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Letter to Editor

Comment on "Sensory Ganglionopathy in Hansen's Disease: Report of a Patient and Review of Literature"

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Leprosy is a chronic infectious disease that predominantly affects the skin and nerves, mainly the small fibres and peripheral nerves. It is rarely known to affect the ganglions, which is usually a result of progression of the nerve damage or a reactive process in a classic case of Hansen's. Pure neuritic leprosy presents with thickened peripheral nerve trunk, tenderness (with or without), and sensory impairment associated with the absence of skin lesions and negative skin smears for acid fast bacilli. The same would be expected in pure ganglionic leprosy, if suspected.

We hereby wish to raise a few points regarding diagnosis and management of a patient of sensory ganglionopathy which was attributed to Hansen's disease described by Chaudhary et al in the case report: "Sensory Ganglionopathy in Hansen's Disease: Report of a Patient and Review of Literature".

Keywords : Sensory Ganglionopathy, Pure Neuritic Leprosy, Hansen's Disease

Sir,

We read with interest the article titled "Sensory Ganglionopathy in Hansen's Disease: Report of a Patient and Review of Literature", where the authors discussed a case of polyganglionopathy which was attributed to leprosy (Chaudhary et al 2022). As described in the case report, sensory ataxia, pseudo athetosis, areflexia, mild weakness of hands and unrecordable sensory nerve conduction in the patient were indicators of polyganglionopathy. In the author's own description, there were no cutaneous or peripheral neuropathy features that are classically seen in leprosy. There is no mention of the reason why leprosy was suspected in the first place and despite the progression of the disease to involve ganglions, it spared the skin and peripheral nerves. The diagnosis of leprosy was made based on the slit skin smear positivity. If pure ganglionic leprosy was suspected like the pure neuritic type, then it is unlikely for the smear to be positive (Rao & Suneetha 2016). The criteria for clinical diagnosis of pure neuritic leprosy requires thickened peripheral nerve trunk, tenderness (with or without), and sensory impairment associated with the absence of skin lesions and negative skin smears for acid fast bacilli.

Leprosy per se is known to cause mainly small fiber and temperature-sensitive neuropathy

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and does not involve ganglions (van Brakel et al 2005). It doesn't lead to generalized areflexia, loss of position and joint sense, or ataxia. In the few reports where these features were seen, they were preceded by characteristic features of leprosy and ganglionopathy was a reactive process (Bafna et al 2020, Khadilkar et al 2007, Rice et al 2016).

Oddly enough, the Magnetic Resonance (MR) neurography of the patient was found to be normal. MR imaging in sensory ganglionopathy generally shows swelling, increased signal on T2-weighted images, or enhancement in the dorsal root ganglia, as well as degeneration of the posterior columns in the spinal cord (Amato & Ropper 2020). These changes have been reported in all studies of Hansen's disease presenting with sensory polyneuropathy that are quoted in the case report as well (Bafna et al 2020, Khadilkar et al 2007, Rice et al 2016). Apart from MRI, the cerebrospinal fluid examination is usually recommended in patients with sensory ganglionopathy to elucidate the aetiology, which was not performed in this case.

The sensory ganglionopathy was attributed to leprosy, and there was no peripheral nerve involvement, thus, there should not have been any motor weakness such as reduced strength of small muscles of the hands (grip strength of 70%). The nerve conduction study (NCS) should also demonstrate pure sensory abnormality with preservation of latency and velocity (Amato & Ropper 2020) but the NCS of the described case showed abnormality in motor conduction as well. Ganglionopathy is usually uniform and not length dependent, it may be diffuse, patchy, sparing distal lower limbs, or lacking a distal-to-proximal gradient, but in this case, a symmetrical pattern progressing distally to proximally was present which is very unusual.

Glucocorticoids are commonly employed for the treatment of sensory polyneuropathy since it is commonly an autoimmune phenomenon. Thus, the improvement in the described patient might be attributed to the steroids rather than the multidrug therapy. In fact, the patient should have been initiated on higher dose of steroids (1 mg/kg/day) for faster and complete response.

No detail is provided on bacteriological/ morphological index on slit skin smear positivity found and what was its status after completion of one year of multidrug therapy.

So, to conclude, the attribution of the ganglionopathy in the described patient to Hansen's disease seems erroneous and requires further investigation and elaboration. There are many key features that are against pure sensory ganglionopathy without peripheral nerve involvement as described. It is highly unlikely for Hansen's disease to affect the ganglion, more so prior to the involvement of the skin or peripheral nerves. Bilaterally symmetrical pure neuritic leprosy has not been described. Moreover, demonstration of acid-fast bacilli on slit skin smear (not substantiated by histopathology of skin biopsy) goes against pure neuritic Hansen.

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