Original Article

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 clinch 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 (B_{12} 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, meningovascular 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.[1-4] Late NS has decreased and early neurological involvement is being increasingly reported.[2] 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.[2,4,5] 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/

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Submitted: 10-Jan-2021 **Revised:** 26-Jan-2021 **Accepted:** 12-Feb-2021
Published: 17-Jun-2021

DOI: 10.4103/aian.AIAN_28_21

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
Cases 1 to 6- Cognitive decline/encephalopathy						
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
Case 7: Meningitis with hearing impairment						
7	55	M	HA, decreased hearing, diplopia	4 months	Neck stiffness, bilateral LR palsies, SN deafness	Meningitis with hearing impairment
Case 8: Cerebellar ataxia						
8	67	M	Progressive imbalance	1 year	Gait and limb ataxia	Cerebellar ataxia
Case 9: Myelopathy						
9	40	M	Paraparesis, urinary hesitancy	2 weeks	Paraparesis- power 4 LLs, hyperreflexia, upgoing plantars, spastic gait	Myelopathy
Case 10: Asymptomatic neurosyphilis (Condyloma lata)						
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

Table 3: Investigations, treatment and outcomes of all patients

No.	Age	Sex	MRI	Serum TPHA	CSF examination				Rx	Outcome	Time for Improve-ment
					P	S	Cells	VDRL			
Cases 1 to 6- cognitive decline/encephalopathy											
1	65	M	Atrophy	+ve	35	77	19	+ve	Cf	Still depend-ent	Has not improved
2	62	M	Normal	+ve	70	70	10	+ve	CP	Improved	3 months
3	53	M	Atrophy	+ve	41	78	60	+ve	Cf	Improved	3 weeks
4	45	M	Temporal, parietal, thalamic infarcts	+ve	28	92	30	+ve	CP	Improved	6 months
5	42	F	Normal	+ve	61	36	5	+ve	Cf	Improved	4 weeks
6	42	M	Atrophy	+ve	76	42	20	+ve	CP	Improved	6 months
Case 7: meningitis with hearing impairment											
7	55	M	Pachy-meningeal enhancement	+ve	85	50	24	ND	Cf	Improved	6 weeks
Case 8: cerebellar ataxia											
8	67	M	Cerebellar atrophy	+ve	59	68	12	+ve	CP	Improved	6 months
Case 9: Myelopathy											
9	40	M	C2-3 & T2-11 cord signal with candle-gutter appearance	+ve	40	70	7	+ve	Cf	Improved	2 months
Case 10: Asymptomatic neurosyphilis (Condyloma lata)											
10	41	F	Normal	+ve	72	66	10	+ve	Cf	Improved	NA

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

showed generalized cerebral atrophy. His serum B_{12} 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 performance on three-stage command, short-term memory impairment, easy distractibility and irritability. His cranial nerves, motor and sensory system were normal and he had gait ataxia. The MRI brain showed generalized atrophy. The hemogram, biochemical tests and thyroid function were normal and CSF examination revealed 60 lymphocytes with normal proteins and sugar. Considering his history of alcohol abuse, he received intravenous thiamine and vitamin B_{12} along with intravenous acyclovir with no improvement. The fourth subject was a 45-year-old male who got admitted with progressive cognitive decline and seizures. He had a history of progressive behavioral change (withdrawn, apathetic, irritable) and forgetfulness over 6 to 8 months before admission. He had also suffered six episodes of tonic-clonic seizures during this period and was initiated on phenytoin for the same. He developed walking difficulty and urge incontinence which was gradually worsening. He became dependent on family for activities of daily living such as bathing, dressing and feeding. 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 postero-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. The fifth patient, a 42--year-old lady presented with a 2- week history of confusion and behavioral change. She had mild fever 3 days before admission which caused further worsening of her cognitive abilities. On examination, she was alert but confused and disoriented to time and place. She had

