Neurosyphilis, A True Chameleon of Neurology

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Abstract

Background: Neurosyphilis (NS) is a rarely encountered scenario today. Manifestations are heterogeneous, and their characteristics have changed in the antibiotic era. A differential diagnosis of NS is not commonly thought of even with relevant clinical-radiological features, as it mimics many common neurological syndromes. Objectives: To study the manifestations of NS in the present era and the process of diagnosis. Method: The data of ten patients with NS was collected and analyzed. Their background data, clinical features, investigations, the process of reaching the diagnosis, management and outcomes were recorded. Observations and Results: The manifestations of NS in our cohort included six patients with cognitive decline/encephalopathy and one each with meningitis with cranial nerve palsies, cerebellar ataxia, myelitis and asymptomatic NS. The presence of Argyll Robertson pupil helped to clinical diagnosis in one patient. Treponemal tests were ordered in two patients only after alternative etiologies were looked at, to begin with, whereas in six patients treponemal test was requested as a part of standard workup for dementia/ataxia. Conclusions: NS dementia and behavior changes are mistaken for degenerative, vascular, nutritional causes, autoimmune encephalitis or prion disease. Meningitis has similarities with infective (tubercular), granulomatous (sarcoidosis, Wegener’s), collagen vascular disease and neoplastic meningitis, and myelitis simulates demyelination or nutritional myelopathy (B12 deficiency). Rarely, NS can also present with cerebellar ataxia. Contemplate NS as one of the rare causes for such syndromes, and its early treatment produces good outcomes.

Keywords: Asymptomatic neurosyphilis, cerebellar ataxia, dementia, meningitis, myelitis

INTRODUCTION

Neurosyphilis (NS) has become rare, but we still encounter patients periodically. Since the last 20 years, there has been a gradual rise in NS.1,2 Traditionally described presentations are meningitis, meningo-vascular syphilis, tabes dorsalis and dementia. The organs involved and clinical manifestations in patients presenting in the present era are similar to the ones described in the past, but not as classical and not so severe.1,3,4 Late NS has decreased and early neurological involvement is being increasingly reported.3,5 Our degree of suspicion for NS is low, and other alternative causes of such presentations which are common are initially thought of in the workup.1,3,4,5 We present our experience to describe features of NS in the present era, the alternative differential diagnoses we thought of, and the process as to how the diagnosis was reached.

MATERIAL AND METHODS

The retrospective data of patients diagnosed with NS over a period of the past 3 years were collected and analyzed. Appropriate permission was obtained from the institutional ethics committee. All of the patients who were included in this study had a positive serum treponema pallidum hemagglutination test (TPHA). All of the patients underwent cerebrospinal fluid (CSF) examination. The diagnosis of NS was established on the basis of a positive cerebrospinal fluid (CSF) venereal disease research laboratory test (VDRL)/TPHA and/or CSF lymphocytic pleocytosis with pertinent clinical features. The demographics and clinical manifestations at the onset and after treatment were collected. The results of all relevant tests performed which included neuroimaging, CSF studies, and all tests performed during the process of diagnosis and management were recorded. All treatment they received and their outcomes after therapy were noted. All our patients were tested negative for HIV (ELISA).

OBSERVATIONS AND RESULTS

Ten patients were found to have NS of which nine had neurological symptoms and signs, whereas one had asymptomatic NS. The clinical presentations we encountered in our cohort were divided into five neurological syndromes: Cognitive decline/
encephalopathy, meningitis, cerebellar ataxia, myelopathy and asymptomatic NS. The type of syndrome and the number of patients in each category and sub-category is outlined in Table 1. Each neurological syndrome with its patient details and course, investigations, management and outcomes of all patients are summarized in Tables 2 and 3. The case summaries of all the patients are described in detail hereafter.

