中文題目：年輕男性胸痛，容易遺忘的鑑別診斷：心肌橋

英文題目：Myocardial Bridging: An easily forgotten cause of acute chest pain in a young male

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## Introduction

Myocardial bridging is a congenital variant of the coronary artery vasculature running through the myocardium vessel tunnels rather than a typical epicardial course. Traditionally, it had been considered to be a benign condition but several recent studies have reported that it can have lethal complications including acute coronary syndromes, coronary spasm, ventricular septal rupture, arrhythmias , transient ventricular dysfunction and sudden death. We report a young male with myocardial bridging presented with atypical chest pain, ST elevation on electrocardiogram and elevated cardiac enzyme.

## Case presentation

A 26-year-old Taiwanese man with no significant past medical history, presented with a sudden onset of non-radiating pain in his left chest without precipitating and relieving factor. He works as an engineer and denied alcohol consumption, cigarette smoking history. His family history was not contributory. There was no other constitutional symptoms including fever, malaise, anorexia, nausea, vomiting or headache, but he mentioned diarrhea days before chest pain.

Upon emergent department arrival, his vital signs and chest X ray were normal but electrocardiogram (ECG) showed ST elevation at precordial lead V2-V5. Laboratory data revealed no leukocytosis but elevated CRP (50 mg/L) and elevated Troponin I (2 ng/ml) . Under the suspicion of ST- elevation myocardial infarction (STEMI), aspirin and ticagrelor with heparin was prescribed and emergent coronary angiography was arranged. There was no significant stenosis in coronary arteries but myocardial bridging was disclosed at segment 7 of left anterior descending artery.

During hospitalization, intermittent chest pain was complained but was relieved spontaneously or by acetaminophen use. Serial ECG follow up showed persistent ST elevation at precordial leads and troponin-I rised to a peak level of 4.8 ng/ml.

The echocardiography results were unremarkable, with ~~a~~ normal left ventricle (LV) size and function (LVEF : 55.92%). There was neither regional wall motion abnormality nor pericardial effusion. After beta-blocker was prescribed, the symptoms relieved and the patient was discharged uneventfully.

## Discussion

Myocardial bridging is a congenital anomaly described as tunneling of a coronary artery through the myocardium rather than running a typical epicardial course. Its prevalence ranges from 0.5% to 29.4% when detected by angiography and range from 5.4% to 85% by autopsy. Myocardial bridging is generally confined to the mid left anterior descending artery, as demonstrated in our patient. It is less frequently located in the circumflex artery, and is occasionally seen in the right coronary artery.

The mechanism lies in the disturbance of blood flow through the tunneled artery. There is enhanced myocardial compression in which the vessel enters into the myocardium, mainly occurs during systole and is resolved in the diastolic phase, leading to a disturbance of blood flow to the myocardium. Symptoms and complications of myocardial bridging are associated with the degree of systolic narrowing, depth of tunneled artery, number of tunneled segments, and high heart rate.

ECG rarely show any abnormalities in these patients, but our patient's ECG disclosed persistent ST elevation in precordial leads. Coronary angiography remains the standard diagnostic tool of myocardial bridging. The "milking effect" demonstrated systolic compression of the tunneled artery and subsequent antegrade flow with expansion of the vessel diameter during diastole. Other diagnostic tools include intravascular ultrasound, intracoronary doppler sonography, cardiac computed tomography, cardiac magnetic resonance imaging.

First-line therapy includes beta-blockers and calcium-channel blockers which focused on decreasing the compression of intra-myocardial arteries via decreasing systolic contraction and prolonging the diastolic phase. Ivabradine has also been shown to have an effect on myocardial bridges. Pure vasodilating agents such as nitrates should be avoided, as they intensify systolic compression of the tunneled segment, leading to retrograde flow and thus worsening

symptoms. If symptoms persist despite medical therapy, intracoronary stenting or surgical myotomy can be further considered.

## Conclusion

Myocardial bridging is an increasing but often forgotten cause of angina. Here we report a young male presented with atypical chest pain, ST elevation on electrocardiogram and elevated cardiac enzyme and myocardial bridging was noted on coronary angiography. It reminds us of patients who have low clinical suspicion for atherosclerosis, presenting with anginal equivalents should undergo coronary angiography for immediate differential diagnosis and treatment since myocardial bridging may cause severe complication such as myocardial infarction, cardiac arrhythmias and sudden cardiac death.