MUMPS MYOCARDITIS AS A CAUSE OF NEONATAL CARDIOGENIC SHOCK

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Abstract- The mothers may be infected during pregnancy with infectious agents. Mumps induced myocarditis, especially endocardial fibroelastosis, was previously a common disease of infants but is rare now. A 25 day old male infant admitted to the intensive care of our hospital because of cardiogenic shock. Further studies revealed ischemic electrocardiograms, poor ventricular function, and positive results by polymerase chain reaction (PCR) technique for mumps virus. Regarding this case, although is very rare but mumps myocarditis should be included in the differential diagnosis of left ventricular dysfunction in neonatal period.

Acta Medica Iranica, 46(2): 156-158; 2008

Key words: Cardiogenic shock, myocarditis, mumps, newborn

INTRODUCTION

Intrauterine infection can harm the fetus seriously if transferred from the mother to the fetus during pregnancy. The infected mothers are often unaware of their illness and passing it to the fetus. Several infectious agents may cause severe fetal damage (1, 2). Fetal heart is one of the organs that can be affected by this organism in critical perinatal period. Mumps induced myocarditis, especially endocardial fibroelastosis was previously a common disease of infants, often resulted in congestive heart failure and death (1).

During the past two decades because of wide spread immunization against common preventable diseases such as mumps a dramatic decline in the incidence of cardiac involvement by mumps had been occurred (3-5). Therefore, unlike the pattern of infection of the enteroviruses, which shows periodic peaks in infection rates, the decline in incidence of mentioned cardiac involvement seems to reflect the decreased prevalence of mumps virus in the population (1, 6, 7).

During pregnancy, as far as mumps are concerned, the risk of fetal involvement seems much lower than usually believed; however a few documented reports in the medical literature show that risk is a reality (4, 5). Although it is very rare, especially at this time, we report an infant who admitted in critical condition with cardiovascular collapse which further investigation revealed mumps as ethologic factor.

Received: 4 Sept. 2006, Revised: 5 Nov. 2006, Accepted: 11 Mar. 2007

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CASE REPORT

A 25 day old male newborn infant was admitted to our neonatal intensive care unit because of tachypnea and respiratory distress. He was born via

normal vaginal delivery at 38 weeks of gestations with birth weight of 3250 gr, and Apgar scores of 8 and 10 at 1 and 5 minutes, respectively.

His mother was an 18-year-old primigravida in good health without any significant problems during pregnancy except a self-limited febrile illness about three weeks prior to delivery. She had not regular prenatal follow up. Either his maternal or family history were unremarkable. There was also no family history of cardiac disease.

He was developed recurrent coughing in first week of life which accompanied with irritability, tachypnea, diaphoresis and difficulty in breast feeding subsequently. He had also one episode of cyanosis for about few seconds one day prior to admission. There were not any other significant problems. He had not any medical visit since birth. At time of admission he was alert but appeared pale with mottled skin in severe respiratory distress with respiratory rates of 80/minutes, nasal flaring, and subcostal retraction without cyanosis. His pulse rate was 170/minutes which were accompanied with prolonged capillary refilling time. Initial systolic blood pressure was 55 mmHg. Four limbs pulse oximetries were around 92-95% without obvious differences. The patient had narrow pulse pressure and gallop rhythm was detected on cardiac examination without any cardiac murmur. His liver edge was palpable just 2-3 cm below costal margin with no splenomegaly.

Initial lab data were hemoglobin 12 g/dL, white blood count 9200/mm^3 with 72% neutrophil, and 16% lymphocyte, CRP^+, and other blood works without significant abnormal results. The initial chest roentogram revealed cardiac enlargement with increased pulmonary vascular markings. Electrocardiography was in favor of tachycardia, and left ventricular hypertrophy with strain pattern (Fig. 1). Color Doppler echocardiography was suggestive of anatomically normal heart but indicative of severe left ventricular dysfunction, mild mitral regurgitation, with ejection fraction of 22% and dyskinetic motion of interventricular septum, and patent ductus arteriosus (PDA) (Fig. 2).

All bacterial cultures were negative. Screening for inherited metabolic disorders was unremarkable. Other paraclinical studies were disclosed creatinine kinase was 113 IU/L (normal: 30-170) with MB fraction of 28 IU/L (normal < 24). Troponin T was negative and Troponin I was within normal limit (1.5 IU/ml; normal up to 3.1). Further report of viral studies for TORCH diseases and enteroviruses were negative. The infant showed serologic evidence of mump infection with positive polymerase chain reaction (PCR) of blood. Maternal blood was positive for mumps IgM antibodies which returned to negative after recheck 3 months later.

After initial stabilization he was treated with combination of dopamine and dobutamine, furosemide, and digoxin with impression of cardiogenic shock. The patient also treated with carnitine for two days and captopril due to ventricular dysfunction. He received intravenous immunoglubin (IVIG) for three times with impression of post viral cardiac involvement. At fourth days of admission his condition became considerably better that breast feeding was started for him. The infant was discharged from hospital in stable condition at 32 days of age with Lanoxin, furosemide, and captopril. Later follow up at cardiologic clinic revealed ejection fraction of 30% at cardiac echocardiography with appropriate growth and development regarding of his age at eight months of age.