**Case 1 to 6: Cognitive decline/encephalopathy**

We had six patients presenting with cognitive decline/encephalopathy with/without behavior change. A 65-year-old male presented with progressive behavioral change followed by cognitive impairment for the past 2 years. The family complained that he had started becoming aggressive and abusive, which was followed by cognitive impairment. There was no history of seizures, headache, focal symptoms or fever. The examination revealed no signs of meningism. The neurological assessment revealed markedly agitated and restless behavior. His poor attention made a detailed cognitive assessment difficult. There was no language or visuo-spatial dysfunction. There were no signs of parkinsonism or gait apraxia. The magnetic resonance imaging (MRI) brain

| Table 1: Types of neurological syndromes, number of patients in each category and sub-category |
|-----------------------------------------------|-----------------|-----------------|
| Type of neurological syndrome | Number in sub-category | Number in each category |
| Cognitive decline/encephalopathy | 6 | |
| Gradual onset cognitive decline with behavioral changes | 2 | |
| Gradual onset cognitive decline with seizures and ischemic strokes | 1 | |
| Gradual onset cognitive decline with parkinsonism | 1 | |
| Rapid onset dementia | 1 | |
| Subacute encephalopathy | 1 | |
| Meningitis with hearing impairment | 1 | |
| Cerebellar ataxia | 1 | |
| Myelopathy | 1 | |
| Asymptomatic neurosyphilis (Condyloma lata) | 1 | |
| Total | 10 | |

| Table 2: Demographics, clinical features of all patients and the type of neurological syndrome (as per categories described in Table 1) |
|-----------------------------------------------|-----------------|-----------------|-----------------|-----------------|
| Case no. | Age | Sex | Symptoms | Duration | Neurological examination | Type of Neurological syndrome |
|-----------------------------------------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| 1 | 65 | M | Behavior changes, cognitive decline | 2 years | Poor attention, disoriented to time and place, impaired recent memory | Gradual onset cognitive decline with behavior change |
| 2 | 62 | M | Behavior changes- grandiose, paranoid, memory loss | 4 months | Impaired recent memory and executive functions | Gradual onset cognitive decline with behavior change |
| 3 | 53 | M | Rapid onset cognitive decline, irritability | 3 weeks | Poor attention, impaired 3 stage command and recent memory | Rapid onset dementia |
| 4 | 45 | M | Cognitive decline, behavior changes, memory loss, walking difficulty, seizures | 6 months | Impaired 3 stage command and recent memory, poor motivation | Gradual onset cognitive decline with seizures and ischemic strokes |
| 5 | 42 | F | Confusion, behavior changes, mild fever | 2 weeks | Disoriented to time and place, impaired recent memory | Subacute encephalopathy |
| 6 | 42 | M | Cognitive decline, slowness of activities | 1 year | ARP, executive, and visuospatial dysfunction, rigidity, bradykinesia | Gradual onset cognitive decline with parkinsonism |
| 7 | 55 | M | HA, decreased hearing, diplopia | 4 months | Neck stiffness, bilateral LR palsies, SN deafness | Meningitis with hearing impairment |
| 8 | 67 | M | Progressive imbalance | 1 year | Gait and limb ataxia | Cerebellar ataxia |
| 9 | 40 | M | Paraparesis, urinary hesitancy | 2 weeks | Paraparesis- power 4 LLs, hyperreflexia, upgoing plantars, spastic gait | Myelopathy |
| 10 | 41 | F | No neurological symptoms, h/o condyloma lata | NA | Normal | Asymptomatic neurosyphilis |

M: male, F: female, h/o: a history of, HA: headache, ARP: Argyll Robertson pupil, LR: lateral rectus, SN: sensorineural, LLs: lower limbs, NA: Not applicable

