SHORT REPORT

The accessory deep peroneal nerve: a pitfall for the electromyographer

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Abstract

The presence of an accessory deep peroneal nerve may alter the usual clinical and electrophysiological characteristics of peroneal nerve lesions. Three cases of such an anomalous innervation and a peroneal nerve lesion are presented.

The accessory deep peroneal nerve is a common anatomical variant that arises from the superficial peroneal nerve and innervates the extensor digitorum brevis of the foot.[1 2] Awareness of this anomalous innervation is important for the correct evaluation of peroneal nerve lesions. We report a case of deep peroneal palsy in the presence of an accessory deep peroneal nerve, a case of accessory deep peroneal nerve palsy, and a case of superficial peroneal palsy in the presence of an accessory deep peroneal nerve.

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Case reports

Case 1

In April 1990 a 55 year old man developed a complete left foot drop which hardly improved over the next few months. Examination in July 1990 showed an impairment in dorsiflexion of the left foot. The left extensor digitorum brevis was well developed and showed visible contraction when he was asked to extend his toes. Inversion, eversion, and plantar flexion of the foot were normal. The electrophysiological findings are presented in the table.

Case 2

In April 1990 a 19 year old man was injured with a penknife on the lateral aspect of the distal third of his left leg. The wound was closed within a few hours and the patient did not complain about anything afterwards. In July 1990 examination showed appreciable atrophy of the left extensor digitorum brevis. No visible contraction of the left extensor digitorum brevis was elicited when the patient was asked to extend his toes. Inversion, eversion, dorsiflexion, and plantar flexion of the foot were normal (table).

Case 3

In February 1990 a 23 year old man developed a haematoma on the lateral aspect of the upper third of his right leg and complained about parethesia of the dorsum of his right foot. He was referred to our department in October 1990. Examination showed sensory loss over the dorsum of the right foot and appreciable atrophy of the right extensor digitorum brevis. No visible contraction of the right extensor digitorum brevis was elicited when he was asked to extend his toes. Eversion of the right foot was mildly impaired. Inversion, dorsiflexion, and plantar flexion of the foot were normal (table).

Table *Results of nerve conduction study and electromyographic examination*

		Case No 1	2	3
Nerve conduction study				
Peroneal nerve:				
Superficial	Evoked potential (μV)	7·6	19	No response
	Conduction velocity (m/s)	42	41	No response
Common*	Evoked potential (mV)	5·68	0·27	0·02
	Conduction velocity (m/s)	—	—	—
Deep†	Evoked potential (mV)	No response	No response	No response
	Conduction velocity (m/s)			
Accessory deep‡	Evoked potential (mV)	5·31	1·35	0·02
	Conduction velocity (m/s)	49	46	—
Electromyographic examination				
Anterior tibialis	Rest	Fibrillation	Normal	Normal
	MVC	No MUAP	Normal	Normal
Extensor digitorum longus	Rest	Fibrillation	Normal	Normal
	MVC	Single MUAP	Normal	Normal
Extensor digitorum brevis	Rest	Normal	Fibrillation	Normal
	MVC	Normal	No MUAP	Single MUAP
Peroneus	Rest	Normal	Normal	Normal
	MVC	Normal	Normal	Polyphasic MUAPs

Stimulating electrode at knee*, over dorsum of ankle†, and behind lateral malleolus‡, with recording electrode in extensor digitorum brevis in all three instances.
MUAP = motor unit action potential.
MVC = Maximum voluntary contraction.

Discussion

The accessory deep peroneal nerve is a comparatively common variation in the innervation of the extensor digitorum brevis, which is usually innervated by the deep peroneal nerve. It arises from the superficial peroneal nerve, runs along the posterior border of the peroneus brevis muscle, and subsequently winds around the lateral malleolus to enter the lateral border of the extensor digitorum brevis.[3] The accessory deep peroneal nerve usually innervates the lateral portion of the extensor digitorum brevis,[2] but in some cases this muscle is exclusively innervated by the accessory deep peroneal nerve.[4] This anatomical variant has been reported in 19–24% of subjects.[5-7]

The clinical and electrophysiological findings in case 1 clearly indicated the presence of a left

deep peroneal palsy. The tibialis anterior and extensor digitorum longus were clinically and electrophysiologically impaired whereas the peroneii were spared. Nerve conduction study of the superficial peroneal nerve was also normal. The unusual and unexpected finding for a deep peroneal palsy was the absence of any clinical or electrophysiological abnormalities in extensor digitorum brevis. This apparent inconsistency was resolved by stimulation over the posterolateral aspect of the ankle just behind the lateral malleolus, which elicited a normal compound muscle action potential in the muscle. The final diagnosis was therefore a lesion of the left deep peroneal nerve in the presence of a left accessory deep peroneal nerve innervating the extensor digitorum brevis, as in the two cases reported by Gutmann.[8]

In case 2 the tibialis anterior and extensor digitorum longus were normal whereas the left extensor digitorum brevis was partially denervated. The absence of any potential evoked in this muscle by stimulating the deep peroneal nerve might have led to the incorrect diagnosis of a distal lesion of the deep peroneal nerve. Nevertheless, the presence of a small potential elicited in the left extensor digitorum brevis by stimulating behind the lateral malleolus suggested an anomalous innervation of the muscle by an accessory deep peroneal nerve. Furthermore, the very small potential evoked in the left extensor digitorum brevis by stimulating the common peroneal nerve compared with that elicited by stimulating the accessory deep peroneal nerve suggested the presence of a conduction block in the accessory deep peroneal nerve. The final diagnosis was therefore a lesion of the left accessory deep peroneal nerve at the lateral aspect of the distal third of the leg at the site of the injury.

The clinical and electrophysiological findings in case 3 strongly suggested the presence of a right superficial peroneal palsy. The right peroneus muscles showed numerous polyphasic potentials and no sensory potential was recorded from the right superficial peroneal nerve. The unusual and unexpected finding was the presence of clinical and electrophysiological abnormalities in the extensor digitorum brevis. This apparent inconsistency was resolved by stimulating behind the lateral malleolus which evoked a small potential in the muscle. The involvement of the extensor digitorum brevis was therefore related to the presence of an accessory deep peroneal nerve arising from the superficial peroneal nerve. The final diagnosis was a lesion of the right superficial peroneal nerve at the upper third of the leg at the site of the haematoma in the presence of an accessory deep peroneal nerve innervating the extensor digitorum brevis.

In conclusion, the presence of an accessory deep peroneal nerve may alter the usual clinical and electrophysiological characteristics of peroneal nerve lesions. Unexpected findings in electromyographic examination of the extensor digitorum brevis should raise the possibility of an anomalous innervation of the muscle by an accessory deep peroneal nerve, which would be then confirmed by stimulating over the posterolateral aspect of the ankle behind the lateral malleolus.

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