West Nile virus myocarditis causing a fatal arrhythmia: a case report

Anurag Kushawaha*, Sunil Jadonath and Neville Mobarakai

Address: Department of Medicine, Division of Infectious Diseases, Staten Island University Hospital, 475 Seaview Avenue, Staten Island, NY 10305, USA
Email: AK* - anurag rk@hotmail.com; SJ - sjadonath@siuh.edu; NM - dsallery1@verizon.net
* Corresponding author

Published: 27 May 2009
Received: 28 January 2009
Accepted: 16 March 2009
Cases Journal 2009, 2:7147 doi: 10.1186/1757-1626-2-7147
This article is available from: http://casesjournal.com/casesjournal/article/view/7147
© 2009 Kushawaha et al; licensee Cases Network Ltd.
This is an Open Access article distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/3.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Abstract
West Nile Virus is one of the most frequently reported etiologies of viral encephalitis in the USA. West Nile Virus infections among hospitalized patients manifests most commonly as neuro-invasive disease. West Nile Virus has also been reported to cause myocarditis. Arrhythmia is not an uncommon occurrence in viral myocarditis. As cases of West Nile Virus increase, it is important that the index of suspicion also increase for this uncommon complication. Physicians who are caring for West Nile Virus-infected patients need to be aware of the possibility of West Nile Virus-related myocarditis. The question arises whether a patient with an established diagnosis of West Nile Virus-related encephalitis should be under continuous cardiac monitoring, bearing in mind the rare, but fatal, complication of cardiac arrhythmia secondary to viral myocarditis. We present a case report of a 65-year-old man who initially presented with fever, blurry vision, and decreased oral intake who subsequently suffered a fatal arrhythmia; further laboratory tests and autopsy findings revealed the patient likely had developed encephalitis and myocarditis secondary to West Nile Virus infection.

Case presentation
A 65-year-old man of Scottish-American descent presented to the emergency department (ED) in late summer 2008 complaining of fever, cough, and decreased oral intake for the past seven days. His symptoms were associated with blurry vision, nausea, and weakness. Past medical history included diabetes and hypertension. He was a former smoker (quit 4 years ago) and denied travel history or sick contacts.

In the ED, he was noted to be alert, conversant, and oriented. His temperature was 100.8 F, blood pressure was 133/76 mmHg, and heart rate was 104 bpm. Physical examination was unremarkable. The eyes were normal to inspection with normal visual acuity, extraocular movements were intact, with no evidence of scleral icterus or conjunctival erythema. Admission labs were notable for a sodium level of 125.2 mEq/L (reference range 135–146 mEq/L) and creatinine of 1.6 mg/dL (reference range 0.7–1.5 mg/dL). The WBC count was 7.6 × 10³/µL (reference range 4.8–10.8 × 10³/µL). Chest roentgenogram demonstrated no acute infiltrates. Electrocardiogram (EKG) showed normal sinus rhythm at a rate of 83. Blood cultures were
drawn and the patient was started on intravenous ceftriaxone and azithromycin.

On day 1, the patient became slightly confused and disoriented at times. He still complained of blurry vision and was noted to have a fever of 103.4 F. Antibiotics were changed to ceftriaxone, vancomycin, and the antiviral acyclovir was also added.

On day 2, computed tomography of the head showed chronic inflammatory changes with no acute bleed, mass effect, or shift. Lumbar puncture was conducted and revealed colorless cerebrospinal fluid (CSF), 4 RBCs/mm³, 29 WBC/mm³ (68% lymphocytes, 31% neutrophils, 1% monocytes), glucose of 115 mg/dL (serum level, 219), and a protein of 195 mg/dL. Bacterial and fungal stains were negative. Herpes simplex virus (HSV) polymerase chain reaction (PCR) of the CSF was negative. Serum Lyme antibodies were negative. Blood cultures were negative. Vancomycin was stopped, while ceftriaxone and acyclovir were continued. A transthoracic echocardiogram revealed ejection fraction of 50–55% with abnormal left ventricular relaxation and a dilated left atrium. The serum sodium level was noted to be 128 mEq/L.

