CASE REPORT: MYOCARDITIS IN WEST NILE VIRUS INFECTION

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Abstract. West Nile virus (WNV) myocarditis has been documented pathologically in birds and mammals but has rarely been reported in human clinical syndromes. We describe myocarditis associated with WNV.

CASE REPORT

A 69-year-old man presented to the emergency department (ED) in late summer of 2004 complaining of diffuse weakness and fever for 2 days. Past medical history included Waldenstrom macroglobulinemia in remission and a remote history of treated prostate cancer. He was a non-smoker with occasional alcohol consumption. He had no ill contacts or recent travel. He did not recall mosquito bites, but walked daily along an irrigation ditch. After a brief physical examination, he was released.

He returned the following day with a severe headache with neck stiffness, fever, chills, diarrhea, and nausea. On returning to the ED, he was noted to be alert, conversant, and oriented. His temperature was 38.2°C, blood pressure was 119/59 mm Hg, and heart rate was 90 bpm. Physical examination showed mild nuchal rigidity, trace pretilial edema, but no pulmonary edema, S3, or jugular venous distention (JVD) elevation. A WBC count was 4.8 × 10^3 (reference range, 4.0–10.6), and platelets were 106 × 10^9/L (reference range, < 0.5 ng/mL). Chest x-ray and non-contrast computed tomography (CT) scan of the head were normal.

Admission electrocardiogram (ECG) revealed normal sinus rhythm at a rate of 83 with occasional premature ventricular contractions (PVCs). All intervals were appropriate, and there were no ST or T wave abnormalities. Cerebral spinal fluid (CSF) showed 65 RBCs/mm^3, 1 WBC/mm^3, glucose of 54 mg/dL (serum level, 98), and protein of 41. Bacterial and fungal stains were negative. He was given ceftriaxone and ampicillin. An HIV Western blot, hepatitis panel, monospot, and cytomegalovirus (CMV) indirect immunofluorescence stain for pp65 were negative. Antinuclear antibody (ANA), rheumatoid factor, and anti-neutrophilic cytoplasmic antibody (ANCA) were negative. Both serum and CSF specimens were negative for West Nile virus (WNV) antibodies. Herpes simplex virus (HSV) polymerase chain reaction (PCR) assay of CSF was negative, and a magnetic resonance image (MRI) was not consistent with herpes encephalitis. Tests for toxoplasmosis, mycoplasma, and leptospirosis were negative.

On Day 2, he became hypoxic and developed progressive generalized weakness and declining mental status. An erythematous macular rash developed on the trunk. Cardiac monitoring showed intermittent atrial flutter/fibrillation, which resolved spontaneously, as well as occasional PVCs. There was no ST elevation, T wave changes, or other evidence of ischemia or infarct. A transthoracic echocardiogram (TTE) showed normal biventricular function with an ejection fraction of 60%.

Cardiac enzymes peaked on Day 2. The troponin was 352.7 ng/mL, and creatine phosphokinase (CPK) was 1435 U/L (MB fraction, 2.3%) and remained elevated through Day 10. A repeat TTE on Day 4 found new global hypokinesis and an ejection fraction of 45–50% and was without wall motion abnormalities. At this time, the new cardiac dysfunction and elevated cardiac enzymes were felt to be secondary to acute myocarditis.

On Day 9, he developed lower extremity paresis and respiratory failure requiring mechanical ventilation. A repeat WNV serology from the same day was now positive with IgM of 2.8 and IgG of 0.24. His ECG continued to show supraventricular tachyarrhythmias without evidence of infarct or ischemia. His continued intermittent arrhythmias did not require medical therapy, and there was no associated hypotension or need for vasoactive pressors. His neurologic status declined, and ultimately the decision was made to withdraw support on Day 17.

DISCUSSION

We report a case of myocarditis associated with WNV. In our patient, the manifestations included cardiac arrhythmias, new global myocardial dysfunction, and elevated cardiac enzymes—all of which were consistent with diffuse myocardial damage. Based on these findings and the lack of evidence for acute ischemia or infarct, viral myocarditis was felt to be the most likely diagnosis. The presence of neurologic involvement, positive WNV serology, and absence of other prominent infections indicate that this is WNV-associated myocarditis. The family declined post-mortem examination, so no pathologic evidence exists. Other than autopsy, no definitive test for viral myocarditis exists. Currently, the “gold standard” for myocarditis is the endomyocardial biopsy, which unfortunately has quite low sensitivity and specificity.1

There have been numerous reports of myocarditis secondary to WNV in a variety of mammalian species2–4 and birds,5–7 indicating a predilection for myocardial involvement. Findings include multi-focal myocardial necrosis and lymphohistiocytic myocarditis.8 While this patient’s myocarditis likely did not lead directly to his demise, the severity of involvement was never correlated through pathologic examination. Although WNV myocardial involvement has only been described in two autopsy reports,9,10 cardiac sequelae have been reported after infections with other flaviviruses.10–12

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The implications of this case are widespread. WNV is now the most frequently reported etiology of viral encephalitis in the United States. As cases of WNV accumulate, it is important that the index of suspicion increase for this rare complication. Physicians caring for WNV-infected patients need to be aware of the possibility of WNV-related myocarditis. Also, WNV should be considered with any case of acute myocarditis, particularly during summer and fall when mosquitoes are most prevalent. Autopsy and pathologic study of human WNV cases could help elucidate the pathogenesis of this disease.

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REFERENCES