Original Research Paper

Volume-9 | Issue-11 | November - 2019 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

Neurology

ACUTE DORSAL MYELITIS WITH ACUTE HEPATITIS -B NOT RESPONDED TO STEROID INITIALLY RESPONDED TO PLASMAPHARESIS- FIRST RARE CASE REPORT .

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ABSTRACT Many potential etiologies for acutemyelitis have been described. Clinical, immunological, and radiological findings of noncompressive myelopathies are reviewed, as are how thesefindings can be used to distinguishbetween demyelinating, infectious, other inflammatory, vascular, neoplastic, and paraneoplastic etiologies. .(1).Acute transverse myelitis(ATM) is a rare clinical entity characterized by sudden onset of sensori-motor paralysisdue to complete spinal cord dysfunction with truncal weakness ., It may be precipitated by a number of conditions, especially acute viral and post vaccinations. Though hepatitis B vaccination can result in transverse myelitis, its occurrence following acute hepatitis B infection has been described in few case reports worldwide.We report the case of acute transverse myelitisassociated with hepatitis B infection while no response to intravenous steroid.,responded to plasmapharesis. No other demonstrable clinical and laboratory evidence for any other disorder raise the probability of other etiology in this case.

KEYWORDS : ATM-Acute Transeverse Myelitis

INTRODUCTION -

Acute transverse myelitis (ATM) is a uncommon neurological disorder characterized by weakness of limbs due to inflammation of the spinal cord . The inflammatory process often extends longitudinally over three or more segments and functionally transects the entire substance of the spinal cord leading to paraplegia. This condition has diverse etiologies and has a well-known association post vaccinations especially hepatitis B vaccine [3].. Acute transverse myelitis following acute hepatitis B virus infection has been rarely described in the literature. This case highlights the first case report of acutetransverse myelitis being first clinical feature of extra-hepatic manifestation of acute hepatitis B infection. . In this report we discuss the significance of this newassociation of transverse myelitis and acute hepatitis B infection as sole manifestation.(3)

CASE REPORT-

A 42 year old male nonsmoker,nonalcoholic presented with sudden onset of weakness and loss of sensation over both lower limbs and impairement in turning on bed for 3 days. The weakness was symmetric, involving distal and proximal muscles and had progressed since few hours after onset .Power was grade 0/5 proximal and distal lower limbs. There was no history suggestive of bladder involvement initially .No history of cranial nerve deficits, autonomic dysfunction ,seizure or brainstem involvement.

On admission MRI spine with contrast showed long segment diffuse T2hyperintensity involving dorsal cord and conus from D8 level onward without contrast enhancement while MRI BRAIN was normal.Hb 13.35%,WBC-11600,S.Cr-1.2, electrolytes ,ECG ,thyroid tests,chest xray were normal.S.ANA was negative.CSF study showed wbc 02 ,protein94,glucose 81 mg/dl.CSF culture was negative.Nerve conduction study was normal.Neurotropic viral profile including adenovirus,EBV PCR,HHV6,HHV7,Human parachnovirus,Parvo B19,HPeV,VZV,CMV DNA,HSV-1 and HSV- 2 DNA,TB DNA PCR were negative.Serum Anti-NMO antibody was negative.HIV,HCV spot were negative.

HBsAg was positive.Hepatitis B core antibody(Anti-HBc)-IgM was positive,while quantitative HBV DNA PCR was 172 IU/ml.Serum Hepatitis Be Ag (HBeAg) and hepatitis Be antibody (Anti-HBe)were nonreactive suggestive of acute hepatitis B with low viral load.Liver function test,serum albumin and prothombin time were with in normal limit.

Antineutrophilic cytoplasmic antibody(ANCA) includes p-ANCA,C-ANCA,GBM were negative.

Following discussion we started him on pulses of methyl prednisolone injection 1gm daily for five days. After five days of steroid, he became restless with mild breathing difficulty with low grade fever.Counts were increased .no improvement in power was noted after 5 days.Therefore in view of sepsis due to steroid we stopped oral steroid and immediately started plasmapharesis.After first cycle patient condition became stable .After completion of three cycles of plasmapharesis his power of right lower limb was grade 2/5 and left lower limb was 3/5.he still had difficulty in turning on bed.because of his stable condition we again started oral steroid with strict observation .Following 1 week he was stable and achieved power of grade 3/5 in bilateral lower limbs.

