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CASE REPORT

Bilateral Trophic Ulcer – A Case Chronic Inflammatory Demyelinating Polyneuropathy

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ABSTRACT

The difficult ulcer to manage is a tropic ulcer for a surgeon. But the compounding problem is when the known etiology of the neurosensory loss is not found. Here is a case who had sensory loss with absences' of diabetes mellitus, Hansen's etc. which was found to be case of chronic inflammatory demylenating polyneuropathy. And entity every surgeon must know.

KEY WORDS: Hansen's disease, Diabetes mellitus, inflammatory demylenating polyneuropathy.

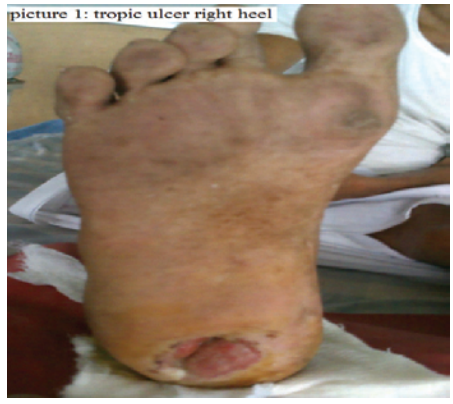
Case report

A 70yr old male presented with complains of multiple non healing ulcers for the past 4 yrs. Started as a blister bilaterally in both heel gradually progressed to an ulcer. Which was treated and the blister opened with discharge and resulted in an ulcer.

Patient has decreased sensation over bilateral foot and impaired hearing present. No other cranial nerve involvement, weakness present. No exposure to chemicals, toxins. Patient is not a diabetic or hypertensive. No history suggestive of Hansen's disease. Prostatic symptoms present. He does not consume alcohol and does not smoke, he consumes a vegetarian diet. No history of similar illness in the family.

On examination patient his higher mental functions normal, there was Pallor, no pedal edema.

Local examination Right thigh ulcer measuring 6X5 cm with punched out edges with pale granulation tissue, [figure 1]. Left heel Ulcer of size 3X4X2 cm with pale granulation tissue.


picture 1: tropic ulcer right heel

with the available history and examination findings diagnosis of peripheral neuropathy with bilateral trophic ulcer was made and evaluated for the same. There was paresthesia

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over right foot and ankle jerk diminished reflex present. No peripheral nerve thickening or hpyopigmented patch. No muscle wasting seen [1].

The following differential diagnosis was considered for the above symptoms 1. Diabetic peripheral neuropathy 2. Toxin induced 3.Sub acute degeneration of the cord 4.Amyloidosis vasculitis 5. Hereditary sensory motor neuropathies.

Base line investigations revealed Hb of 8.9 gms a microcytic hypochromic type. Serum B12 levels are normal. VDRL, HIV, HBsAg, was negative. Nerve conduction study was performed which revealed severe sensory motor poly radiculo neuropathy involving both upper limbs (demyelinating type) and both lower limb nerves were not able stimulate [2] and doppler of the lower limb arterial study was normal. PSA, ANA, Urine for Bence Jones Protein , RA Factor was negative.

CSF analysis was within normal limits except for increased protein (500miligm /dl), Sural nerve biopsy was done which is suggestive of small fiber neuropathy and negative for leprosy amyloidosis, vasculitis and for hereditary neuropathies [3-4]. A genetic work up is nessarry which is not available at present in our institute [5]. PTA revealed presence of bilateral moderate sensoineural deafness. Pus culture sensitivity was done. Revealed pseudomonas sensitivity to ciprofloxacin

A diagnosis of CIDP was made. Which needs a conservative management [6] with steroids and wound care with daily dressing. Appropriate antibiotics started and iron supplementation given.

Discussion

This patient was a case of CIDP – a chronic inflammatory demylenating polyneuropathy. It is a diagnosis of exclusion. And nerve biopsy is warranted to establish a diagnosis. So a patient with sensory loss with trophic ulcers must be carefully examined and found the cause of his neurological defect.

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