Case Report

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Zona zoster; an atypical presentation with severe pre-lesion pain: a case report

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ABSTRACT

The Varicella Zoster virus can initially cause chickenpox in humans and then become latent. It can later reactivate for various reasons and manifest as shingles in dermatome areas, characterized by redness, rash, and neuropathic pain. In this case, we want to highlight a Zoster case with atypical symptoms resembling acute coronary syndrome, but without the presence of a rash. The patient is a 78-year-old female who presented to the emergency department with complaints of chest pain resembling acute coronary syndrome.

Keywords: Differential diagnosis, atypical zona, acute coronary syndrome

INTRODUCTION

Varicella zoster virus (VZV) is an alpha herpesvirus with a double-stranded DNA genome. VZV infects humans, and its primary targets are T lymphocytes, epithelial cells, and ganglia. The primary infection results in chickenpox, during which VZV becomes latent in ganglionic neurons. After the primary infection, VZV, which is a neurotropic virus, becomes latent in neurons in the peripheral autonomic ganglia. Latent VZV can reactivate and cause herpes zoster (shingles) spontaneously or by following one or more triggering factors. Zoster typically manifests as a painful or itchy cutaneous vesicular rash that appears in a characteristic dermatomal distribution.[1,2]

Zoster can lead to chronic pain (postherpetic neuralgia) as well as other serious neurological complications such as meningoencephalitis, myelitis, cranial nerve palsy, and vasculopathy. It can also result in ophthalmic complications (keratitis, retinopathy) and gastrointestinal complications (ulcers, hepatitis, pancreatitis), among others.[3,4]

In this case, we wanted to highlight a case of zoster presenting with atypical symptoms resembling acute coronary syndrome but without any rash.

CASE

A 78-year-old female patient presented to the emergency department with complaints of pain in the left retrosternal area radiating to the back, which started 1-2 hours prior to arrival. The pain did not alleviate with movement or change in position and was constant. Her medical history included a lobectomy of the right lower lobe and radiotherapy for lung carcinoma 5-6 years ago. Recent follow-up consultations at the Medical Oncology Clinic did not reveal any recurrence or residual findings. Laboratory tests and imaging were normal. She had recently undergone eradication treatment for *H. pylori* infection following an endoscopy for dyspeptic symptoms, and it was the 9th day of treatment. Physical examination revealed normal lung sounds and equal expansion of both hemithorax's with no pathological sounds. Cardiac examination was unremarkable, peripheral circulation was normal, and blood pressure measured from both arms was within the normal range. Oxygen saturation was not low. The ECG was in sinus rhythm without any pathology, and the chest X-ray showed no abnormalities. Blood tests for troponin were normal, and there were no abnormalities in other blood parameters. The patient was admitted to the internal medicine clinic for further evaluation and management. She received treatment with NSAIDs, tramadol, and paracetamol for ongoing pain. A cardiology consultation was requested, but cardiac pathology was not suspected. Consultations were also requested from the physical medicine and rehabilitation and psychiatry departments, and a preliminary diagnosis of fibromyalgia syndrome was made. The patient was started on Duloxetine and Tramadol for pain

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management. On the 3rd day of follow-up, a physical examination revealed erythematous, pruritic, and painful vesicular eruptions consistent with dermatomes extending from the left anterior axillary line to the left posterior vertebral line. A dermatology consultation confirmed the diagnosis of herpes zoster, and the patient was immediately started on acyclovir treatment at a dose of 10 mg/kg three times a day. Topical steroid and vitamin B12 were added to the treatment. The patient completed a 10-day course of treatment. Due to severe and refractory pain, Gabapentin was initiated, and pain control was achieved before discharge.