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showed generalized cerebral atrophy. His serum B₁₂ levels and TFT (thyroid function test) were normal and CSF examination revealed 19 lymphocytes with normal proteins and sugar. Another 62-year-old male attended our clinic with a 4-month story of memory impairment and behavioral change. On questioning, he admitted to having noticed mild generalized headache. Behavioral change was in the form of grandiose ideas and paranoia. Cognitive testing revealed impaired recent memory and executive functions with no focal/long tract signs. The computed tomography (CT) brain, serum B12 levels, and TFT were normal and CSF examination showed 10 lymphocytes and 70 mg% proteins. The third patient was a 53-year-old alcoholic male who presented with rapid onset cognitive decline and irritability over 3 weeks. There was no history of myoclonus, fever, headache or vomiting. The examination revealed impaired comprehension, three words recall and poor motivation. There was no focal weakness and his planters were extensor. The MRI brain showed infarcts in the right temporal and parietal lobes and bilateral posterior-medial thalami [Figure 1a]. The CSF analysis showed proteins at 28 mg%, sugar at 92 mg% (parallel blood sugar 132 mg%), and 30 cells (100% lymphocytes). The CSF polymerase chain reaction (PCR) for mycobacterium tuberculosis and herpes simplex virus was negative. CSF IgG Index was elevated at 8.7 (reference 0.28-0.66) indicating intrathecal IgG production. The CSF neurotropic virus panel which tests antibodies against Varicella zoster, Cytomegalovirus, Epstein Barr, dengue, Japanese encephalitis, West Nile and Chandipura virus was negative. The serum scrub typhus IgM was negative. In these four patients, a serum/CSF VDRL was sent as a part of standard dementia workup and it turned out to be positive which triggered our sending off of tests such as serum TPHA and/or CSF VDRL. 

No.: Patient number, M: male, F: female, MRI: magnetic resonance imaging, C2-3: cervical second and third vertebral level, T2-11: thoracic second to eleventh vertebral level TPHA: treponema pallidum hemagglutination test, +ve: positive, CSF: cerebrospinal fluid, P: proteins, S: sugar, VDRL: venereal disease research laboratory test, Rx: treatment, CF: intravenous ceftriaxone 2 g/day for 14 days, CP: intravenous crystalline penicillin 24 million units/day for 14 days, ND: not done, NA: not applicable.
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no meningeal signs; her fundi were normal and there were no lateralizing signs. This acute febrile encephalopathy was investigated to find raised white cell counts at 15,400/cu mm with mildly reduced platelet counts at 1.28 lakhs/cu mm. Her dengue and rickettsial serology and hepatitis B and C serology were negative. The MRI brain was normal. Like the previous patients, a CSF VDRL was sent as a part of the standard workup to look for uncommon causes of encephalopathy, and as it was positive at 1:8 titers, it prompted us to send her serum TPHA as well as VDRL.

The sixth case was a 42-year-old teacher, who presented with a history of cognitive decline and slowness in activities for the past 1 year. He gradually became dependent for daily activities. There was no history of ulcer, fever, rash or Koch’s in the past. On neurological examination, the most important sign was Argyll Robertson pupils (ARP) [Figure 2]. Higher mental function assessment revealed executive dysfunction with visuospatial disorientation. He had signs of symmetrical parkinsonism with predominant gait involvement. MRI brain showed mild cerebral atrophy. The ARP was a big giveaway to expedite treponemal work up.

Further workup of these patients with CSF examination showed raised proteins in three of them at 61, 70 and 76 mg%, low sugar in two patients at 36 and 42 mg% and CSF lymphocytosis in five, with cell counts in the range of 10 to 60 cells/cu mm. One patient who had a normal CSF cell count had a positive VDRL in the CSF. Three patients received intravenous (IV) ceftriaxone 2 g/day and three received crystalline penicillin (CP) 24 million units/day for 14 days. The sixth patient received a repeat course of CP after 3 months. All of these patients, except for the first one made a good enough recovery to be able to perform all their daily activities. The third and the fifth patient, who had presented with rapid onset dementia and subacute encephalopathy respectively, improved over 3–4 weeks, whereas three out of the six patients who presented with gradual onset cognitive decline +/- behavior change, improved gradually over 3–6 months. The first patient has started improving but is still dependent.

Case 7: Meningitis with hearing impairment

A 55-year-old male presented with a 4 months history of generalized headache, reduced hearing in both the ears and double vision. On examination, he was fully conscious, oriented with no signs of meningism. He had bilateral lateral rectus palsies and sensorineural hearing loss in both the ears. The rest of the examination of the cranial nerves, motor, sensory system and co-ordination was normal. There were no systemic features. His hemogram and renal and liver function were normal. The MRI brain with contrast showed pachy-meningeal enhancement [Figure 1b]. The CSF examination revealed proteins at 85 mg%, sugar at 50 mg% (with simultaneous venous sugar at 124 mg%) and 24 cells/cu mm (90% lymphocytes and 10% polymorphs). The CSF Gram stain, Ziehl Nielson stain, adenosine deaminase levels, tuberculosis (TB) PCR, gene expert for TB and cytology for malignant cells were negative. A further workup for meningitis revealed normal antinuclear antibody (ANA) blot, C and P antineutrophil cytoplasmic antibodies (ANCA), ACE and IgG4 levels. At this stage, we sent a serum TPHA which was strongly positive. A repeat CSF with an intention to perform CSF VDRL was advised to the patient, but he was not willing for the same. He received intravenous ceftriaxone.

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Figure 1: (a) Diffusion-weighted imaging (DWI) and fluid-attenuated inversion recovery (FLAIR) axial MRI brain sequences of a patient who presented with gradual onset cognitive decline with seizures. Images show infarcts in the right temporal and parietal lobes and bilateral postero-medial thalami. Bilateral periventricular and right frontal lobe FLAIR hyperintensities are also seen (b) The MRI brain axial sequences including T1-weighted (T1W), T1W contrast, T2-weighted (T2W), and FLAIR of another patient who had meningitis with hearing impairment. The MRI shows pachy-meningeal enhancement in bilateral anterior frontal regions.
2 g daily for 2 weeks with complete recovery of headache and lateral recti palsy and a significant improvement in hearing in a period of 6 weeks.

**Case 8: Cerebellar ataxia**
A 67-year-old male presented with progressive imbalance while walking for the last 1 year, which was more prominent while walking through narrow passages, without any side predilection. He had no history of weakness or sensory loss. On neurological examination, his higher mental function was normal. His tone, power and reflexes were normal. There were significant gait ataxia and mild incoordination in the upper and lower limbs. The biochemical tests, TFT and vitamin B12 were normal. A syphilis workup was sent when the cause could not be determined in the initial tests. The serum VDRL test as well as TPHA were highly positive in serum as well as in CSF (titer 1:640). The CSF showed lymphocytic pleocytosis (12 cells/cu mm) and protein was elevated at 59 mg/dL. The MRI brain revealed cortical and cerebellar atrophy. IV CP was initiated at 24 million units per day for 14 days. Thereafter, intramuscular benzathine penicillin at 2.4 million units, once per week was injected for 3 weeks. At 6 months, the patient showed a significant recovery in gait and limb ataxia. The patient has been able to perform all activities of daily living with mild residual gait ataxia.

