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Isolated Hyperacute T-Waves in West Nile Encephalitis Indicating Atypical Variant of Stress-Induced Cardiomyopathy

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Abstract

Several cardiac outcomes have been reported with West Nile-encephalitis; however, the underlying pathophysiology remains complex. We present a 42-year-old female, with multiple sclerosis, whose neurological symptoms and respiratory decline were finally explained by the diagnosis of West Nile-encephalitis. During her admission, the isolated peaked T-waves indicated the underlying stress-induced cardiomyopathy. The absence of all other causes of hyperacute T-waves, their subsequent resolution with the resolution of infection and improvement in wall motion abnormalities, further supported the association. This case highlights the importance of considering hyperacute T-waves in an approach towards the diagnosis of WNV-encephalitis related atypical variant of stress-induced cardiomyopathy.

Keywords

West Nile Virus encephalitis, WNV, Hyperacute T-Waves, Takotsubo Cardiomyopathy, Atypical/Inverted Variant of Stress-Induced Cardiomyopathy, CMP

1. Introduction

West Nile virus (WNV) has been established to present with a spectrum of cardiac manifestations, including atrial fibrillation and atrioventricular (AV) node block in the setting of myocarditis and autonomous dysfunction [1]. The association of sinus bradycardia and a prolonged corrected QT (QTc) interval resulting in asystole has also been reported with WNV encephalitis [2]. Hyperacute T-waves have been associated with acute transmural myocardial infarction, hyperkalemia, LVH, and in some cases acute myopericarditis [3], however, their

significance with an atypical (inverted) variant of stress-induced cardiomyopathy or WNV infection has never been established. We present a case with EKG findings of hyperacute T-waves in the setting of West Nile Virus encephalitis, highlighting one of the possible clues to the diagnosis of stress-induced cardiomyopathy in critically ill patients

2. Case Presentation

A 42-year-old female with a history of seizures, multiple sclerosis, migraines, and anxiety presented to the emergency department with progressive left upper extremity weakness, lower extremity myalgias and altered mental status for 2 days, accompanied by headache and low-grade fever. She denied any history of sick contacts, traveling or changes in her medications.

2.1. Vitals and Physical Examination

On presentation, her blood pressure was 122/60 mmHg, a heart rate of 67 beats per minute, and saturating 99% on room air. Neurological examination revealed akathisia, decreased tone in the left lower extremity, 2+ reflexes in left upper extremity, 4+ patellar and mute plantar reflexes bilaterally. Ankle clonus noted in both lower limbs. Kernig's and Brudzinski's sign was negative. The rest of the examination was unremarkable.

2.2. Investigations and Work-Up

An electrocardiogram (EKG) on day one showed a normal sinus rhythm and normal intervals. Labs were notable for hypokalemia (potassium 2.9 mmol/l), thrombocytopenia (Platelets 93000/mcl) and elevated C-reactive protein (CRP), other work-up as shown in Table 1. Chest x-ray revealed mild pulmonary venous congestion with elevated left diaphragm.

2.3. Clinical Course

Neurology was consulted in the emergency department for the evaluation of stroke given left upper extremity weakness, which was ruled out with normal brain imaging. She was restless and agitated a night earlier and was kicking in bed while asleep. These findings along with hyperthermia and the use of multiple serotonergic medications including buspirone, fluoxetine, and trazodone raised concerns for serotonin syndrome. The patient had not been taking any medication for multiple sclerosis or headaches for a few years. Upon admission, all serotonergic medications were held, meanwhile, levetiracetam was resumed for seizure prophylaxis.

Given the constellation of symptoms with a 2-day onset into acute left arm pain and weakness, altered mentation, and akathisia, an infectious process was brought into consideration. A lumbar puncture was performed and based on the findings suggestive of viral meningitis, IV acyclovir was started. The Infectious Disease team was taken on board with concerns of aseptic meningitis, however,

Table 1. Pertinent workup during hospitalization.