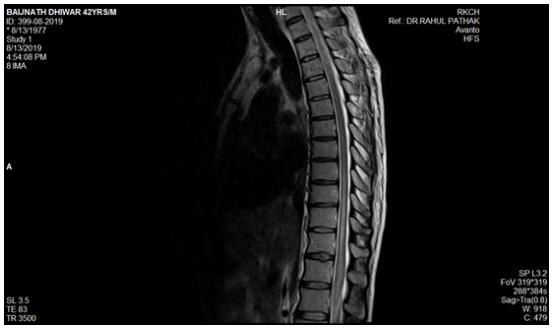


Fig.1: T2 weighted MRI of spine; intramedullary hyperintensity was noted in the spinal cord extending from D8 to Conus.

DISCUSSION-

Acute transverse myelitis is characterized by sudden onset neurologic deficits that develop over a period of hours and progress over the next few days to weeks . Spinal cord dysfunction is usually complete producing the transverse myelopathy. There is sensorimotor weakness of the limbs and trunkdepending on the segments involved with or without sphincter,autonomic involement.Case report of HBsAg - carrier describes an atypical case of SAH, transverse myelitis and nephrotic syndrome (5).Viral agents can be the underlying cause of acute myelopathy such as HCV, HBV,HAV,HIV, CMV, VZV, HSV,and human T-cell lymphotropic virus type 1 (HTLV-1).

Interestingly as vaccine against HBV is also frequent cause for transverse myelitis. Postvaccinal and post-infectious transverse

myelitis, many mechanisms have been proposed including molecular mimicry , polyclonal B cell activation,immune complex deposition and epitope spreading leading to developement of autoimmunity.

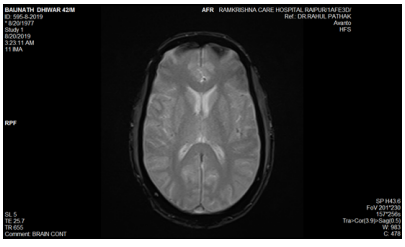
In our case patient was diagnosed with acute hepatitis B with low infectious state as positive IgM-anti HBc and nonreative HBeAg and anti-Hbe.

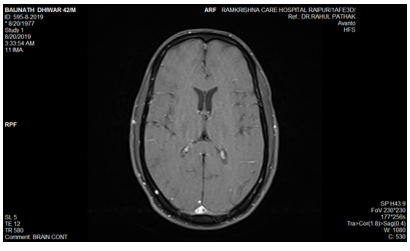
Intravenous immunoglobulin has no role in the treatment of ATM(3).In view of acute hepatitis B infection , the opinion available were steroid ,immunosuppression and plasmapharesis. Considering the degree of functional disability and after discussion with the patient, we started steroids with carefulmonitoring of the liver functions and viral load. Following initiation of steroid, neurologic deficits deteriorated slightly and patient became tachypnic.Therefore immediately we started plasmapharesis .After three cycles of plasmapharesis patient motor weakness improved to grade 3/5 with normal breathing pattern.Patient was discharged with residual weakness with advice on regular followup.

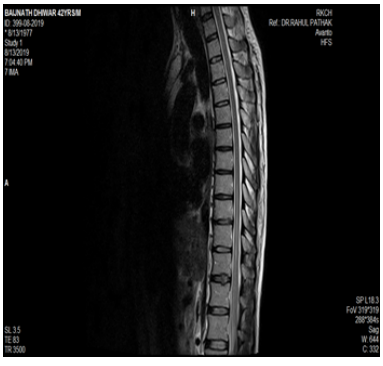
Our case is unique and probably first case in india as rare case of ATM with acute hepatitis B with low infectivity ,not responded to steroid which is usually a first line treatment responded to plasmapharesis.

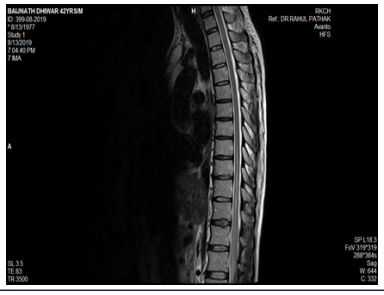
CONCLUSION

Although rare our case suggest that in all cases of acute transverse myelitis screening of hepatitis B should be part of workup. Treatment options are steroid and plasmapharesis ,therefore this case suggests immediately to start plasmapharesis if steroid fails.









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