CASE REPORT

Backpack palsy with Horner's syndrome

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SUMMARY

Traumatic injuries to the brachial plexus are typically high impact and can be debilitating, life-changing injuries. Backpack palsy is a rare but well-established cause of brachial plexus injury, arising as a result of heavy backpack use. We present an unusual case of backpack palsy with Horner's syndrome.

BACKGROUND

Backpack palsy can result in acute severe sensory loss and motor disability. The true incidence of backpack-associated neural compression is unknown, but is estimated to be between 29.7 and 53.7 per 100 000 military individuals each year.^{1–3}

CASE PRESENTATION

A 33-year-old fit and well male in the military was on a training exercise carrying a 50 kg backpack, 10 kg of webbing, 5 kg helmet and personal body armour, and a 7 kg rifle. After 6 hours of carrying the load, he developed tingling and numbness of his right arm. He continued training for a further 4 hours before removing the load, at which point he realised that he could not move his right arm.

He presented to our emergency department with paralysis and paraesthesia of the right upper limb. On examination, there was symmetrical muscle bulk and no fasciculations. He reported no pain and there was complete loss of sensation in dermatomes C6–T1. His right arm had decreased tone and 1/5 power from nerve roots C5–T1 with absent reflexes. All pulses were present bilaterally and the left arm was normal. Additionally, it was noted that he had miosis, ptosis and enophthalmus of the right eye, as shown in [figure 1](#). The patient had no previous symptoms of peripheral neuropathy.

INVESTIGATIONS

Plain radiographs of his cervical spine showed no fracture or cervical rib. MRI showed no gross abnormality of soft tissues in the region of the right brachial plexus and no evidence of spinal cord injury. In the cervical spine, there was normal vertebral body height, alignment, bone marrow signal and exit foramina.

TREATMENT

The patient was treated conservatively with intravenous steroids and daily physiotherapy. Over 4 days, he started to recover right arm function ([figure 2](#)). He was discharged with oral steroids and was seen regularly by physiotherapists.

OUTCOME AND FOLLOW-UP

One week postinjury the patient had improved sensation to 4/10 in the C4 and C5 dermatomes and shoulder abduction to 90°. Four weeks postinjury he regained flexion and extension at the elbow, and at 8 weeks he had shoulder abduction to 120°. At 4-month follow-up, sensation was normal up to T1 and he had normal finger movements. Ten months postinjury he was back to routine activities of daily living but not yet full military duties.

DISCUSSION

Brachial plexus injuries are seen in 1%–2% of patients with polytrauma in the UK and occur in almost 5% of motorcycle polytraumas.⁴ The most common mechanism of injury is traction, for example, during a motorcycle accident when the head is thrown back and shoulder downward.³ The plexus can also be damaged by compression or penetrating wounds.^{3,5}

The brachial plexus is formed from the confluence of spinal nerves C5–T1, with variations across the population to include contributions from the C4 and T2 roots. The plexus emerges posterior to the retropharyngeal space between the middle and anterior scalene muscles. It extends from the spinal cord to the axilla, and along its 15 cm length it can be divided from proximal to distal into roots, trunks, divisions, cords and branches ([figure 3](#)). The terminal branches innervate all muscles and skin of the upper limb. The superficial location of the brachial plexus between the cervical spine and the mobile upper limb leaves it vulnerable to injury.

Brachial plexus injuries can be classified in several ways, the most clinically useful of which is into supraclavicular (roots and trunks), retroclavicular (divisions) or infraclavicular (cord and branches) lesions. Supraclavicular lesions can be further subdivided into upper (C5 and C6), middle (C7) and lower (C8–T1) plexus injuries.⁶ It is of prognostic importance to specify the injury's location relative to the dorsal root ganglion. Preganglionic lesions are less likely to recover spontaneously since an avulsion injury proximal to the dorsal root ganglion involves the central nervous system, which does not undergo Wallerian degeneration and has no potential to regenerate.⁵ Postganglionic lesions have similar pathophysiology to other injured peripheral nerves in that these lesions have the potential to recover by regeneration.

Backpack palsy (also known as cadet palsy or rucksack paralysis) was first described by Daube in 1969. He presented a case series of 17 military personnel with unilateral motor and sensory disturbance of the upper limb after carrying heavy

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Figure 1 Miosis and ptosis of the right eye.

backpacks during service in Vietnam, although more recent cases have also been described.^{7,8} The pathogenesis of backpack palsy is considered to be direct compression of the plexus from the straps of the backpack causing a conduction block from temporary demyelination.⁶ Backpack palsy has also been described following civilian leisure activities, such as mountaineering, hiking and even in boy scouts.^{9–11} A second backpack related neuropathy recently reported was isolated axillary nerve palsy caused by a pressure effect of the strap on the subscapular artery.¹² There are no previous case reports of backpack palsy with Horner's syndrome.

