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Case Report

Abdominal pseudo hernia: a rare sequelae of complicated herpes zoster

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ABSTRACT

Herpes zoster (HZ) caused by Varicella Zoster Virus causes several complications, the most common being PHN (Post Herpetic Neuralgia). Here we report a case of phantom hernia following HZ involving T10-T11 dermatome. Close follow up will ensure full recovery to avoid unnecessary surgical intervention.

Keywords: Herpes zoster complications motor deficit, Muscle paralysis, Abdominal pseudo hernia

INTRODUCTION

Herpes zoster (HZ) is a disease with unilateral vesicular eruptions with radicular pain limited to the dermatome of single segment ganglion, due to reactivation of dormant Varrilella Zoster Virus infection, in the sensory ganglia. Incidence is 3-4 per 1000 people per year in United Kingdom. This incidence is depends on the age and immune status, not on the sex ; incidence more after 60 years of age with a lifetime risk of 10% to 30% that rises to 50% among those living to ≥85 years . No major seasonal variation. HZ infection is frequent among the patients with malignancy, organ transplantation and HIV infections.[1,2]

After a primary varicella-zoster virus (VZV) infection (termed "varicella" or "chickenpox"), the virus establishes latency in dorsal root and cranial nerve ganglia can lie dormant for many decades. Herpes zoster (HZ), also known as "shingles," results from reactivation of VZV and its spread from a single ganglion to the neural tissue of the affected segment and the corresponding cutaneous dermatome. Reactivation is thought to result from waning cell mediated immunity. It typically presents with painful unilateral vesicular dermatomal rash, that causes acute morbidity lasting between 2 weeks and 4 weeks.[1,2]

VZV passes centripetally from skin & mucous membrane lesions to the sensory ganglia via sensory nerves. Reactivation of VZV is by trauma, sunburn, stress & old age. After a period of latency, subclinical reactivation and viremia, newly synthesized VZV transported along the sensory nerves and unilateral dermatome skin lesions appears. If the VZV infection spread through the posterior nerve root, local leptomeningitis, CSF pleocytosis, segmental myelitis can occur, if inflammation in the anterior horn motor neurons & nerve root, local palsies follows the skin lesions.[1,2]

Factors affecting cell-mediated immunity like older age group, immune suppression [HIV up to 10 times, leukemia, lymphoma, metastatic malignancies, autoimmune disorders, diabetic, Chronic obstructive

pulmonary disorder (COPD), treatment with cytotoxic drugs or therapies] increases the risk of HZ infection. Potential risk factors for zoster are as follows diet low in micronutrients, smoking, alcohol and stress. In iatrogenic immune suppression or age-related immunosenescence, with depleted immunity, viral replication ensues, resulting in ganglionitis and extensive infection and destruction of neurons and supporting cells.[3]

Post-herpetic neuralgia (PHN), is the commonest complication, followed by cranial Ramsay Hunt syndrome, i.e. facial paralysis and lesions of the ear (zoster oticus) and peripheral nerve palsies, encephalitis and myelitis, and stroke. Systemic dissemination may accompany the skin changes with involvement of the lung, liver, and brain. Visceral dissemination is associated with a mortality rate of 5% to 15%, with most deaths attributable to varicella pneumonitis. The neurologic complications of HZ may include acute or chronic encephalitis, myelitis, aseptic meningitis, polyradiculitis, retinitis, autonomic dysfunction, motor neuropathies, Guillain-Barré syndrome, hemiparesis, and cranial or peripheral nerve palsies. More common complications include bacterial super infection by *Staphylococcus aureus* or *Streptococcus pyogenes*, scarring, and hyper pigmentation.[4]

Motor nerves may be involved in 5%–15% of HZV infections. By use of electromyography, it is possible to show that muscles are involved in 50% of cases. Obvious paresis typically improves over time and may respond to physical therapy. Satisfactory recovery of muscle function decreases with the age of the patient and the severity of the paralysis at onset.[5]

CASE REPORT

A 65 year old, security supervisor was referred from surgical department to our skin OPD for a second opinion for a painless mass in the left flank of abdomen.

