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Postherpetic Neuralgia Induced Asystole - A Deadly Consequence of a Common Disease

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Abstract

Postherpetic neuralgia is a well-studied and somewhat common sequala of herpes zoster leading to intense pain episodes. Pain-induced vasovagal episodes can rarely lead to asystole, which is a fatal potential complication. It is crucial to have a high clinical suspicion of asystole when a patient presents with severe herpetic neuralgia leading to syncope. This is a unique cause of a 67-year-old Caucasian female who presented with syncope caused by severe postherpetic neuralgia with the eventual finding of pain-induced asystole episodes.

Keywords: *Postherpetic neuralgia, herpes zoster, cardiac pauses, asystole*

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Introduction

Herpes Zoster is a clinical condition caused by the reactivation of latent varicella zoster virus following initial infection. Postherpetic neuralgia is a neuropathic sequela of herpes zoster characterized by pain that can persist for months to years after the resolution of the initial herpes zoster rash. It is estimated that approximately 1 million cases of herpes zoster occur annually in the US and 5-20% of people experience postherpetic neuralgia as a sequelae.[1,2] The risk of postherpetic neuralgia increases with age, occurring in 20% of people aged 60-65 years who have had acute herpes zoster and more than 30% of people aged 80 years or older.[3] The intense pain characterized in postherpetic neuralgia can lead to vasovagal episodes causing syncope and other responses with less clear pathogenesis such as pain-induced asystole. Recognizing this rare but possible sequela of postherpetic neuralgia is vital for the timely management of a potentially life-threatening condition.

Case Report

We report a case of a 67-year-old Caucasian female with a history of asthma, prior TIA, and shingles who presented to our facility after a syncopal episode. The patient reported she had recently been diagnosed with shingles and had been treated with valacyclovir and prednisone. Since the time of initial diagnosis, she had been experiencing intermittent episodes of severe postherpetic neuralgia which was being treated with gabapentin.

The patient presented to our facility following a syncopal event following a severe postherpetic neuralgia episode in her left upper face, leading to loss of consciousness. As a result of the episode, she sustained a head laceration with various nasal and spinal fractures. A computer tomography scan of the head revealed no acute intracranial processes. Vital signs were stable upon admission and labs were significant for WBC 11.7.

During the hospital admission, the patient was found to experience sustained episodes of asystole ranging from 6 to 10 seconds but with no associated loss of consciousness. It was found that these paroxysms coincided in chronicity with the patient's severe postherpetic neuralgia episodes. Following the asystole episodes and resolution of her pain, she would return to normal sinus rhythm according to telemetry monitoring. Of note, this patient's pain episodes with subsequent asystole exclusively transpired at night during her hospitalization. The neurology team was consulted, and seizure etiology was ruled out. The dose of gabapentin was increased, and narcotics were added to achieve adequate pain control. Despite controlling baseline pain, the patient continued to experience episodes of severe

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postherpetic neuralgia and asystole. Sustained cardiac pauses, oftentimes sustained for longer than 10 seconds, prompted a recommendation from Cardiology for placement of a temporary pacemaker. To ensure safe discharge and avoid catastrophic outcomes caused by asystole such as cardiac arrest resulting in death, a temporary pacemaker and close follow-up was scheduled. The pacemaker was removed 2 weeks later and a 14-day Holter monitor was placed to confirm the cessation of asystole episodes. According to continued cardiology follow-up, the patient did not experience further pain episodes and subsequently no further episodes of asystole associated with postherpetic neuralgia.

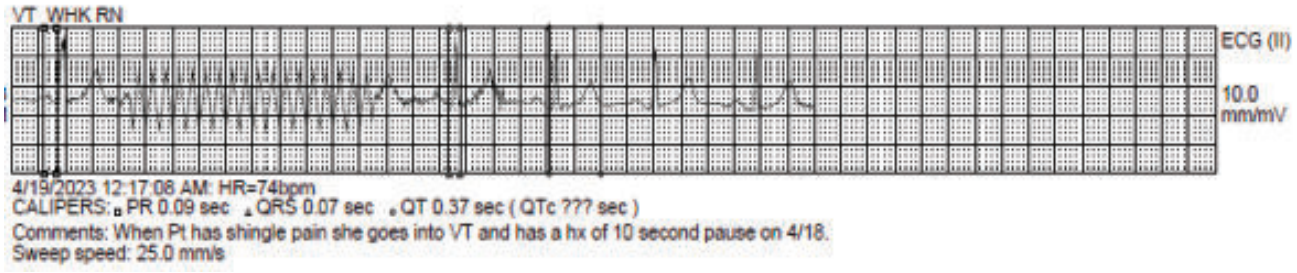

Figure 1: Telemetry reading showing the onset of ventricular tachycardia during pain episode.

