Recurrent Myelitis Associated With Hepatitis C Infection

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DISCUSSION:
We report a patient with recurrent myelitis in association with recently diagnosed hepatitis C infection. Serums and CSF antibodies against hepatitis C virus were detected. Initial evaluation for other causes for myelitis failed to show another etiology. Sarcoidosis is unlikely given the normal levels of serum and CSF of tumor necrosis factor (TNF) and normal chest CT. Multiple serologies were also potentially excluded with negative viral and bacterial markers, negative serologies for syphilis, negative VDRL, and positive VDRL against the brain. Adverse events did not lead to viral hepatitis B and C and no evidence of other gain-infections. Anti-ganglioside antibodies were also negative. Hepatitis C is also known to cause several extra-hepatic complications including vasculitis and cryoglobulinemia (2,3) and vasculitis and hemorragic strokes (1-3). Acute disseminated encephalomyelitis was also reported as a complication (4). Myelitis, although rare, has been reported (5,6). In this case report we present a patient with recurrent myelopathy with CSF and serologic evidence of hepatitis C infection.

METHODS

CASE REPORT:
A 59 year-old African American female with previous history of hypertension and IV drug abuse presented with 2 week history of back and neck pain, bilateral lower extremity paresthesias involving the right limb more than the left and numbness of the left hand. She denied any abnormalities in bowel or bladder function or fever. Her neurological exam revealed right lower limb weakness 4/5, decreased sensation to vibration and pain dadiata in bilateral lower extremities but no clonus. Her levels were normal. She had decreased sensibility in bilateral lower extremities. She also had decreased sensation in bilateral lower extremities. Spinal fluid revealed protein 41, glucose 69, WBC 3 with 2% of segmented, 45% of lymphocytes. Normal ACE level, normal cytology, but increased gamma globulins and myeloid basic protein. Cultures for acid fast bacilli and fungi were negative. Serums for HCV were positive, but HCV RNA was not detected in the cerebrospinal fluid. Nolte et al. described a 61 year-old man with sensory ataxia and myelopathy with HCV RNA positive in the CSF. However, some other reports only showed positive antibodies against HCV.

Our patient had positive HCV antibodies in the CSF which is consistent with the idea of a possible immune mediated mechanism. The impact of HCV specific treatment with interferon in myelopathy is not established. Our patient is being currently treated, but no conclusions can be made at this point. Positive NMO antibodies identified later are suggestive of NMO. However, she did not have any visual complaints and the visual evoked potential studies were normal. However, given the extensive spinal lesion on MRI and lack of lesions typical for MS on brain MRI, neuromyelitis optica (NMO) IgG antibodies were also examined and tested to the enhanced unit. At that time it was decided to start treatment for hepatitis C.

She had no visual symptoms during any of her admissions and clinical neuro-ophthalmologic evaluation and visual evoked potential studies were normal. However, given the extensive spinal lesion on MRI and lack of lesions typical for MS on brain MRI, neuromyelitis optica (NMO) IgG antibodies were checked and found to be positive in September 2006.

CONCLUSIONS:
1. Hepatitis C should be in the differential diagnosis for recurrent myelopathy.
2- There are no reports of the role for interferon alpha in the treatment of myelitis related to hepatitis C Virus.

REFERENCES: