary according to patient characteristics and underlying etiology. Collectively, these limited cases underscore the rarity of this association and suggest that multimodality imaging plays a crucial role in elucidating the anatomical and clinical significance of concurrent CAE and MB, warranting further investigation in larger cohorts.

Conclusion

The rare coexistence of coronary artery ectasia (CAE) and myocardial bridging (MB) in the same left anterior descending artery segment is illustrated in this case. This report is the third recorded case of contemporaneous CAE and MB in a single coronary segment, suggesting that this coexistence can occur even in the absence of obstructive coronary artery disease and may manifest clinically in younger people. Multivessel ectasia indicates a possible more extensive vascular problem rather than just an anatomical variance. These results underline the significance of thorough angiographic evaluation and tailored clinical monitoring for patients with combined coronary anomalies, as well as the need for larger studies to clarify the associated pathophysiological mechanisms and long-term effects of this rare condition.

Consent to Publish

Institutional approval for publication of this case report was obtained from Khatam Al-Nabieen Research Ethics Committee, and the ethics approval code is (AF, knu.edu.af.rec 17, 2025-02-10). The patient gave written informed consent for this case report and its associated images to be published.

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Disclosure

The authors report no conflicts of interest in this work.

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