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Invited review

Neuropathies of the foot

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Abstract

Compared with the common neuropathies affecting the hands (carpal tunnel syndrome and ulnar neuropathy), neuropathies of the feet have received less attention in the past. This is partly because of the rarity of these disorders as well as the lack of reliable electrophysiological tests for them. Over the years, nerve conduction tests for various nerves of the feet have been reported, and at this time techniques for all the nerves of the feet are available to the electromyographer.

This review will provide up-to-date information on the current status of the research and issues relating to the neuropathies of the foot, with an emphasis on the most useful tests and the caveats for clinical neurophysiologists.

Keywords: Burning feet syndrome; Distal sensory neuropathy; Foot neuropathy; Interdigital neuropathy; Morton's neuroma; Small-fiber neuropathy; Tarsal tunnel syndrome

1. Introduction

Compared with the common neuropathies affecting the hands (carpal tunnel syndrome and ulnar neuropathy), neuropathies of the feet have received less attention in the past. This is partly because of the rarity of these disorders as well as the lack of reliable electrophysiological tests for them. Over the years, nerve conduction tests for various nerves of the feet have been reported (Oh, 2003), and at this time techniques for all the nerves of the feet are available to the electromyographer.

This review will provide up-to-date information on the current status of the research and issues relating to the neuropathies of the foot, with an emphasis on the most useful tests and the caveats for clinical neurophysiologists.

Neuropathies of the feet can be divided broadly into two main categories: polyneuropathy and mononeuropathies. Polyneuropathy of the feet is predominantly a sensory neuropathy.

2. Sensory polyneuropathy (neuropathy) of the feet

Sensory neuropathy of the feet is sometimes referred to as burning feet syndrome (BFS) because one of the common causes of this disorder is distal sensory neuropathy (DSN). The causes of BFS are multiple, ranging from plantar fasciitis to distal sensory neuropathy. In this article, we used the narrower definition of BFS, i.e., DSN confined to the feet representing sensory neuropathy of the feet.

2.1. Distal sensory neuropathy

DSN has become a major management problem in many neuromuscular disease clinics (Wolfe et al., 1999; Periquet et al., 1999). It is common and usually benign in elderly patients, and is characterized by symmetrical painful paresthesia in the feet and lower legs. This entity is often labeled small-fiber neuropathy (SFN as defined in 2.2) (Holland et al., 1998; Jamal et al., 1987), distal SFN (Gorson and Ropper, 1995; Stewart et al., 1992), or painful neuropathy (Periquet et al., 1999), because pain, which is mediated by unmyelinated fibers (C fibers) or small myelin-

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ated fibers (A-∂ fibers), is the symptomatic hallmark. However, SFN is a misnomer because many DSN patients have a large-fiber neuropathy (LFN).

The functions of nerve fibers depend on their size: unmyelinated C fibers and small myelinated (A-∂) fibers are responsible for pain whereas large myelinated fibers (A-β and A-α) are responsible for proprioception, vibratory sensation, reflexes, and motor strength (Oh, 2003). Depending on whether small or large fibers are involved, DSN can be classified into two types: SFN and LFN. SFN is characterized by impaired pinprick sensation and LFN, by vibration loss, position sense loss, and absent or decreased reflexes. In Periquet's series, 51% of cases were classified as having LFN and 38%, SFN (Periquet et al., 1999), In Herrman's series, there were an equal number of cases of LFN and SFN (Herrmann et al., 2004). In our series, two-thirds of patients had LFN (Oh et al., 2001a,b,c). The distinction between the two types of DSN is important since the underlying cause is more likely to be identifiable and nerve conduction abnormalities are more common in LFN (Periquet et al., 1999; Herrmann et al., 2004; Nodera et al., 2002).

The cause of DSN has been unknown in a majority of cases in all previous studies including our own. In two large series in neuropathy with normal routine nerve conduction, causes were determined in 35–37% of cases, the most common being diabetes mellitus (Oh et al., 2001a,b,c; Herrmann et al., 2004). In Periquet's study, causes were found in only 19% of cases, the most common being monoclonal gammopathy (Periquet et al., 1999). This difference is most likely due to the variation in case selection criteria: no diagnosis accounting for neuropathic pain was required as a selection criterion at the time of referral (Periquet et al., 1999). Thus, some obvious causes such as diabetes mellitus or alcoholism were most likely excluded. The next most common causes are rheumatoid diseases and vitamin B12 deficiency. In our recent unpublished data on 116 patients with "pure" DSN, a cause was found in 66% of cases (Young et al., 2006). The most common causes were diabetes mellitus and glucose intolerance.

