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Letter to the Editor

Response to The Combination Therapy of Oral Steroids and Nerve Decompression for Ulnar Neuritis in Leprosy - Usefulness in Prevention of Progression of Hand Deformities

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Dear Editor,

We read with great interest the article titled "The Combination Therapy of Oral Steroids and Nerve Decompression for Ulnar Neuritis in Leprosy - Usefulness in Prevention of Progression of Hand Deformities", where the authors have tried to emphasise the role of early introduction of surgical decompression for recovery of ulnar neuritis (Sajid et al 2022). According to the results, of the 247 eligible patients, 210 did not respond to 12 weeks of steroid therapy–193 such patients, treated with steroids and nerve decompression responded to combined treatment.

Although a surgical intervention by nerve decompression has been reported to be useful after 3 months of non-response to steroids in certain cases of neuritis and nerve function impairment (NFI) in prior studies, there are several lacunae to decipher the usefulness of this procedure in all the 193 said steroid-non-respondent patients. First and foremost, there is no clarity on what spectrum of leprosy patients were included in the study and whether these patients were in reaction and had a grade 1 or a grade 2 disability at the time of initial presentation. This is important as not all types of neuritis in leprosy require or are amenable to surgical decompression. The use of decompression surgery is rather controversial. Most clinicians believe that treatment with corticosteroids will decompress the nerve. Few conditions where decompressive surgery should be considered:

1. Nerve abscess.
2. No improvement or worsening in neuropathy treated adequately with or without reaction.
3. Control of severe pain.
4. Contraindications to the use of corticosteroids.

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5. Severe adverse reactions to corticosteroids.
6. Ulnar nerve dislocated or subluxated in the groove at the elbow.

The sizes of cellular infiltrate in tuberculoid and borderline tuberculoid (BT) leprosy are greater than in lepromatous leprosy (LL). The infiltrate and the accompanying oedema causes compression of the nerves at 'sites of predilection' causing severe nerve injury, equivalent to crush injury, resulting in Wallerian degeneration of the nerve. This explains the more severe nerve damage observed in BT leprosy as compared to LL. Nonetheless, even in LL extensive Wallerian degeneration and axonal degeneration can take place insidiously.

The development of ulnar neuritis can occur through different patho-mechanisms, and hence considering the "adequate therapy" should rather be based on the pathogenesis of nerve involvement than applying a blanket treatment for all patients. There are other medical management options that are in conventional use alongside or in steroid non-responders for neuritis, these include thalidomide, clofazimine, azathioprine and cyclosporine among others. In a study done to assess the efficacy of minocycline in steroid-nonresponsive neuritis patients of leprosy, all 11 patients demonstrated stabilization of the nerve functions: with 9 (81.82%) showing either complete restoration or improvement in NFI. All patients with active neuritis also showed improvement in neuropathic pain and nerve tenderness (Narang et al 2017). While the authors have mentioned that there are no clearcut guidelines on deciding when to intervene with surgical management, it is essential to have a thorough awareness of the pathogenesis of neuritis, spectrum of leprosy, bacillary load, whether the neuropathy is reversible or not and the deformity that is to be dealt with during a surgical intervention.

All the cases were started with 40 mg prednisolone/day and the dose of prednisolone was tapered down 5 mg every 8th day and in 8 weeks it reached 5 mg/day which is an arbitrary dosage schedule that follows neither the WHO nor the NLEP guidelines. Evidence says that a longer course of steroids is better than a short course, especially in borderline lepromatous patients, and studies showed that there was a lower relapse rate after a 20 week, rather than a 12-week course of steroids (Rao et al 2006). The authors have adopted a similar standard of surgical approach for all 193 patients of ulnar neuritis in an attempt to stop or prevent the progression of hand deformities without a mention of what grade of deformity or what degree and type of neuropathy (demyelinating/ axonal/ combined) the patients had at the time of presentation. The inclusion criteria of the study are subjective complaints of numbness, paraesthesia in little finger and ring finger with or without compromise of hand function and an exclusive involvement of ulnar nerve. It is difficult to explain the isolated involvement of the ulnar nerve in 247 patients without having any objective tests like nerve conduction study with sympathetic skin response (NCS-SSR) performed for the same as leprosy usually targets multiple nerves especially in the multibacillary spectrum of disease. The idea of surgical intervention in all patients without an understanding of localisation of the symptoms, site or length of involvement using a NCS or ultrasonography seems a bit exaggerated. The improvement of sensory complaints in the patients both touch and pain is difficult to explain given the consistent results of previous studies showing an improvement of pain but hardly any improvement in the sensory losses (Rao & Siddalinga Swamy 1989). Eighteen of the 193 patients had deterioration of motor function post-decompression the reason for which has not been elucidated. The adverse effects of decompressive surgery have not been adequately described.

Timing of neuritis also holds importance as acutely inflamed nerves should be put to strict rest as any trauma would further aggravate the inflammation. In addition, rest also reduces reactive fibrosis. Complications of decompressive surgery are painful scars, wound problems, hematoma, infection, and damage to nerves, arteries or tendons; hence it should be considered an exception rather than a rule. Evidence from RCTs for the effectiveness of decompressive surgery is scarce. A Cochrane review of studies of decompressive surgery in leprosy showed that there was very low-quality evidence from two RCTs insufficient to draw robust conclusions about the effect of decompressive surgery for treating nerve damage in leprosy (van Veen et al 2009). Two trials examining the added benefit of surgery over steroids for neuritis of less than six months duration did not show significantly better outcomes with steroids plus surgery than steroids alone in the long term (Pannikar et al 1984, Ebenzer et al 1996). Some studies have reported beneficial results from decompressive surgery most common being pain relief (Husain & Mishra 2008). Nerve function impairment recovery is dependent on the duration and severity of the neuropathy before diagnosis. It is necessary to determine the characteristics of patient populations or nerve types most likely to respond favourably to decompressive surgery.

Future RCTs should be carefully planned to determine the value and efficacy of decompressive surgery and medical treatment in combination compared to medical treatment alone. Non-clinical factors like costs and the effect on quality of life should be given more consideration in new studies because they are crucial indicators for both participants and policymakers.

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