Initial investigations:	
CT scan	Stable periventricular white matter changes
Electroencephalogram	A moderate nonspecific generalized cerebral dysfunction. There were no epileptiform discharges or seizures recorded.
MRI brain	Increased demyelinating plaques since prior scan in 2015
MRI cervical spine	Numerous new demyelinating plaques throughout the cervical spine
MRI upper extremities	There is bilateral symmetric muscle edema without fatty infiltration/atrophy.
Upper limb arterial doppler	Normal left arm pressures and waveforms.
Total Lymphocyte	8400/mcl
ESR	12
CRP	6 mg/l
Urine toxicology	Cannabinoids positive
CSF analysis	Elevated protein and WBC count of 128 (53% PMNs, 42% lymphocytes). Lyme CSF, CSF VDRL were negative. CSF ACE levels within normal limits.
CSF Myelin basic protein	67.90 ng/mL
Lyme antibodies, HIV antibodies	Negative
Serum Vit B1, Vitamin B12, aldolase, serum copper, Serum immunoglobulins	Within normal limits
Immunological work-up	Negative
Blood, urine and sputum cultures	Negative
Cardiac work-up	
ECHO (Day 4)	LVEF 50%. Moderate basal hypokinesis with preserved to hyper-contractile apical function. Normal RV systolic function, as shown in Figure 2(a).
ECHO (Day 14)	EF 55% - 60%, mildly hypokinetic segments in basal region (improved from previous study), Figure 2(b).
High sensitivity troponin	34 pg/ml → 37 pg/ml → 47 pg/ml

given thrombocytopenia, Lyme disease work-up including CSF antibodies was ordered and doxycycline was started.

For the increased plaque burden on MRI and IgG in CSF, a 5-day course of intravenous immunoglobulin (IVIG) was started by neurology for MS flare and acyclovir was discontinued on day 3 after the CSF Biofire was negative for HSV. Negative Lyme serologies led to doxycycline discontinuation. On day 4 of admission, the patient was upgraded to the ICU due to worsening mentation and inability to protect the airway. She developed acute hypoxic respiratory failure, which was thought to be secondary to hospital-acquired pneumonia versus aspiration, given nasogastric tube was placed a day earlier and a repeat chest X-ray showed worsening bibasilar infiltrates (Figure 1). Antibiotics were escalated and the patient was intubated.

With no evidence of clinical and neurological improvement, a 5-day course of methylprednisolone was initiated by neurology for autoimmune pathology. The

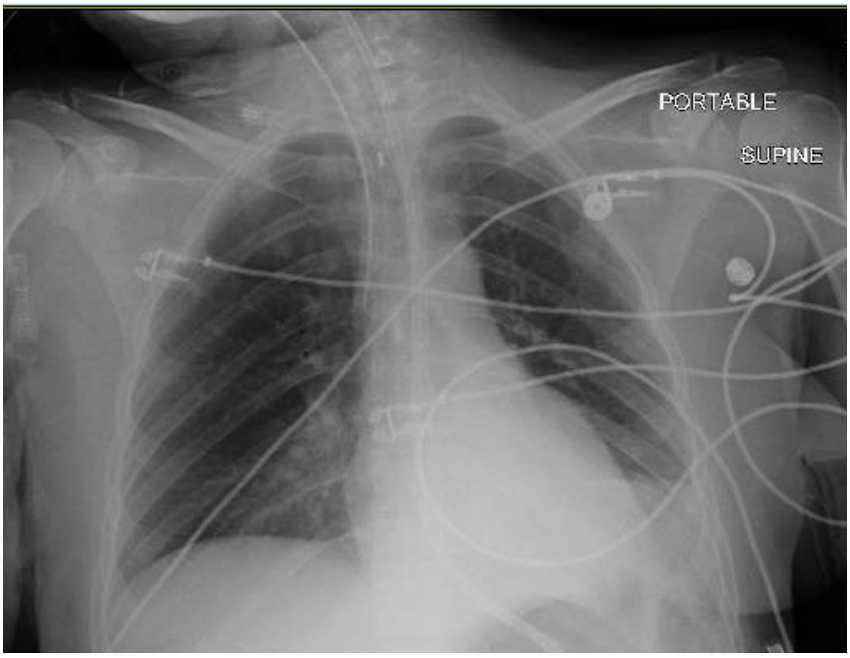

Figure 1. Chest X-ray with worsening bibasilar infiltrates.

