Fatal Mumps Myocarditis

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SUMMARY

The case of a 9 year old boy with acute renal failure and myocarditis as complications of mumps is reported. The cardiac rhythm and conduction disorders which appeared after admission were refractory to treatment and the outcome was fatal. On necropsy, minimal interstitial nephritis and myocarditis were seen, confirming the clinical diagnosis.

Additional Indexing Words:
Fatal myocarditis Mumps Interstitial nephritis

Mumps is an acute systemic disease of specific viral etiology with well-known symptoms and complications and usually a benign course. In addition to parotitis, orchitis, pancreatitis and meningoencephalitis, myocarditis can sometimes be seen.1)-12) Although transient ECG changes can be encountered during the course of the illness, clinical symptoms and signs of myocarditis are rare.2)-4),11)

Few cases of fatal mumps myocarditis have been reported in the literature.5)-11) Clinical cases of mumps associated with nephritis are also rare13),14) and such cases associated with fatal mumps myocarditis have not been reported. Therefore, the present case of mumps with myocarditis and minimal interstitial nephritis progressing to acute tubular necrosis with a fatal course is of interest.

CASE REPORT

A 9 year old boy who had previously been healthy was admitted to hospital with severe cardiac and renal failure. He had contracted mumps
during a school epidemic. The initial presentation was characterized by fever and bilateral parotid swelling. Upon resolution of the swelling he became dyspneic and fatigued and was admitted to a local hospital with vomiting and abdominal pain. He was referred to our center after 4 days of uncontrolled heart failure.

On examination, the patient appeared to be acutely ill. His physical development was within normal limits. His body temperature was 36°C, pulse rate 78/min and irregular and blood pressure was 120/70 mmHg. Peripheral cyanosis, venous engorgement and minimal bilateral parotid swelling were observed. Respiration was dyspneic, the abdomen was tender and the liver was palpable 11 cm below the right costal margin. Peripheral pulses were palpable and the central venous pressure was initially 26 cm H₂O. There were no abnormal findings on the rest of physical examination. In the hematologic examination hemoglobin was 11 g/dl, hct 34%, the white-cell count was 34,000/mm³ with 77% neutrophils. The platelet count was 240,000/mm³. On urinalysis the sediment contained 3-4 WB cells and 7-8 tubular epithelial casts. Blood urea nitrogen was 117 mg/dl, creatinine was 8.1 mg/dl, uric acid 13 mg/dl, phosphorus 9.7 mg/dl, plasma amylase activity was 17,000 SU, tenfold normal levels. Serum sodium, potassium, chloride, SGOT, SGPT, calcium, alkaline phosphatase and proteins were normal. The tests for antinuclear antibodies, anti DNA, LE cells, C₃, C₄ and immune complex were normal. The specimens for culture were negative and the complement fixation test for mumps antibodies was positive at a titer of 1/20.

The abdominal ultrasonography revealed diffuse hepatomegaly and pancreatic enlargement. Renal structure and collecting systems were normal.

There was minimal cardiomegaly on telecardiogram. The ECG initially
showed a nodal rhythm with aberrant conduction. Later, paroxysmal atrial tachycardia, ventricular extrasystoles and ventricular tachycardia were observed (Fig. 1). M-mode and two-dimensional echocardiography were performed upon admission. The left ventricular diastolic dimension was slightly increased with normal wall thickness, but with a marked decrease in the amplitude of the left ventricular posterior wall. The mitral valve excursion and the E point of the anterior mitral leaflet to the ventricular septum were normal. Fractional shortening and ejection fraction of the left ventricle were decreased.

Severe heart failure due to myocarditis and secondary acute tubular necrosis were the dominant clinical features. The nodal rhythm at 75 bpm with aberrant conduction which was present on admission continued. Later,
attacks of supraventricular tachycardia which were reversible with verapamil were observed. Ventricular extrasystoles and tachycardia present at the final stage of the illness were not controlled by antiarrhythmic therapy. Acute renal failure developed due to acute tubular necrosis secondary to the reduction of effective blood volume and interstitial nephritis. Peritoneal dialysis was performed as the anuria and azotemia worsened. The patient's cardiac status and clinical course deteriorated and he died on the 13th day of his illness and the third day following admission to the hospital.