**Case 9: Myelopathy**
A 40-year-old male complained of bilateral lower limbs tingling, difficulty in walking for 2 weeks and urinary hesitancy 3 days before admission. There were no upper limb symptoms. The general examination was normal. The neurological examination revealed normal cognition and cranial nerves. The power was normal in the upper limbs and 4/5 in the lower limbs, deep tendon reflexes were brisk and planters were bilaterally extensor. The sensory system was normal and gait was spastic. The MRI spine revealed a hyperintense signal in the cervical spine at the C2–3 level and another much longer one in the thoracic spine, mainly in the posterior portion of the cord from the T2 to T11 level, with contrast enhancement, giving a candle-gutter appearance. There was not much cord swelling seen [Figure 3]. The MRI brain was normal. The hemogram, biochemistry, serum B12, serum copper level and serum anti-aquaporin-4 IgG and HIV were normal. The CSF proteins and sugar were normal with seven lymphocytes/cu mm, 72.5 mg% proteins and a normal CSF sugar level. There were significant gait ataxia and mild incoordination in the upper and lower limbs. The biochemical tests, TFT and vitamin B12 were normal. A syphilis workup was sent when the cause could not be determined in the initial tests. The serum VDRL test as well as TPHA were highly positive in serum as well as in CSF (titer 1:640). The CSF showed lymphocytic pleocytosis (12 cells/cu mm) and protein was elevated at 59 mg/dL. The MRI brain revealed cortical and cerebellar atrophy. IV CP was initiated at 24 million units per day for 14 days. Thereafter, intramuscular benzathine penicillin at 2.4 million units, once per week was injected for 3 weeks. At 6 months, the patient showed a significant recovery in gait and limb ataxia. The patient has been able to perform all activities of daily living with mild residual gait ataxia.

**Discussions**
The portions of neuraxis that get involved by NS today have remained the same as traditionally described, but the symptoms are varied and are different from the traditional descriptions.[2-5]

As far as the first case scenario is concerned, our patients presented with cognitive decline, irritability, personality and behavioral change. One of them also had features of parkinsonism and another suffered seizures and showed infarcts on his brain MRI. Classical syphilitic dementia [general paresis of insane (GPI)] in the mid- twentieth-century literature is described as personality change, disinhibition, emotional lability, paranoia, illusions, delusions of grandeur, hallucinations and inappropriate behavior, followed by cognitive deficits involving multiple lobes which progress over many years.[1,6] Recent descriptions of NS with cognitive impairment are mild or atypical and masquerade
dementias with cortical deficits, dementia with extrapyramidal features, encephalitis and subacute delirium.\[7,8\] There are recent descriptions of just dementia and dementia with parkinsonism.\[7,9\] Seizures were seen in only one of our patients, though they are reported in one-fourth of patients along with features of cognitive decline and other signs of meningitis in a series from South India.\[10\] Seizure is a well-established feature of NS and infarcts occur as a part of meningo-vascular syphilis.\[1,2,7\] Whenever a patient presents with cognitive impairment with/without parkinsonian features, seizures and ischemic strokes, we think of etiologies that we frequently confront, such as degenerative dementia (dementia with Lewy body disease- if associated with parkinsonism, Alzheimer’s- if recent memory, visuospatial and language dysfunction are the main deficits, frontotemporal dementia- if behavioral change or language dysfunction predominate), autoimmune encephalitis, prion diseases, vascular dementia (if acute/subacute worsening with gait disturbance), nutritional, metabolic or toxin exposure. NS mimics many such diseases and has to be thought of as a differential of these syndromes.\[4\]

One case had a gradual onset of cerebellar ataxia with gait and limb affection. Such a presentation with cerebellar ataxia is rare but certain case reports have been published in the literature.\[9,11\]

One patient had meningitis with cranial nerve palsies and the differentials thought of initially were TB, vasculitis, sarcoid and malignancy, after which we sent off the TPHA test which finally yielded the diagnosis. Cranial nerve palsies form a part of the clinical manifestations of syphilitic meningitis.\[1,12\]

Even in the myelitis patient, serum B$_2$ and copper levels, AQ-4 IgG, autoimmune profile and CSF cytology were performed to begin with, thinking of one of these etiologies. As these tests turned out negative, serum and CSF VDRL were ordered, clinching the diagnosis. Spinal cord involvement in NS is well known. The classical tabes dorsalis has become rare and other manifestations described in the literature are syphilitic meningomyelitis, syphilitic spinal pachymeningitis, spinal vascular syphilis, syphilitic polyneuropathy and spinal cord compression due to vertebral gumma or aortic aneurysm.\[1,7,13\] Syphilis needs to be kept in mind as an etiology of acute, subacute as well as chronic myelopathy.\[1,8\]