On day 3, the patient was noted to be lethargic and disoriented with a fever of 104.5 F. An electroencephalogram demonstrated abnormal moderate generalized background slowing consistent with diffuse encephalopathy. Antimicrobials were continued.

On day 4, during morning rounds, the patient was his usual self, but continued to be lethargic, yet arousable and responsive to questions, with no new complaints. Ten minutes later, he was found unresponsive and pulseless. The electrocardiogram revealed asystole. As the patient was not on telemetry monitoring nor was a routine electrocardiogram ordered for that morning, there was no other electrocardiogram to compare, besides the admission EKG. Cardio-pulmonary resuscitation was unsuccessful.

Subsequently, the CSF that had been sent to the Board of Health, was reported to be positive for West Nile Virus (WNV) genome by PCR. IgM antibodies for WNV were also reported positive in the CSF. Serum IgM antibodies for WNV were not carried out. An autopsy was conducted. The central nervous system examination revealed microglial nodules, sparse perivascular lymphocytic infiltrate, and focal leptomeningeal monovascular infiltrate consistent with viral meningoencephalitis. West Nile Virus genome was also detected in the brain tissue by PCR. The cardiovascular examination demonstrated global slowing consistent with diffuse encephalopathy. The presence of neurologic involvement, positive WNV genome by PCR from the CSF and brain, and absence of other major viral infections, supports that WNV-associated myocarditis causing a fatal arrhythmia as the likely cause of the patient’s demise. The implications of this case are significant. West Nile Virus is one of the most frequently reported etiologies of viral encephalitis in the USA. West

Arrhythmia is not uncommon in viral myocarditis, including supraventricular tachycardia, atrial ectopic tachycardia, ventricular premature beats, ventricular tachycardia, and ventricular fibrillation [1]. Friedman et al., described 12 patients (ten patients < 18 years old), mostly without obvious myocarditis, who had biopsy findings consistent with myocarditis. Of these, eleven had ventricular tachycardia and one had multiform ventricular premature beats [2]. Wiles et al., made similar observations of 33 patients evaluated for ventricular ectopic rhythm using endomyocardial biopsy, three had focal lymphocytic myocarditis [3]. Take et al., described nine adults with complete heart block with viral myocarditis, which was permanent in two cases [4]. Currently, the “gold standard” for myocarditis is endomyocardial biopsy which unfortunately has low sensitivity and specificity [5]. There have been numerous case reports of myocarditis secondary to WNV in several mammalian species [6] and birds [7], indicating a predilection for myocardial involvement. These findings include multi-focal myocardial necrosis and lympho-histiocytic myocarditis [8]. Although the WNV genome PCR from the myocardium was negative, WNV myocardial involvement has only been described in few autopsy reports [9]. During a 1999 WNV epidemic in Russia, hydropericarditis with flabbiness of the cardiac muscle was described among 40 fatal cases. However, the number of patients with these cardiac findings and their ages were not specified [10]. Cardiac sequelae have been reported after infections with other flaviviruses [11].
Nile Virus infections among hospitalized patients manifest most commonly as neuro-invasive disease [12]. In recent outbreaks of WNV, overall mortality has been shown to be higher for the elderly, the presence of profound weakness, deep coma, failure to produce IgM antibody, impaired immunity, and coexisting illnesses, such as hypertension and diabetes mellitus [13] (both of the latter conditions the patient had.) As cases of WNV increase, it is important that the index of suspicion also increase for this uncommon complication. Physicians who are caring for WNV-infected patients need to be aware of the possibility of WNV-related myocarditis. The question arises whether a patient with an established diagnosis of WNV-meningoencephalitis should be under continuous cardiac monitoring, bearing in mind the rare, but fatal, complication of cardiac arrhythmia secondary to viral myocarditis. Also, WNV should be considered with any case of acute myocarditis, particularly during the summer and fall when mosquitoes are most prevalent.