patient remained in the ICU, and multiple failed attempts at extubation were made, with subsequent tracheostomy formation. The mentation had improved by that time.

On day 12 of admission, the neurological exam was found consistent with a cervical cord lesion given normal mental function and intact cranial nerve examination, quadriparesis, (upper limbs affected more than lower limbs), lower motor signs in her upper limbs with hypotonia, and hyporeflexia and upper motor neuron signs in lower limbs with hypertonia, hyperreflexia, and upgoing plantar bilaterally. Difficulty in weaning from mechanical ventilation was also thought to be because of the cervical lesion affecting the diaphragm. MRI cervical spine was ordered, which showed some improving inactive demyelinating plaques. MRI of the brachial plexus bilaterally was also unremarkable. The serum antibodies for neuromyelitis optica were reported negative as well.

The etiology of the clinical presentation still remained unclear. It was this time when West Nile serology was sent, which came out positive on day 15 of admission. It was finally concluded that patient had acute encephalopathy and myelitis secondary to neuroinvasive West Nile virus in the context of positive IgM and IgG antibodies in the serum. However, patient's clinical condition had significantly improved as compared to the initial presentation and she was down-graded to intermediate care with supportive care, pressure support during the day and full vent overnight. She was transferred to long-term acute care facility on day 19 of admission, with subsequent complete recovery after a few weeks.

2.4. Case Highlight

During the ICU stay, patient was found to have persistent asymptomatic brady-

cardia with hyper-acute T waves (Figure 2). This led to further cardiac work-up (Table 1), with transthoracic echocardiogram (TTE) on day 4 revealing basal variant of stress-induced cardiomyopathy (Figure 3). On hospital Day 18, with improvement in neurological deficits and clinical improvement, there was complete resolution of hyperacute T waves on the EKG findings (Figure 4). The repeat TTE showed improved wall motion (Figure 5).

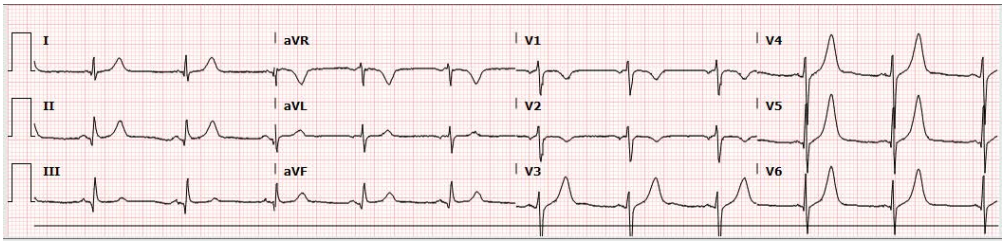

Figure 2. EKG on day 13, hyper-acute T waves.

