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Acute Painful Peripheral Diabetic Neuropathy: A Life Limiting Morbidity and it's Treatment.

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ABSTRACT

Background: Diabetes is a pandemic disease and the painful neuropathy is one of the most debilitating complications. It is characterized by constant, sudden onset burning pain along with paresthesia and intermittent sharp, shooting pain, usually involving the extremities. It handicaps the daily activities of the patient, interferes with sleep and also affects the mood. Case: We report the case of a 30-year-old male, with Type 1 DM who presented with complaints of painful neuropathy in the lower extremities. **Conclusion:** Painful neuropathy is an increasing threat and is often not detected or misdiagnosed due to inconsistent definitions and lack of diagnostic criteria. The knowledge of this condition in very important not only to diagnose it but also to appropriately manage a case, from the widely available pharmacological options.

Keywords: Acute painful diabetic neuropathy, APDN, Diabetes mellitus.

INTRODUCTION

Peripheral neuropathy is one of the most common long-term complications in both Type 1 and Type 2 Diabetes Mellitus.[1] Neuropathic pain associated with peripheral neuropathy is seen in at least one of six diabetic patients.[2] The presentation can range from asymptomatic to debilitating. The symptoms can be either negative like loss of sensation and strength or positive like shooting and pricking pain. The most bothersome symptom being neuropathic pain and paresthesia.[3] The prevalence of painful diabetic neuropathy (PDN) is much more common in patients with clinically significant diabetic peripheral neuropathy (DPN).[1,4] However, once it is diagnosed it presents with its own distinct problem in management and should be considered a syndrome clinically different from Diabetic painful neuropathy.[5]

CASE REPORT

A 30-year-old man presented to outpatient department Saraswathi institute of medical sciences, with an ulcer on the medial side of dorsal surface of his right foot. It was approximately 3x3 cm in size [Figure 1]. The ulcer was debrided and covered with hydrocolloid dressings. We conducted a Random blood sugar test and an oral glucose tolerance test and the patient was diagnosed with a Type 1 Diabetes after positive for anti-GAD antibodies. At the time of presentation his Hemoglobin A1c (HbA1c) was 15g/dL, most likely due to poor compliance and no lifestyle modifications. He was started on a NPH (or lente) insulin as well as basal insulin and regular insulin at meal times. After 5 months he presented again to us with severe intermittent pain over the soles of both his foot. The ulcer had also not healed. He complained of the pain spreading and involving his lower limbs up to the level of the knees. On examination there was severe discomfort with touch sensation indicating hyperesthesia. There was no sensory or motor deficit but the Achilles tendon reflex was absent. Nerve conduction studies were unremarkable. Also, there were no signs or symptoms of diabetic retinopathy or nephropathy. However, the patient was having impotence along with early satiety due to gastroparesis for the past couple of months, indicating marked autonomic dysfunction. He was started on gabapentin 300 mg on day one and increased to a maximum of 1800 mg taken in 3 divided doses, however it was ineffective even after

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2 weeks. Later oxycodone (20 mg) and duloxetine (60 mg) were added which eventually resulted in pain relief. In addition, metoclopramide was started to treat the gastroparesis and Tadalafil for the treatment of the erectile dysfunction associated with Diabetes. Strict lifestyle modification and a meticulous sliding scale insulin regimen was advised which led to a downward trend in the HbA1c levels of the patient.

Table 1: Mechanisms of neuropathic pain[7]

Peripheral Mechanisms	Central Mechanisms
Alteration in sodium channels	Sensitization of central receptors
Alteration in calcium channels	Sprouting of Aβ into lamina II of the dorsal horn
Changes in neuro-peptide expression	Decreased inhibition by the descending pathways
Sensitization of peripheral receptors	
Changes in peripheral blood flow	
Atrophy, degeneration or regeneration of axons	
Destruction of small fibers	

Factor	Contradiction
Glaucoma, orthostatic hypotension, heart problems	TCAs
Hepatic disease	Duloxetine
Oedema and weight gain	Pregabalin, gabapentin
Problems with balance	TCAs

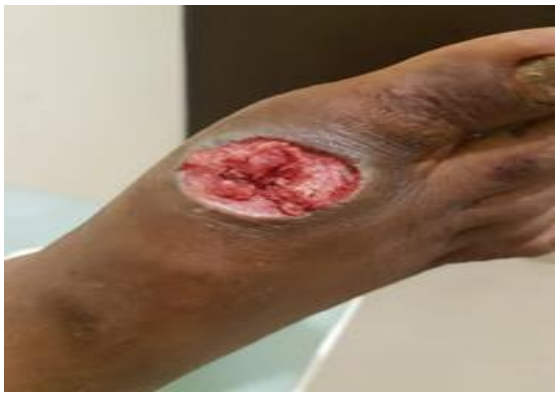

Figure 1: Patient's right foot showing the 3x3 cm ulcer with a pink base and no signs of gangrene.

