

# Myocardial Bridge Syndrome: A Case Report and Literature Review

Mei-lian Cai (✉ [1142653454@qq.com](mailto:1142653454@qq.com))

The Second Affiliated Hospital of Guilin Medical University <https://orcid.org/0000-0003-1333-7801>

Wei Zhang

Guilin First People's Hospital <https://orcid.org/0000-0002-7388-0796>

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## Case Report

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# Abstract

**Background:** Myocardial bridging (MB) is a congenital coronary artery malformation. Various clinical symptoms related to MB have been reported, however, subxiphoid pain has rarely been reported. Moreover, it is rarer for electrocardiography (ECG) to show such obvious ischaemia in patients with MB.

**Case presentation:** We report a 36-year-old man who presented with subxiphoid pain. Electrocardiography showed ST segment depression and T-wave inversion with symmetry in leads V2 to V6. Emergency coronary angiography (CAG) confirmed that it was caused by MB.

**Results:** CAG showed MB of the left anterior descending (LAD) artery with a 1.5 cm intramural segment, and that the vascular lumen was compressed 80% during systole. At the same time, gastroscopy showed peripyloric and antral ulcers. The patient received calcium channel blockers (CCBs) and peptic ulcer-related drug treatment then, the symptoms disappeared.

**Conclusion:** The ECG showed obvious myocardial ischaemia. MB may have been one of the reasons for ischaemia. When the heart contracts, the MB compresses the mural coronary artery and further narrows its lumen. The longer and thicker the MB is, the greater the angle between myocardial fibres and blood vessels, the more severe the mural coronary artery stenosis, the more severe the distal myocardial ischaemia.

## Case Report

A 36-year-old man presented with subxiphoid pain for 3 days that was aggravated for 13 hours on July 3, 2021. Three days prior, the patient suffered from subxiphoid pain that was related to his daily activities and the pain was a paroxysmal distending pain that was located in the lower 2 transverse fingers of the subxiphoid area and lasted from a couple of seconds to 5 minutes with no connection to eating. The symptoms were relieved after rest, and they were not accompanied by palpitations, shortness of breath, dizziness, acid regurgitation, belching, or haemoptysis. He did not receive treatment in the previous three days. The symptoms worsened 13 hours prior. The patient went to the emergency department for treatment. He underwent electrocardiographic examination. The electrocardiography results showed the following: sinus rhythm, left ventricular high voltage, ST segments with significant depressions on the V2 to V6 leads, and T-wave inversions with symmetry (Fig. 1a). Preliminary diagnosis: Coronary heart disease (CHD), Acute coronary syndrome (ACS)? He was transferred immediately from the emergency department to our department for emergency coronary angiography (CAG). He had smoked one pack a day for 10 years. He denied the use of any alcohol and did not have other known chronic diseases. There were no similar diseases or any other genetic history of disease in his family.

The patient's vital signs were normal upon admission. On physical examination, the patient's body temperature was 36.5°C, pulse rate was 59 beats/min, respiratory rate was 20 breaths/min, blood pressure was 113/73 mmHg, weight was 66 kg, and height was 1.70 metres. The patient had a clear mind, clinostatism, and no cyanosis of the lips. The abdomen was flat and soft, with slight tenderness in

the subxiphoid, without rebound pain, and the bowel sounds were normal. No other unusual findings were present on his physical examination.

Admission diagnosis: The causes of chest pain that needed to be investigated were as follows: CHD, acute coronary syndrome (ACS) and gastritis. Emergency CAG showed that there was MB of the left anterior descending (LAD) artery with a 1.5 cm intramural segment, the vascular lumen was compressed 80% during systole, and there was a TIMI glow of grade 3 (Fig. 2a–2b). No plaques were observed, occlusion was observed in the left circumflex artery (LCX), and the retention of contrast medium was observed. No plaques were observed in the right coronary artery and the bloodstream was unobstructed. The patient refused intravascular ultrasound (IVUS) and fractional flow reserve (FFR) evaluation. Auxiliary examination results from the emergency department showed no detectable changes in the abdominal colour ultrasound, standing abdominal plain film, myocardial enzymes, myoglobin, serum troponin T, routine blood tests, coagulation function, liver and kidney function, serum glucose, or electrolytes. After CAG, re-examination of myocardial enzyme and serum troponin T were normal. The electrocardiogram (ECG) showed no change from before. No abnormalities were found in thyroid function, glycosylated haemoglobin, or echocardiography. Gastroscopy showed peripyloric and antral ulcers, and antral biopsy showed moderate chronic nonatrophic gastritis with erosion. The carbon 14 exhale test was negative.

