

Hemidiaphragmatic paralysis: A rare complication of cervical herpes zoster

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Abstract

Herpes zoster, a sequel of the reactivation of the varicella zoster virus, usually presents with cutaneous eruptions associated with intense pain and burning sensation in the affected dermatomes. Motor weakness, however, can sometimes complicate herpes zoster. In this report we present a case that had diaphragmatic motor weakness as a sequel of herpes zoster lesions in the neck.

Key words: Herpes Zoster, Postherpetic Neuralgia, Hemidiaphragmatic Paralysis

Herpes zoster is a relatively common disease and affects predominantly middle-aged and elderly people. Apart from segmental distribution of cutaneous eruption and neuralgic pain, it also causes motor weakness; though this can readily be obscured by intense pain of the disease. Hemidiaphragmatic paralysis due to phrenic nerve involvement is a rare complication of cervical herpes zoster when the disease affects C3, 4, and 5 dermatomes. Here, we describe a case of hemidiaphragmatic weakness that developed soon after the onset of herpetic rashes in the neck and had complete recovery within one year.

Case report

A 60-year old gentleman presented to Medical Referral Clinic of Patan Hospital with 2 weeks' history of skin rashes in the right side of the neck and upper part of right anterior chest wall. The skin lesions were fluid-filled in the beginning, and there was intense pain with burning sensation over the rashes. After about one week the lesions dried up forming crusts and ultimately pigmented scar over the skin. The burning pain, however, was persistent even after the healing of these rashes. One week after the onset of these rashes the patient experienced sudden onset of shortness of breath which was gradually progressive over the days. There was no associated fever, cough, sputum production, chest pain, or wheeze. The patient's current medication includes Atenolol which he was taking for high blood pressure for last 5 years. Apart from this, review of the cardiovascular, gastrointestinal, and nervous systems revealed no significant problems. Similarly, he had no remarkable medical illnesses in the past including no allergy to drugs.

On examination his vitals were normal with blood pressure 130/80 mm Hg. There were multiple healing lesions with crusts and scabs in the right side of the neck and upper part of right anterior chest wall (Fig 1). There was decreased excursion of the right chest wall; percussion note was dull on the base of the right lung, with decreased vocal resonance and breathe sound on the same area. Examination of the head, neck, cardiovascular system, and abdomen was unremarkable. Laboratory investigations revealed normal haematological and biochemical findings. The dome of the right hemidiaphragm was elevated on chest x-ray (Fig 2), a completely new finding from the previous x-ray taken five months ago (Fig 3). Ultrasound of the chest and abdomen did not reveal any evidence of pleural or sub diaphragmatic fluid collection. CT scan of the chest revealed compressive atelectasis of the right lower lobe, without lymph node enlargements or mediastinal lesions. The fiberoptic bronchoscopy was normal.

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Fig. 1: Healing herpetic rashes in rt.side of neck and upper chest wall



Treatment was initiated with topical calamine lotion for rashes and paracetamol and codeine for pain; amitriptyline was added to control the postherpetic neuralgic pain. No active intervention was done for the breathlessness, which decreased slowly during the follow up. Follow up x-ray at one year showed normalization of the previously elevated right hemidiaphragm. (Fig 4). Fluoroscopy, though not available at the time of initial presentation, one year later showed normal excursion of the right hemidiaphragm both during inspiration and expiration, which was equal to that in the left side. However, the burning pain was still persistent at the end of the one year, though the severity was much reduced.

Figs 2, 3, and 4: Chest x-rays showing elevated Rt. hemidiaphragm at presentation (Fig. 2), and normal hemidiaphragm 5 months before presentation (Fig. 3) and 1yr after presentation (Fig. 4).



Discussion

Herpes zoster is characterized by sharp, severe, radicular pain and vesicular rashes that gradually convert into crusts, dry scabs and ultimately into pigmented scars. It is caused by varicella zoster virus, a human specific herpes virus. Though viral isolation, direct immunofluorescence assay, and polymerase chain reaction are used in confirming the diagnosis, the appearance of herpes zoster rash is sufficiently distinctive to make an accurate clinical diagnosis.¹

Infection by the virus at an early age results in chickenpox, the virus then passes through a period of latency in cranial-nerve and dorsal-root ganglia, and frequently reactivates decades later to produce herpes zoster, and other neurological complications like postherpetic neuralgia, motor paresis and even severe infections like myelitis and encephalitis.² Decrease in the cell-mediated immunity due to old age, certain malignancies, and human immunodeficiency virus

(HIV) infection and use of various immunosuppressive treatments increase the risk of herpes zoster and its sequel.¹ Postherpetic neuralgia (pain persisting after 30 days of onset of rashes or cutaneous healing) occurs in about 8-70 percent of patients with herpes zoster; ¹ people above 50 years of age are 15 times more likely to develop this complication. In general, postherpetic neuralgia is a self-limited condition; and only less than 25% and 5% of patients experience this pain at six months and at one year respectively.³ The pathogenesis of postherpetic neuralgia is unclear but supposed to be the activation of the latent virus in nerve ganglia causing inflammation of the nerves (neuritis) that innervate the corresponding dermatomes. Our patient had typical zosteriform skin rashes and persistent burning pain of postherpetic neuralgia. The pain, however, was persistent even after one year, though the severity was much reduced.

Motor nerve involvement in herpes zoster is not uncommon; its prevalence varies from 0.5% to 31% in different series. The paresis in herpes zoster has certain characteristics: it occurs almost always after the onset of cutaneous rash, has unilateral distribution, and the interval between the skin eruption and the onset of muscle weakness is generally 2 weeks.⁴ There are reports that have pointed out arm weakness and diaphragmatic paresis after cervical zoster, leg weakness and bowel and bladder dysfunction after lumbosacral zoster, and facial palsy after herpes zoster of the external ear or hard palate (Ramsay Hunt Syndrome).²

In this case, sudden appearance of shortness of breath, new onset elevated hemidiaphragm on chest x-ray with negative work up and a recent history of cervical herpes zoster led us to think the cause of elevated hemidiaphragm to be diaphragmatic paralysis due to zoster-induced phrenic nerve involvement. Moreover the presence of postherpetic neuralgia in the C3, 4, and 5 dermatome supported our notion of cervical herpes zoster as the cause of hemidiaphragmatic paralysis. The time between the development of the rashes and the onset of shortness of breath due to hemidiaphragmatic weakness was one week in our patient.

Diaphragmatic weakness in cervical herpes zoster is the result of the lesion in phrenic nerve that originates from cervical spinal cord segments C3, 4, and 5. However it is not clear exactly what region of the nerve (anterior horn cells, rootlets, roots, or the nerve itself) is affected by the virus. The possible pathogenetic mechanisms include either the direct damage of the anterior horn cells by the virus or diffusion of the viruses into anterior nerve roots from the dorsal root ganglion, the usual location of the latent virus.⁵

The prognosis for herpes zoster associated motor paresis is relatively good; complete or near complete recovery has been reported in 50-70% of patients.⁶ However, serious complication like hypoventilatory

respiratory failure has been described due to cervical herpes zoster induced unilateral diaphragmatic paralysis. According to one report only in 3 out of 18 cases, the hemidiaphragmatic paralysis resolved within 18 months of herpes zoster infection.⁷ In our case, the recovery was uneventful and complete resolution occurred at one year as evidenced by normal excursion of diaphragm on fluoroscopy and disappearance of elevated hemidiaphragm on chest x-ray.

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