A detailed history and clinical examination was done. He was a diabetic on oral hypoglycaemic agents. Smoker, non alcoholic. He was operated for uncomplicated bilateral direct hernia 10 years ago with uneventful post operative periods. He gives history of pain (sharp, shooting) and erythematous grouped vesicles in the left T10-T11 dermatome 5months ago, diagnosed as herpes zoster. Baseline investigations done and all are within normal clinical limits. He was treated with oral acyclovir in the standard prescribed dose along with neuroleptics and supportive vitamins for one week. Vesicles resolved & leaving behind hyper pigmented atropic scars in the dermatome. In the ensuing weeks he developed burning sensation and hyperesthesia with sharp shooting pain occasionally relieved by taking medications.

He had swelling in the operated sites following excessive day time standing and heavy manual works, disappearance of the swelling in the morning after a night sleep for last one year. For the last one month he noticed similar type of swelling in the left flank scar area also, which is appearing in day time after heavy manual work, disappearing after a night sleep. Swelling increases after cough, sneeze, urination and defecation. No history of any injury, No pain or tenderness. This was soft, non mobile, non tender swelling of 20 cm x15cm with well defined sloping margins and reduced on pressure or lying down posture. Superficial sensory & motor reflex were absent in the left T10-11 dermatome. Cough reflex was present. Routine blood parameters were within normal limits. Glycemic status was under control.

A diagnosis of the recurrent bilateral direct inguinal hernia with HZ induced pseudo hernia in flank was made clinically. Follow up is being done. He was advised to come for the hernioplasty after 3 months.

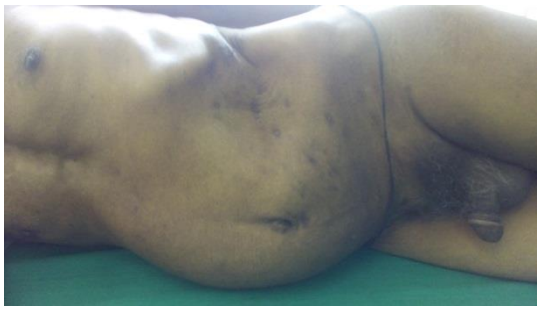

Figure 1: Zoster affected areas showing localized muscle weakness.

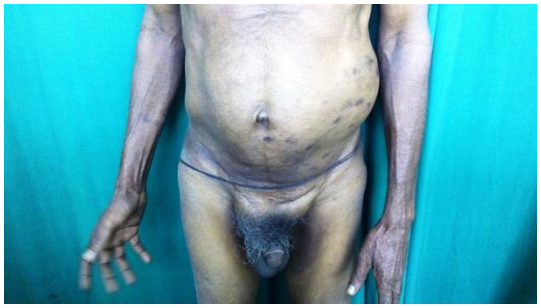

Figure 2: Showing hyper pigmented atrophic scars along Left T10-T11 Thoracic dermatome with pseudo hernia. Note the recurrent bilateral direct inguinal hernia.

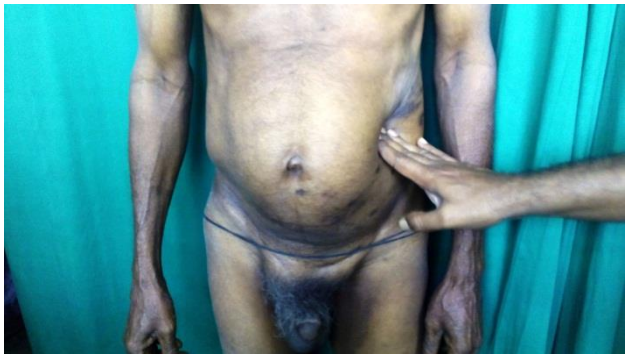

Figure 3: Cough impulse / compressibility in Left T10-T11 thoracic dermatome. Note the linear, old surgical scars in both inguinal regions.

