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Herpetic shoulder paresis in a Chinese elderly patient

一位華裔老年病人所患的疱疹性肩非全癱瘓

A patient with left shoulder girdle weakness secondary to herpetic myotomal paresis is reported. Needle electromyography revealed denervational discharge from the left supraspinatus, deltoid, and brachioradialis muscles, compatible with a radiculopathy that was relevant to his myotomes affected by zoster infection. The patient was managed with range-of-movement and strengthening exercises as well as pain relief for post-herpetic neuralgia. Further studies are required to determine whether antiviral treatment can limit the extent of motor deficit and hasten recovery. Zoster paresis should be one of the differential diagnoses of girdle muscle weakness.

本病例報告的病者患上疱疹性肌節非全癱瘓，以致左肩帶無力。針極肌電圖檢查顯示，左棘上肌、三角肌和肱橈肌出現失去神經支配的情況，與患者因為肌節受帶狀疱疹感染而患上的神經根型頸椎病症狀吻合。醫院為患者安排各種肌肉移動和強化的練習，並替病人舒緩帶狀疱疹後神經痛。帶狀疱疹性非全癱瘓應是帶狀肌無力的一種鑒別診斷症狀，須進一步研究抗病毒治療能否控制肌肉運動不良的範圍，以及加快病人的復原速度。

Introduction

Herpes zoster, or shingles, is a common vesicular eruption due to reactivation of latent varicella zoster virus in the dorsal sensory ganglia. Herpes zoster occurs during the lifetime of 10% to 20% of all people.¹ The annual incidence ranges from one to two cases per 1000 in the young to 10 times this figure in the elderly.² Whilst the usual presentation is principally that of a painful cutaneous dermatomal rash, other neurological complications have been described. These include post-herpetic neuralgia, meningoencephalitis, cranial arteritis, myelitis, and segmental paresis.³⁻⁶ We report a patient with shoulder weakness secondary to herpetic myotomal paresis.

Case report

An 85-year-old man with a history of Parkinson's disease, gouty arthritis, and mild dementia was admitted with a 3-day history of weakness of his left upper limb. There was no history of trauma to his left shoulder, head, or neck region. He was a social drinker only. He had never smoked and had no history of cerebrovascular disease.

Neurological examination on the first day of admission revealed left shoulder weakness in abduction (4/5), adduction (4/5), flexion (4/5), extension (4/5), and external and internal rotation (4/5). No muscular atrophy or fasciculation were observed. The power of the rest of his left upper limb was normal. Biceps, triceps, and brachioradialis reflexes were preserved. His left upper limb sensation was intact. The rest of his

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neurological examination was unremarkable. Blood tests revealed a normal blood picture, and renal and liver function. Calcium and creatinine kinase levels were unremarkable. Blood glucose level was normal and a low potassium level (2.9 mmol/L) was promptly corrected after admission.

The day following admission, erythematous papules appeared over the patient's left shoulder, and upper and lower arm with vesicular formation. The lesions mainly corresponded to the left C4, 5, and 6 dermatomes. There was also deterioration of left shoulder power, namely abduction (3/5), adduction (3/5), flexion (2/5), and extension (2/5). A mild decrease in elbow flexion (4/5) was also noted. The power of left elbow extension and distal upper limb was unaffected. No other neurological deficits were elicited including preserved sensation. A diagnosis of acute herpes zoster affecting C4 to 6 dermatomes complicated by myotomal zoster paresis was made. Oral valaciclovir 1 g 3 times daily for 7 days was commenced.

During the second week, skin rash and vesicular eruptions reached a peak in severity. Judicious skin care with regular dressing by nurses was provided and secondary infection was prevented. Left upper limb power remained unchanged.

By the third week, the vesicles had gradually resolved with no appreciable post-herpetic neuralgia. Unfortunately, limb girdle power further deteriorated with abduction (2/5), adduction (2/5), flexion (1/5), and extension (2/5). Muscular fasciculation was absent. Elbow flexion and supination were slightly weak (4/5). Forearm pronation, wrist flexion and extension, as well as finger power were all normal. The biceps, triceps and brachioradialis reflexes were still preserved. There was no sensory deficit.

By the fourth week, the patient complained of post-herpetic neuralgia in his left upper limb. It was controlled by amitriptyline, later replaced by gabapentin due to increased daytime somnolence. Upper limb power remained unchanged. Electrophysiological studies revealed normal sensory nerve action potentials in the hands. Needle electromyography showed denervational discharge from the left supraspinatus, deltoid, and brachioradialis muscles, compatible with a radiculopathy that was relevant to the myotomes affected by the zoster infection with incomplete denervation.

