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A RARE CASE OF PRIMARY HIV INFECTION PRESENTING AS MYELOPATHY

Chunduri Venkata Viswa Vikranth* Andhra Medical College, *Corresponding Author

ABSTRACT An HIV infected person develops a broad range of neurological manifestation throughout the disease course, and most of them will develop during the advanced stages of the disease because of low CD4 count and secondary infections. Vacoular myelopathy is the most common form of myelopathy in a HIV infected person presenting late in the disease course. Early HIV infection generally presents with nonspecific symptoms resembling mononucleosis and flu. But this case is a rare presentation of early HIV infection causing myelopathy.

KEYWORDS : HIV myelopathy, primary HIV infection, early HIV infection.

Case report

A 13-year-old orphan girl staying in a welfare hostel presented with sudden onset of weakness of all four limbs of one-day duration, involving both proximal and distal muscles; more in the lower limbs than the upper limbs

The weakness was associated with neck pain, paresthesias of hands & feet and girdle-like sensation at the level of lower part of the chest. She also gave a history of autonomic disturbances in the form of initial urinary retention followed by urinary incontinence.

There is no history of speech or language disturbance, with intact higher mental function and cranial nerve function. There are no features of any fever, vomiting, headache suggestive of meningitis. She also did not have any history of trauma or history of other systemic illness.

On examination, she is an averagely built and poorly nourished individual having pallor. There are no palpable lymph nodes, or visible rashes, skin and appendages appear normal. The lung and heart sounds were normal on auscultation and palpation of abdomen yielded normal findings.

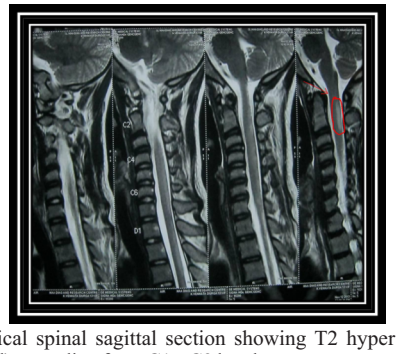
On central nervous system examination; her higher mental functions and cranial nerves were normal. Motor system examination showed normal bulk with increased tone(spasticity) in both lower limbs. Power of her upper limbs was decreased with 4/5 at the shoulder joint in all the direction of movements (flexion, extension, adduction and abduction) with normal power (5/5) at the elbow and wrist joint. The power of the lower limb was grossly decreased 2/5 at the hip (flexion, extension, abduction, adduction), knee 2/5 (flexion and extension) ankle 2/5 (flexion and extension).

The deep tendon reflexes/ muscle stretch reflexes were normal in the upper limb but were grossly exaggerated in the lower limb with ill sustained ankle clonus. The plantar response showed bilateral plantar extensor with a positive Babinski sign.

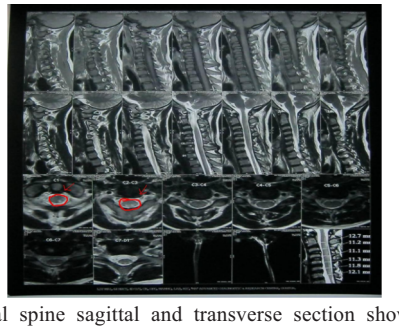
Sensory system examination showed decreased posterior column sensations in both lower limbs with normal pain and temperature sensations. Romberg's sign is positive (could be tested only after a week). The cerebellar examination was normal and there are no signs of meningeal irritation.

Laboratory investigation showed Hb- 8gm% microcytic and hypochromic blood picture with no abnormal cells, WBC count 7300cell/mm3 with 74% polymorphs, platelet count 2.3 lakh/mm3, ESR- 40 mm in 1st hour. Renal and liver function tests were normal. HIV serology performed using fourth generation ELISA showed both HIV 1 and 2 positive, later confirmed with HIV RNA detection. The CD4 count was 534 cells/mm3. HBsAg, HCV serology was negative and VDRL was of low tiers. CSF analysis showed 8cells/mm3 with 50% lymphocytes and 50% neutrophils, protein 43mg/dl, glucose 37mg/dl and ADA was 6.6 IU/L. serum B12 levels was 312pg/ml (normal 200-900 pg/ml). imaging studies chest x-ray, ultrasound abdomen and CT brain were normal.

