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Clinical and Electrophysiological Characteristic of Anomalous Innervations in the Lower Extremities

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Abstract

Introduction: Anomalous innervations of the extremities are common and influence the interpretation of electrophysiologic studies and clinical features of those with peripheral nerve lesions. The aim of this study was to describe the most common anomalous innervations in lower extremities and to point out their clinical repercussions.

Methods: Article has an analytical character and review of literature, including some personal articles.

Results and Discussion: Anomalous innervations of the upper and lower extremities are common and influence on the interpretation of electrophysiological data during electromyoneurography. Namely, in the course of an electrodiagnostic investigation of a peripheral nerve lesion, the examiner may be confronted with unexpected findings in contradiction with the clinical picture. In this review, a description is given of the most common innervation anomalies in lower extremities.

Conclusion: As anomalous innervations of the extremities are common and influence the interpretation of electrophysiological studies in normal subjects and those with peripheral nerve lesions. Detailed anatomical knowledge is essential for accurate interpretation of physical examination, electrophysiological findings, diagnosis, prognosis and reducing the risk of iatrogenic injuries during surgical procedures.

Keywords: Anomalous innervations; Lower extremities; Electromyoneurography

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Introduction

Double innervation, abnormal innervation and communications among nerves are causes of anomalies of innervation. The different anatomical anomalies of peripheral nerves occur with various frequencies in the population [1,2]. The most widely recognized are Martin-Gruber Anastomosis (MGA) in the upper and Accessory Deep Peroneal Nerve (ADPN) in lower extremities.

Anomalous innervations of the upper and lower extremities are common and influence the interpretation of neurophysiological studies in normal subjects and clinical features of those with peripheral nerve lesions [3]. Namely, in the course of an electrodiagnostic investigation of a peripheral nerve lesion, the examiner may be confronted with unexpected findings in contradiction with the clinical picture. Awareness of such anomalies may be important in order to avoid misdiagnoses during electrodiagnostic study, such as a conduction block involving the ulnar nerve or carpal tunnel syndrome or axonal lesion of the peroneal nerve [1-6].

On the lower extremity the most widely recognized is Accessory Deep Peroneal Nerve (ADPN) [7]. From the late 1960s, this anomalous variation has been reported to occur in as many as 28% of people [8-10]. This anomaly has an autosomal dominant pattern of inheritance in man [11-13].

The accessory deep peroneal nerve

According to standard textbooks of anatomy the peroneal nerve is derivate from the L4-S1 nerve roots, which travel from the lumbosacral plexus and eventually the sciatic nerve. Within the sciatic nerve, the fibers forming the peroneal nerve run separately from those that become the tibial nerve.

More distally, the sciatic nerve bifurcates above the popliteal fossa into the common peroneal and tibial nerves. The common peroneal nerve first gives rise to the lateral cutaneous nerve of the knee, and divides into the deep and superficial peroneal nerves.

The deep peroneal nerve innervates the peroneus tertius muscle and the dorsiflexors of the ankle and toes, including the tibialis anterior muscle, extensor digitorum longus and extensor hallucis longus, and extensor digitorum brevis [2,14,15].

The Accessory Deep Peroneal Nerve (ADPN) has been regarded as an anomalous nerve derived from the superficial peroneal nerve or its branch and supplies motor innervations for Extensor Digitorum Brevis (EDB) and sensory innervations for the lateral part of the ankle and foot regions [8]. ADPN arises from the superficial peroneal nerve on the lateral aspects of the leg, descends along the posterior border of the peroneus brevis muscle near to the Achilles tendon and sural nerve and winds around the lateral malleolus [5,9].

ADPN was reported initially by Ruge in 1878, and the first anatomical description was provided by Bryce (1891, 1901). Winckler published in 1934 a more detailed analysis of this nerve and reported a more frequent occurrence in man (7 of 19 legs) [8].

The EDB is usually innervated exclusively by the deep peroneal nerve, however, in some cases, one or both of the EDB muscles are innervated by the ADPN nerve (partially or exclusively), and could be detected by nerve conduction studies (Figure 1)[15,9,10-12].

It was reported that ADPN was present in 12% to 35% of the population [16], but it was found that there is a wide variation of prevalence of ADPN among different studies [16-21]. One a meta-analysis study assessed the overall pooled ADPN prevalence of 18.8%, the electrophysiological pooled ADPN prevalence of 13.6%, and the anatomical pooled prevalence of 39.3% [21]. This could be explained by the differences between studies regarding the studied population and the techniques used in the assessment of ADPN, whether anatomical or electrophysiological studies [12,16,21].

