Idioventricular Rhythm in a Case of West Nile Encephalomyelitis

Ronald Espinosa[1*], **Kimberly Schelb**[2] **and Antonio Liu**[3]

[1]*Department of Family Medicine, California Hospital Medical Center/University of Southern California, USA*

[2]*Ross University School of Medicine, USA*

[3]*Department of Neurology, Loma Linda School of Medicine, USA*

[*]**Corresponding author:** Ronald Espinosa, Department of Family Medicine, California Hospital Medical Center/University of Southern California, Los Angeles, California, USA, Tel: 12137482411; E-mail: ronald.f.espinosa@gmail.com

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Abstract

Background: *West nile virus* (WNV), since its first appearance in the USA in 1999 has been a leading cause of viral encephalitis. There is one case report of associated fatal arrhythmia.

Case: Presented is a 65 year old Hispanic male without significant past medical history presenting with West Nile Encephalomyelitis with subsequent quadriplegia and developed recurrent idioventricular rhythm requiring a permanent pacemaker.

Conclusion: In the setting of West Nile Encephalomyelitis, cardiac monitoring for idioventricular rhythms and cardiac intervention should be considered.

Keywords: *West nile virus*; Idioventricular; Arrhythmia; Myocarditis; Encephalopathy; Pulseless electrical activity

Case Presentation

65 year old Hispanic male without known significant past medical history presents with 3 weeks of delayed mentation and body aches that began after attending an outdoor swap meet in hot weather. He developed a severe headache that resolved with acetaminophen but he became more lethargic over the next two weeks. The patient was found disoriented by family staring at himself in the mirror. Initial review of symptoms was positive for nonproductive cough, nonbloody diarrhea and nausea. During the initial hospital admission, he developed signs of meningitis and sepsis including photophobia, headache, fever and nuchal rigidity as well as acute hypoxia. Discussion with patient and family he is typically an active individual with normal exercise tolerance. The patient's medical and surgical history was negative. Family history was notable for coronary artery disease in his father and uterine cancer in his mother. There was no recent travel or known sick contacts. The initial physical exam was notable for slow responses to questions and mild bilateral lower extremity edema. Subsequent neurological exam notable for improved orientation from 2-3 with normal gait, muscle strength, and muscle sensation testing. He was placed on meningitic doses of ceftriaxone, vancomycin and acyclovir. Lumbar puncture was unsuccessful secondary to patient agitation. The Initial laboratory evaluation notable for mild transaminitis. Chest roenterogram demonstrated no acute findings.

Electrocardiogram showed sinus tachycardia. He was intubated for acute respiratory failure and airway protection which was thought to be secondary to aspiration pneumonia and encephalopathy. Computed tomography angiography was negative for pulmonary embolism. The patient was stabilized and transferred to another hospital for insurance purposes two weeks later. Magnetic Resonance Imaging of the brain was performed and was negative for acute intracranial process. WNV serum titers of IgG and IgM were positive, 6.76 and 6.44 respectively. Dengue, syphilis, coccioidomycosis, cryptococcus and Guillain Barre testing were negative. He developed acute renal failure and eventually required dialysis which was likely secondary due acute tubular necrosis from ischemia from the initial hypotensive insult at the first hospital. The patient's neurologic status worsened with development of quadriparesis shortly after transfer. His hospital course was stable until about 4 weeks after the initial presentation, when he developed idioventricular rhythm with rate approaching the 30's, which resolved immediately upon suctioning.

Chest roentgenogram, electrocardiogram and arterial blood gas were negative. Basic metabolic panel was essentially unchanged. One week later, the patient developed severe idioventricular episodes also to the 30's with sinus pauses of up to 8 seconds occurring at 0317. A code blue was called for pulseless electrical activity and symptomatic bradycardia. Return of spontaneous circulation was achieved after 13 minutes with single doses atropine and epinephrine. Creatinine kinase and troponins were negative. Arterial blood gas was consistent with mild metabolic acidosis. Transthoracic echocardiogram showed hyperdynamic LV with estimated EF of 65%, and moderate RV dilation. Chest computerized tomography scanning was negative. There was no significant valvular disease. Given the severity and

resultant idioventricular rhythm requiring permanent pacing. Previous studies describe encephalitis having a 1030% mortality rate and limb strength recovery taking around 68 months usually resulting in persistent weakness [1,2]. Our patient demonstrates his encephalomyelitis improving at a much faster rate than previously reported, with muscle strength testing of his distal upper extremities improving from 0/5 to 3/5 at the end of his inpatient course. Given his neurological improvement during hospitalization, his signifcant cardiac symptomatology necessitated more active and aggressive cardiac monitoring and intervention.