The last case scenario was asymptomatic neurosyphilis by virtue of no symptoms/signs and reactive CSF VDRL. This scenario is much more common than symptomatic NS. Although one-third of the syphilis patients show CSF abnormalities, only a fraction of these present with clinically significant manifestations.\[1,2\]

Clinical presentations of NS have changed in the antibiotic era and the classical presentations seen in the past such as tabes dorsalis, GPI and syphilitic gumma have become rare.\[4,14\] We are confronting atypical presentations or milder versions of classical manifestations.\[4,7,15\] Meningovascular syphilis, which was less common in the preantibiotic and pre-HIV era, has become more common than parenchymatous syphilis. This shift has been thought to be due to prior exposure of syphilis patients to antibiotics for other concomitant infections, thus, precluding patients from progressing to more severe forms, i.e., parenchymatous syphilis.\[16\] A rising trend of syphilis has been observed in the recent years in India as well as in the West.\[17,18\] MRI findings depend on the mode of presentation, i.e., meningeal enhancement in meningitic form, vasculitic infarcts in the meningo-vascular form, cerebro atrophy in gradually progressive dementia, cerebellar atrophy in cerebellar ataxia and spinal cord signal intensity in myelitis form, as seen in our series. Syphilitic gumma has become rare, and we did not encounter any in our study. They are commonly seen as dural-based lesions that can mimic meningiomas.\[18\]

Not only can NS mimic other common neurological diseases, but it can also rarely coexist with them. The simultaneous presence of NS and tubercular meningitis (TBM) has been reported.\[19\] It is extremely important to suspect such an association with TBM in our Indian setting. So, to speak, Hickam’s dictum and Occam’s razor can both occur with NS.

Cognitive decline, behavioral and personality change, encephalopathy, meningitis, cerebellar ataxia and myelitis have many common differentials in today’s clinical practice. One should suspect NS as one of the possibilities during the workup of these clinical scenarios. Testing for syphilis needs to be performed routinely in all typical as well as atypical case scenarios as no clinical findings and imaging features are specific. More time lost amounts to more tissue destruction in this preventable and treatable disease. Another important point to remember is that asymptomatic NS is the most common form, and it can occur at all stages, so screening at the primary care level may help diagnose NS cases even before tissue destruction has begun. We also recommend not to rely only on serum VDRL and include other specific treponemal tests as well as dark ground microscopy whenever a high degree of suspicion arises.\[20\] In all our cases, HIV was nonreactive, though the history of homosexuality and HIV must be ruled out in every case as false positivity as well as concomitant illnesses can occur with NS.\[21\]

Our first patient with gradual onset cognitive decline with behavioral change who was presented to us 2 years after the onset of symptoms did not improve even after adequate treatment. Two other patients with gradual onset cognitive decline (one with seizures and ischemic strokes and another with parkinsonism) and the patient with cerebellar ataxia consulted the neurologist only 6 months to 1 year after becoming symptomatic and they took a period of 6 months to get better. Patients with rapid onset dementia, subacute encephalopathy, and myelopathy got diagnosed within 2–3 weeks of presentation and hence improved within a span of 3 weeks to 2 months of therapy. The meningitis patient got diagnosed only after 4 months of his hearing loss and he improved within 6 weeks after receiving ceftriaxone. This patient improved quickly in spite of 4 months delay in...
diagnosis. However, an important point to note in this patient is that this patient had only meningitis and cranial nerve palsies, no brain parenchymal involvement unlike the previously discussed patients who improved very slowly. Hence, we draw the conclusion that a longer duration from symptom onset to treatment and parenchymal involvement is associated with a poor response. A similar observation has been reiterated in the literature.[7]

**Conclusion**

If you face a scenario of dementia, encephalopathy, organic neuropsychiatric disorder, meningitis, cerebellar ataxia or myelitis, think of neurosyphilis as one of the causes if you are at a loss to come to a confirmed etiological diagnosis.

