

# Myocardial bridge in a child without hypertrophic cardiomyopathy: A case report

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## ABSTRACT

In children, myocardial bridge is an inborn coronary anomaly and is usually found in the patients with hypertrophic cardiomyopathy or left ventricular hypertrophy. Myocardial bridges were long thought to be a benign anatomical variant, even in patients with obstructive and non-obstructive hypertrophic cardiomyopathy. However some patients suffer from myocardial infarction, arrhythmia and even sudden cardiac death. In the present report, we describe a 7-year-old girl with the manifestation of recurrent precordial discomfort, and subsequently diagnosed as having an isolated myocardial bridge in the distal segment of left anterior descending coronary artery using 64-slice computed tomography coronary angiography.

**Key words:** myocardial bridge, hypertrophic cardiomyopathy, child, 64-slice computed tomography.

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## INTRODUCTION

Myocardial bridging occurs when a segment of the coronary artery takes an intramural course. This phenomenon was first mentioned by Reyman<sup>1</sup> in 1737, and the artery coursing within the myocardium is called a tunneled artery. It is characterized by systolic compression of the tunneled segment, which remains clinically silent in the vast majority of cases. However, some patients had suffered from myocardial ischemia and infarction, even sudden cardiac death due to the presence of a myocardial bridge.<sup>2,3</sup>

## CASE REPORT

A 7-year-old girl was admitted to our department with the manifestation of recurrent episodes of precordial discomfort without any premorbidities. The episodes lasted up to 1 minute and accompanied by pallor, sweating, nausea and vomiting. Physical examination revealed normal vital signs (temperature 36.7°C, pulse rate 92/m & respiratory rate 24/m) and the cardiac examination revealed a 2/6 grade systolic blowing murmur in the pericordium. The laboratory tests had no any significant finding on admission.

The electrocardiogram on admission showed pathologic Q waves in V2, V3 leads, with T wave inversion in V1-V4 leads, and no electrocardiographic signs of chamber hypertrophy, ischemia, or a prolonged QTc interval. During hospitalization, we recorded the electrocardiogram of a witnessed episode of precordial discomfort, and the electrocardiogram demonstrated ST segment elevation in the limb leads I and aVL. (Figure 1)

Furthermore the cardiac markers performed during the episode were significantly increased, especially the sensitive troponin I was elevated to 27.012 ug/L (normal range 0 - 0.06 ug/L) Echocardiography revealed normal cardiac & coronary artery anatomy with normal left heart function, but the left ventricular found enlarged with no regional wall motion abnormality.

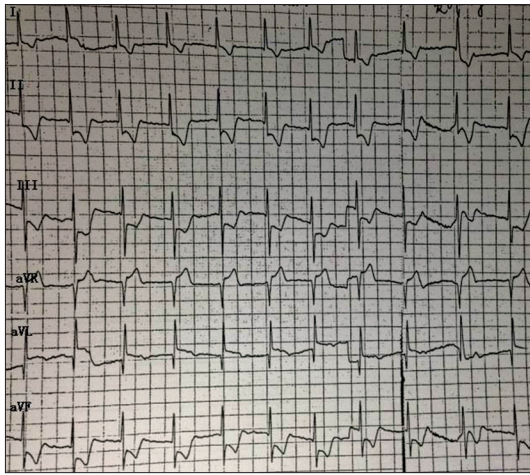
The patient had two episodes of precordial discomfort during hospitalization. During the second episode, the patient suffered from cardiogenic shock, acute left heart failure, and was transferred to Intensive Care Unit. The bedside echocardiography showed that the left ventricle was dilated with regional ventricular septum motion abnormality. After 3 days' treatment of assisting

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**Figure 1:** The electrocardiogram manifestation of precordial discomfort onset



**Figure 2:** The arrow showed the presence of myocardial bridging located at the distal segment of left anterior descending coronary artery, which was detected by multidetector-row computed tomography coronary angiography

breathing with ventilator, cardiac agents (digoxin), diuretics (furosemide) and some other treatments, the vital signs got stable and she was back to our department. A 24-hour electrocardiographic evaluation showed ventricular ectopic beats, no ST-T segments change, with normal heart rate variability. The multidetector-row computed tomography coronary angiography revealed the presence of myocardial bridging at the distal segment of the left anterior descending coronary artery, with the length of 36.7mm, and depth of 0.7mm (Figure 2). After we made the definitive diagnosis, the patient left the hospital due to some uncertain factors, so she did not get any special treatment.

