CASE REPORT

Myocardial bridge: bridging the differential diagnosis

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SUMMARY

A 50-year-old man with a history of vasospastic angina diagnosed 3 months prior presented with recurrent episodes of substernal chest pain. His chest pain was characterised as 'squeezing', lasting 10 min and typically occurred on awakening. Medications included isosorbide, nitroglycerin and ranolazine; however, due to lack of insurance he was unable to obtain these medications. On admission, patient was given a full-dose aspirin and nitroglycerin, which acutely worsened his chest pain. ECG did not reveal ischaemic changes and initial troponin was negative. A left heart catheterisation was conducted, revealing a myocardial bridge of the mid-left anterior descending artery. Myocardial bridge, due to its rarity, is often overlooked as an aetiology for angina, myocardial ischaemia, acute coronary syndrome, syncope and cardiac death. This case highlights the importance of broadening the differential to include myocardial bridge in the work-up for chest pain, especially if nitrates worsen symptoms.

BACKGROUND

A myocardial bridge is an anatomical variant of the coronary arteries, first described by Reyman in 1737.¹ It is characterised by an epicardial coronary artery tunnelling under the myocardium, causing compression of this segment during systole.^{2,3} Before the advent of intravascular imaging, myocardial bridges were only identified at time of autopsies. However, technological advances in coronary angiography and intravascular ultrasound have led to increased reporting and better understanding of the pathophysiological process involving this disorder.

The reported prevalence of myocardial bridges from angiography ranges from 0.5% to 12%.³ Most commonly it is localised to the middle segments of the left anterior descending (LAD) coronary artery with 18% associated with diagonal branches and 40% associated with marginal branches.²⁻⁴ Rarely, it is detected in the right coronary or left circumflex arteries.⁴

Myocardial bridges produce clinical manifestations by three hypothesised methods: (1) direct compression of the tunnelled coronary artery via myocardial contraction, (2) accelerated atherosclerosis in the proximal segment relative to the myocardial bridge and (3) 'branch-steal' phenomenon of systolic retrograde flow and overall flow reduction.⁴ As a result, multiple case reports suggest symptomatic myocardial bridges may cause angina, myocardial ischaemia, acute coronary syndrome, coronary spasms, exercise-induced dysrhythmias,

transient ventricular dysfunction, syncope and even sudden cardiac death.⁵ Appreciation of this under-recognised phenomenon can lead to appropriate treatment; but more importantly, as seen in this case, prevent exacerbation of symptoms which can be life threatening.

CASE PRESENTATION

The patient is a 50-year-old man who presented with chief complaint of recurrent substernal chest pain. Onset of this chest pain began approximately 4 months prior, described as 'squeezing' pain over the left thorax, rated as 10/10, non-radiating and each episode lasting approximately 10 min. Associated symptoms included intermittent dyspnoea and diaphoresis. Patient denied any alleviating or exacerbating factors and pain was irrespective of exertion. On admission, his chest pain was worse than previous, causing him to have an unwitnessed syncopal episode. He denied any fever, chills, cough, palpitations, abdominal pain, chest trauma, tongue biting, urinary incontinence, sick contacts or travel. Of note he was admitted to an outside hospital 3 months prior with similar chest pain and was diagnosed with vasospastic angina. Work-up at that time included routine lab work, serial troponin, a baseline ECG and cardiac CT. He was discharged on isosorbide, nitroglycerin and ranolazine; however, due to lack of insurance, the patient did not fill these medications. He denied any tobacco, alcohol and intravenous drug use. He was not currently sexually active, was previously married and had started a new desk job at an insurance company a week prior.

On admission, the patient was haemodynamically stable. Physical examination revealed regular heart rate, no murmurs, rubs or gallops. Lungs were clear to auscultation and there was no tenderness to palpation over chest wall. Pertinent laboratory findings included brain natriuretic peptide (BNP) test 19.5 pg/mL, troponin <0.10 ng/mL, creatinine phosphokinase (CPK) level 118 U/L, thyroid stimulating hormone (TSH) 1.28 IU/mL, normal white blood cell count and renal function. ECG demonstrated normal sinus rhythm with no acute ischaemic changes (figure 1). The patient was given full-dose aspirin 325 mg and nitroglycerin 0.4 mg sublingual and transferred to telemetry floor. Shortly after, the patient developed worsening, crushing chest pain. Telemetry strip was unchanged.

