Abdominal muscle paralysis in herpes zoster

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ABSTRACT

Herpes zoster or Shingles, an inflammatory viral disease caused by varicella has classical clinical presentation with herpetic morphological pattern exhibited along one or more dermatomes. The self limiting skin disease is accompanied by pain and burning sensations leading to post herpetic neuralgia. The sensory component of the disease is so prominent that the motor involvement is often overlooked. It was not known till recently that profound muscle weakness can be a part of this syndrome. We report 2 cases of herpes zoster who had prominent appreciable motor weakness of the abdominal muscles following the disease. The weakness however has a better prognosis and the lesions in both of our patients healed in 6 months without leaving any residual paralysis

Keywords: Motor paralysis, herpes zoster.

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Herpes zoster (HZ) or "Shingles" infection is manifested by the appearance of a circumscribed vesicular eruption of the skin and mucous membrane in a dermatomal distribution. It is associated with inflammation of the dorsal root ganglion of the involved segment and often of the immediately adjacent spinal segments.[1] In the presence of the appropriate cutaneous manifestations, it is not difficult to attribute segmentally distributed sensory changes and pain to HZ. It is less well known that the motor system can be affected and that profound muscle weakness can also be part of the zoster syndrome. In this paper, we describe two cases of lower dorsal HZ with abdominal muscle paralysis and review the literature about this subject.

Case Reports

Patient 1. A 63 year-old diabetic Saudi man presented with a burning sensation and pain segmentally distributed on the lower part of the left side of the chest of 4 days duration. One day before presentation he noticed erythema with the appearance of small grouped vesicles along the dermatomes on an erythematous base. The diagnosis of HZ was made, baseline investigation such as complete blood count, erythrocyte sedimentation rate and liver profile were normal, and he was treated with oral acyclovir. The lesions healed in 4 weeks, leaving a scar in that area. Intense burning and hyperesthesia with sharp shooting pain was the most agonising symptom. He was given oral carbamazepine and amitriptyline with partial relief. One week later, he noticed an abnormal swelling on the left side of the abdomen, in the area of the rash, just below the costal margin. This swelling was moving with respiration.

On examination, a prominent bulge of 10-15cm in diameter was present on the left side of the trunk, in the area of T9 - T11 dermatomes. There were

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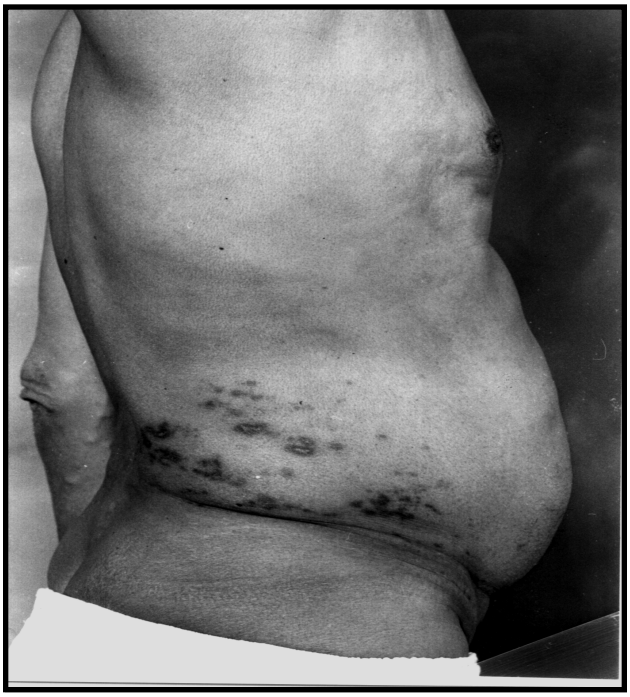

Figure 1a - Bulging of the right flank (T9-T10).

