

Herpes zoster complicated by phrenic nerve palsy and respiratory compromise

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Abstract:

Background: *Herpes zoster* can be associated with severe neurological complications.

Case presentation: In this article, we describe the case of a 54-year-old man with *herpes zoster* affecting his right upper chest and neck region complicated by phrenic nerve palsy and respiratory compromise. The diagnosis of *herpes zoster* was made based on the classic appearance of the rash and associated neuropathic-type pain. The diagnosis of phrenic nerve palsy was made by chest x-ray and ultrasound.

Conclusion: Clinicians should be aware of the possibility of phrenic nerve palsy occurring in patients who have *herpes zoster* affecting the region of C3,4,5 dermatomes. Although symptoms of unilateral diaphragmatic paresis are usually mild, in patients with obesity or comorbid lung disease, new onset phrenic nerve palsy can lead to significant respiratory compromise.

Keywords: *Herpes zoster*, neurologic complication, phrenic nerve palsy, case report.

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Introduction

Herpes zoster occurs as a result of reactivation of latent *varicella zoster* virus, often due to age-related immunosenescence, HIV infection, or other immunosuppressive condition. It typically presents as a painful vesicular rash with a dermatomal distribution. Although the rash is usually benign and self-limited, the virus is neurotropic and devastating neurological complications can develop ranging from an intractable pain syndrome (post-herpetic neuralgia) to disabling stroke or limb weakness.

In the following report, we describe a case of phrenic nerve palsy associated with cervical distribution zoster - a rare but potentially serious complication of *herpes zoster*.

Case study

A 54 year old male presented with hypoxia and severe episodic pains over his right upper chest and neck region. He had well-controlled HIV infection; his recent CD4 count was >1,000 cells/ml, his viral load was undetectable, and he was on a stable anti-retroviral regimen including tenofovir, lamivudine and efavirenz for more than 5 years.

Three weeks prior to this presentation, he first noticed a painful vesicular rash over his right upper chest and neck region. He was treated with acyclovir and the vesicles gradually healed over with scars. However, the pain persisted, he began feeling generally weak and unwell, and he presented to hospital.

On examination, there were scattered lesions over his right lateral neck and upper chest region characteristic of scars from *herpes zoster* (figure 1).

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Figure 1: Characteristic skin lesions of herpes zoster involving dermatomes corresponding to dorsal ganglia of nerve roots of C3, 4, and 5. Anterior (motor) ganglia from these same nerve roots innervate the diaphragm

He was diaphoretic and appeared weak and unwell. His temperature was 37.5°C, blood pressure 130/90 mmHg, heart rate 100, respiratory rate 24, and oxygen saturation,

while breathing room air, fluctuated between 82% to 92%.

His chest x-ray documented elevation of the right hemi-diaphragm (figure 2).



Figure 2: Chest x-ray documenting marked elevation of the right hemi-diaphragm consistent with right phrenic nerve palsy

Ultrasound examination confirmed that the right diaphragm was elevated and did not move as expected with inspiration - a finding consistent with phrenic nerve palsy. His electrocardiogram documented normal sinus rhythm; there were no ST-T wave changes concerning for myocardial ischemia.

He was managed symptomatically with analgesics and over the next few days his pain reduced and his overall condition improved. Unfortunately, he passed away suddenly approximately 2 weeks following his presentation to hospital. The cause of death was unknown, however assumed to be a sudden cardiac death perhaps related to myocardial infarction or arrhythmia.

Discussion

There are many well described neurologic complications related to *varicella zoster* reactivation including facial nerve

palsy (Ramsay hunt syndrome), meningoencephalitis and CNS vasculopathy with increased risk of stroke.¹ Neurological syndromes typically occur 2 – 4 weeks after the skin rash suggesting an immunologic component.

Zoster paresis (weakness) may occur when there is extension of involvement of the dorsal (sensory) nerve roots to the anterior (motor) nerve roots.¹ Weakness generally occurs in the muscle groups corresponding to the involved dermatomes.

Arm weakness or diaphragmatic weakness can occur with cervical distribution, leg weakness with lumbar distribution, and urinary retention with sacral distribution zoster.

Phrenic nerve palsy is a rare complication of *herpes zoster* involving the cervical dermatomes around C3,4,5.² Although symptoms of unilateral diaphragmatic paresis are

usually mild, in patients with obesity or comorbid lung disease new onset phrenic nerve palsy can lead to significant respiratory compromise.³

The weakness associated with *herpes zoster* is generally self-limited and tends to improve on its own with time. Approximately 50% have complete functional recovery.¹

Unfortunately, our patient died suddenly and unexpectedly within 2 weeks of his diagnosis. Progressive and disseminated viral infection was an unlikely because of an initial trend towards improvement, however, there is increasing evidence linking episodes of *herpes zoster* to an increased risk of cerebrovascular or cardiovascular events.⁴

Conclusion

Clinicians need to be aware of the potential for neurological complications that can occur after an episode of *herpes zoster*, generally affecting nerves roots related to the dermatomal distribution of the rash. Other than supportive care, there is currently no known effective treatment for these complications, so prevention is the best recourse. As new varicella vaccines are becoming increasingly available, awareness of the potential for neurolog-

ical complications may improve advocacy for universal access to the vaccines.⁵

Conflict of interest

None declared.

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