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**Conflicts of interest**

There are no conflicts of interest.

**References**

1. Berger JR, Dean D. Neurosyphilis. Handb Clin Neurol 2014;121:1461-72.
2. Ghanem KG. REVIEW: Neurosyphilis: A historical perspective and review. CNS Neurosci Ther 2010;16:e157–68.
3. Hooshmand H, Escobar MR, Kopf SW. Neurosyphilis. A study of 241 patients. JAMA 1972;219:726-9.
4. Madhusudhan M. Neurosyphilis. Neurol India 2009;57:233-4.
5. Skalnaya A, Fominykh V, Ivashchenko R, Averchenkov D, Grazhdantseva L, Frigo N, et al. Neurosyphilis in the modern era: Literature review and case series. J Clin Neurosci 2019;69:67-73.
6. Waddington K, Thomas R, Willis M. General paralysis of the insane. Pract Neurol 2011;11:366-9.
7. Kayal AK, Goswami M, Das M, Paul B. Clinical spectrum of neurosyphilis in North East India. Neurol India 2011;59:344-50.
8. Timmermans M, Carr J. Neurosyphilis in the modern era. J Neurol Neurosurg Psychiatry 2004;75:1727-30.
9. Sabre L, Braschinsky M, Taba P. Neurosyphilis as a great imitator: A case report. BMC Res Notes 2016;9:372.
10. Sinha S, Harish T, Taly AB, Murthy P, Nagarathna S, Chandramuki A. Symptomatic seizures in neurosyphilis: An experience from a university hospital in south India. Seizure 2008;17:711-6.
11. Garg D, Vibha D, Pandit AK, Srivastava AK. Neurosyphilis presenting as pure cerebellar ataxia: An atypical manifestation. BMJ Case Rep 2019;12:e231058.
12. Jay CA. Treatment of neurosyphilis. Curr Treat Options Neurol 2006;8:855-92.
13. Sharma C, Nath K, Kumawat BL, Khandelwal D, Jain D. Erb's paraplegia with primary optic atrophy: Unusual presentation of neurosyphilis: Case report and review of literature. Ann Indian Acad Neurol 2014;17:231-3.
14. Sethi S, Das A, Kakkar N, Banga SS, Prabhakar S, Sharma M. Neurosyphilis in a tertiary care hospital in north India. Indian J Med Res 2005;122:249-53.
15. Ganesh R, Stanley A, Ganesh N, Venkatram MK, Alagappan R. Prevalence of neurosyphilis at Government Rajaji Hospital, Madurai, India. Int J STD AIDS 1994;5:290-2.
16. Chahine LM, Khoriati RN, Tomford WJ, Hussain MS. The changing face of neurosyphilis. Int J Stroke 2011;6:136-43.
17. Sethi S, Mewara A, Hallur V, Prasad A, Sharma K, Raj A. Rising trends of syphilis in a tertiary care center in North India. Indian J Sex Transm Dis AIDS 2015;36:140-3.
18. Marra CM. Neurosyphilis. Continuum (Minneap Minn) 2015;21:1714-28.
19. Wang L, Zhang Z. Simultaneous neurosyphilis and tuberculous meningitis in a patient with systemic lupus erythematosus: A case report. Int J Clin Exp Med 2018;11:8766-9.
20. Nayak S, Acharjiya B. VDRL test and its interpretation. Indian J Dermatol 2012;57:3-8.
21. Johns DR, Tierney M, Felsenstein D. Alteration in the natural history of neurosyphilis by concurrent infection with the human immunodeficiency virus. N Engl J Med 1987;316:1569-72.