MRI spine shows T2 long segment hyperintensity extending from C1 to C3 spinal segments with selective involvement of dorsal column and pyramidal tracts



A MRI cervical spinal sagittal section showing T2 hyper intensity (circled in red) extending from C1 – C3 level



MRI cervical spine sagittal and transverse section showing T2 hyperintensity (circled in red) at the level of C1 and C2-C3

DISCUSSION

About 7% of AIDS patients show clinical features of myelopathy during lifetime (McArthur 1987; Guiloff et al. 1988) and 50% of the patients of AIDS show pathological changes in postmortem studies (Henin et al. 1992). Of all the myelopathies caused by HIV vacuolar myelopathy is most common and widely recognized entity. Other causes of myelopathy in AIDS are caused by tumors, secondary infections, vascular lesion (Guiloff, Roberto. 1997).

The pathological incidence of myelopathy in HIV is higher than the clinical incidence because of coexisting peripheral nerve or intracranial disease, absence of clinical signs in cases with mild or moderate pathological change and failure to make the clinical diagnosis of vacuolar myelopathy in cases without weakness or spasticity or without the typical combination of pyramidal and posterior column signs (Guiloff, Roberto. 1997).

Vacuolar Myelopathy is the most common chronic myelopathy associated with HIV infection.

HIV-associated vacuolar myelopathy occurs during the late stages of

HIV infection when CD4 lymphocyte counts are very low, often in conjunction with AIDS dementia complex, peripheral neuropathies, and opportunistic infections or malignancies of the central or peripheral nervous system (eg, cytomegalovirus, progressive multifocal leukoencephalopathy, lymphoma).

However, It is well recognized that patients with AIDS can present with myelopathy unrelated to a tumour, opportunistic infection, or vascular disease. Because in such cases it is uncommon to obtain pathologic correlation and because we seldom determine the actual cause of the clinical and MR findings, the term "AIDS-associated myelopathy" has been used to reflect this uncertainty

Primary HIV infection refers to the very early stages of HIV infection or the interval from initial infection to the time that antibody to HIV is detectable. Diagnosing patients with primary HIV infection is a clinical challenge. The symptoms of primary HIV are nonspecific, and the diagnosis commonly is missed at initial presentation. (Ridzon R et al. 1997)

Neurological symptoms of Acute Retroviral syndrome include Aseptic meningitis, Radiculitis, Cranial nerve palsies and very rarely presents with features of Acute transverse myelitis.

HIV myelitis which is due to primary HIV infection occurs in earlier phases of disease when the CD4 count normal or near normal, with predominant involvement of posterior column (initially), corticospinal tract and autonomic nervous system (sometimes). MRI shows selective tract involvement in early stages despite clinical involvement of other tracts. (Niu MT et al. 1993; Castellanos F et al. 1994; Hassin-Baer S et al. 1998)

In contrast, vacuolar myelopathy occur in the later parts of the disease process when the CD4 counts are less than 50 cells/mm3, clinically involves all the tracts of the spinal cord and MRI shows diffuse involvement with spinal cord oedema or atrophy. (Woolsey RM et al. 1989, Di Rocco A 1999)

Myelopathy due to opportunistic infections and tumours also occur in later stages of the disease and the corresponding CD4 counts for various opportunistic infections are variable, clinically involves all the spinal cord tracts and MRI spine shows variable imaging findings.

Considering the above discussion my patient came with an acute spastic quadriparesis who is a newly diagnosed retroviral positive with CD4 counts >500 cells/mm3 without any obvious evidence of infectious, nutritional, metabolic or Neoplastic causes of myelopathy, goes in favour of Primary HIV myelitis.

CONCLUSION

Findings in our case are consistent with HIV Myelitis that is caused by virus attacking spinal monocytes and macrophages. Recognizing primary HIV infection in symptomatic patients is essential as early diagnosis provides an opportunity for early linkage to HIV care and may decrease future HIV transmission by newly identified patients, who are particularly infectious during early untreated HIV infection. A serious neurologic disease may present with relatively trivial symptoms and signs therefore, a high index of suspicion must be maintained to detect the disease early in these patients.

A careful neurologic examination to attempt anatomic localization is necessary to guide further laboratory and imaging studies because multiple neurologic diseases often coexist in patients. Close follow-up is needed even if a presumptive diagnosis has been made and change in clinical condition often necessitates a thorough reevaluation.

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