Acute myocardial infarction in a child with myocardial bridge

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INTRODUCTION

Myocardial infarction (MI) is rare in children, and Kawasaki disease is now recognized as the main cause for MI. In this report, we present a child with MI caused by myocardial bridge (MB).

CASE REPORT

A boy of 7.5 years old was admitted to the hospital on September 16, 2008 because of paroxysmal chest pain for 16 hours plus pale and sweat for 2 hours. Two days ago, the boy had a high fever of unknown reason, and the fever diminished after administration of one package of nimesuli prescribed by his parents. Next afternoon, the boy had chest pain for 10-20 minutes with an interval of 1-2 hours, and he didn't receive any treatment. On the third day, the chest pain aggravated with dyspnea, vomiting, sweat, and pale. The boy was transferred to Weifang People’s Hospital Affiliated to Weifang Medical College. He had no history of Kawasaki disease nor familial history of lipid metabolism disorders. Physical examination showed T 37.4 °C, P 118 beats/min, RR 32 beats/min, weight 40 kg, BP 110/90 mmHg, and HR 100 beats/min. He was pale, sound minded, slightly irritable, and had poor spirit and shortness of breath. The breath sound in the right lung was lower than that in the left lung, and there was no rale. The abdomen was soft, the liver was soft at 2.5 cm from the rib and 1.0 cm from the xiphoid. There was no edema in the lower extremities nor positive sign in the nervous system.

Laboratory examinations showed that WBC 9.50×10^9/L, RBC 4.87×10^12/L, Hb 140 g/L, platelet count 472×10^9/L, the relative value of neutrophils 0.75, relative value of lymphocytes 0.17, the relative value of monocytes 0.03; urine, stool (-); erythrocyte sedimentation rate 27 mm/h; C-reactive protein 84.40 mg/L; anti-O 20 IU/mL; blood electrolytes (mmol/L): sodium 134 (130-147), K 3.92 (3.2-5.3), Cl 93.0 (98-102), Na 134 (130-147), K 3.92 (3.2-5.3), Cl 93.0 (98-102).
110), calcium 2.0 (2.1-2.7); liver function (IU/L): alanine aminotransferase 184 (5-40), aspartate aminotransferase (AST) 422 (8-47). Renal function was normal; anticardiolipin antibody IgM (+); routine coagulation tests: prothrombin time 12.8 seconds (8.8-18.8), activity 74% (80%-130%), international normalized ratio 1.09 (0.8-1.2), part of the prothrombin time 33.80 seconds (26-42), thrombin time 17.4 seconds (10.3-17.6), fibrinogen 326 mg/dL; D-dimer 183 ng/mL (0-500); and myocardial enzymes and troponin-I increased obviously (Table 1).

At 2 days after admission, echocardiography showed (1) large heart, a small amount of mitral regurgitation and tricuspid regurgitation; (2) cardiac function 25%, fractional shortening 11%; (3) pericardial effusion. X-ray showed a median amount of pleural effusion in the right thorax cavity and an increased heart shadow. At 6 days after admission, coronary CT angiography showed the patent coronary artery without stenosis; myocardial bridge in the left anterior descending coronary artery (Figure 1); and a small amount of pericardial effusion. Radionuclide myocardial perfusion imaging revealed myocardial ischemia and myocardial infarction signs in the left ventricular apex, a partition near the apex of the heart and the inferior wall.

The boy was initially diagnosed as having (1) acute inferior myocardial infarction and extensive anterior myocardial infarction; (2) fulminant myocarditis; or (3) coronary myocardial bridge.

After admission, the boy was monitored with cardiogram. He was treated with oxygen, thrombolysis, myocardial nutrition, vitamin C (4.0 g per time), dexamethasone (7.5 mg per time), a large dose of gamma globulin, and interferon. At 3 days after treatment, edema occurs in the face and lower limbs. The liver was tenacious and tenderness at 3.5 cm from the rib and 4.0 cm from the xiphoid. Then the boy was administered with cedilanid and diuretics. At the 4 days after treatment, BP decreased to 80-90/50-60 mmHg, and HR decreased to 40 beats/min. He was then administered with 20 mg dobutamine, and at 5 days, BP and HR returned to normal. At 15 days, the results of chest X-ray examination showed that pleural effusion was absorbed, and the heart shadow became normal.

