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BRIEF COMMUNICATION

Peroneal nerve mononeuropathy associated with herpes zoster. A case report

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Abstract
Background Sensory or motor syndromes, especially mononeuropathies, are infrequent complications of herpes zoster.
Case Report We describe a 77-year-old woman who developed a severe right common peroneal nerve mononeuropathy with clinical onset 10 days after a vesicular rash consistent with herpes zoster in the territory of distribution of this nerve.
Conclusion To our knowledge, peroneal nerve mononeuropathy associated with herpes zoster has not been reported previously.

Keywords Herpes zoster · Neurological complications · Peroneal nerve · Mononeuropathy

Introduction

Herpes zoster is an infectious disease due to the reactivation of the varicella zoster virus (VZV), which usually presents with a self-limiting vesicular rash. Postherpetic neuralgia, which can precede the eruption by several days to weeks, is the most frequent neurological complication of herpes zoster. Sensory and motor complications of herpes zoster, mainly segmental paresis, are infrequent: while classical textbooks described 1–5% of cases with cutaneous herpes zoster [1], 3 of 205 patients (1.5%) with herpes zoster developed motor complications in a recent report [2]. Development of mononeuropathies other than facial palsy is even rarer. We report a case of isolated peroneal nerve lesion related with typical herpes zoster.

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Case report

A 77-year-old woman without any previous disease was referred to our Unit by her family physician because of suspected "postherpetic neuralgia." Three months before, she had developed a vesicular rash consistent with herpes zoster together with severe dysesthesia and loss of sensation affecting the dorsal surface of her right foot and the lateral aspect of her right leg. Despite the pain, the patient was not immobilized at any time. Ten days after the onset of the rash, she also complained of difficulty walking due to right foot drop. The vesicular rash had disappeared gradually during the first month despite she had not been treated with acyclovir. She had received gabapentin 600 mg/day (reduced to 300 mg/day because of alopecia and drowsiness) and reported improvement of the pain, but persistence of loss of sensation in the dorsum of the right foot and in the lateral side of the right leg and of foot weakness. Clinical examination showed muscle weakness (according to the Medical Research Council Scale) in the right tibialis (3/5), peroneus longus (3/5), and extensor digitorum brevis (0/5) muscles, and tactile hypoesthesia and hypoalgesia in the right superficial peroneal nerve territory. Knee and ankle reflexes were normal, and cutaneous plantar reflex was flexor. Because our first evaluation of this patient was done 3 months after clinical onset, we did not perform cerebrospinal fluid examination.

An initial electrodiagnostic study was performed 3 months after clinical onset. Needle electromyography showed no abnormalities in the gastrocnemius, abductor hallucis, and paraspinal L5 muscles; absence of denervatory activity and of voluntary

Table 1 Cases reported of peroneal nerve lesion associated to herpes zoster infection

Author, year [ref]	Age/ gender	Clinical features	Needle electromyography findings	Nerve conduction study results
Sachs et al., 1996 [3]	69/F	■ Severe shooting pain radiating from the left buttock to the anterior aspect knee and ankle. ■ Development of a vesicular rash in the lateral aspect of left ankle and dorsal surface of the left foot 10 days after the onset of pain, with decreased sensation to light touch and pinprick and allodinia in the same areas. Treatment with acyclovir (doses not specified) during 1 week. ■ Development of a progressive left leg weakness and urinary retention 11 days after the onset of pain (strength 0/5 in ankle dorsiflexors, evertors, and toe extensors, 3/5 in left knee flexors, toe flexor,s and plantar flexors). ■ Recovery of sensitive symptoms and significant improvement of knee flexion and ankle inversion in 3 months. Persistence of severe weakness in ankle dorsiflexors (1/5) 10 months after onset.	■ 3+ fibrillations and positive sharp waves with absence of recruitment of motor units in left extensor digitorum brevis, tibialis anterior, and extensor hallucis longus (the first motor unit potentials appeared at 8 months in TA, 14 month in EHL and 22 months in EDB) ■ 2+ fibrillations and reduced recruitment of mildly prolonged polyphasic motor units in the left tensor fascia lata. ■ Normality of EMG of L3-S1 paraspinal muscles and short head of biceps femoris.	■ Lack of recording of CMAPs from the left extensor digitorum brevis and tibialis anterior, marked decrease in amplitude of CMAP from the left abductor hallucis. CMAP was first recorded from the TA at 2 months, with amplitude reduced by 73%. ■ Lack of recording of SNAP from the left superficial peroneal nerve, and normal SNAP from the left sural.
Takahama et al., 2007 [4]	72/M	■ Development of right leg pain followed by a vesicular rash in the right leg 6 days later and disseminated vesicular rash 8 days later. Treatment with acyclovir 750 mg/day i.v. for 14 days. ■ Development of right drop foot and numbness of the right foot 10 days after pain onset; and development of numbness and pain on the left side of the face, difficulty closing the left eye, and drooping of the left angle of the mouth 23 days later.	■ Neurogenic pattern in the right peroneal muscle.	■ Absence of motor evoked response in the right peroneal nerve and motor latency delay in the left facial nerve.
Leo et al., 2009 [5]	79/F	■ Acute onset of sciatic pain in the right lower limb and paresthesias over the right buttock. ■ Development of right foot droop 4 days later (inability for the dorsiflexion of the right foot and great toe, which did not improve 9 months later) and a petechial eruption on the sacrum and along the anterior surface of the right leg and foot in the L4-L5 dermatomes 6 days later. ■ The patient was on treatment with prednisone because of temporal arteritis, and had 3 prior cases of herpes zoster and 2 of temporary Bell palsy. Treatment with acyclovir 600 mg/day i.v. for at least 1 week (not stated) and with pregabalin 75 mg/day.	■ Not stated.	■ Absence of motor evoked response in the right peroneal nerve.
Boylu et al., 2010 [6]	68/M	■ Development of subacute onset right foot drop 10 days after vesicular rash around the right knee and the popliteal fossa (strength 2/5 in tibialis anterior, 2/5 in extensor hallucis longus, 2/5 in peroneus longus and 2/5 in gastrocnemius medialis muscles). Treatment with acyclovir (doses not specified) during 1 week ■ Impaired sensation distal to the right knee except for the safenous nerve region and decreased right ankle reflex. ■ Recovery almost complete of muscle weakness at the end of 3rd month (strength +4/5 in tibialis anterior, extensor hallucis longus, and peroneus longus muscles).	■ Fibrillations and positive sharp waves and polyphasic motor unit potentials with increased amplitude and reduced recruitment on the muscles innervated by tibialis anterior and common peroneal nerve distal to the right knee.	■ Lack of evocation of CMAP of the right common peroneal and SNAP of the right superficial peroneal nerves. ■ Prolonged distal motor latency and decreased CMAP amplitude of the right tibial nerve. ■ Prolonged distal sensory latency and decreased SNAP amplitude of the right sural nerve.