INVESTIGATIONS

Orthostatic vitals did not meet criteria for orthostasis—supine blood pressure: 117/75, heart rate 99 and standing blood pressure 123/79, HR 100.

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Figure 1 Admission ECG revealing normal sinus rhythm, right axis deviation due to left posterior fascicular block. No acute ST segment elevation or depression.

Complete routine laboratory work-up was essentially within normal limits. Chest radiograph did not reveal acute cardiopulmonary pathology. No consolidation, pleural effusion or cardiomegaly was noted. Serial troponin x3 every 8 hours were within normal limits. Serial ECGs with chest pain did not reveal acute ischaemic changes. Echocardiogram demonstrated left ventricular ejection fraction 55%–60%, normal chamber sizes and trace aortic and tricuspid regurgitation. The cardiac CT report was obtained from outside hospital, indicating 30% stenosis of proximal LAD artery and tortuous left coronary arterial supply. Since the cardiac CT images were not obtained, the patient underwent diagnostic left heart catheterisation (LHC) revealing two myocardial bridges of the LAD artery with mild spasms that were not inducible with hyperventilation or intravenous phenylephrine.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis for substernal chest pain is broad, involving multiple organ systems including cardiac, pulmonary, gastrointestinal, musculoskeletal and endocrine. History and physical examination is essential in narrowing this differential. Furthermore, laboratory abnormalities can aid in supporting a clinical finding. In this case, life-threatening aetiologies such as pulmonary embolism, aortic dissection, acute coronary syndrome, pericardial tamponade and mediastinitis were ruled out as they did not correlate with the clinical picture. Benign causes of chest pain such as pneumonia, bronchitis, pericarditis, gastrointestinal reflux, pancreatitis, biliary colic, costochondritis, muscle strain and hyperthyroidism were also excluded.

Interestingly, the patient was diagnosed with vasospastic angina 3 months prior and was prescribed nitrates to relieve his pain. Based on review of his outside records and monitoring on telemetry while he was hospitalised, there was no evidence of transient ischaemic ST changes during his episodes of chest pain. In addition, coronary artery spasms were not induced by hyperventilation or intravenous phenylephrine during the LHC. Finally, the administration of nitrate made patient's chest pain significantly worse, which argued against the diagnosis of vasospastic angina based on current criteria.⁶

The LHC revealed two myocardial bridges of the mid-LAD artery with at least 70%–80% stenosis during systole, which was thought to be the aetiology of the patient's symptoms (figure 2).

The patient's syncopal episode was thought to be due to a vasovagal response secondary to pain. The differential for syncope includes orthostasis, cardiac arrhythmias, cardiac valvular disease and carotid hypersensitivity. The patient did not demonstrate a positive response for orthostasis, defined as a drop in systolic blood pressure >20 mm Hg from supine to standing position.⁷ Echocardiogram did not reveal significant valvular disease and telemetry did not display significant arrhythmia.

TREATMENT

The cardiology service was consulted during hospitalisation. Patient was monitored on telemetry and treated symptomatically with low-dose beta-blockers.

OUTCOME AND FOLLOW-UP

Patients' current medications of isosorbide, nitroglycerin and ranolazine were discontinued. Subsequently, he was started on aspirin 81 mg daily, atorvastatin 40 mg daily and metoprolol tartrate 12.5 mg twice daily. Follow-up with cardiology was arranged. He was compliant with his medications and described improvement in intensity and frequency of his chest pain.

DISCUSSION

Though a true gold standard for diagnosing myocardial bridges is lacking, coronary angiography remains the most common technique. Angiographic diagnosis depends on the presence of >70% reduction in the minimal luminal diameter during systole and persistent, >35% reduction in minimal luminal diameter during mid-to-late diastole.^{4,5} This finding is often referred to as the 'milking effect'.⁵ Adjuvant interventional modalities, such as intravascular ultrasound, can aid in diagnosis with the characteristic finding of the 'half-moon' sign, an echolucent area present between the bridged coronary segment and epicardial tissue that persists throughout the cardiac cycle.⁵ Furthermore, measurements using fractional flow reserve can be used to evaluate haemodynamically significant stenosis of the myocardial bridge.⁵ Non-invasive diagnostic methods most commonly include multiple-slice cardiac CT as it provides both structural and physiological assessment of the myocardial bridges.⁵

Figure 2 Left heart catheterisation in right anterior oblique, caudal view. (A) Diastolic imaging revealing patent left anterior descending (LAD). (B) Systolic imaging revealing two areas of transient stenosis (arrows) of the mid LAD.

