Histologic and Angiographic Imaging of Acute Shock Dengue Myocarditis

Imagen histológica y angi cardiográfica de miocarditis aguda por dengue

To the Editor,

A 65-year-old woman, resident of Mexico City, began to complain of myalgia, headache, general malaise, and fever 3 days after returning from a 5-day stay in Acapulco. Two days later, she experienced syncope and was taken to hospital. She was conscious, her blood pressure was 70/30 mmHg, respiratory rate was 24 per minute, and her temperature was 38.6°C; no skin lesions or edema where seen, heart sounds were normal, and lung fields clear.

Her electrocardiogram showed sinus rhythm; chest x-ray was normal. A complete blood count revealed normal hemoglobin, normal white blood cells with 90% neutrophils, and mild thrombocytopenia. Blood urea nitrogen and creatinine were normal; hyponatremia and hypokalemia were also present (126 and 3.6 mEq/L, respectively).

Volume replacement was started with saline and norepinephrine infusion was also required. Clarithromycin, cefepime, and oseltamivir were started. The patient continued to have a fever of 38.7°C and increased hemoglobin and hematocrit (17.9 g/dL and 53%) and leucopenia (3000/μL with 71% neutrophils, 17% lymphocytes, and 12% monocytes) were observed. All culture samples were negative.

On the sixth hospital day, she suddenly developed oppressive chest pain and dyspnea. Heart sounds revealed S3 gallop rhythm and generalized lung rales. An electrocardiogram showed sinus tachycardia, new ST depression in V1 through V6, and ST elevation in aVR and aVL. Chest x-ray revealed an enlarged cardiac silhouette and pulmonary edema. Creatine kinase-MB fraction was elevated (38 mg/dL), while troponin I was normal (1.6 μg/L).

The patient was started on noninvasive ventilation, digoxin, furosemide and ivabradine. Six hours later she developed bidirectional ventricular tachycardia, sustained monomorphic ventricular tachycardia, and polymorphic ventricular tachycardia, which were attributed to digitalis intoxication and were resolved with diphenylhydantoin. Endotracheal intubation was required and cardiac catheterization was performed. Left ventricular end-diastolic pressure was 38 mmHg, left ventriculography demonstrated severe diffuse hypokinesis with an ejection fraction of 14% and grade II mitral regurgitation (Figure 1). Coronary angiography revealed normal coronary arteries; left ventricular endomyocardial biopsy shows necrosis of myocardial fibers (Figure 2).

Dengue virus-specific IgM and IgG antibodies were positive. Oseltamivir was replaced with ribavirin. A diagnosis of severe dengue myocarditis was made and steroid treatment was initiated with methylprednisolone (500 mg i.v. bolus followed by 250 mg tid for 3 days).

On the tenth day, her electrocardiogram showed sinus rhythm, giant T wave inversion, and prolonged QT interval; chest x-ray showed reduction of cardiac silhouette and a marked decrease of pulmonary interstitial edema. Catecholamine infusion was stopped on the eleventh day. On the following day, the

Figure 1. Clear furrowing of the left ventricle border, a peculiar striation known as “myocardial waffling”, which is more apparent in anteroapical segments and is considered a sign of myocardial necrosis.
A

B

C

Figure 2. Left ventricle endomyocardial biopsy (Masson 40x) shows necrosis of myocardial fibers (arrow in A) with inflammatory cell infiltration and marked interstitial edema causing fiber separation (A), perivascular inflammatory infiltrate (arrow) with a necrotic fiber (B), and perivascular lymphocytic infiltrate (thick arrow in C), and vacuolization of myocardial fibers corresponding to cellular edema (narrow arrows in C).

These authors found that poor functional class, positive histological findings, and the absence of beta-receptor antagonist therapy are predictors of high mortality.

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