## DISCUSSION

Myocardial bridge is a band of cardiac muscle overlying the epicardial coronary artery, and the affected coronary

termed as tunneled artery. Descriptions of myocardial bridging in children are rare, and its prevalence in the normal pediatric population is unknown. Nearly all reports of myocardial bridge in children occur in association with hypertrophic cardiomyopathy, with a prevalence of 28% among children with diagnosed hypertrophic cardiomyopathy<sup>4</sup>. There is an ongoing debate whether hypertrophic cardiomyopathy-associated myocardial bridge is an independent risk factor for ischemia and sudden cardiac death in children or simply an indicator of the severity of left ventricular hypertrophy. To our knowledge, the patient in our case, who suffered from cardiogenic shock and acute left heart failure, is the youngest patient with myocardial bridge without hypertrophic cardiomyopathy, and it may confirm the idea that myocardial bridge is a risk factor for serious cardiac events in the form of different clinical symptoms.

Baurassa et. al<sup>5</sup> in 1976 described the angiographic appearance of myocardial bridge, termed a “milking effect”, as at least 50% systolic compression of coronary segment on selective coronary angiography. It is the gold standard for detection of myocardial bridge. Multidetector-row computed tomography (MDCT) coronary angiography is being increasingly used in the diagnosis of myocardial bridge. Lubarsky et al evaluated the prevalence and characteristics of myocardial bridging in a large series of patients and to assess the relation between atherosclerosis and myocardial bridge<sup>6,7</sup> and this might validate MDCT coronary angiography for the diagnosis of myocardial bridge<sup>8</sup>. In our case, the existence of myocardial bridging at the distal segment of the left anterior descending coronary artery was also successfully diagnosed using MDCT coronary angiography. It is of note that the high-quality three-dimensional reformations of MDCT coronary angiography enable the assessment of the length, depth and precise location of the tunneled coronary segment<sup>9, 10</sup>. However, although catheter angiography, intravascular ultrasound (IVUS) and intracoronary Doppler ultrasound (ICD) can be used to identify the morphological and functional features of myocardial bridging, these procedures are invasive.<sup>7, 11, 12</sup>

Treatment for symptomatic patients with myocardial bridging varies. Medical treatment as a first-line therapy includes nitrates, beta-blockers and calcium antagonists. In patients with severe angina and clinically relevant ischaemia, surgical treatment such as myotomy and coronary artery bypass grafting is considered. Coronary stent implantation may be the treatment of choice for patients whose diseased state is complicated by infarction or recalcitrant ischaemia, in spite of the frequent occurrence of restenosis and major periprocedural complications invasive.<sup>7, 11, 14, 15</sup>

## CONCLUSION

Accordingly, this case is unique because the patient presented at relative young age with myocardial bridge without hypertrophic cardiomyopathy. We may conclude that myocardial bridge is a risk factor for serious cardiac events in the form of different clinical symptoms, and MDCT coronary angiography, as a useful noninvasive imaging method, can be used in the diagnosis of myocardial bridge. And for the patient with recurrent precordial discomfort, myocardial bridge should be a differential diagnosis (coronary atherosclerosis, Angina & Myocardial Ischemia) even in the absence of left ventricular hypertrophy. We agree with others<sup>13</sup> that large multicenter clinical databases are required to identify criteria that justify the link between clinical signs or symptoms and the myocardial bridge as the primary culprit and which move beyond the current empirical approach to the clinical management of this frequent coronary anomaly.

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### Authors Contribution:

**SK Yadav** - Designed the study, collected the laboratory data, analysed the data, drafted the manuscript & reviewed the manuscript; **Xue Zhou** - Contributed to study design, study the laboratory tests; **Ling He** - Reviewed the manuscript; Qi-jian Yi - Edited and reviewed the manuscript.

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