The ADPN has more than one clinical importance [8,12,16,21,22]. Studying the ADPN can complicate the clinical picture and disturb the interpretation of the electrophysiological studies of common peroneal, deep peroneal, and superficial peroneal nerves lesions and injuries, as well as, ADPN neuropathy [8,12]. Namely, superficial peroneal nerve and its branches (including ADPN) are risk for iatrogenic damage while performing arthroscopy, local anesthetic block, surgical approach to the fibula, open reduction and internal fixation of lateral malleolar fractures, application of external fixators, elevation of a fasciocutaneous or fibular flaps for grafting, surgical decompression of neurovascular structures, or miscellaneous surgery on leg, foot and ankle [12,17].

Tibial-to-peroneal nerve communication

Innervation of the EDB by the tibial nerve is rarely reported as a normal variant [23-25], Phillips and Morgan in 1993 [25] described the findings of tibial-peroneal nerve communication on the basis of intraoperative nerve conduction studies. Stimulation of the tibia1 nerve produced a contraction from the peroneus longus muscle and a nerve action potential in the distal peroneal nerve [5,26,27]. A prior tibial-peroneal communication was reported but no details given [5,27,28]. Linden and Berlit [28] this nerve communications named „all tibial foot“, and this rear anomalous innervation were described in several other case reports [29-32]. Yamashita et al. [31] emphasized

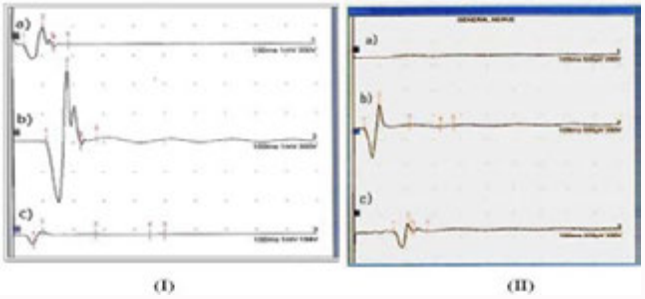

Figure 1: Muscle extensor digitorum brevis partially (I) and exclusively (II) innervated by accessory deep peroneal nerve.
a) Action potential evoked when stimulating the deep peroneal nerve at the ankle; b) action potential evoked when stimulating the common peroneal at the knee; c) action potential evoked when stimulating the accessory deep peroneal nerve.

that this communication between the tibial and deep peroneal nerves include sensory fibers. Swerdloff and Stewart in their recently paper [32] reported frequency of this anomalous as 11% (8 cases out of 72 subjects), and communication was present in both legs in half of the subjects.

Anatomical variations of the sural nerve

The Sural Nerve (SN) is a sensory nerve supplying the skin of the lateral side of the foot lateral and posterior part of the inferior third of the leg. In most of cases, it is formed by the union of the Medial Sural Cutaneous Nerve (MSCN), a branch of the tibial nerve, and the Lateral Sural Cutaneous Nerve (LSCN), a branch of the common fibular (peroneal) nerve. The site of union of the MSCN and LSCN to form the sural nerve is highly variable. It may be in the popliteal fossa, the distal third of the leg, or at the ankle [33,34].

In a clinical setting, the SN is widely used for both diagnostic (biopsy and nerve conduction velocity studies) and therapeutic purposes (nerve grafting) and the LSCN is used for a sensate free flap [34-36]. Bilateral asymmetry in the pattern of sural nerve formation is the rule rather than the exception, and a site of union is variable. So, anatomical variants in the formation and course of the SB are common in the population [34]. A detailed knowledge of the anatomy of the SN and its contributing nerves are clinically is very important, and clinicians, especially surgeons, should be aware of these variations to avoid iatrogenic injury to the nerve during operative procedures [37,38].

Conclusion

Anomalous innervations of the lower extremities, as well as upper one, are common and influence the interpretation of electrophysiological studies and clinical features of those with peripheral nerve lesions. The most common innervation anomaly in lower extremities is accessory deep peroneal nerve. Detailed anatomical knowledge is essential for accurate interpretation of physical examination, electrophysiological findings, diagnosis, prognosis and reducing the risk of iatrogenic injuries during surgical procedures. If these variations are not given enough attention, errors and other consequences will be inevitable.

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