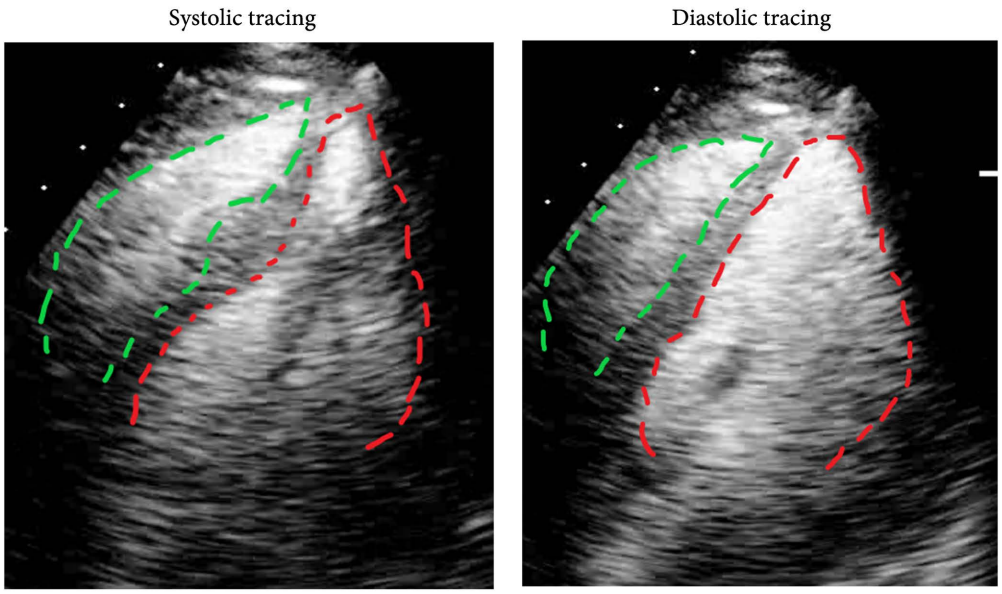

Figure 3. Initial echocardiogram on admission. Echocardiogram with left ventricular (red dotted line) and right ventricular tracing (green dotted line).

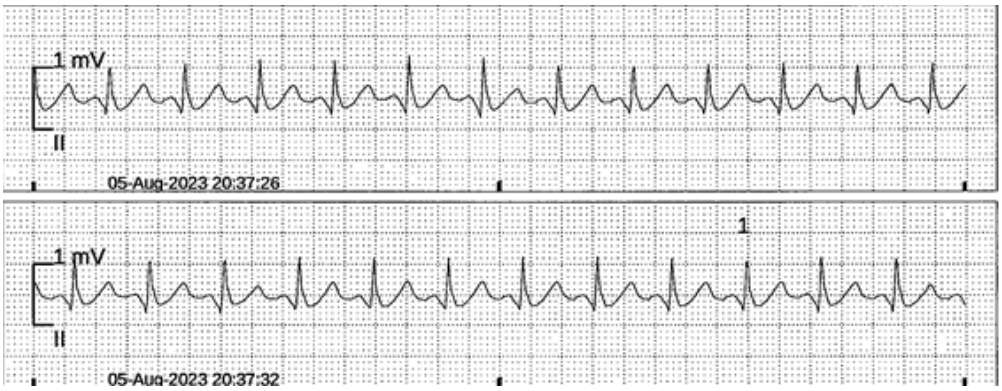

Figure 4. Resolution of hyper-acute T waves on day 20 of admission.