DISCUSSION

The clinical presentation of varicella and zoster, which typically includes a distinctive generalized or unilateral dermatomal vesicular rash, serves as the main basis for diagnosis. However, sometimes the diagnosis can be made in the presence of additional symptoms, such as facial paralysis,[5] meningitis,[3] paralysis,[6] gastrointestinal involvement,[7] or as in our case, in the absence of rash and only with pain symptoms (zoster sine herpete).[8] There are two common ways to find out if VZV has been activated and to confirm the diagnosis: polymerase chain reaction (PCR) for finding VZV DNA and enzyme-linked immunosorbent assay (ELISA) for measuring anti-VZV antibodies, specifically anti-VZV immunoglobulin (IgG and IgM) levels.[8]

Antiviral treatment is recommended for immunocompromised patients, those over the age of 50, and those with severe rash or rash on the face or eyes, as well as patients with any complications of herpes zoster. Oral acyclovir, valacyclovir, or famciclovir are commonly used for treatment.[9,10] Treatment is typically given for 7-10 days and reduces the time to cessation of new lesion formation, crusting of the lesions, and relief of acute pain.[11] Hospitalized patients or those with neurological complications are treated with intravenous acyclovir for 7-10 days, as intravenous treatment has been shown to reduce the risk of internal organ involvement and cutaneous dissemination. Initiation of antiviral treatment within 3 days of rash onset is recommended, but if new lesion formation continues in later days, treatment should still be initiated. While antiviral treatment reduces the acute pain associated with herpes zoster, it has not been reliably shown to reduce the risk of postherpetic neuralgia (PHN).[12] Prednisone reduces acute pain and improves clinical outcomes in patients with herpes zoster,[13] but it does not decrease the risk of PHN,[14] and caution should be exercised when using steroids due to the elderly age and the presence of additional diseases like diabetes and hypertension in this patient population. Antiviral treatment should always be given to all patients receiving steroids.[12]

Zoster pain symptoms can be treated with non-steroidal anti-inflammatory drugs (NSAIDs) or acetaminophen in primary care. Local lidocaine can reduce pain, but it should only be used on intact skin due to the risk of irritation. For more severe pain, opioids and opioid agonists (such as oxycodone or tramadol), anticonvulsants (such as gabapentin or pregabalin), or tricyclic antidepressants (such as nortriptyline) can be used.[12]

Postherpetic neuralgia (PHN) is the most serious complication of herpes zoster and is seen in approximately 15% of cases. Age is the most important risk factor for PHN and rapidly increases after the age of 50.4 In addition to age, the risk is also increased in immunocompromised patients, organ transplant recipients, patients receiving chemotherapy for cancer or with autoimmune diseases, individuals with HIV infection, and patients with various chronic diseases.[15] Treatment of PHN is generally challenging, and significant improvement in pain is not seen in most patients. The treatments used are symptomatic rather than targeting the underlying cause of pain. Topical lidocaine, topical capsaicin, gabapentin, pregabalin, and tricyclic antidepressants are commonly used in treatment.[16] The tolerability of topical treatments is often difficult due to pain and rash. Additionally, all systemic medications have potential side effects that may be challenging for elderly patients, and dosages need to be closely monitored for each patient. Referral to a pain specialist may be necessary for patients.

It has been shown that the zoster vaccine is safe and effective in healthy individuals, but it is not yet included in routine vaccination programs.[12]

Due to the acute onset of chest and back pain in our case, acute cardiac emergencies such as acute coronary syndrome, acute valve diseases, and aortic dissection, as well as acute respiratory emergencies such as pneumonia, pneumothorax, and pleural effusion, were ruled out through physical examination, laboratory tests, and imaging investigations. In the literature, there are cases of herpes zoster presenting with chest pain and without the presence of rash.[17] As a result, cases presenting without a rash, like the one in our case, can be missed or misdiagnosed, and patients may not receive timely antiviral treatment. Sometimes, this can keep the varicella-zoster virus (VZV) active, which can cause persistent herpetic neuralgia and then damage the nerves in a way that leads to postherpetic neuralgia (PHN).[18] More seriously, continuous reactivation of VZV can result in fatal complications such as encephalitis, cerebrovascular disease, and paralysis.[19,20] Compared to the pain experienced by classical herpes zoster patients, it has been shown that the pain in patients with zoster sine herpete is more severe both initially and one to three months after the onset of symptoms.[18]

CONCLUSION

In summary, the symptoms and signs of zoster can be subtle, and when other causes are ruled out, zoster sine herpete should always be included in the differential diagnosis for patients with unexplained and more severe radicular-neuropathic pain. A detailed medical history should be obtained from these patients, and the possibility of zoster without a rash should be kept in mind.

ETHICAL DECLARATIONS

Informed Consent: All patients signed and free and informed consent form.

Referee Evaluation Process: Externally peer-reviewed.

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