The patient underwent physiotherapy with range-

of-movement exercises to his left shoulder. Functional electrical stimulation to prevent atrophy was carried out on the left shoulder deltoid muscles after resolution of skin lesions. The patient was discharged from hospital after 10 weeks. On discharge, left upper limb power had not improved and referral was made to the Geriatric Day Hospital for continuation of shoulder exercises and electrical stimulation therapy.

Treatment at the Geriatric Day Hospital was completed in the seventh month. Left shoulder girdle power showed obvious improvement: abduction (4/5), adduction (5/5), flexion (4/5), and extension (4/5). The power of left elbow flexion and extension were full with normal biceps, triceps, and brachioradialis reflexes. Post-herpetic neuralgia had also improved significantly.

Discussion

Herpes zoster occurs following reactivation of varicella zoster virus.⁷ The reactivation is thought to migrate in a retrograde manner along the sensory nerve to cutaneous tissue.⁸ It is relatively common and can affect healthy and immunocompromised individuals. Although no age-group is spared, it is more common among the elderly with a peak incidence between the ages of 50 and 70 years.⁹ The exact incidence in Hong Kong has not been established. Several precipitating factors for herpes zoster have been proposed and include localised trauma, underlying malignant disease, diabetes mellitus, and chronic steroid therapy.¹⁰⁻¹² Cigarette smoking is also thought to be a risk factor for herpes zoster reactivation.¹³ Immunocompromised individuals are at risk of reactivation, and infection is usually more severe and extensive.^{14,15}

Herpes zoster is complicated by motor involvement in 0.5% to 5% of cases.¹⁶ Sites of involvement, in descending order of frequency, are the thorax, neck, face, cervical, and lumbosacral area.¹⁷ Although the thoracic region is the most common site, a patient presenting with segmental paresis is rare.¹⁸ The highest rate of occurrence is found in facial paralysis following cranial herpes zoster, such as Ramsay Hunt and Horner's syndromes. It accounts for 46% of the general population and 80% of hospitalised patients with herpes zoster and motor paralysis.¹⁹ Segmental zoster paresis is rare and usually affects the upper limbs more than the lower limbs. A local review of 93 in-patients revealed three cases with segmental paresis, giving an incidence of 3.2%.¹² All three had segmental cervical paresis that resulted in ipsilateral upper limb weakness.

The interval between the appearance of a rash and the onset of muscle weakness varies. It most commonly occurs within days of the cutaneous rash but a delay of several months has been reported.^{20,21} The motor paresis of the patient reported here started early and weakness occurred 3 days before the skin lesions appeared, creating a diagnostic challenge.¹² Motor involvement usually follows the dermatomal distribution of zoster eruptions but in some cases may spread to widely separated segments.²² Needle electromyography usually shows denervation changes and is useful to confirm motor nerve involvement.^{23,24} Additional imaging such as magnetic resonance imaging (MRI) may be indicated in selected cases to rule out the presence of any structural lesions before confirming a diagnosis of motor herpes.

The exact pathogenesis of muscular paralysis following herpes zoster infection remains unclear. The dermatomal-myotomal association suggests a possibility of viral spread from the dorsal root ganglion to the anterior horn cells or anterior spinal nerve roots.²⁵ The direct spread of virus in return induces inflammatory involvement of the motor nerve that results in neurological deficit.²⁶ Electrophysiological studies have confirmed complete denervation of muscle with delayed reinnervation. This suggests that recovery depends on axonal regeneration from a proximal site.^{27,28} Motor neuron cell death may explain the poor prognosis associated with some cases of herpes zoster. Magnetic resonance imaging evidence of glial scar formation in cervical cord has been reported in one patient following resolution of post-herpetic polyradiculitis.²⁹ This may explain why up to one third of patients do not have a complete recovery.

The long-term prognosis for herpes zoster paresis is good.³⁰ Complete or near-complete recovery of muscular function occurs in about two thirds of patients. The time of recovery varies but is usually between 1 and 2 years.³¹ All of the three local cases recovered within a period of 3 to 9 months.¹² The treatment for segmental paresis includes analgesia for post-herpetic neuralgia, protection of the weakened muscles, maintenance of range-of-movement exercises, and a programme of graduated strengthening exercises.³² Functional electrical stimulation can be tried to prevent muscular atrophy before reinnervation occurs.

Conclusion

Albeit rare, zoster paresis should be one of the differential diagnoses of unilateral girdle muscle

weakness. The diagnosis of zoster paresis may be made when typical skin lesions develop. Electrophysiological study can confirm the diagnosis. If the diagnosis is doubtful, further investigations such as MRI can rule out other structural causes of motor weakness. Management involves prescription of range-of-movement and strengthening exercises as well as judicious pain relief for post-herpetic neuralgia.³³ More importantly, functional assessment and rehabilitation are required as in other cases of neuromuscular diseases. This is particularly important for older patients. Further studies are needed to determine whether antiviral treatment can limit the extent of motor deficit and hasten recovery.

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