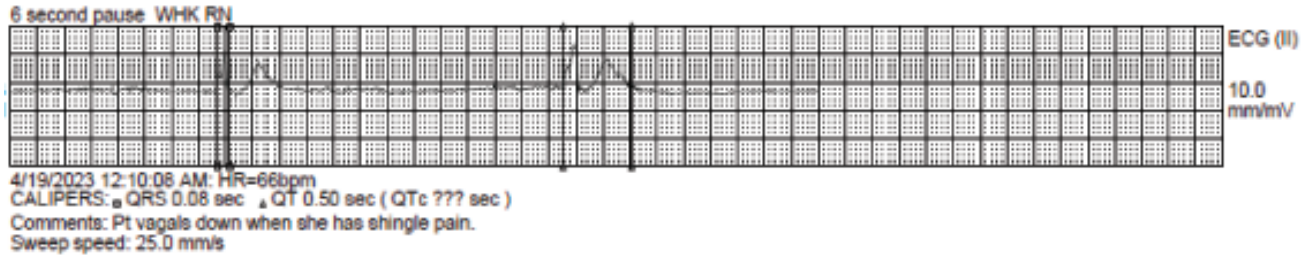

Figure 2: Telemetry reading demonstrating 6 seconds of sustained asystole caused by postherpetic neuralgia-induced pain with the return to normal sinus rhythm.

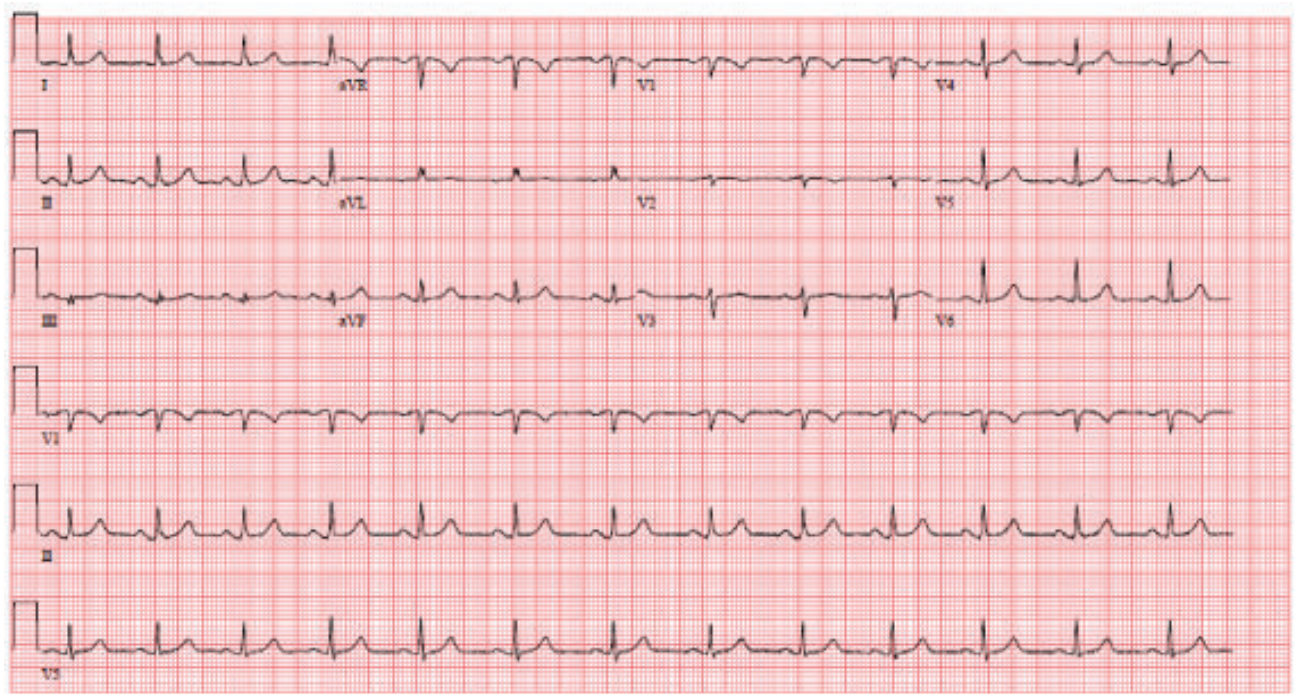

Figure 3: *EKG following placement of temporary pacemaker showing the regular rate and normal sinus rhythm.*