Definite diagnosis: 1. Myocardial bridge syndrome (the proximal of the left anterior descending) 2. Peripyloric and antral ulcers. During his hospitalization, he asked the Gastroenterology Department and cardiothoracic surgery for consultation. The patient was treated with diltiazem (90 mg, qd), indobufen (100 mg, bid), atorvastatin (20 mg, qn), pantoprazole (20 mg, q12 h), rebaperate (100 mg, tid), and Kangfuxin (10 ml, tid). He was advised to quit smoking. He refused to undergo surgical unroofing. After 7 days, the patient's subxiphoid pain disappeared. Re-examination of the ECG indicated sinus rhythm, left ventricular high voltage, less ST segments with significant depressions on leads V2 to V6, and less T-wave inversions with symmetry (Fig. 1b). The patient improved and was discharged on July 14, 2021.

## Discussion And Conclusions

The patient suffered from subxiphoid pain with connection to daily activities, and ECG showed evident myocardial ischaemia. Therefore, he was diagnosed with ACS. In addition, similar ECG changes were seen for MB and hypertrophic cardiomyopathy (HCM). Echocardiography showed normal results, which excluded HCM.

MB is a congenital coronary artery malformation that was discovered 100 years ago. It was first reported by Cranicianu in 1922 [1]. Myocardial bridging is a common finding (5%-86%) depending on the imaging modality/autopsy series [2]. The detection rate of coronary angiography (CAG) is 2%-6%, that of coronary computed tomography angiography (CCTA) is 19%-22%, and that of autopsy is 33%-42%. The CCTA data are closer to the autopsy data. Therefore, CCTA is more sensitive than CAG, and intravascular imaging and can be used for general diagnosis [3]. The middle segment of the left anterior descending

coronary artery is the most frequent location of MB; however, bridging on the circumflex branch of the left coronary artery and right coronary arteries has also been reported [4]. Cai M et al. found that a case of MB was located in the second right ventricular branch of the right coronary artery [5].

The current methods to diagnose myocardial bridges include CAG, CCTA, IVUS, and cardiac magnetic resonance imaging (MRI). CAG shows compression of the artery caused by the MB with narrowing of the lumen and diastolic relaxation. CAG clearly shows signs of coronary artery ischaemia compressed by myocardial bridges during systole, while the coronary artery compression is relieved during diastole. The haemodynamics of a bridging segment may be assessed with the mean FFR measurement. Compared with conventional coronary angiography, computed tomography (CT) enables direct visualization of coronary arteries, including the surrounding tissue, and thus allows for the depiction of tunnelled segments even when there is only minimal or no systolic compression and no change in the vessel course [6]. The depiction rate of MB is greater with 64-section CT coronary angiography than with conventional coronary angiography. The degree of systolic compression of MB significantly correlates with the tunnelled segment depth but not the length [6]. MRI shows the relationship between the mural coronary artery and MB and judges whether the coronary artery blood supply function at the MB is abnormal through the functional imaging of the MB.

MB usually has a benign prognosis; however, it can lead to dangerous complications, including ischaemia and acute coronary syndromes, coronary spasm, ventricular septal rupture, arrhythmias, coronary thrombosis, ventricular dysfunction and sudden death [7]. YoungJae Ki reported a rare case of MB presenting as myocardial ischaemia-induced cardiac arrest, and he highlighted MB as one of the important causes of sudden cardiac death. They considered vasospasm, which was confirmed by a transient ST elevation on ECG, to be a trigger factor. The patient survived [8]. In addition, some patients also have vascular endothelial dysfunction and microvascular disease complications, resulting in myocardial ischaemia. Some studies have shown a prevalence of MB of 28%- 50% in HC patients [9–12]. MB has been linked to sudden death, and patients with combined HCM and MB have a worse prognosis [12].

The direction of myocardial fibres of the myocardial bridge is at a nearly at a right angle to the long axis of the vessel at the anterior and posterior descending branches, while it is at a smaller angle at the anterior right ventricular branch and the anterior left ventricular branch. In the cross section of the mural coronary artery, the lumen is small, and the wall is thin, which is more obvious when the muscle bridge is thick. The mural coronary artery segment does not easily form atherosclerosis, and atherosclerosis is often seen in the intima of its proximal and distal vessels. Due to the above anatomical characteristics, when the heart contracts, the MB compresses the mural coronary artery and further narrows its lumen. The longer and thicker the MB is, the greater the angle between myocardial fibres and blood vessels, the more severe the mural coronary artery stenosis, the more severe the distal myocardial ischaemia, and the development of myocardial infarction becomes more likely. The degree of systolic compression of MB significantly correlates with the tunnelled segment depth but not the length [6]. Systolic vessel compression has been shown to persist into mid-to-late diastole [13].