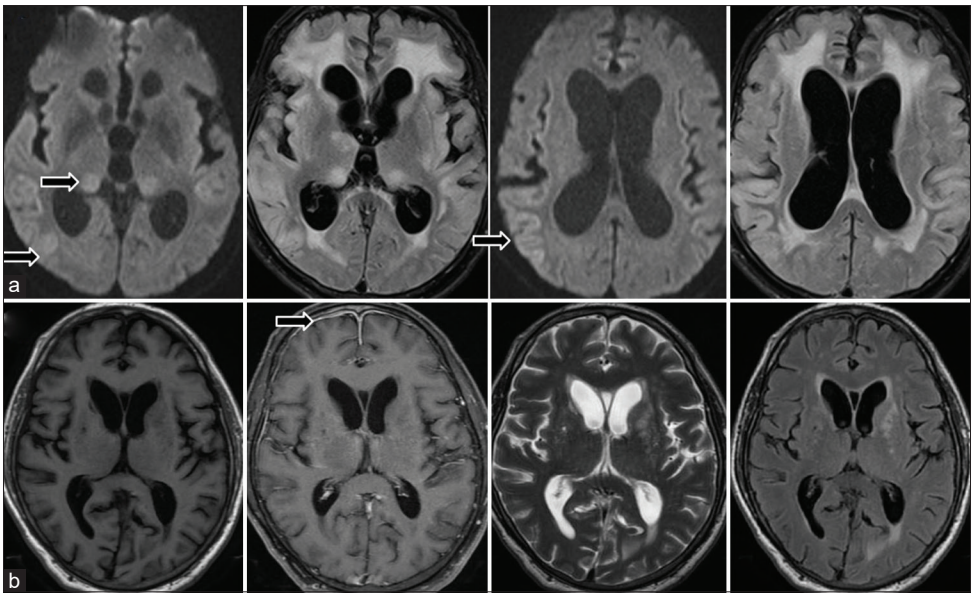

Figure 1: (a) Diffusion-weighted imaging (DWI) and fluid-attenuated inversion recovery (FLAIR) axial MRI brain sequences of a patient who presented with gradual onset cognitive decline with seizures. Images show infarcts in the right temporal and parietal lobes and bilateral postero-medial thalami. Bilateral 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

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 4months 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

2 g daily for 2 weeks with complete recovery of headache and lateral recti palsies 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 B_{12} 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 B_{12}, serum copper level and serum anti-aquaporin-4 IgG and HIV were normal. The CSF proteins and sugar were normal with seven lymphocytes and CSF viral panel and oligoclonal band were normal. After extensive investigations to find the cause of myelitis failed, a serum and a CSF VDRL were sent, which were detected reactive and the serum TPHA turned out strongly positive. He received IV ceftriaxone 2 g daily for 14 days with which his symptoms gradually improved over 2months.

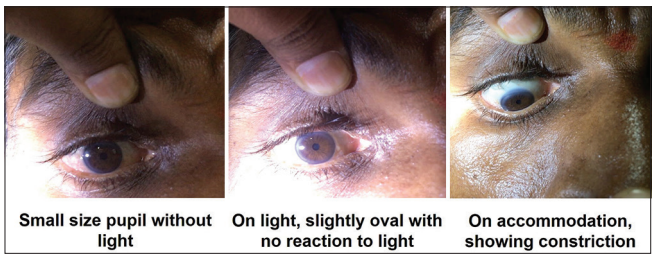

Figure 2: Argyll Robertson pupils (ARP) in a patient with neurosyphilis who presented with gradual onset cognitive decline with parkinsonism

Case 10: Asymptomatic neurosyphilis (Condyloma lata)

A 41-year-old female was referred from the anesthesia department for the evaluation of a positive serum VDRL test. She was being worked up as she was posted for piles surgery under spinal anesthesia. She was a diagnosed case of condylomata lata. She was asymptomatic from a neurological point of view and her neurological examination and MRI brain were normal. Her CSF VDRL was reactive with 10 lymphocytes/cu mm, 72.5 mg% proteins and a normal CSF sugar level. The patient was treated as a case of secondary asymptomatic neurosyphilis with IV ceftriaxone 2 g daily for 14 days.

Discussion

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

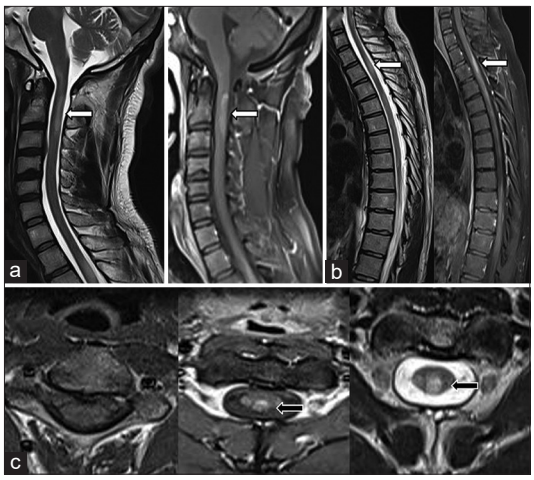

Figure 3: The MRI spine (a and b- T2W and T1W contrast sagittal, c- T1W, T1W contrast, and T2W axial) showing a hyperintense signal in the cervical spine at the C2–3 level and a much longer one in the thoracic spine, mainly in the posterior portion of the cord (black arrow) from the T2 to T11 level, with contrast enhancement, giving candle-gutter appearance (white arrows). There was not much cord swelling seen

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 meningovascular 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_{12} 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 poliomyelitis 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 meningovascular form, cerebral 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.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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