Hepatitis A Leading to Myocarditis

Sir,

Many different viruses have been implicated in causing myocarditis, including hepatitis C virus.1 We had a case of hepatitis A infection complicated by myocarditis, which made this patient sick and prolonged his recovery period. His serology was positive for hepatitis A but no biopsy or viral isolation was done. As no other cause for myocarditis was evident, hepatitis A virus was presumed to be its cause.

A 19-year-old student was admitted with low-grade fever for 4 days, recurrent vomiting and decreased urine output for 2 days. He had no other systemic complaints. He had no history of alcohol or drug intake. There was no past history of jaundice or any significant medical illness. On examination, pulse was 86 / minute, blood pressure 112 / 80 mmHg. He had significant icterus and no swelling. His abdominal examination revealed mild tender hepatomegaly. His neurological, chest and cardiovascular examination was unremarkable. A provisional diagnosis of viral hepatitis was made. His laboratory parameters showed Hb 15.4 g/dl, TLC 9200/mm³, Platelets 41000/mm³, ALT 7080 u/L, total and direct bilirubin 9.2 and 6.9 mg/dl, INR 1.7, BUN 53.7 mg/dl, creatinine 6.1 mg/dl, Na 131 meq/L, and K 4.7 meq/L. His IgM anti-HAV was positive and serology for hepatitis B, hepatitis C, HIV, Dengue and Leptospira was negative. His smear and rapid malaria test was negative. Ultrasound of abdomen showed hepatomegaly (17 cm) and mild splenomegaly (14 cm) without evidence of portal hypertension. His blood and urine culture were negative. He was managed with hemodialysis for oliguric renal failure, and other supportive treatment for liver failure. On 5th post admission day, he became restless, complaining of left sided chest pain, breathlessness and had tachycardia of 190 / minute. He had pulmonary edema, hypotension and was intubated and put on ionotropic support. A cardiology opinion was taken. ECG showed sinus tachycardia of 180 / minute and 2-D Echocardiography revealed compromised global left ventricular (LV) systolic function with EF (ejection fraction) of 35% with entire septum and posterior wall being severely hypokinetic. A diagnosis of acute myocarditis was made and cardiac enzymes including troponin was sent, which came positive. He was managed conservatively with Digoxin, Dobutamine, L-carnitine and later Angiotensin converting enzyme (ACE) inhibitor was added. On eighth post admission day, he started showing clinical improvement. Gradually, he was weaned off all supports and was discharged home with increasing urine output. On follow-up after 1 month, all his investigations are within normal limits.

On literature search, few cases of myocarditis are reported in association with hepatitis A from Japan2 and France3 and this is the first case report from our country. Many more cases may have gone undetected because of lack of awareness of this association and also because in many cases hepatitis A infection remains subclinical. With increasing number of case reports, we feel that hepatitis A should also be enlisted in the viral causation of myocarditis along with hepatitis C.

Roshni Jagtap*, R Sethi**, T Jeloka***
*Clinical Assistant, Department of Nephrology; **Senior Consultant, Department of Cardiology; ***Senior Consultant, Department of Nephrology, Aditya Birla Memorial Hospital, Pune.  Received : 5.2.2008; Accepted : 4.4.2008

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