Pyrex Journal of Educational Research and Reviews

Vol 2 (4) pp.34-36 August, 2016 Copyright © 2016 Pyrex Journals Author(s) retain the copyright of this article http://www.pyrexjournals.org/pjerr ISSN: 2985-8879

Review Paper

Tarsal Tunnel Syndrome in Iraqi Patients with Type 2 Diabetes Mellitus

Safaa Hussein Ali

Email: safaalshammary@yahoo.com

Al Mustansiriya College of Medicine, Baghdad, Iraq.

Accepted 18th August; 2016

Abstract

Background: Diabetic patients often present with distressing symptoms such as pain and burning dysthesia in the feet. The present study was designed to estimate the point prevalence of tarsal tunnel syndrome (TTS) in a population of subjects with diabetes and to identify the most valid electrodiagnostic test for discriminating TTS from diabetic peripheral polyneuropathy (DPN) in different stages of severity of DPN. Diagnosis of TTS in patients with DPN is important, as therapeutic interventions directed toward relief of TTS may be effective irrespective of diffuse neuropathy. Method: In this study a group of 35 normal volunteers who had no neurological complaints or foot trauma were selected, and 78 patients having longstanding diabetes mellitus with complaints of distressing pain. burning sensation and paresthesias in the feet were selected for electrophysiological tests where bilateral tibial distal motor latencies to both the AHB and ADQP, for the medial and lateral plantar nerves, respectively, stimulating the tibial nerve proximal to the tarsal tunnel at the medial malleolus. medial and lateral plantar sensory responses were studied. Results: In the present study we observed that 60 (77%) of diabetic cases showed abnormal findings, e.g. prolonged distal motor latency, decreased amplitude of M-response, low or absent sensory response suggesting TTS. Conclusion: This study shows that TTS may be present in a significant number of diabetic patients with subjective neuropathic symptoms in the feet. It is essential to investigate more localized reasons like TTS that may mimic diabetic neuropathy and it should be considered in diagnostic workup and the management of diabetes mellitus.

Keywords: (tarsal tunnel syndrome, diabetes mellitus, medial plantar nerve , nerve conduction study, entrapment neuropathy).

INTRODUCTION

Patients with a long standing history of diabetes mellitus often complain of distressing pain, numbness, paraesthesia and burning sensation of the feet and heel, sensation of tightness, cramping pain, and worsening of symptoms with prolonged standing or walking (Trepman and Kadel, 2000). These features could in fact, be due to compression of the posterior tibial nerve or its medial, lateral or calcaneal branches in the proximal and/or distal tarsal tunnel (Keck, 1962; Lam, 1962). Evaluation of suspected TTS is greatly simplified if one side is symptomatic and the other side is normal. This situation

allows for side-to-side comparison studies (Ibrahim, 2013).

The term was first coined by Keck and Lam in separate articles (Keck, 1962; Lam, 1962). However, the first description of the clinical feature of tarsal tunnel syndrome (TTS) is attributed to Von Malaise in 1918. To assess TTS electro physiologically the following tests are required: distal motor latency (DML), distal sensory latency (DSL), sensory conduction velocity (SCV) and a study of the amplitude of sensory nerve action potential (SNAP) of medial and lateral plantar nerves (Mondelli, 2004)

SUBJECTS AND METHODS

The study was conducted in Al Yarmouk Teaching Hospital's section of Neurophysiology, at the period from January 2014 to March 2016. An informed consent was obtained from all normal subjects and patients, and the Hospital Ethics Committee approved the study. The control group consists of 35 healthy volunteers (21 males and 14females). The age of this group ranged between 28 and 57 years with a mean age of (42.8±8.8) years. All subjects were healthy and symptom-free, with neither history nor clinical evidence of lumbosacral radiculopathy. Those with a previous history of lumbar laminectomy, foot trauma, or alcoholism were excluded.

A group of 78 patients with a history of diabetes of up to twenty years duration were selected. All the patients complained of pain, burning, numbness and paraesthesia of the heel or feet. All patients were diagnosed and referred by diabetologists and neurologists in the neuromedicine departments of Al Yarmouk Teaching Hospital.

Nerve conduction studies were performed using standard techniques of supramaximal percutaneous stimulation with a constant current stimulator and surface electrode recording, maintaining skin temperature at 32°

In both control and diabetes mellitus patients, the tibial nerve was stimulated 1 cm posterior to the medial malleolus and the recording electrode was placed on the abductor hallucis (AH) and abductor digiti quanti pedis (ADQP) muscles. The reference electrode was kept on the big toe while the ground was put on the dorsum of the foot. The distance between stimulating and recording electrode was kept at 10 cm. The measurements were made with a flexible tape while the ankle was in a neutral position (90°).

Medial plantar SNAPs were recorded orthodromically by placing a surface active electrode on the sole at a distance of 8 cm from the base of the big toe along the line from the mid-point of the big toe to the mid-point of the heel. The reference electrode was positioned 3 cm proximal to the active electrode. The surface electrodes were round silver discs, 1.0 cm in diameter. Ring electrodes were used on the big toe to stimulate the medial plantar nerve. A ground electrode was placed over the medial plantar surface between the stimulating and recording electrodes. The amplitude of the response was measured from peak to peak.

Latency was measured from the onset of the stimulus to the beginning of the major negative deflection for maximum nerve conduction velocity (NCV) and to the peak of the major negative deflection for negative-peak NCV. Response duration was measured from the beginning of the major negative deflection to the baseline return of the last component. Studies were regarded as abnormal when the NCVs were more than 2 SDs below normal mean values and when the durations of SNAPs were more than 2 SDs above normal mean values.

NIHONKOHDEN MEB – 9400 A/ K EMG/ EP measuring system was used for the recording of sensory, motor nerve conduction parameters, late responses and muscle activities.

