Myocarditis with Myocardial Infarction Like Findings in A 3-year Old Girl

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A 3 year-old Japanese girl had an acute onset associated with vomiting. The electrocardiogram (ECG) indicated changes similar to those of acute myocardial infarction (MI); there was no past history of kawasaki disease.

Selective coronary angiography taken on the 28th day of illness revealed no abnormality. Thallium 201 scintigraphy was also performed and it revealed that the area of absent myocardial uptake was in the anterior wall. In serological findings, antibody titers against Coxsackie B-3 virus had risen significantly; therefore acute myocarditis caused by Coxsackie B-3 virus infection was diagnosed.

Electrocardiographic findings simulating MI are not common in young children but they are relatively well known in Kawasaki disease. The authors describe a case of myocarditis with similar changes of MI on ECG.

CASE REPORT
Case A.O., 3 year-old girl.

History
The patient vomited suddenly and turned pale during her breakfast on May 19, 1984. She was taken to a local medical center, where no definite abnormalities were found on chest X-ray, even though she continued to vomit. She was admitted to another hospital at 11 p.m. since vomiting had not stopped. The next day she was transferred to Tokyo Women’s Medical college Daini Hospital because of findings of arrhythmia and bradycardia with shock.

Status on Admission
Body weight: 12 kg, height: 93.1 cm, body temperature: 35.6°C. History indicated normal delivery and development. Upon admission, consciousness was clear; cyanosis was observed on the lips and nail beds. Eyelids showed slight edema. Heart rate was 40-45/min associated with arrhythmia and respiratory rate was 48/min with slight dyspnea. The systolic blood pressure was 82 mmHg. The auscultation of the heart and lungs revealed no abnormal physical findings. The liver was palpable 3 cm below the costal margin but the spleen was not palpable. There were no abnormal neurological signs.

Laboratory Examinations
On admission, enzymes were markedly elevated in the blood; creatinephosphokinase (CPK) of 690 I.U/l, lactate dehydrogenase (LDH) of 2961 I.U/l, glutamic oxaloacetic transaminase (GOT) of 948 K.U.. Leucocytes of 9900/mm³ with 66% neutrophils (11% band forms), 32% lymphocytes and 2% monocytes, hemoglobin of 11.9 g/dl, platelet of 23.4 x 10⁴/mm and C-reactive protein (CRP) of (±) were found. Serum electrolytes, triglycerides, cholesterol, urea nitrogen and glucose were within normal limits. There was no abnormality in urinalysis. Arterial blood analysis (FiO₂ 0.4) was pH 7.51, PaO₂ 65 mmHg, PaCO₂ 29 mmHg.

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Japanese Circulation Journal Vol. 50, December 1986 1275
As seen in Fig. 1, a chest X-ray film showed slight cardiac enlargement with a cardio-thoracic ratio of 53%, pulmonary congestion with pleural effusion significantly on the right pulmonary field compared with the left side. ECG on admission (Fig. 2) showed complete atrio-ventricular block (A-V block). ST-T wave segments were depressed in leads II, III and aV_F, elevated.
in leads I, aVL, and V1-V4, abnormal Q waves appeared in leads V1-V4, these findings suggested myocardial infarction of the anterior wall and the septum.

M-mode echocardiogram on the fourth day of illness revealed neither aneurysm nor abnormality of origin of coronary arteries but did reveal poor movement in the apex (ejection fraction of 64-72%).

During Hospitalization
Treatment started initially with atropine sulfate and oxygen but the pulse rate did not change as expected. Isoproterenol of 0.5 mg/hr was administered intravenously with diuretics and an anticoagulant (urokinase at 10000 U/kg/day); the pulse rate increased to 80-90/min; the systolic blood pressure improved 90-100 mmHg. Complete A-V block diminished after five hours.
from the time of admission; sinus rhythm with sporadic ventricular extrasystole (PVC) appeared. From this period gallop rhythm was heard on the apex on auscultation.

Electrocardiographic course on chest leads are shown in Fig. 3. On the second day of illness, abnormal Q waves appeared and ST-T wave segment was elevated on leads V₁-V₄. On the fifth day abnormal Q waves were still present but ST-T wave segments returned to the baseline. On the eighth day, q waves appeared on lead V₅. The ECG on the 32nd day of illness became almost normal except for slight low voltage of T wave on leads V₄, V₅, and further, after 7 months it revealed a quite normal ECG.

At the time of admission the patient was in an unstable condition and needed tranquilizing, but she became calm when sinus rhythm recovered and she could take water orally by herself and could cry loudly by the fourth day of her illness. PVC sometimes appeared when she moved. On the eighth day of her illness PVC was not apparent. At this point oxygen was stopped, and further urokinase was also stopped, while aspirin and furosemide were started orally.

Selective coronary angiogram on the 28th day after onset showed neither anomaly nor obstruction of the coronary arteries (Fig. 4).

On the 35th day of illness Thallium 201 scintigram showed the area of absent myocardial uptake in the anterior wall (Fig. 5). This was thought to be the state of MI of the anterior wall, associated with the findings of the Technetium 99m scintigram.

The serum antibodies against Coxsackie virus were measured by complement fixation reaction (Table I). The antibody titer of Coxsackie B-3 rose compared with acute and convalescent serum samples, and this case was diagnosed as myocarditis due to Coxsackie B-3 virus.

The patient was discharged from hospital on the 60th day after onset. After a year chest X-ray, ECG and echocardiogram have not shown any abnormalities, but Thallium 201 scintigram still reveals almost the same size of absent myocardial uptake.

**DISCUSSION**

This patient was first believed to have had myocardial infarction caused by anomalous origin of coronary arteries because her past history revealed neither Kawasaki disease nor symptoms of a common cold before onset. However typical changes of ECG and serum enzymes demonstrated charactaristic MI. Furthermore, angiocardiography was made, and no abnormalities were found. Meanwhile, antibodies to Coxsackie B viruses were serologically measured by complement fixation reaction (although the neutralization test is more sensitive, it needs sufficient...
quantity of samples to be measured accurately.). As there was serological evidence of infection with Coxsackie B-3, this patient was diagnosed as having Coxsackie B-3 virus myocarditis. Such a case is extremely unusual in childhood and has not been reported before, to our knowledge.

There is “nontransmural myocardial infarction” (coronary angiogram reveals no abnormality at all) in the classification scheme of MI in adults. In a subgroup classified as nontransmural MI, the incidence of Coxsackie B viral infection is 14%, reported by O’Neill et al. There are also case reports on adults that suggest MI caused by Coxsackie B myocarditis. However, a causal relationship between infection with Coxsackie B virus and MI has not clearly been established. Woods et al. suggested that a mechanism such as autoimmunity may in fact cause similar changes of MI as Coxsackie infection. While, Burch et al. suggested in experimental models that myocardium injured by coronary atherosclerosis and hypertension could make the subject more susceptible in a virus attack, and therefore demonstrate symptoms that simulate MI. It is our opinion that a severe myocardial disorder caused by Coxsackie B virus might appear similar to MI, as demonstrated by the case presented.

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