

Postherpetic pseudohernia of abdominal wall: A case report

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Abstract Herpes zoster is a common disease of the dorsal root ganglia and sensory nerve fibers, clinically characterized by unilateral painful vesicular eruption of the skin in a dermatomal distribution. Segmental motor weakness is a rare complication that occurs in 0-5 percent of patients and is associated with an excellent prognosis for recovery. We herein, present a case of herpes zoster complicated by segmental motor paresis of abdominal musculature resulting in pseudohernia. This rare complication of herpes zoster, with an excellent prognosis for recovery, should be recognized by dermatologists and surgeons as it can save costly consultations and evaluations.

Key words

Herpes zoster, pseudohernia.

Introduction

Herpes zoster is caused by reactivation of a latent varicella-zoster virus and almost affects 10-20% of the general population.¹ Being a neurotropic virus it has predilection for the posterior root ganglia and that is why the majority of neurological complications are sensory. At times, motor complications occur in the corresponding segments to the involved sensory dermatomes.² This may lead to abdominal wall weakness and hence can present with abdominal wall hernias.³ The first case report of motor paresis following herpes zoster was published in 1886 by Broadbent.⁴ We report another case of dermatomal herpes zoster with subsequent abdominal muscle weakness and a formation of pseudohernia.

Case Report

A 55-year-old man, farmer by profession,

presented in dermatology OPD with a sudden painless bulge over left lumbar area, a week later to development of a painful vesicular rash on left side of his abdomen. There was no comorbidity and negative previous history of surgery over abdomen. Clinical examination revealed a lump of around 20cm x 15cm over left lumbar area with normal overlying skin except for healing rash of zoster and postinflammatory hyperpigmentation (**Figure 1**). It was doughy in character, nontender, and compressible, had ill-defined margins and enlarged on straining. A relevant abdominal and systemic examination was normal.

An ultrasound examination of the swelling and of the abdomen showed no abnormality. Nerve conduction studies could not be performed due to lack of facility. Clinical diagnosis of pseudohernia of abdominal wall due to motor paresis of abdominal musculature was made as there was positive temporal relationship between the lump formation and onset of herpes zoster and with on abdominal pathology. Patient was counselled and educated about the disease and its good prognosis and advised to wait and see

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Figure 1 Lateral and oblique view of pseudo-hernia along with resolving herpes zoster rash in T11 and T12 dermatomes.

the progression. He was requested to follow up after every four weeks. He was advised NSAIDs for mild postherpetic neuralgia. On first follow-up visit, there was significant objective reduction of pseudo-hernia.

Discussion

Herpes zoster is caused by a neurotropic virus from *Herpesviridae* family of DNA viruses. It affects one or several adjacent dorsal root ganglia and sensory nerves. Clinically it presents as painful and unilateral vesicular rash in the corresponding sensory dermatomes, although rarely, damage to the anterior horn cells at same level, resulting in motor muscular weakness, is possible. Motor complications can be somatic, including cranial like Ramsay-Hunt syndrome and peripheral like segmental paresis of the limbs, diaphragm or abdominal musculature and visceral, involving the gastrointestinal and urinary tract resulting in colonic pseudo-obstruction and bladder dysfunction⁵.

Pseudo-hernia represents a limited protrusion of the abdominal wall without an actual weakness of muscles. It can be seen in unilateral paralysis of abdominal muscles, resulting in relaxation of the anterior abdominal wall and resultant bulging of flanks because of the abdominal pressure. Pseudo-hernias have been reported after

ventral nerve root damage secondary to excision of radiculopathies, intradural tumour, Lyme disease, poliomyelitis and syringomyelia.⁶

The exact mechanism of abdominal muscle paresis following herpes zoster infection is poorly understood. The spread of active inflammation from the dorsal root ganglion to the anterior horn cells and anterior nerve roots, and involvement of the motor nerve are postulated to be involved.^{7,8} Cioni *et al.*⁹ found ganglionic lesions combined with degeneration of the related sensory and motor roots and severe neuritis on pathological examination, findings which can easily explain the clinical and electrophysiological signs.

The reason for this extremely rare complication is felt to be the dual innervation of the musculature of abdominal wall. Symptoms of segmental muscle paresis usually appear within 2 to 6 weeks after the beginning of herpes zoster, and the involved myotomes correspond to the involved dermatomes.

Diagnosis is usually suspected by clinical evidence of herpes zoster associated with abdominal wall or flank bulging. Physical examination shows reduced or absent segmental reflexes.² To confirm the diagnosis, a nerve conduction study must be done; electroneuromyography is used for this purpose.¹⁰ MRI with gadolinium-DTPA can help to define the extent of the inflammation, and to exclude local entrapment of spinal nerve roots, which is known to be a precipitating factor of herpes zoster.^{11,12} Differential diagnosis includes lumbar hernias, that can occur through the inferior lumbar triangle of Petit or the superior triangle of Grynfeltt, and other conditions such as diabetic truncal neuropathy, syringomyelia, polyradiculoneuropathy, Lyme disease and prolapsed L1-L2 intervertebral disc.

The prognosis for this motor weakness is good, with complete recovery in 55-75% of patients within 6-12 months of the onset, but some patients remain with permanent weakness.^{5,6,13}

Conclusion

Postherpetic pseudohernia should be considered when a patient develops clinical signs and symptoms of motor paresis that coincide with or follow a herpes zoster infection resulting in abdominal wall herniation or bulging. Recognizing this rare complication is important, because it is a potentially reversible disease with a favourable prognosis and does not require surgical intervention.

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