Journal of Tissue Viability xxx (2016) 1–4

Contents lists available at ScienceDirect

Journal of Tissue Viability

journal homepage: www.elsevier.com/locate/jtv

Case report

Lacking the 'protective label' of diabetes: Phenytoin-induced distal symmetrical peripheral neuropathy. A clinical case report

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ARTICLE INFO

Article history:
Received 17 December 2015
Received in revised form
19 May 2016
Accepted 23 June 2016

Keywords:
Peripheral neuropathy
Phenytoin
Amputation
Podiatry
Charcot

ABSTRACT

This report documents an unusual case of distal symmetrical peripheral neuropathy (DSPN) in an otherwise healthy patient without diabetes mellitus (DM) presenting to a podiatric wound care clinic. The development of gas gangrene coupled with Charcot neuroarthropathic changes ultimately resulted in a potentially life-saving transmetatarsal (TMT) amputation. Causation of, or at least a contributor to, the DSPN was likely phenytoin usage for epileptic seizures. Long-term use of phenytoin can lead to axonal shrinkage and random clusters of nerve demyelination [1]. Clinical standards for DM-induced DSPN indicate that annual comprehensive neurological assessment to detect nerve function deterioration is warranted [2]. This can aid in identifying patients at high risk of diabetic foot ulceration. However, oftentimes, patients exhibiting medication-induced neuropathy are not assessed to determine severity of the neuropathy nor are they educated about ulcer prevention in the same manner as patients with DM. This report advocates for a standardized threshold of diagnostic and preventative investigation for neuropathy of all aetiologies; diabetic, traumatic, viral, medication-induced and idiopathic.

1. Introduction

Diabetes mellitus (DM) is rarely documented in a good light. However, patients with DM who attend a podiatry care centre are likely to undergo annual diagnostic testing for vascular and neurological pathologies, whereas those without DM are not. Therefore, in a clinical care setting, labels such as 'diabetic foot' or 'diabetic neuropathy' could be interpreted as positive in terms of the higher frequency of diagnostic assessment. There are standardized guidelines and care pathways in place for the clinician to implement in the care of the diabetic foot such as those by the National Institute for Health and Clinical Excellence [3] (NICE). However, the diabetic foot is not the only foot type at risk for neuropathy. Astute clinicians must be cognisant that patients with a history of spinal trauma, alcohol abuse or those who have been administered chemotherapeutic agents may exhibit lower limb neuropathy also. Although the use of the latter is commonly associated with nerve dysfunction accompanied by dysesthesia [4], the link between lower limb neuropathy and other widely used medications is less well known. Phenytoin, a commonly prescribed antiepileptic drug (AED), is one such medication. This is an unusual case of total DSPN due to long-term phenytoin use.

2. Patient description

The patient was male and in his mid-50's. He had a history of a single epileptic seizure (tonic-clonic) in 2009 for which he was taking long-term phenytoin (300 mg/day) with prophylactic folate supplementation. The patient was prescribed phenytoin initially and as of the time he presented into the centre of podiatric care, he had never been prescribed another AED. He had complete distal symmetrical peripheral neuropathy (DSPN) and documented peripheral vascular disease (PVD). In late 2014, the patient underwent amputation of his left 2nd and 3rd toes. Full closure of this amputation site was never documented. This patient had no history of DM, was a non-smoker and had no history of recreational drug use nor alcohol abuse. His weekly alcohol intake was approximately 2–5 units. He had no co-morbid disease aside from the PVD.

3. Case history

This patient had been referred to our clinic by his public health nurse (PHN) who had been redressing the non-healing digital amputation site. Compounding this case, the patient had stood on a nail prior to presentation in our clinic, creating a second plantar

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http://dx.doi.org/10.1016/j.jtv.2016.06.006
0965-206X/

ulceration directly under his left 3rd metatarsal head. The plantar skin of the left foot was chronically macerated with recurrent cellulitic episodes. Approximately 3 weeks prior to this presentation, the patient had finished a 7 day course of Amoxicillin (2 g/PO Q12h) as prescribed by his GP. The patient had been supplied with an offloading Aircast boot from our facility but repeatedly attended weekly clinics in regular loafers. Regarding the Aircast, the patient stated that he 'wore it all the time' at home. The patient had weekly podiatry appointments at our clinic with wound redressing carried out in the interim by his PHN. The diagnosis of PVD had been made some months prior at a vascular-specialist clinic through assessment of ABPI/TBPI. Total distal-symmetrical poly-neuropathy (DSPN) had been previously diagnosed at our centre of podiatric care by assessment with a 10 g monofilament, 28HZ tuning fork, 2-point discrimination test, sharp/blunt test and Tiptherm apparatus. Proprioception testing was also conducted. Both Achilles' reflexes were absent, even upon reinforcement with Jendrassik's maneover. Both patellar reflexes were present with no reinforcement required. This patient yielded the maximum neuropathy disability score (NDS) of 10/10.