Sternheim D et al. pointed out that for patients without clinical symptoms, treatment should focus on risk factor correction, including the appropriate treatment of concomitant coronary artery disease and the mitigation of potential inducements. The predisposing factors include coronary artery spasm, smoking, use of excitatory substances (cocaine), physiological stress and an insufficient functional exercise reserve [3]. It is estimated that the predisposing factors of the patient are smoking and coronary artery spas.

Medical management with negative inotropic and negative chronotropic agents is considered first-line therapy [14].  $\beta$  Receptor blockers can slow down the heart rate, increase the diastolic filling time and reduce the arterial pressure in the tunnel section. Calcium channel blockers (CCBs) are also commonly used to treat symptomatic myocardial bridges, especially for patients with myocardial infarction. CCBs are preferred in patients with receptor blocker contraindications (such as bronchospasm). In addition, CCBs cause vasodilation and may be beneficial to patients with vasospasm. If the ischaemia of myocardial bridge patients cannot be alleviated by drugs, percutaneous transluminal coronary intervention (PCI) is not impossible, but there are some disputes. Because the radial pressure of the myocardial bridge on the stent is significantly greater than that of atherosclerosis, there is a high risk of coronary perforation (up to 6.3% in the study), stent rupture (reported in individual cases), in-stent restenosis (75% in BMS and 25% in DES) and in-stent thrombosis (reported in individual cases). After strict screening of patients, PCI could be considered in patients who had poor drug treatment effects, a short life expectancy or surgical contraindications [15].

Surgery is an effective treatment for symptomatic MB patients for whom drug treatment is ineffective, and these surgical techniques include coronary artery bypass grafting (CABG) and surgical unroofing. CABG is more suitable for patients with deep (> 5 mm) or long (> 25 mm) myocardial bridges [16, 17]. There is a risk of ventricular wall perforation after myocardial bridge resection, but Hemmati P et al. confirmed that surgical unroofing is a safe option for patients with isolated MB [18].

In this case, in addition to the myocardial ischaemia caused by MB, subxiphoid pain was also related to gastrointestinal ulcers.

## **Declarations**

### **Follow-up and Outcomes**

Two months after discharge, a repeat gastroscopy showed that the peripyloric and antral ulcers were cured. At the 11-month outpatient follow-up, the patient stated that he was taking his medicine regularly and paying attention to his diet, and he had no further attacks of subxiphoid pain.

### **Study Limitations**

The patient refused IVUS and FFR evaluations, so we could not better evaluate his myocardial ischaemia. The most important limitation of this study is its lack of long-term follow-up, and the subsequent inability

to assess the patient's prognosis.

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## **Conflicts of interest**

The authors declare that they do not have any conflicts of interest.

## **Ethics approval**

Written informed consent was obtained from the patient.

## **Consent to participate**

The work was approved by all the authors for participation.

## **Consent to publications**

The work has been approved by all the authors for publication.

## **Availability of data and material**

Some or all data and material generated or used during the study are available in a repository or online (provide full citations that include URLs or DOI).

## **Code availability**

Some or all codes generated or used during the study are available in a repository or online (provide full citations that include URLs or DOI).

## **Author contributions**

Conceptualization: Mei-lian Cai, Data curation: Mei-lian Cai

Investigation: Mei-lian Cai, Resources: Mei-lian Cai

Supervision: Wei Zhang, Writing- original draft: Mei-lian Cai

Writing-review & editing: Mei-lian Cai

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## **Abbreviations**

MB = Myocardial bridging, ECG = electrocardiogram, CAG = coronary angiography, LAD = left anterior descending, CCB = calcium channel blocker, CHD = coronary heart disease, ACS = acute coronary syndrome, LCX = left circumflex artery, IVUS = intravascular ultrasound, HCM = hypertrophic cardiomyopathy, CCTA = coronary computed tomography angiography, CT = computed tomography, MRI = magnetic resonance imaging, CABG = coronary artery bypass grafting, PCI = percutaneous transluminal coronary intervention.

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## Figures

### Figure 1

a: Electrocardiography showing ST segment depression and T-wave inversion with symmetry in leads V2 to V6.

b: Electrocardiography showing less ST segment depression and less T-wave inversion with symmetry in leads V2 to V6.

## Figure 2

The a and b represent coronary angiography showing MB over the proximal of the left anterior descending (LAD) with a 1.5 cm intramural segment, the vascular lumen was compressed 80% during systole, and the compression was relieved during diastole.