KANSAS JOURNAL of MEDICINE

Not Just a Rash: Herpes Zoster-induced Progressive Cardiac Block

Ahmad Mahdi, M.D.[1], Grace Nassim, M.D., MPH[1,2], Mahmoud Mahdi, M.D.[1], Freidy Eid, M.D., FACC[1,3]
[1]University of Kansas School of Medicine-Wichita, Wichita, KS Department of Internal Medicine
[2]Wesley Medical Center, Wichita, KS
[3]Cardiovascular Care, P.A., Wichita, KS

Received Feb. 14, 2023; Accepted for publication April 24, 2023; Published online June 20, 2023
https://doi.org/10.17161/kjm.vol16.19522

INTRODUCTION

Varicella-zoster virus (VZV) is a neurotropic virus that causes primary infection as chickenpox, which typically occurs in childhood, followed by a latent phase that can reactivate as shingles later in life.[1,2] After the primary infection, the virus spreads from epidermal and mucosal lesions to local sensory nerves. It stays latent afterwards in the dorsal ganglion cells of sensory nerves. However, viral reactivation can occur due to stress or immune deficiency which results in shingles.[3-5] Upon reactivation, it replicates in neuronal cell bodies, resulting in virions to shed from their carrier cells through the nerve to the skin area that is innervated by its ganglion. In the skin, it results in localized inflammation and blistering. The resulting pain is due to inflammation of the nerves affected by the virus.[6]

While VZV primarily affects the skin and nervous system, it has been implicated in cardiovascular complications such as heart block. Varicella-zoster virus (VZV)-related heart block is an infrequent but potentially serious complication of VZV infection.[7] The incidence of VZV-related heart block was estimated to be less than 1% of all VZV infections. However, the risk of developing this complication is increased in immunocompromised patients, such as those with HIV or undergoing chemotherapy, as well as in elderly individuals. Symptoms of heart block can include dizziness, fainting, shortness of breath, and chest pain, and anyone experiencing these symptoms should seek medical care immediately.[8]

This case is a report of a patient that presented with a progressive conduction abnormality in the setting of shingles reactivation.

CASE REPORT

A 59-year-old male with a past medical history of hypertension, dyslipidemia, chronic kidney disease, gastroesophageal reflux, and childhood infection with chickenpox presented to the emergency department for esophageal pain. The patient reported burning pain in his esophagus and chest. The pain began five days prior to presentation and radiated directly beneath his left breast. The patient's symptom onset was accompanied by shortness of breath with exertion. He denied palpitations, dizziness, or lightheadedness. The pain was associated with a reported red, itchy, burning rash without blisters over the left chest. He had not received the shingles vaccine and denied a history of shingles. Social history was unremarkable. There was no family history of premature sudden cardiac death.

On admission, the patient's heart rate was 38 bpm, blood pressure was 132/61 mmHg, respiration rate was 22 breaths/min, and oxygen saturation was 95% on room air. On physical examination, the patient had a left-sided thoracic vesicular rash on an erythematous base (Figure 1). Otherwise, his lungs were clear on auscultation, no cardiac murmur was detected, and no mucosal or skin lesions were found. Chest x-ray was unremarkable. He was given 2 mg of intravenous morphine, then 0.4 mg of sublingual nitroglycerine for his worsening chest pain. He was started on valacyclovir and placed under contact precautions. He was placed on telemetry.

High sensitivity troponin was negative twice during trending (49 ng/mL and 42 ng/mL, respectively). An electrocardiogram showed a second-degree heart block with 2:1 conduction and intermittent left bundle branch block (LBBB) with no signs of acute ischemia (Figure 2). During his stay, an echocardiogram showed normal ejection fraction of 65-70% with moderate concentric hypertrophy, moderate aortic stenosis, mildly dilated left atrium and grade 2 diastolic dysfunction.

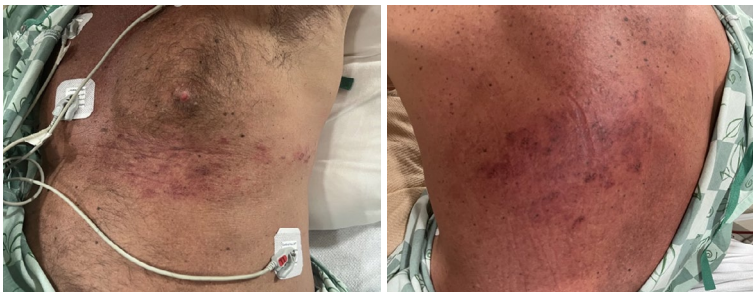

Figure 1. Vesicular rash was noted on an erythematous base in a dermatomal distribution.