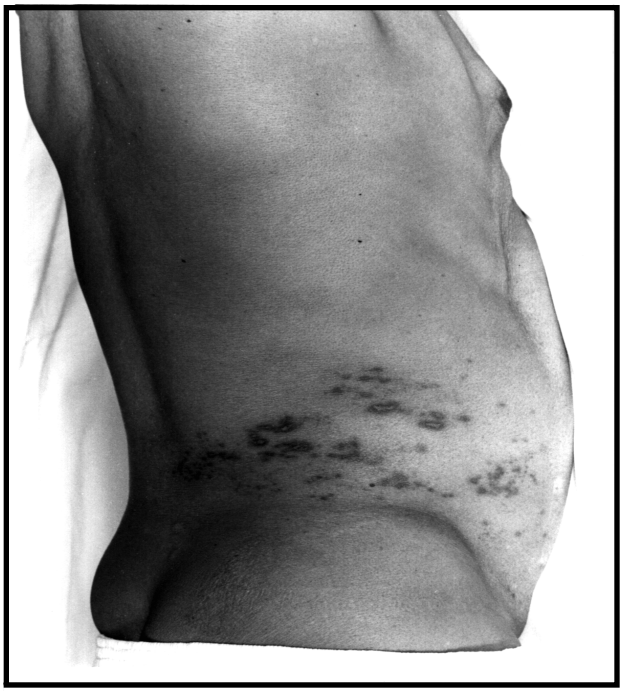

Figure 1b - Resolution after 6 months.

hyperpigmented scars and scabs on the surface of the bulge. The area was hyperesthetic to touch. The overlying skin was lax. The bulge did not increase on coughing and was not reducible. Abdominal reflexes were absent on the left side.

Three weeks after the appearance of the bulge, nerve conduction and electromyographic (EMG) studies were done. The right and left 9th to 11th intercostal nerves were stimulated by surface electrodes over the intercostal spaces, at mid-distance between the vertebral column and the sternum. The motor evoked responses were recorded over the abdominal recti muscles. There was no recordable response on the left side. Magnetic stimulation of the thoracic spinal cord over T10 induced a very small response of less than 0.1mV amplitude on the left, while on the right side, it reached 0.8mV. Concentric needle electromyography of the left paraspinal muscles revealed 2+ fibrillation potentials and sharp waves at rest at D10 - D11 levels. Over the the next 6 months, his condition improved progressively and weakness had resolved when the patient was examined 7 months later.

Patient 2. This 58 year-old Saudi male with type II non-insulin dependant diabetes presented with paresthesia and pain of one week duration. He developed grouped vesicular lesions on an erythematous base along the right side of the trunk (T9-T10 dermatomes). Diagnosis of Herpes Zoster was made and he was treated with analgesics and acyclovir. Two months later at a follow-up visit, a bulge of the right flank was noted in the area of the rash (Figure 1a). The features of this bulge were similar to those of Patient 1. The patient did not come for the planned investigations (EMG, magnetic resonance imaging of the spinal cord). Six months later when he came for a follow-up visit the bulge had disappeared (Figure 1b).

Discussion. Sir W. Broadbent, more than a century ago, was probably the first author to report motor involvement in HZ. Taylor (1895) reported the first case of paralysis of the abdominal muscles following "Shingles".[2] However, up to 1991, only 17 cases could be found in the literature.[3] On 101 patients with HZ studied by Grant and Rowe in 1961, 5 had motor paralysis of the limbs.[4] This proportion of 5% was also found in the largest series of HZ ever published, where Thomas and Howard (1972) found 61 cases with paralysis in 1205 cases studied.[5] Of these, 2 cases of abdominal wall paralysis occurred in the 277 cases of lower thoracic zoster (T8 - T12), resulting in a percentage of only 0.7%. On the contrary, the highest percentage of paresis occured in cephalic zoster totaling 28 cases on 227 (12%). Most of these were geniculate zoster infections, the so-called Ramsay-Hunt syndrome.

Denny-Brown et al[1] showed that the pathological process in HZ is primarily a unilateral segmental poliomyelitis, 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. Electromyographic studies, as well as the few studies performed on other such

patients,[6] 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. In addition, recently published magnetic resonance images of the spinal cord in a patient with L1 segmental zoster paresis have shown abnormal contrast enhancement of anterior and posterior nerve roots at the affected segment.[7] We wanted to perform such an MRI on our second patient but unfortunately he did not come on his appointment day.

Our two patients recovered within 6 months, as most of the patients with paralytic HZ do. In Thomas and Howard series,[5] only 15% of the patients had residual paralysis after one year. These cases are presented to arouse the awareness about this not so uncommon complication and its benign as self resolving outcome.

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