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*Editorial*

# Are we missing neurosyphilis?

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**Quick Response Code:**

The current issue of the Journal carries a study by Nair on the prevalence of neurosyphilis (NS) among those with latent syphilis (LS). This paper is quite timely, meticulously done, and informative.[1] It analyses retrospective data from a single center in Kerala, India. It looks at the proportion of patients with NS among patients who were clinically classified as LS. The study shows that 4% of patients with clinical LS had features of NS in cerebrospinal fluid. Although authors mention it as "only eight persons (4%)," it has to be taken as quite significant, considering the population at risk, the long asymptomatic period of the disease, and the poor health-seeking behavior of at-risk individuals.

After an initial reduction in the incidence of syphilis following the syndromic treatment approach, the recent trend is suggesting an upsurge. This indirectly means we will have more late syphilis in the future.

Central nervous system (CNS) invasion by Treponema pallidum is known to occur early in syphilis. Lying dormant for years, the disease could get activated when the person's immunity suffers temporary setbacks. The disease process involves endarteritis obliterans, in addition to the body's immune mechanism launching a counterattack. This results in diffuse permanent damage. Depending on the site of involvement, the clinical presentation varies. Frontal lobe involvement causes personality changes such as delusions of grandeur during the initial inflammatory process and later results in dementia when the damage is complete; the parietal lobe involvement manifests as Jacksonian seizures initially that get replaced after paralysis that's usually bilateral and haphazard. These observations of paralysis occurring in an insane person led to the time-honored term general paralysis in the insane.

Similar pathology involves the spinal cord, almost always the posterior nerve root ganglion. Inflammation of these first-order sensory neurons causes lightning pain and visceral crisis. Still, as the damage becomes permanent, they stop conveying all senses routed through them. The posterior column of the spinal cord undergoes wasting as these neurons originate from the posterior nerve root ganglion outside the spinal cord. These neurons act as 2$^{nd}$ order neurons, relaying messages received from the outside ganglion. Thus, there is no wasting despite the absence of function. All these changes progress slowly; unless the patient/relatives are intelligent enough, they can overlook the changes until it's too late.

Hence, ignoring or not identifying early CNS involvement and undertreating syphilis with doxycycline or benzathine penicillin, which will not reach cerebrospinal fluid (CSF) in adequate concentration, can lead to NS after many years, at least in a few.

This emphasizes the importance of screening for NS, even among those presenting with early manifestations. While this is impossible in all clinical scenarios, at least those with LS should be routinely screened for NS.

The most common age group in this study is 21–30, suggesting that asymptomatic NS in this group can lead to symptomatic NS after many years, which can be missed as a more common neurological morbidity of older people.

The recent guidelines by the Centre for Disease Control Atlanta suggest CSF Study only for symptomatic patients, probably considering the difficulty of performing and interpreting CSF studies.[2] Nair opines that CSF study should be done only in "centers where it is routinely done." In contrast, we believe that there is sufficient evidence from this study to consider recommending CSF analysis in all cases of asymptomatic NS. To that extent, the study by Nair is an eye-opener. Overall, this study is very relevant, and we consider that more studies in this area are necessary.

## REFERENCES

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