4. Physical examination

The patient presented to our clinic for weekly review of his left foot ulcerations. On this day, the entire dorsum of the left foot was oedematous and cellulitic. The surface temperature was hotter than the contralateral limb. The patient stated that he 'could not fit his foot properly into his shoe' and reported that the foot had 'blown up' three days prior. The clinical presentation was similar to acute Charcotic reaction or osteomyelitis. Left dorsalis pedis (DP) and posterior tibialis (PT) pulses were palpable and monophasic with Doppler. The plantar wound dressings were damp with a green tinge and were malodorous with extensive bloody strike-through. Upon removal of dressings, the underlying skin was extremely macerated with significant callus build-up (Fig. 1).

After cleansing and debridement of any devitalized tissue, a probe-to-bone test of the plantar ulcer was positive, reaching a depth of over 3.5 cm with little to no soft tissue resistance (Fig. 2). Lateral compression of the left metatarsal arch resulted in a flow of sero-sanguinous exudate from the plantar wound.

Both wounds were dressed with Inadine and Medipad and off-loaded with 10 mm semi-compressed felt padding. The cellulitic border was traced and time-stamped (Fig. 3). The patient was then referred straight to the emergency department of the main city hospital with a letter highlighting clinical findings and the urgency of this acute lower limb presentation.

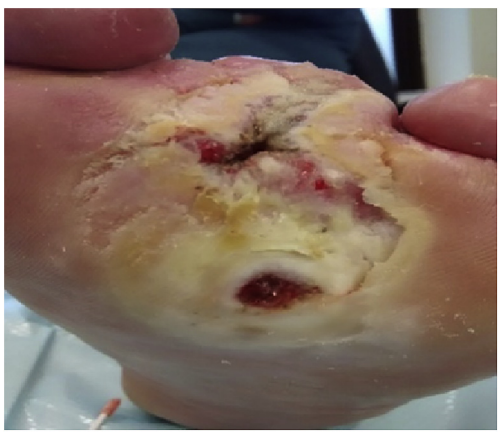

Fig. 1. Macerated callus formation surrounding the plantar wound.

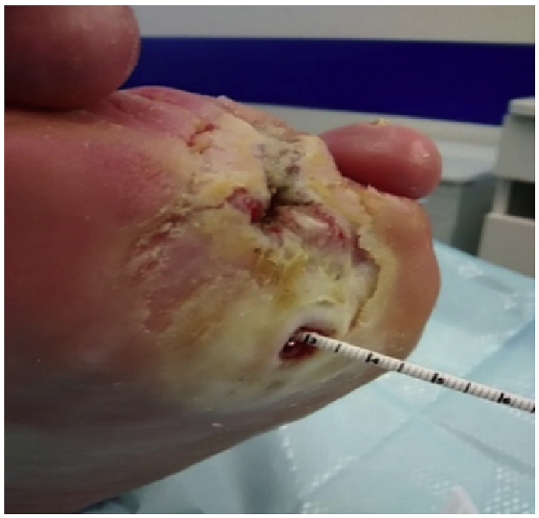

Fig. 2. Positive probe-to-bone test.

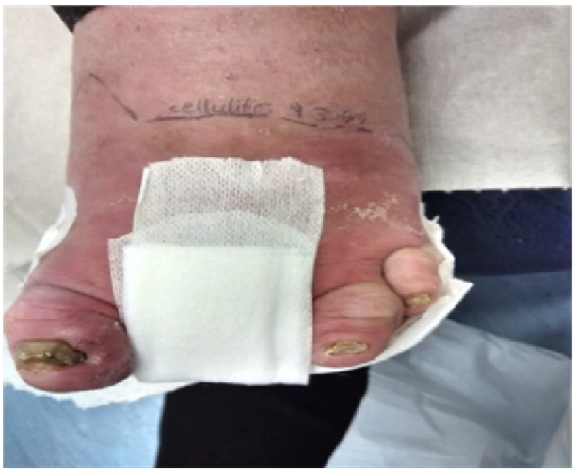

Fig. 3. Border of cellulitis.

5. Results of other investigations

During the podiatric assessment, a deep tissue swab was taken for culture analysis. Lab results confirmed the presence of infecting pathogens gram negative pseudomonas aeruginosa. Radiology investigation from the main hospital found an oblique fracture through the base of the 1st metatarsal shaft with medial deviation of the distal bone (an isolated Lisfranc's fracture; Fig. 4a). There were some bony fragments adjacent to the medial cuneiform suggestive of Charcot-like transformation (Fig. 4b). In the distal soft tissue there was a large collection of gas (Fig. 5). A standard fasting blood glucose analysis at the hospital (after acute sepsis had abated) confirmed a nil diabetes diagnosis in this patient at 6.6 mmol/L.

6. Treatment plan and outcome

Due to the severe infection that was clinically evident upon presentation into our podiatric care facility, it was expected that the patient would be admitted to hospital for intravenous antibiotics as a minimum intervention. The patient was admitted to the main city hospital immediately for urgent surgical intervention. Primarily a

Please cite this article in press as: Mahon AM, Lacking the 'protective label' of diabetes: Phenytoin-induced distal symmetrical peripheral neuropathy. A clinical case report, Journal of Tissue Viability (2016), http://dx.doi.org/10.1016/j.jtv.2016.06.006