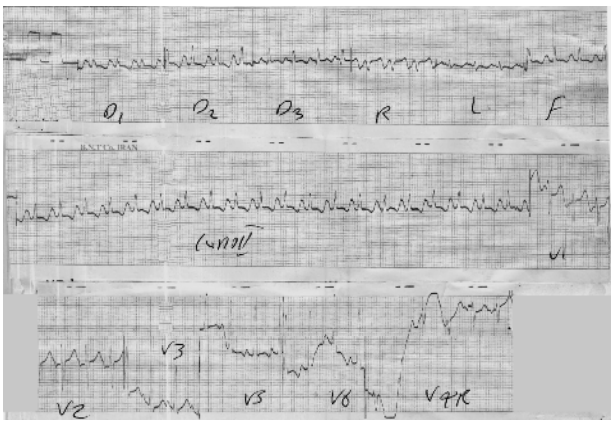
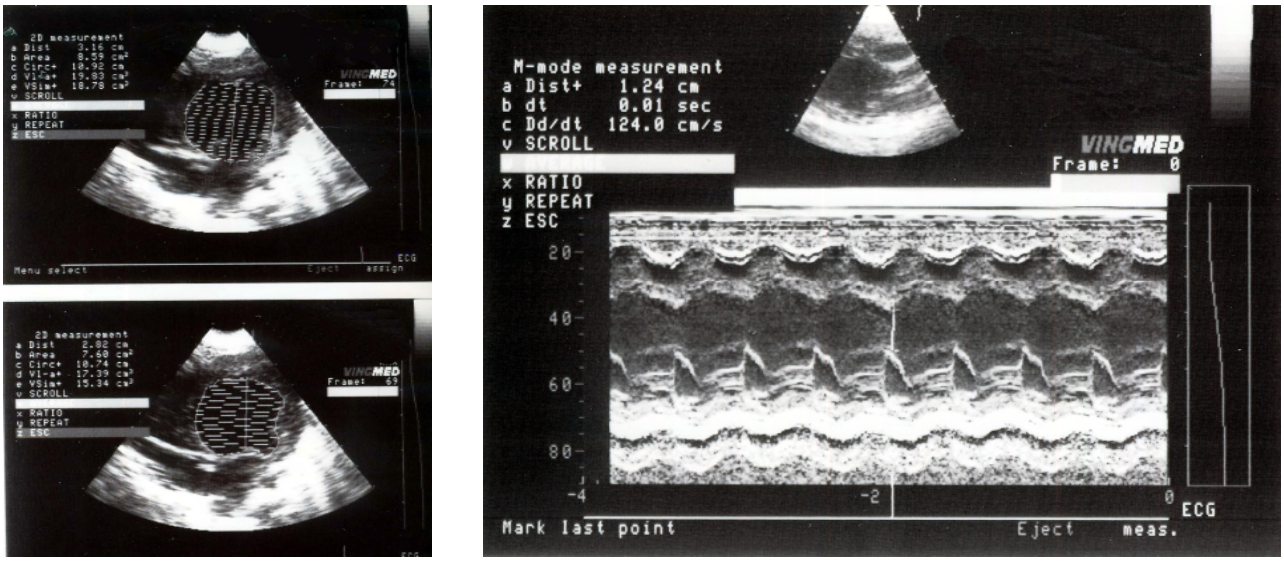


Fig.1. Electrocardiography of the patient; which shows heart rate of 150/minute, hypertrophy of left ventricle accompanied with strain pattern.



A B

Fig. 2. Echocardiography of the patient. **A**. 2 dimensional echocardiography in 4 chamber view in systole and diastole which demonstrates the ejection fraction (EF) of around 20% measured by Simpson method; **B**. M-mode echocardiography which illustrates left ventricular enlargement, increasing distance between mitral valve and interventricular wall in diastole which is more than normal limits.

DISCUSSION

Acute heart failure presented as cardiogenic shock in the early neonatal period is rare. In general it is due to asphyxia, severe septicemia, metabolic disorders, and congenital cardiac malformations or rarely because of viral myocarditis (6, 7).

Although myocarditis has been reported as a rare condition, it is recognized as a serious consequence of viral infection in neonatal period (1, 4). Viral infections is increasingly reported in cases of sudden unexplained death in infancy, raising the possibility being more common than previously among those infants (1, 6, 7). Neonatal viral myocarditis is suggested by an ischemic electrocardiogram, raised cardiac enzymes, and left ventricular dysfunction on echocardiography with normal coronary arteries. However severe respiratory distress may occur with cardiovascular collapse in infancy (1, 6).

Mumps virus considered potential cause of infantile endocardial fibroelastosis which was common before widespread immunization resulted in congestive heart failure and even death (1). Now a days many women are immune against this disease, which in a study in Germany 96% of pregnant women have shown detectable antibodies against mumps virus (3). Although complications with mumps virus are rare and myocarditis, one of them, reports more common than diagnosed because of vague and benign manifestations, generally transient abnormalities of cardiac rhythm and conduction (8, 9). However, there are a few reports of fulminant or fatal mumps myocarditis in ages other than neonatal period (10-14). Our case was presented with cardiogenic shock which echocardiography was suggestive of myocarditis with mild improvement in follow up. We did not find any stigmata of possible ethologic factors except serologic evidence of mumps infection. Although a review of the literature shows that clinical mumps is rare and usually benign in neonates, we should consider mumps virus as potential cause in newborn infants (3, 15, 16).

We found a few case reports of congenital mumps infection in review of the literatures which presented in neonatal period by respiratory distress complicated with pulmonary hypertension, congenital pneumonia, and thrombocytopenia (17-19).

In conclusion, neonatal viral myocarditis may be rare but should be considered as cause of cardiovascular collapses among infants. However, complications such as myocardial involvement due to prenatal mumps are not common, but should be regard as etiologic issue.

Acknowledgement

We thank GT Khotaiee MD, pediatric infectious diseases specialist for her great help in the interpretation of the result of viral studies of the patient and his mother.

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