List of abbreviations
F, degrees Fahrenheit; mmHg, millimeters of mercury; bpm, beats per minute; mEq/L, milliequivalents per liter; mg/dL, milligrams per deciliter; /μL, cells per microliter; RBC/mm³, red blood cells per cubic millimeter; WBC/mm³, white blood cells per cubic millimeter; WNV, West Nile Virus; PCR, Polymerase Chain Reaction; CSF, Cerebrospinal fluid; ED, Emergency Department; EKG, Electrocardiogram; HSV, Herpes simplex virus.

Consent
Written informed consent was obtained from the patient’s next of kin/ family for publication of this case report. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests
The authors declare that they have no competing interests.

Authors’ contributions
AK conducted literature review and interpreted the patient data regarding WNV encephalitis and was a major contributor in writing the manuscript. SJ reviewed the patient’s course while in the hospital. NM participated in care of the patient as the infectious disease consultant and was a major contributor to the manuscript.

References
1. Garson A, Smith RT, Moak JP et al.: Incessant ventricular tachycardia in infants. J Am Coll Cardiol 1987, 10:619-626.
2. Friedman RA, Kearney DL, Moak JP et al.: Persistence of ventricular arrhythmia after resolution of occult myocarditis in children with ventricular ectopic rhythm. J Am Coll Cardiol 1994, 24:780-783.
3. Wiles HB, Gillette PC, Harley RA et al.: Cardiomyopathy and myocarditis in children with ventricular ectopic rhythm. J Am Coll Cardiol 1992, 20:359-362.
4. Take M, Sekiguchi M, Hiroe M et al.: Long term followup of EKG findings in patients with acute myocarditis proven by endomyocardial biopsy. Jpn Circ J 1982, 46:1227-1234.
5. Feldman AM, McNamara D: Myocarditis. N Engl J Med 2000, 343:1388-1398.
6. van der Meulen KM, Pensaert MB, Nauwynck Hj: West Nile virus in the vertebrate world. Arch Viral 2005, 150:637-657.
7. Gibbs SE, Ellis AE, Mead DG, Allison AB, Moulton Jk, Howerth EW, Stallknecht DE: West Nile Virus detection in the organs of naturally infected blue jays (Cyanocitta cristata). J Wildl Dis 2005, 41:354-362.
8. Wunschmann A, Shivers J, Carroll L, Bender J: Pathological and immunohistochemical findings in American crows naturally infected with West Nile virus. J Vet Diagn Invest 2004, 16:329-333.
9. Omalu BI, Shakir AA, Wang G, Lipkin WI, Wiley CA: Fatal fulminant pan-meningo-polioencephalitis due to West Nile virus. Brain Pathol 2003, 13:465-472.
10. Platonov AE, Shipulin GA, Shipulina OY et al.: Outbreak of West Nile Virus infection, Volgograd Region, Russia, 1999. Emerg Infect Dis 2001, 7:128-132.
11. Obeyesekere I, Hermon Y: Arbovirus heart disease: Myocarditis and cardiomyopathy following dengue and chikungunya fever. Am Heart J 1973, 85:186-194.
12. LoRanee B, Tsuchida T, Hans S: Meningoencephalitis in a child complicated by myocarditis, quadriaparesis and respiratory failure. Pediatr Infect Dis J 2006, 25:853-856.
13. Nash D, Mostashari F, Fine A et al.: The outbreak of West Nile Virus infection in the New York City area in 1999. N Engl J Med 2001, 344:1807-1814.

Do you have a case to share?
Submit your case report today
• Rapid peer review
• Fast publication
• PubMed indexing
• Inclusion in Cases Database

Any patient, any case, can teach us something

www.casesnetwork.com