Table 1 (continued)

Author, year [ref]	Age/ gender	Clinical features	Needle electromyography findings	Nerve conduction study results
Valle-Arcos et al. (current case)	77/F	■ Acute onset of vesicular rash, severe dysesthesia, and loss of sensation affecting the dorsal surface of her right foot and the lateral aspect of her right leg. ■ Development of right foot drop 10 days after the clinical onset. ■ Clinical examination 3 months later showed strength 3/5 in the right tibialis and peroneus longus muscles and 0/5 in extensor digitorum brevis; and tactile hypoesthesia and hypoalgesia in the right superficial peroneal nerve territory. ■ Muscle strength remained identical 9 months after clinical onset.	■ No abnormalities in the rectus femoris, biceps femoris, gastrocnemius, abductor hallucis, and paraspinal L5 muscles. ■ Absence of denervatory activity and of voluntary activity in the right extensor digitorum brevis. ■ Mild denervatory activity consisting in fibrillations, and polyphasic motor unit potentials with normal amplitude and moderately reduced recruitment in the right tibialis and peroneus longus.	■ Normality of amplitudes and latencies of SNAP of the right sural and posterior tibial nerves. ■ Normality of amplitude and latency of CMAP and of MNCV in the right posterior tibial nerve. ■ Lack of evocation of SNAP in the right superficial peroneal nerve. ■ Lack of evocation of CMAP by stimulation of the right deep peroneal nerve in the extensor digitorum brevis, and normal latencies and moderate decreased amplitude in the tibialis anterior.

CMAP compound muscle action potential, *EDB* extensor digitorum brevis, *EHL* extensor hallucis longus, *EMG* electromyography, *MNCV* motor nerve conduction velocity, *SNAP* sensory nerve action potential, *TA* tibialis anterior, *VZV* varicella-zoster virus

activity in the right extensor digitorum brevis; scarce denervatory activity consisting in fibrillations, and polyphasic motor unit potentials with normal amplitude and moderately reduced recruitment in the right tibialis and peroneus longus muscles. Nerve conduction studies showed (1) normality of amplitudes and latencies of the sensorial action potentials (SNAP) of the right sural and posterior tibial nerves; (2) normality of amplitude and latency of the compound muscle action potential (CMAP) and of motor nerve conduction velocity in the right posterior tibial nerve; (3) SNAP in the right superficial peroneal nerve could not be evoked; and (4) CMAP by stimulation of the right deep peroneal nerve was not evoked in the extensor digitorum brevis, and showed normal latency and moderate decreased amplitude in the tibialis anterior (Table 1). Electrodiagnostic studies performed 3, 6, 9, and 12 months after the clinical onset were identical with the exception of the disappearance of the denervatory activity in the right tibialis anterior and peroneus lateral longus (proximal and paraspinalis muscles were not evaluated in the three follow-up electrodiagnostic studies). Muscle weakness and sensory loss persisted through the time despite rehabilitation therapy.

Discussion

The reported patient suffered from a typical cutaneous herpes zoster, which was followed by pain and sensory loss in the right superficial peroneal nerve territory and motor weakness corresponding to the territory of innervation of both the right deep and superficial peroneal nerve. Electrodiagnostic studies showed data consistent with a predominantly axonal lesion affecting exclusively the common peroneal nerve.

To our knowledge, isolated lesion of peroneal nerve associated with herpes zoster has not been previously reported. A PubMed search crossing the terms "zoster" and "herpes zoster" with "peroneal" or "peroneal nerve" or "peroneal nerve palsy" retrieved six items, being valid only four, because the other two were not related with this issue (Table 1). In the other patient reports, the lesion of peroneal nerve was associated to contralateral facial palsy [4], ipsilateral tibial nerve neuropathy [6], concomitant L4-L5 roots involvement [3, 5], and a possible intramedullary involvement [3].

The reason for the isolated lesion of peroneal nerve in our patient is not well understood. Because according to the current knowledge of the pathogenesis of VZV infections, VZV could be transported anterogradely from dorsal root ganglion neurons through spinal nerve fibers to the skin [7], it seems tentative to speculate with the hypothesis of a possible relationship with anterograde transport to more distal regions in the peripheral nerve system.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Informed consent Informed consent was obtained from the individual participant included in the study.

Ethical standard The authors declare that they acted in accordance with ethical standards laid down in the 1964 Declaration of Helsinki.

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