Idioventricular rhythms are defned as three or more ventricular beats with a rate less than 100, but typically fall within a range 2040 beats per minute via automaticity of the ventricular myocytes. Some common causes are myocardial ischemia, infarction, sinoatrial failure, atrioventricular failure, cardiomyopathy, hyperkalemia, medication induced and hypoxia. Review of the patient's medical record did not show signifcant hypoxia. No calcium channel blockers or digoxin was given, but the patient was on a stable dose of metoprolol 25 milligrams twice daily for two weeks without any episodes of bradycardia. The patient's electrolytes the day prior and afer the second code blue did not show hyperkalemia. Cardiac enzymes and electrocardiograms were negative on three occasions (Table 1).

Causes of Idioventricular Rhythm
Cardiac: Myocardial infarction, myocardial ischemia, sinoatrial or atrioventricular failure/block, cardiomyopathy
Metabolic: Hyperkalemia
Medication: Digoxin, nodal blocking agents
Respiratory: Sleep apnea, hypoxia
Idiopathic

Table 1: Causes of idioventricular rhythm [10].

Since the migration of the WNV to New York in 1999 as reported by Roehrig [3], the Centers for Disease Control have documented a total of 41762 cases with 1765 deaths [4]. There have also been reports of hypercapnic respiratory failure in the setting of WNV requiring intubation [5] (Figure 1).

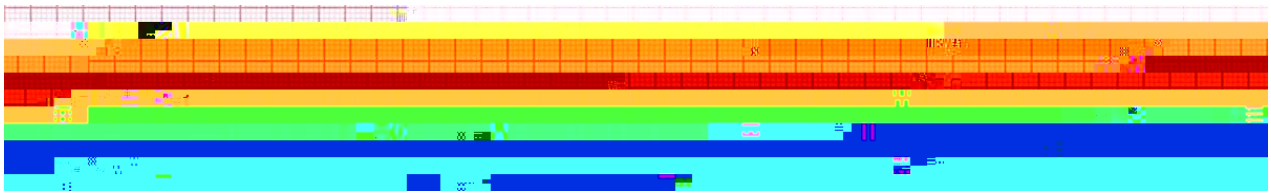

Figure 1: Patient's telemetry strip on admission.

Myocardial involvement has been demonstrated in mammals [6]. It has been reported that cardiopulmonary manifestations were the most common complication of WNV that resulted in death [7]. Kushawaha et al. [8] describe a case report of WNV myocarditis causing a fatal arrhythmia in 2009. In a WNV outbreak in Russia in 1999, there were reported 40 postmortem cases of myocardial involvement [9,10].

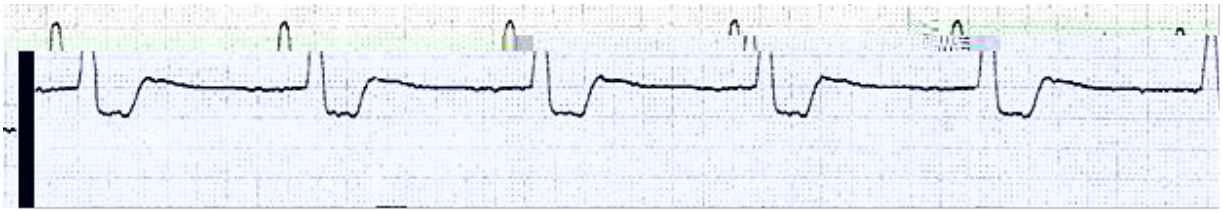

Figure 2: Patient's telemetry strip of idioventricular rhythm.

Further studies are needed to determine if WNV affects not only myocardium but the sinoatrial and HisPurkinje systems (Figure 2).

A team based approach to active cardiac monitoring and aggressive intervention should be considered in cases of WNV, as well as awareness of the possible rapid improvement of WNV encephalomyelitis within the acute hospital stay.

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