Flail Foot- Common Presentation Of An Uncommon Disease

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Abstract

Neurosyphilis is classically associated with meningovascular syphilis in the acute-subacute stage and tabes dorsalis & dementia paralytica in later stages. But cases presenting as demyelinating polyradiculoneuropathy are uncommon. A 27 yr old male presented with ascending polyneuropathy with electrodiagnostic testing consistent with Guillain-Barre Syndrome (GBS).He had CSF VDRL TPHA positive, supporting a diagnosis of neurosyphilis associated with GBS.This case highlights the importance of considering syphilis in any patient presenting with acute polyradiculoneuropathy.

Keywords: Guillain-barre syndrome, VDRL, CSF, Neurosyphilis

Introduction

The incidence of syphilis has been drastically falling In India due to the early diagnosis and better treatment options[1]. Apart from the classical presentations, syphilis presenting as acute polyradiculoneuropathy in immunocompetent patients is rare [2,3] .We report a case of acute polyradiculopathy in an immunocompetent patient who turned out to be syphilitic.

Case Presentation

A 26 yr old male presented with insidious onset left heel pain and swelling, which subsided with analgesics. 1 week later he developed bilateral dull aching calf pain which was aggravated by walking for 2 months followed by bilateral foot drop Left > right . He had to support and lift the left leg while climbing stairs. He felt unsteady while standing on one leg while dressing. Two weeks later, he noted difficulty in making food bolus , opening bottle lids and wringing towels with right hand. His symptoms gradually worsened over 2 weeks. There were no sensory symptoms , neck pain, bladder symptoms, recent fever, vaccinations, or travel history.He had unintentional weight loss(6 kg in 6 months) with preserved appetite. There were no prior episodes in the past. He had occasional ethanol intake , smoking,and high risk sexual behaviour for the past 1 year(multiple protected and unprotected , heterosexual and homosexual contacts with known and unknown partners; with last contact being unprotected 1.5 months back). There was no significant family history .

On admission, his blood pressure was 120/80 mmHg and pulse rate was 78/min. There was no pallor , lymphadenopathy or edema . Few erythematous macules were present over the chest with few discrete hyperpigmented macules over bilateral soles and palms.

Higher mental functions were normal with a MOCA score of 30/30 .

Cranial nerve examination was normal. Motor system examination showed reduced tone in bilateral ankles ; power- grade 4- in left infraspinatus, flexor carpi ulnaris, flexor carpi radialis and weak small muscles in both hands. He had grade 4- in bilateral hip flexors, grade 4+ in bilateral hip extensors, grade 3 in bilateral adductors , grade 1 in left dorsiflexor, grade 2 in right dorsiflexor, grade 2 in bilateral plantar flexors with weak bilateral toe grip and bilateral toe flexors . All reflexes were retained with normal sensory examination. He had bilateral high steppage gait and no peripheral nerve thickening.

The routine blood tests, including complete blood count, hematocrit and kidney function were unremarkable. Liver function test showed- SGOT/ SGPT - 39/56 IU/ ml and rest were normal. In view of the new onset neuropathy, the following investigations were done: ESR 26 mm/ hr;.Thyroid function test (TSH- 1.793 mIU/L, T3- 1.1 ng/ ml, T4 - 7.34 mcg/ dl) ;. FBS 102 mg/ dl; PPBS 71 mg/ dl; HBA1c 6.4%; serum ACE 27.8 mIU / L(Normal); VDRL, HBsAg, Retro I & II, AntiHCV negative; Total cholesterol 166 mg/ dl, Triglycerides 105 mg/ dl, LDL 98 mg/ dl; URE- 10-12 pus cells, with trace albumin; cANCA & pANCA negative; ANA 1/ 80 dilution positive (nuclear speckled

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pattern) with profile negative. Anti Ganglioside antibody panel was negative. The CSF study showed pleocytosis with 76 cells (100% lymphocytes), protein 109.40 mg/ 100 ml and sugar 58 mg/ 100 ml(GRBS 98 mg/ dl) . CSF VDRL & TPHA were positive. The serum VDRL was repeated in dilution (1:32) and it was positive. CSF lyme titres were negative .

Nerve conduction study showed bilateral peroneal CMAP amplitude reduction with poor proximal pick up, bilaterally reduced tibial and ulnar CMAPs with conduction blocks and prolonged F wave latency in bilateral peroneal nerves, thus fulfilling the Haddens criteria for AIDP.

Needle EMG of the selected muscles of the left upper and lower extremities showed decreased recruitment with normal amplitude and normal duration MUAPs. There was no abnormal spontaneous activity.

He was started on injection Ceftriaxone 2 gm twice daily with injection Dexamethasone 4 mg twice daily. MRI Brain was normal (done to rule out meningovascular syphilis). MRI spine showed mild cervical cord atrophy with no intrinsic cord hyperintensity or meningeal enhancement.

Dermatology opinion was sought and was continued on ceftriaxone for 2 weeks. At the end of one week, he had subjective improvement with bilateral dorsiflexion power in grade 3. He was discharged and kept under follow up.

Discussion

Our patient had a new onset symmetrical distal more than proximal motor weakness of two months duration with CSF VDRL positivity . The absence of rash, retained reflexes and absence of other neurologic features like cranial neuropathy, dementia, myelitis with a normal MRI Brain & spine rules out a late syphilis. The pleocytosis of 76 cells and protein elevation in this case suggests the possibility of neurosyphilis over GBS. However it remains unclear whether the patient's polyradiculopathy was demyelinating in nature, or whether syphilis was the primary inciting factor.

There have been several studies describing GBS-like symptoms in a patient with syphilis [4,5]. While penicillin is the mainstay of treatment, it remains unclear whether immunotherapy (immunoglobulins or plasmapheresis) is necessary. Also our patient did not have any other classical clinical features of neurosyphilis , which points to the fact that acute polyradiculoneuropathy can be one of the earliest manifestations of syphilis[6]. We followed up with the patient for further neurological symptoms as early seronegative HIV couldnt be ruled out .

In summary, this is a case of clinical and electrodiagnostic GBS without other preceding confounders strongly suggesting syphilis as the underlying aetiology.

Conclusion

This case emphasises the need to include syphilis in the differential of any patient presenting with an acute ascending paralysis concerning GBS, and highlights the need to understand the variety of syphilis presentations as the incidence increases.

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