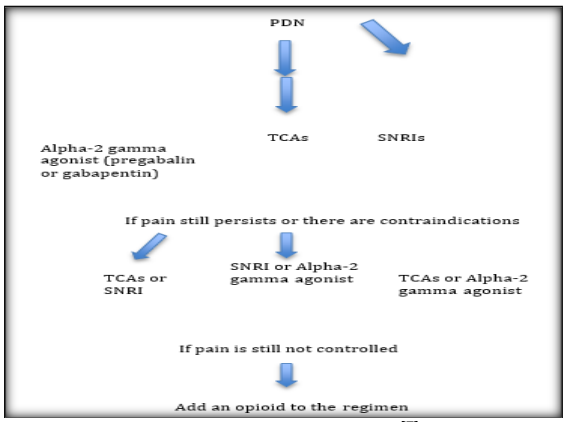

Figure 2: Treatment algorithm for PDN[7]

DISCUSSION

International Association for the Study of Pain defines neuropathic pain as 'pain arising as a direct consequence of abnormalities in the somatosensory system in people with diabetes.[6] Patients usually describe their pain as burning, electrical shock, shooting or lancinating pain (often like 'stabbing' or 'knife-like' pains) along with agonizing tingling and allodynia (triggering of pain from stimuli that would not normally cause pain).[7] They often express different subjective sensations of altered temperature perceptions and an achy and cramp like sensation in their feet and legs, almost like 'walking on marbles'.

Yet another form of symmetrical polyneuropathy is Acute painful neuropathy, which presents with fewer neurological signs. According to S. Tesfaye et al,[7] it is seen when there is any form of rapid change in the insulin levels in the body. It can either be a sudden improvement in glycemic control also known as 'insulin neuritis' or a drop-in control as is seen in young patients suffering from Type 1 DM, like after an episode of diabetic ketoacidosis.

According to a study conducted by Halawa MR et al,[8] there was a prevalence of 65% PDN in 1039 patients with Diabetes Mellitus. Of three large, clinic-based studies from Europe, the prevalence of diabetic polyneuropathy varied from 23 to 29%.[9,10]

There are a lot of causes that have been postulated as the pathophysiology behind PDN [Table 1].[7] Other causes also include altered foot skin microcirculation,[11] reduced intra-epidermal nerve fiber density and autonomic dysfunction.[12,13] The diagnosis of this condition is clinical, based on the patient's description of pain; usually distal and symmetrical getting exacerbated at night. Other tests like nerve conduction studies and quantitative sensory testing can also contribute and are helpful and also contribute to exclude other causes like entrapment syndromes. Even though pain is a very subjective feeling, different scales have been used to assess the severity of the symptoms. These include, the 11 point Likert scale (0=no pain and 10= worst pain), neuropathic pain symptom inventory,[14] the LANNS pain scale,[15] the McGill Pain Questionnaire etc.[16]

Since PDN also gravely affects the quality of life (QoL) of a patient, specific QoL scales such as NeuroQol,[17] Norfolk QoL Scale and Neuropathic Pain Impact on QoL questionnaire (NePIQoL) are also used to quantify the impact it has on the daily activities of the patient.[18,19] A lot of other scales are also used to study the impact PDN has on the mood, behavior and sleep patterns of the patient.

Our patient also presented with increased agitation and difficulty in sleeping, including drowsiness in the daytime. A lot of pharmacotherapy as well as lifestyle modification techniques are available for management of this condition [Table 2].[7] Strict control of HbA1C is a very important factor in the

long-term outcome of acute painful diabetic neuropathy. However, the change should not be very rapid as it itself can precipitate Diabetic peripheral neuropathy.

Tricyclic anti-depressants (TCAs) are usually used as treatment of DPN. Other therapies like Serotonin Norepinephrine reuptake inhibitors (SNRIs) are more helpful in treating painful diabetic neuropathy and are approved by the US Food and Drug Administration (FDA) and European Medicine Agency for this use. According to various trials, Duloxetine in the dose of 60-120 mg/day and Venlafaxine in 150-225 mg/day can greatly help to reduce pain symptomatology. The use of first-generation anti-convultants like carbamazepine is not very helpful however drugs like Gabapentin and Pregabalin in doses of 150-600 mg/day have been proven to be very efficacious in controlling neuropathic pain. A lot of physicians are hesitant to prescribe opioids because of fear of addiction. But tramadol, for example, has a lower abuse potential. Topical capsaicin (0.075%) applied 3-4 times/day can also help to relieve the neuropathic pain.

Peripheral Mechanisms Central Mechanisms

Alteration in sodium channels Sensitization of central receptors Alteration in calcium channels Sprouting of Aβ into lamina II of the dorsal horn

Changes in neuro-peptide expression Decreased inhibition by the descending pathways Sensitization of peripheral receptors Changes in peripheral blood flow Atrophy, degeneration or regeneration of axons Destruction of small fibers Non pharmacological therapies like acupuncture,[20] low intensity laser therapy etc.[21] are alternate therapies that can be tried.

CONCLUSION

Painful diabetic neuropathy is an increasing burden on society which needs to be studied in detail and tackled with the utmost precision so as to decrease the suffering of the patient. Our patient reported significant improvement in symptoms, as better sleep along with increased levels of concentration in the daytime. It is an uncommon form of diabetic neuropathy which can be life limiting. Knowledge of this condition is important for timely diagnosis and management.

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