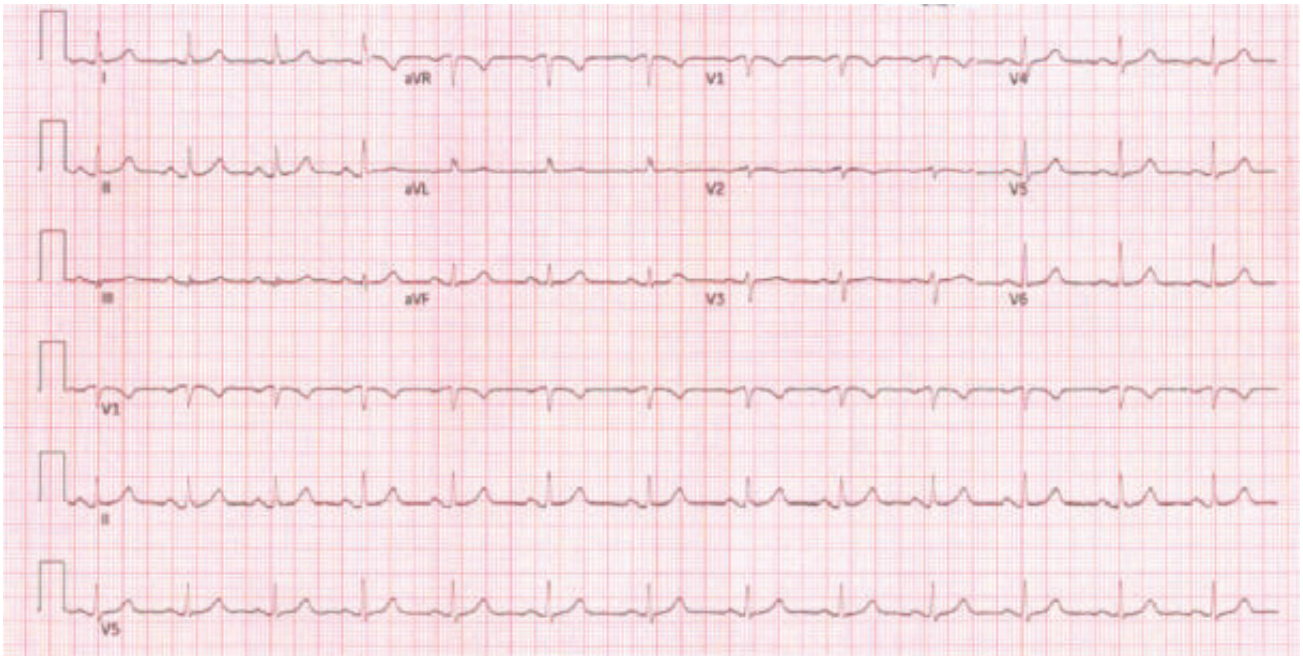

Figure 4: *EKG one week prior to removal of pacemaker showing the regular rate and normal sinus rhythm.*

Discussion

Postherpetic neuralgia is a painful consequence of acute herpes zoster infection. Acute herpetic neuralgia is pain that occurs within 30 minutes of rash onset. The term herpetic neuralgia is used to describe pain that persists 120 days after the initial onset of the rash. [4] No clear direct association has been found between post-herpetic neuralgia and asystole although an indirect explanation would account for this. The mechanism involves reflex syncope leading to bradycardia and in severe cases, asystole.

There are various possible mechanisms to explain pain-induced syncope. Reflex syncope can be further classified as vasovagal, situational, carotid sinus syncope, and atypical reflex syncope. Vasovagal syncope is the most common type. The cardioinhibitory mechanism induced by pain is

mediated by a surge of parasympathetic activation which in turn leads to bradycardia, pauses, or atrioventricular blocks. A vasodepressor mechanism leads to decreased activation of the sympathetic system, leading to hypotension. In our case, the mechanism of the patient's asystole episodes is thought to be due to episodes of intense pain leading to an exaggerated cardioinhibitory response resulting in asystole and cardiac pauses.

For cases such as these related to cardioinhibitory responses, treatment should first be directed towards controlling the pain to avoid initiation of the response. Recognizing prodromal symptoms is vital to prevent these episodes but oftentimes not always possible. If sufficient pain control is unable to be ascertained, additional measures such as a temporary pacemaker can be utilized to counteract the pain-induced syncope and asystole. A temporary transvenous cardiac pacemaker implantation was described first by Khero and Mullins in 1971 to treat the reflex cardiac syncope. [5]

In terms of permanent pacemaker implantation, the available literature is controversial as spontaneous recovery in postherpetic neuralgia is possible although the risk of relapses remains.[6] The decision for permanent pacemaker implantation should be an individualized decision dependent on the risk of fatal outcomes. In this case, a permanent pacemaker was deemed unnecessary as the patient's pain was well controlled with her home pain medication and she ceased to undergo prolonged episodes of asystole following discharge. A permanent pacemaker was not regarded to be needed while inpatient due to the patient's relatively young age and the fact that not every pain episode led to asystole.

In summary, a temporary pacemaker combined with adequate pain control should be considered as an initial treatment modality in patients with postherpetic neuralgia who begin to experience asystole and syncope. If the asystole episodes continue to persist, a permanent pacemaker combined with medical treatment may be the safest treatment option. Prompt diagnosis with appropriate subsequent management of this potentially life-threatening condition is paramount to avoid fatal outcomes.

Conclusion

Pain-induced asystole as a consequence of postherpetic neuralgia has the potential to lead to fatal outcomes, including death, if not met with prompt diagnosis and definitive management. Clinicians' awareness of this rare but highly fatal complication is imperative to prevent devastating outcomes. A multidisciplinary approach and early cardiology intervention are pivotal to ensure safe patient outcomes.

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