In this case, a previous cardiac CT was completed but the report did not comment on a myocardial bridge and the images could not be obtained for independent review. Therefore, coronary angiography was elected to definitively assess severity of vasospasm. Given no evidence of vasospasms on angiography, no transient ischaemic ST changes during chest pain and nitrates worsening chest pain, it was concluded that the patient was misdiagnosed with vasospastic angina. One limitation to this case is that it did not assess haemodynamic significance of the myocardial bridges. This could have been attempted using myocardial perfusion imaging or quantitative coronary angiography.⁸ The presence of ischaemia, which is attributed to degree of systolic narrowing, can then guide medical therapy by using the Schwarz classification criteria.⁸ Instead, it was decided to treat the patient with close follow-up to determine symptom improvement.

The pathophysiology of symptomatic myocardial bridge is essential to understand given the diverse, adverse clinical events described previously.⁹ There is a growing consensus that the onset of the clinical factors related to myocardial bridge is due to a complex set of physiological factors, categorised by intrinsic and extrinsic factors, rather than by simple anatomy.⁴ Extrinsic factors include epicardial arterial compression, sympathetically driven myocardial contraction and coronary vasoconstriction.⁴ Intrinsic factors include interval interactions among aortic pressure, diastolic flow and perfusion time, and transmural perfusion gradients. Impaired diastolic flow can result in a branch steal phenomenon creating a depressurisation of septal

branches leading to an intrabridge decrease in perfusion.⁴ This results in high systolic wall shear stress proximal to myocardial bridge entrance due to the collision of retrograde and antero-grade perfusion leading to constrictive vascular remodelling and early atherosclerosis. Another proposed mechanism of plaque formation proximal to the myocardial bridge is from the heterogeneous stress fields resulting from the motion of severe vessel angulation.⁵

The management of symptomatic myocardial bridges consists of beta-blockers as they relieve haemodynamic disturbance by reducing heart rate, improving diastolic coronary filling and reducing contractility/compression of the coronary artery.^{5,9} Additionally, non-dihydropyridine calcium channel blockers are beneficial in instances of coronary vasospasm due to their vasodilatory effects.^{5,9} Ranolazine, which inhibits cardiac late sodium channel and thus improves myocardial relaxation and perfusion, can be used as monotherapy or in combination with first-line therapies.⁴ Nitrates are contraindicated as they exacerbate symptoms by reducing intrinsic coronary wall tension and subsequently increase reflex sympathetic stimulation of contractility.³ In the setting of atherosclerosis, antiplatelet and statin therapy is prudent to reduce atherosclerotic burden.³ A recent meta-analysis revealed that in patients refractory to medical therapy surgical treatments (ie, myotomy or coronary artery bypass) appeared to be more effective in controlling angina symptoms compared with percutaneous coronary intervention (PCI) stent

Reminder of important clinical lesson

placement.⁹ Stent implantation had lower efficacy due to its high restenosis rates.⁹

With this case, we hope to highlight the importance of including myocardial bridge in the differential diagnosis of acute chest pain. Furthermore, future areas of studies relating to this pathology are necessary to obtain a better understanding of its clinical significance. Primarily, it would be beneficial to know the prevalence of symptomatic myocardial bridges. This would allow appropriate awareness when formulating a differential diagnosis if prevalence was significant. Finally, though beta-blockers remain first-line therapy, no large-scale study has been conducted to determine the clinical endpoints of morbidity or mortality.

Learning points

- ▶ Myocardial bridge should be considered on the differential diagnosis of substernal chest pain (angina).
- ▶ Clinical manifestations of symptomatic myocardial bridge are due to direct compression of tunnelled coronary artery, accelerated atherosclerosis of proximal segment prior to the bridged artery or 'branch-steal' phenomenon of systolic retrograde flow.
- ▶ Mainstay treatment includes beta-blockers and non-dihydropyridine calcium channel blockers.
- ▶ Nitrates can acutely worsen symptoms produced by myocardial bridges.

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