Postmortem examination of the needle necropsy material of heart muscle and kidney was performed. Microscopically, sections of the heart showed focal myocardial degeneration and mononuclear cell infiltration in the interstitial tissue and in the focal areas of myocardial necrosis. Focal myofibrillar degeneration and fragmentation were present. Most myocardial fibers were hypertrophied (Figs. 2, 3). Stains of the myocardium for fat (oil-red), glycogen (PAS) periodic acid-schiff and PAS diastase, amyloid and iron were negative. There was minimal focal interstitial mononuclear cell infiltration in kidney necropsy material and the liver showed chronic passive congestion secondary to congestive heart failure.

**DISCUSSION**

In 1918, Pujol first suggested that myocarditis might be a complication of mumps. He reported 3 cases with clinical evidence of myocardial involvement. In 1945, Rosenberg first described the electrocardiographic abnormalities in a patient who developed complete heart block while convalescing from epidemic parotitis. Two years later, Rosenberg also described a case which at first showed incomplete atrioventricular block, later complete heart block; the rhythm returned to normal on the 77th day after the onset of symptoms. After these 2 cases, Rosenberg studied 104 adult cases with mumps and found 16 cases (15.4%) with electrocardiographic abnormalities. These were mainly in the ST segments and T waves. Myocarditis commonly occurred between the 5th and 10th day after the onset of mumps and electrocardiographic changes usually disappeared within between 2 and 35 days, but in 2 cases did not do so for 3 to 5 months.

Bengtsson and Örndahl studied 564 cases of mumps (243 in adults and 321 in children), and reported electrocardiographic changes in 25 of them (4.4%); myocarditis was more common in adults and was frequently found in patients with mumps meningoencephalitis. Of 25 cases, 20 showed ST depression and inverted or flat T waves. Two patients had atrioventricular block and one had multifocal extrasystoles. These authors also showed that
in most cases these changes appeared during the first week and usually regressed after a further week. However, they persisted in some cases for several months.

Thus mumps myocarditis is a transient, benign and not uncommon disease. Fatal mumps myocarditis is rare. According to our knowledge, only 7 cases have been reported. In 1932, Manca first described the necropsy findings in a case, a 21 year old soldier who died 14 days after the onset of illness. He found acute, interstitial and fibrinous myocarditis. In 1962, Krakower and Roberg reported the case of a 4 year old girl who died of heart failure 55 days after the onset of illness. Roberts and Fox reported a patient who died of severe heart failure 8 months after the onset of an acute illness that could have been mumps. In the autopsy of this patient, diffuse interstitial myocardial fibrosis and focal necrosis were found. Kussy reported on a 9 year old boy who died suddenly 3 days after falling ill. Aram et al have described 2 sisters with mumps myocarditis. One died suddenly on the 50th day in hospital. Brown and Richmond, Baandrup and Mortensen also reported cases of fatal mumps myocarditis.

Based upon the clinical and laboratory findings noted above, the present illness was mumps and severe cardiac failure was due to myocarditis that was determined by electrocardiographic and echocardiographic findings.

Diuretics were given initially and digitalis was avoided since the pulse rate was normal. Later, as renal function deteriorated, dopamine was administered to increase cardiac output. The disturbances of rhythm and conduction were treated with appropriate antiarrhythmic agents and steroid therapy was started. However, the patient's status deteriorated and he died on the third day of admission to the hospital in spite of peritoneal dialysis that was performed because of renal failure.

In our opinion, the course of the disease is due to the degree of involvement of the interstitial and conductive tissue of myocardium as seen in the case presented.

The postmortem pathologic findings in our patient were similar to those previously reported except for additional renal changes characterized with focal interstitial nephritis.

Clinical cases of mumps with interstitial nephritis are not common, but the incidence of viruria during mumps is very high. In patients with viruria, a transient proteinuria, decrease in creatinine clearance and increase in urea retention were reported. In a patient with manifestations of nephritis, the disease was more severe. A few cases of fatal mumps nephritis have been reported.

Although myocarditis was the main problem in our patient, interstitial
nephritis and acute tubular necrosis worsened the clinical picture. There was no fatal case with mumps myocarditis associated with nephritis in the literature to our knowledge. Thus, it was interesting to detect interstitial nephritis along with myocarditis in the postmortem examination.

References