DISCUSSIONS

Herpes zoster is a distinct clinical syndrome that may present with a segmental zoster paresis. Motor involvement in herpes zoster is due to spread of infection from dorsal root ganglia to the same segment of spinal cord mainly in posterior horn and occasionally to anterior horn cells with consequent lower motor neuron paralysis . Lower motor neuron paralysis may also occur from damage to anterior nerve root where it joins dorsal root ganglia .This type of paralysis usually occurs in first few weeks after onset of skin rash but occasionally precedes or accompany them. Nagabhushana et al. reported weakness of right upper limb muscles (C8-T1) in 60 year male Dutta et al, reported wrist drop, and Chopra et al. reported ulnar nerve paresis. Although thoracic dermatomes are the most commonly affected, paresis of the abdominal muscles has been less frequently reported.[6,7]

Sir W. Broadbent, was documented the motor involvement following the HZ first. Grant and Rowe in 1961, Thomas and Howard (1972), Denny-Brown et al. were documented cases series of motor involvement with HZ. Clinically significant motor deficit is rare. Cephalic, Thoracic, Abdominal, Lumbosacral nerve roots segment have the motor involvement in the descending orders. Slight involvement of the paraspinal muscles up to 35% of instances of thoracic herpes zoster .Motor involvement of the HZ usually after one to five weeks, right side dermatome and myotome involvement is twice as common as on the left. The pathogenesis may be related to direct spread of virus to anterior horn cells, ventral roots or both. Motor deficits with varicella zoster virus documented in the distributions of cranial nerves (e.g. Ramsay Hunt syndrome in geniculate ganglion infection), peripheral motor nerves (paralysis of limbs) and visceral nerves (dysfunction of the bladder). The geniculate ganglion also contains latent VZV derived from facial, aural, and oral lesions of varicella. Reactivation in the geniculate ganglion can lead to facial nerve (VII) paralysis (because sensory and motor nerves are conjoined in nerve VII), as a result of a bystander effect. VZV and HSV account for the majority of cases of Bell palsy (idiopathic facial paralysis. Denny-Brown et al showed that the pathological process in HZ is primarily a unilateral segmental myelitis, very similar to anterior poliomyelitis, except that most of the pathological changes are seen in the posterior part of the spinal cord, the posterior root and ganglion.[5-7]

PubMed literature search and review of publications reveals around 36 documented isolated case reports. Male: female ratio is 4:1, 67.5 years as mean age, right &left side with equal ratio, T11 as commonest dermatome, 89% had zoster rash preceded the abdominal weakness, 80% had complete recovery in the follow up periods around 5 months. 19% had visceral neuropathy also. Self limited nature and good prognosis noted in this condition. Recognition of this complication is important to prevent unnecessary diagnostic studies and procedures. Because visceral neuropathy commonly co-occurs with segmental zoster abdominal paresis, it should be actively investigated and treated. [8]

A nerve conduction study must be done for confirming paralysis. We can rule out morphological defects in the abdominal wall by ultrasonography, electroneuromyography can be used to confirm the axonal motor involvement. Electromyographic studies revealed a denervation pattern of the paraspinal muscles at the affected levels, indicating that the nerve segment involved is proximal to the posterior rami, probably within the anterior horn itself. MRI with gadolinium-DTPA can help to delineate the extent of the inflammation, and to exclude the local entrapment of spinal nerve roots, known to be a predisposing factor of herpes zoster. Recent publication with magnetic resonance images of the spinal cord in a patient with L1 segmental zoster paresis with abnormal contrast enhancement of anterior and posterior nerve roots in L1 segment. In our case, HZ infection caused transient muscle paralysis of abdominal paralysis of abdominal muscles leading to pseudo hernia.[9,10]

Differential diagnosis includes lumbar hernias, that can occur spontaneously through the inferior lumbar triangle of Petit or the superior triangle of Grynfeelt, and other conditions such as diabetic truncal neuropathy, Lyme's disease, polyradiculoneuropathy, syringomyelia and prolapsed L1–L2 inter vertebral disc.[11]

"Phantom" – "Phantasm" – means mental imagery produced by fantasy. Phantom hernia described by Achar, in anterior poliomyelitis, for unilateral bulging on either side of the abdomen due to weakness or paralysis of abdominal wall muscles. Similar paralysis leading to hernia repeated in HZ also.[12]

Recovery without substantial sequelae is within one year, in most of the pseudo hernia following herpes zoster. In fact, there is no a connection between the degree of paralysis and complete recovery, and also there does not seem to be any pharmacologic way of hastening recovery. We have to give assurance closely observe and wait for the complete recovery. Unnecessary surgical intervention should be avoided.

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