RESULTS

A cross-sectional case control study was conducted in 35 normal control and 78 type 2 diabetic patients (22 males and 56 females). The age of the patients ranged from 40–72 years and the duration of diabetes mellitus was 12-20 years. Out of 78 (156 limbs) diabetic cases 18 cases (36 limbs) in spite of symptoms had normal nerve conduction studies [NCS] values. Sixty cases (120 limbs) had both symptoms and abnormal findings [NCS] in lower limbs and 13 patients out of 60 had bilateral complaints and abnormal NCS of medial or lateral plantar nerves.

The remaining 47 had unilateral symptoms and NCS abnormality. Out of 47 (94) limbs of abnormal cases the sensory and motor involvement were present in 29 (58%) limbs, and only sensory abnormal findings were observed in 15(30%) limbs. However, in 3 limbs no response was observed. Thus, our results showed that the distal motor latency and amplitude of M-response was abnormal in 32 out of 47 (68%) type 2 diabetic patients. In 3 out of 32 diabetic patients, no sensory nerve action potential was obtained bilaterally.

DISCUSSION

The type 2 diabetic patients had various types of neuropathic complication and TTS is one of them. In diabetic patients with exacerbation of symptoms with long standing and walking, TTS should be considered as one of the causes. A careful examination is thus warranted while evaluating such patients.

TTS is not difficult to diagnose clinically when DPN is not severe and NCV is moderately abnormal. Electrodiagnostic studies are required to confirm the diagnosis, localize the site of the lesion and detect its pathology, whether demyelinating or axonal degeneration and its severity. In the present study, 23% of the cases with a history of burning feet and a positive Tinel's sign showed no abnormality in the conduction study. However, the remaining 77% of the patients showed abnormality in NCS:

- i). Prolonged distal motor latency
- ii). Decreased amplitude of M-response
- iii). Prolonged distal sensory response
- iv). Absent or low amplitude SNAP.

Controversy exists as to the role tarsal tunnel release plays in the management of the diabetic patient with poor plantar foot sensibility due to the intrinsic swelling of peripheral nerves in diabetes (endoneural swelling secondary to increased sorbitol levels within the peripheral nerve). The reason for nerve swelling is related to the transport system with the diabetic nerve (Parker, 2007). The swelling causes compression that damages the cell membrane of the nerve leading to the impairment of rebuilding of protein, tubulin, which is transported inside the cell (Ashraf, 2009).

The results of the present study facilitate the diagnosis of tarsal tunnel syndrome, which may be helpful in the management of diabetes mellitus associated nerve entrapment. The double-crush syndrome as described by Upton and McComas (Ibrahim, 2013) may be applied to the diabetic patient. This hypothesis states that when multiple "subclinical" nerve compressions exist in a series they may be "additive" and give rise to symptoms, even though each compression, by itself, would not. The "first crush" would be the peripheral neuropathy of diabetes and the "second crush" compression of the tibial nerve at the tarsal tunnel. If patients are carefully selected, the release of the tibial nerve through the tarsal tunnel in the diabetic patient may improve plantar sensibility and help prevent plantar ulceration and a possible lower-extremity amputation.

The probable path physiological explanations given by some researchers for the observed changes are that there are mainly two mechanisms involved:

- a). The metabolic theory: when blood glucose is high, it enters the nerve and is converted to sorbitol, which attracts water, leading to nerve swelling, which causes damage to the nerve.
- b). The transport theory: the reason for nerve swelling is related to the transport system with the diabetic nerve (Parker, 2007).

The swelling causes a compression that damages the cell membrane of the nerve, leading it to the impairment

of rebuilding of the protein, tubulin, which is transported inside the cell (Ashraf, 2009).

The results of the present study facilitate the diagnosis of tarsal tunnel syndrome which may be helpful in the management of diabetes mellitus associated nerve entrapment. As this nerve is analogous to the median nerve in hand, it will not be out of place to select the medial plantar nerve for diagnosis of tarsal tunnel entrapment.

REFERENCES

- Antunes AC, Nobrega JA, Manzano GM. Nerve conduction study of the medial 134 and lateral plantar nerves. Electromyogr Clin Neur 2000;40:135–8. 135
- Ashraf Husain. Entrapment of medial plantar nerve [tarsal tunnel syndrome] in type 2 diabetes mellitus: An electrophysiological study. IJDM 2009, 17.
- Daniels TR, Lau JT, Hearn TC. The effect of foot position and load on tibial nerve 132tension. Ankle Int 1998;19(2):73–8. 133
- DeLisa JA, Saeed MA. The tarsal tunnel syndrome. Muscle Nerve 1983;6:664–670.
- Edwards WG, Lincoln CR, Bassett FH III, Goldner JL. The tarsal tunnel syndrome, Diagnosis and treatment. JAMA 1969;207:716—720.
- Ibrahim Khalil. Alexandria Journal of Medicine. 2013: 49, 95–104. Keck C. The tarsal tunnel syndrome. J Bon Joint Surg 1962;44:180–4. 124
- Lam SJS. A tarsal tunnel syndrome. Lancet 1962;2:1354-5. 125
- Mondelli M, Morana P, Padua L. An electrophysiological severity scale in tarsal 128 tunnel syndrome. Acta Neuro Scand 2004;109 (4):284. 129
- Parker RG. Diabetes and nerve compression. The Dellon Institute for Peripheral 136Nerve Surgery; 2007.
- Trepman E, Kadel NJ. Effect of foot and ankle position on tarsal tunnel 130 compartment pressure. Foot Ankle Int 2000;20(11):721. 131
- Von Malaise. Zur Pathologie der Plantar nerven. Dtsch Z Nervenheilk 126.1918;58:89–104. 127