In DSN, the nerve conduction abnormalities revealed by extensive testing are not reported in cases of "pure" sensory neuropathy in the literature (Table 1). The best available data in this regard were from two studies which included some patients with mild distal muscle weakness in 41% of cases in Wolfe's series (1999) and mild toe dorsiflexion weakness in 25% of cases in Gorson's series (1995). Nerve conduction abnormalities were reported in 87% of 93 cases in Wolfe's series (1999), indicating that the NCS is extremely helpful in confirming the diagnosis of neuropathy. In this series, NCS included median, ulnar, peroneal, and tibial nerve motor conduction and median, ulnar and sural sensory nerve conduction. In his series, abnormal sensory nerve conduction was found in 74% and abnormal motor nerve conduction in 60% of cases. Needle EMG abnormalities in anterior tibialis, medial gastrocnemius, and hand intrinsic muscles were present in 70% of 64 patients; fibrillation potentials were observed in 42% and chronic neurogenic change in 64% of cases. In Gorson's series (1995), nerve conduction abnormality was reported in 55% of 20 cases. However, the NCS in this study was not as extensive as in Wolfe's series (1999): at least one motor and one sensory nerve conduction in the lower extremities and NCS in the upper extremity in 80% of cases. In "pure" DSN without any motor weakness, sural nerve conduction data are available: abnormality in 23% of 133 cases in Nodera's series and in 51% of 117 cases in Periquet's series. In Nodera's series (2002), abnormal sural nerve conduction was more frequent in "clinical LFN" (27%) than in "clinical SFN" (9%). In our recent unpublished data on 116 patients with "pure" DSN, ten nerves were tested (Young et al., 2006). Normal findings in all tested nerves were observed in 14% of cases and abnormal nerve conduction in more than 40% of tested nerves (definitely abnormal NCS), in 77% of cases.

Nerve conduction abnormalities were typical of axonal neuropathy, which was supported by the sural nerve biopsy in a small number of cases; axonal degeneration in 13 of 14 cases (Wolfe et al., 1999) and in nine of 12 cases (Gorson and Ropper, 1995). In the latter series, the biopsy showed axon loss in six cases, small fiber loss in three cases. Among six cases of SFN, four had a selective loss of small myelinated fibers or unmyelinated fibers, which was an expected finding in SFN (Holland et al., 1998).

Our study of near-nerve needle sensory nerve conduction of the plantar nerves in 100 cases revealed that the majority of our patients had axonal neuropathy (Fig. 1)(Oh et al., 2001a,b,c). One-half of our patients and 77% of those with definite NCS abnormalities had axonal neuropathy. This observation is similar to the findings in cryptogenic sensory polyneuropathy and idiopathic small fiber neuropathy (Wolfe et al., 1999; Gorson and Ropper, 1995). It is also noteworthy that in 11 (11%) cases in Oh's study (2001), a demyelinating neuropathy pattern was observed. These cases included three cases of chronic sensory demyelinating neuropathy, one case of MGUS, one of sensory GBS, and one case of vitamin B 12 deficiency. In two of our chronic sensory demyelinating neuropathy cases, who also showed a high CSF protein, immuno-therapy was effective (Oh et al., 1992).

We analyzed the relationship between the clinical features and nerve conduction abnormalities in order to see whether there was any clinical clue to suggest LFN or SFN (Oh et al., 2001a,b,c). Our analysis showed that there was no clinical indicator including pinprick impairment for small-fiber neuropathy, but that impaired proprioception and absent or diminished reflexes were reliable indicators for LFN as defined by the abnormal nerve conduction. These findings are to be expected because proprioception is known to be mediated by large-diameter fibers and provide support for the strict diagnostic criteria for small-fiber neuropathy (Holland et al., 1998; Jamal et al., 1987).