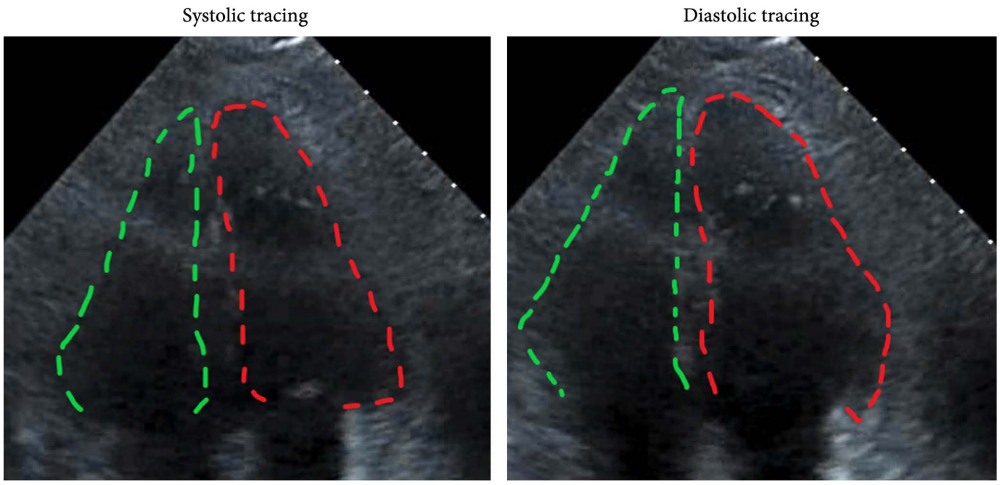

Figure 5. Repeat echocardiogram 14 days after admission.

3. Discussion

Our patient presented with a unique clinical presentation of West Nile encephalitis and myelitis, the presence of west nile IgM antibodies in the serum indicated acute infection, which had a prolonged, yet self-resolving course.

Cardiac involvement is not uncommon in patients with viral encephalitis. Some viruses have been associated with relative bradycardia [4], however, the ability of West Nile virus to affect the sympathetic spinal ganglia has been related to sinus bradycardia and heart block. WNV has also been associated with a wide range of tachyarrhythmia, the persistence of which can need long-term anti-arrhythmic therapy [5]. It has also been recognized to have idioventricular rhythm requiring a permanent pacemaker [6]. With arrhythmias WNV has also been found to be associated with cardiomyopathy [7], however, apical variant of takotsubo cardiomyopathy has seldom been reported. With all these constellations of cardiac manifestations, isolated T wave changes have not been recognized with West Nile virus encephalitis earlier.

Hyperacute T waves have commonly been associated with ST-segment elevation myocardial infarction [8], left ventricular hypertrophy, acute myocarditis, acute pericarditis, and hyperkalemia [9] [10]. Not all hyperacute T waves are pathological or indicative of ischemic pathogenesis or hyperkalemia, they are benign in the early phases of repolarization [11]. The most common EKG findings with takotsubo CMP were found to be ST-segment elevation and T inversion mostly in the precordial leads [12]. Hyperacute T waves, however rarely, are associated with stress-induced CMP.

In our case, the patient began to have a sudden onset of asymptomatic bradycardia with prominent tall peaked T waves several days later after the initial presentation. When the attention was driven to the significantly prominent T-waves, a broader differential was thought given the worsening respiratory status. In the absence of typical symptoms, and cardiac enzyme elevation, it was unlikely for this patient to have a transmural infarction. Along with excluding

ischemic etiology, electrolyte imbalance was keenly monitored with daily labs. Although she presented initially with moderate hypokalemia and hypomagnesemia, she was never found to have electrolyte imbalance concomitantly with the appearance of EKG changes. A thorough medication review did not reveal any medication that could have been thought to contribute to her bradycardia or EKG changes. The transthoracic echocardiogram did not reveal significant left ventricular hypertrophy and there was the absence of pericardial effusion to suggest pericarditis, however, the basal hypokinesis with hypercontractile apex was indicative of an apical sparing variant of basal (inverted) stress-induced cardiomyopathy.

Although a lot has been known about WNV and its cardiac manifestations, our case reflects the unique electrophysiological presentation with isolated peaked T waves, which indicated the underlying atypical variant stress-induced cardiomyopathy associated with infection. The basal or inverted variant is found in <5% of patients and is usually not associated with isolated hyperacute T waves on EKG or severe hemodynamic compromise [13]. This provides an insight into one of the several cardiac manifestations of WNV encephalitis, which should be considered in such diagnostically challenging cases. Our case also highlights the fact that hyperacute T waves should raise suspicion for stress-induced cardiomyopathy in the absence of other causes. Stress-induced cardiomyopathy should be considered a differential diagnosis for such ECG changes in patients with West Nile Virus infection. The hyperacute T-waves resolved with the resolution of infection in our case.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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