Horner's syndrome, classically described as ipsilateral ptosis, pupillary miosis, enophthalmus and hemifacial anhidrosis occurs when there is disruption to the sympathetic chain. The anhidrosis may be undetected or absent as in this case. The sympathetic chain is in close proximity to the lower roots of the brachial plexus, though its fibres ascend from the apex of the lung to the

Figure 2 Day 2 post-injury showing improving right shoulder abduction to 20°.

Figure 3 Diagram of the brachial plexus demonstrating relationship with sympathetic chain. AXI, axillary nerve; MC, musculocutaneous nerve; MED, median nerve; RAD, radial nerve; ULN, ulnar nerve.

orbit, and a lesion anywhere along its length can cause Horner's syndrome.

The aetiology of Horner's syndrome is related to the location of the lesion; either first-, second- or third-order syndromes. First-order syndromes are disorders of the central nervous system, for example, brainstem stroke or spinal cord demyelination. Second-order neurons travel through the brachial plexus from the sympathetic trunk to the superior cervical ganglion. Brachial plexus trauma can therefore cause a second-order Horner's syndrome. Furthermore, Horner's syndrome points to a preganglionic brachial plexus injury.¹³ Third-order Horner's syndromes are commonly due to a lesion of the internal carotid artery.¹⁴

Previous cases of brachial plexus traction injuries with Horner's syndrome have been described, for example in obstetrics, where concurrent Horner's syndrome is a poor prognostic factor for spontaneous recovery of the neonatal limb.¹⁵ There are also cases of the conditions arising as a complication of patient positioning in neurosurgery.¹⁶ Although the presence of Horner's syndrome in our case alerted us to a preganglionic injury, the MRI showed no evidence of this. Instead, the lesion was found to be a temporary demyelinating conduction block typical of backpack palsy without nerve root avulsion.

National Institute for Health and Care Excellence guidelines state that brachial plexus injuries in which the nerves remain intact, as in this case of neuropraxia, can be managed conservatively with physiotherapy.¹⁷ Physiotherapy aims to maintain passive range of motion, reduce pain, prevent stiffness and increase the function of the affected limb. Although steroids were given in this case, there is no conclusive evidence that it improves the outcome of backpack palsy.

Surgery is rarely necessary in backpack palsy as recovery is usually spontaneous.¹⁸ Approximately, 80% of patients make a full recovery of function over 9 months of conservative management, the remaining 20% having a prolonged or incomplete recovery as a result of axonal loss.^{6,18} At 6 months postinjury the rate of natural repair and improvement slows and if there has been no progress clinically, surgery may be considered.⁴

When surgery is required, it will depend on the location and degree of nerve disruption. In complete transection of the postganglionic nerve, direct nerve repair with epineurial micro-sutures may be possible if nerve endings can be approximated with minimal tension. If not, interposition autologous nerve grafts may be employed. Nerve grafting is not an option for

preganglionic injuries and nerve transfer may be required.^{19 20} Nerve transfer involves a healthy donor nerve being redirected and attached to the distal end of the damaged nerve to innervate the affected muscle. Donor examples include the intercostal and phrenic nerves.²¹ Nerve regeneration occurs around 1 mm a day so a prolonged period of rehabilitation will be required. Surgical intervention should take place in a specialist unit with a multi-disciplinary team.²²

Learning points

- ▶ Brachial plexus injuries are a recognised sequelae of heavy backpack use and can result in acute severe sensory loss and motor disability. Backpack palsy has a good prognosis with 80% of cases recovering completely over 9 months.
- ▶ Careful evaluation of the weight carried during training exercises and increased awareness of backpack palsy injuries among military recruits is necessary to avoid this preventable condition.
- ▶ The presentation of concurrent brachial plexus injury and Horner's syndrome should raise a high suspicion of a preganglionic lesion.
- ▶ The treatment of brachial plexus injuries requires a multidisciplinary approach and if surgery is required, this should take place in a specialist brachial plexus injury unit.

Contributors OS and KYW contributed equally. OS, KYW and PS were all involved in drafting, reviewing and final submission of the manuscript.

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