Echocardiography showed normal cardiac function, cardiac function 64%, fractional shortening 35% as well as a little amount of mitral regurgitation, tricuspid regurgitation and pericardial effusion. Myocardial enzymes, liver function, C-reactive protein, and troponin-I returned to normal at 21 days after treatment; at 29 days, ECG indicated that II, III, aVF, V4 - V6 leads had abnormal Q wave, and ST-T changed. The patient was discharged. At 21 days after discharge, ECG indicated that II, III, aVF, V4 - V6 leads had more obvious abnormal Q wave.

<table>
<thead>
<tr>
<th>Cardiac markers</th>
<th>1st day</th>
<th>2nd day</th>
<th>3rd day</th>
<th>5th day</th>
<th>11th day</th>
<th>21th day</th>
<th>29th day</th>
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<tr>
<td>AST (IU/L)</td>
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<td>490</td>
<td>596</td>
<td>246</td>
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<td>960</td>
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<td>1218</td>
<td>1234</td>
<td>994</td>
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<td>98</td>
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<td>28</td>
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<td>CK-MB (IU/L)</td>
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<td>72</td>
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<td>22</td>
<td>18</td>
<td>14</td>
<td>7.0</td>
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<tr>
<td>CTnI (ng/ml)</td>
<td>43.43</td>
<td>26.45</td>
<td>19.43</td>
<td>5.41</td>
<td>1.59</td>
<td>0.05</td>
<td>0.03</td>
<td>0-0.04</td>
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</tbody>
</table>

Table 1. Change of myocardial enzymes and troponin-I (CTnI)

Figure 1. Coronary CT angiography (CTA) of the heart. The arrow in the A and B indicated the location of MB; the arrow in the C indicated cross-sectional coronary artery under cardiac muscle fibers.
DISCUSSION

The major coronary arteries, which are normally distributed over the epicardial surface of the heart, occasionally have a segmental intramyocardial course. During systole, this segment of the vessel is compressed, a condition referred to as milking or systolic “myocardial bridging”. This phenomenon was first recognized more than 200 years ago, was first reported in depth in 1951, and was recognized angiographically in 1960.

The diagnosis of myocardial bridging is largely dependent on angiography. On angiography, bridging is recognized as compression of a segment of the coronary artery during systole, resulting in narrowing that reverses during diastole. It has been thought that most instances of bridging are of less clinical significance. However, reports suggest that severe bridging of the left anterior descending coronary artery can produce myocardial ischemia or sudden death.

The reported prevalence of bridging varies in pathologic and angiographic studies. Necropsy studies have found a mean frequency of myocardial bridging of 25% (range 5%-86%). In one autopsy study, the incidence was 50%. Although all major epicardial coronary arteries can be affected, involvement of the left anterior descending coronary artery is the most common.

The initial ECG of the boy indicated that ST segment elevated 0.3-0.6 mV in the II, III, aVF, V2-V6 leads. Combined with upright T, ST segment wave formed a single curve, and Q wave appeared after the disappearance of the curve. This was in accordance with the Towbin diagnosis.

Cardiac troponin-I, which appears in the blood at 4-6 hours after MI and continues to increase at 7 to 10 days, is a sensitive and specific marker of MI. In this boy, the initial cardiac troponin-I and CK-MB increased by 400 times by 4 hours after MI and continues to increase at 7 to 10 days, is a sensitive and specific marker of MI. This phenomenon was first recognized more than 200 years ago, was first reported in depth in 1951, and was recognized angiographically in 1960.

CARDIAC TROTONIN-I AND CK-MB ARE SLIGHTLY OR MODERATELY INCREASED. HENCE IT IS NOT DIFFICULT TO DISTINGUISH SEVERE MYOCARDITIS FROM MI. KAWASAKI DISEASE, SYSTEMIC LUPUS ERYTHEMATOSUS, AND CORONARY ARTERY DYSPLASIA CAN ALSO CAUSE MI, BUT THE BOY WE PRESENT HERE DID NOT SHOW THE ABOVE DISEASES, AND HE HAD NO FAMILY HISTORY OF HIGH CHOLESTEROL. THIS SUGGESTED THAT THE OCCURRENCE OF MB WAS ASSOCIATED WITH MI.

REFERENCES

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