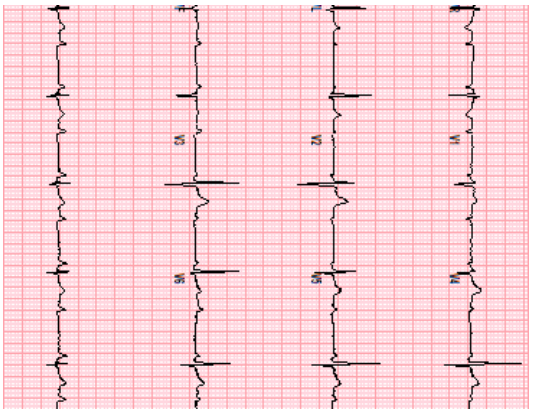

Figure 2. EKG revealed Type II Mobitz II AV Block with 2:1 conduction and intermittent LBBB with no signs of acute ischemia.

The patient had intermittent but frequent LBBBs during his hospitalization suggestive of progressive conduction system disease. Hence, he underwent a Micra™ pacemaker placement without any complications. The rest of the hospital stay was uneventful, and the patient was later discharged to follow-up as outpatient with his cardiologist.

DISCUSSION

The diagnosis of VZV-related heart block is based on a combination of clinical evaluation, electrocardiogram (ECG), and laboratory testing. Clinical evaluation may include a thorough physical examination to assess for symptoms of heart block, such as syncope, dizziness, palpitations, and shortness of breath.[7] The most common type of heart block seen in VZV-related heart block is the Mobitz type II or third-degree

AV block, which is characterized by a complete blockage of electrical impulses between the atria and ventricles. Other ECG findings may include bundle branch block, premature ventricular contractions, and ventricular tachycardia.[9]

Laboratory testing may include serological tests to detect the presence of VZV antibodies or polymerase chain reaction (PCR) assays to confirm the presence of VZV DNA in the blood or cerebrospinal fluid. Elevated levels of inflammatory markers, such as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR), also may suggest an active VZV infection.[10] In some cases, additional testing may be necessary to confirm the diagnosis and guide management. This may include a Holter monitor to assess for the presence of blockages.[11]

Mobitz type II second degree atrioventricular (AV) blocks are caused by a disease of the conduction system distal to the AV node (Bundles of His and Purkinje fibers).[12] Cells do not fatigue progressively, but rather unpredictably and abruptly fail to conduct the supraventricular impulse. The cause can be functional or anatomical.[13,14] Increased parasympathetic tone or disruption of the sodium channels can precipitate the block.[12]

VZV myocarditis could lead to cardiac conduction disturbances and supraventricular, later ventricular, arrhythmias.[15] Progressive decrease in inotropy after the beginning of zoster pain can culminate in bradycardia secondary to complete heart block.[16] A possible and coincident VZV reactivation may occur at both the dorsal root ganglion and the cardiac sympathetic ganglia although no direct neural or vascular connections exist between the two. The progression of conduction abnormality preservation of chronotropic response in our patient may reflect a partial/complete block of the cardiac sympathetic ganglion, or a weakening of resting sympathetic tone.[8] Since there are neither direct neural nor vascular connections between the dorsal root ganglia and the cardiac parasympathetic/sympathetic ganglia, VZV reactivation can occur at the dorsal root ganglion as well as the cardiac sympathetic ganglia. However, VZV-induced conduction pathologies may be reversible if the underlying condition is managed properly.[17]

Treatment for VZV-related heart block typically involves antiviral medications to treat the underlying VZV infection, as well as medication or procedures to manage the abnormal heart rhythm. Acyclovir is the most used antiviral drug, although valacyclovir and famciclovir also may be effective.[18] If the heart block is severe, a temporary or permanent pacemaker may be necessary to regulate the heart's electrical activity.[19]

Ma et al.[16] reported a case of complete heart block induced by VZV reactivation. The suggestive etiology was a persistent elevation of anti-varicella zoster virus IgM antibodies. This led to a progressive slowing of the heart rate following the onset of zoster pain resulting in bradycardia then complete heart block. The patient subsequently had a pacemaker placement. Our patient's VZV-related conduction abnormality may have manifested due to a similar pathophysiology.

CONCLUSIONS

VZV-related heart block is a rare but serious complication of VZV infection that can lead to an abnormal heart rhythm and potentially life-threatening complications. Diagnosis is based on a combination of clinical evaluation, ECG, and laboratory testing, and treatment involves antiviral medications and management of the abnormal heart rhythm. Early diagnosis and treatment are crucial to prevent serious complications and improve outcomes.

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Keywords: herpes zoster virus, shingles, AV block, cardiac arrhythmia, progressive cardiac conduction defect