Transverse myelitis associated with chronic viral hepatitis C

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Myelite transverse associée à une hépatite chronique virale C

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RÉSUMÉ

Pré requis: L'infection par le virus C est une cause rare de manifestations extra hépatiques. La neuropathie périphérique est une complication commune de la cryoglobulinémie mixte. En plus la myélite transverse a été associée à l'infection par le virus C. Cependant la cansalité n'est pas encore bien établie

But : Rapporter une nouvelle observation.

Observation : Un homme âgé de 55 ans a présenté un déficit moteur au niveau des membres inférieurs avec une rétention urinaire. L'examen neurologique a révélé une paraparésie spastique et une ataxie proprioceptive. L'imagerie par résonnance magnétique nucléaire a montré une anomalie du signal au niveau de la moelle épinière de C3 à C5. La sérologie de l'Hépatite C s'est révélée positive. Le diagnostic d'une démyélinisation aigue responsable d'une poly neuropathie sensitivomotrice associée à une hépatite C est établi.

Conclusion : La recherche d'infection par le virus C doit être systématique devant une myélite transverse qui reste sans explication.

Mots-clés

Myélite transverse – l'Hépatite C

SUMMARY

Background: HCV infection could cause several extra hepatic diseases including mixed cryoglobulinemia. Peripheral neuropathy is the most common complication of mixed cryoglobulinemia. In addition to cryoglobulinemia's neuropathy, transverse myelitis had been related to hcv infection.

Aim : But causality of this association is not clearly established.

Case report: A 55-year-old man presented with motor deficiency in lower extremities and urinary retention .Neurological exams showed a spastic paraparesis and proprioceptive ataxia. Spinal MRI revealed a contrast enhancing signal abnormality within the spinal cord extending from Levels C3 to C5. Serology hepatitis C and viremia were positive. Clinical diagnosis of acute demyelinating sensorimotor polyneuropathy associated to chronic hepatitis C was etablished.

Conclusion: Screening of HCV infection must be done in patients with transverse myelitis and no clear aetiology.

Key-words

Transverse myelitis – Chronic viral hepatitis C

Extra hepatic manifestations with chronic hepatitis infection were described, including mixed cryoglobulinemia, membranous proliferative glomerulonephritis, sicca syndrome and thyroiditis (1). In the same way, both peripheral and central nervous system disorders have been described as complications of HCV infection (2,3). Myelitis was described in some cases in patients with chronic HCV infection, but causality of this association is not clearly established (4,5,6,7). We describe a case of a patient with transverse myelitis revealing chronic hepatitis C infection.

CASE REPORT

A 55 –year old man was presented in May 2007 with motors deficiency of lower extremities and urinary retention.
Neurological exams showed a spastic paraparesis and proprioceptive ataxia. Spinal MRI revealed a contrast enhancing signal abnormality within the spinal cord extending from levels C3 toC5. Cerebral MRI was normal. Clinical diagnosis of acute demyelinating sensorimotor polyneuropathy was strongly suspected.
Cerebral spinal fluid (CSF) had normal cytology and biochemistry. CMV, EBV and VIH were negatives in CSF. Laboratory data revealed normal hemogram, liver and thyroid function.
Serology HIV, CMV, HSV, BW, EBV and WRIGT serology were negative, also AAN, PANCA and complement. Serology revealed a negative hepatitis A and B .While hepatitis C serology was positive through ELISA third generation. Viral load was upper to 5.105 uml with genotype 2 .Liver biopsy was not performed.
Cryoglobulinemia was negative .The patient was treated for myelitis with intra venous methyl prednisone 1g/j during five days and was discharged to rehabilitation. Evolution was favourable. The patient saved a spastic walking without assistance. Eight months after the myelitis episode, treatment by pegylated Interferon 2a 180 μg/week with Ribavirin 800 mg/day started for 6 months, with a sustained virological response, as attested by negative viremia at the end of the treatment and six months later. No neurological deterioration was noted during the treatment or after stopping it with a decline of 7 months.

DISCUSSION

We report a case of chronic HCV infection in a patient who developed acute demyelinating polyneuropathy. HCV infection could cause chronic hepatitis, cirrhosis and hepatocellular carcinoma. On the other hand there were several extra hepatic diseases including mixed cryoglobulinemia (8).
Peripheral neuropathy is the most common complication of mixed cryoglobulinemia and result of axonal ischemic damage. Two main pathogenic mechanisms have been suggested, represented by deposits of cryoglobulins in the vasanervrum microcirculation and vasculitis (9). Recently a role of anti neuronal antibodies has been suggested (10). In addition to cryoglobulinemia's neuropathy, transverse myelitis and cognitive impairment have been related to HCV infection (4,5,6,7).
Only four cases of acute transverse myelitis associated with chronic hepatitis C with a negative cryoglobulinemia, have been described in the literature. Nolte et al (7) reported a 61 year old man with chronic HCV infection genotype 1, who developed transverse myelopathy limited to sensory loss. The MRI was normal, but there was an electrophysiological evidence of spinal cord dysfunction. Amrit et al (6) described a case of 46 year old man with hepatitis c viral infection, who developed recurrent myelitis, spinal cord biopsy showed acute demyelination without evidence of vasculitis. Antibodies anti-HCV and HCV-RNA were not detected in the CSF. Neither HCV antigens nor RNA-HCV, were detected in the spinal cord. Zandman-Goddard (5) reported a case of 34 year old man with chronic hepatitis C with positive viremia, who develops a transverse myelitis, anti - HCV and RNA were not looked in CSF. Mester et al (4) describe another case of transverse myelitis associated with central multiple demylinisation, neuropathological exam revealed white matter lesions with areas of demylination and axonal preservation.
In addition to these cases, there are two other reported cases of encephalomyelitis associated with acute HCV infection developed in women patients (11, 12). Evolution was favourable under high dose of corticosteroid in one case, and fatal in the other case, in post mortem, HCV-RNA was detected in brain, liver, pancreas, and blood but not in the CSF.
Pathogenesis of demylination in chronic HCV infection was both: Direct viral invasion and immune- mediated mechanisms. Direct mechanism was supported by the presence of HCV RNA in the CSF and in the brain, indicating that HCV reached the intrathecal compartment (7, 11).
In order to confirm the neurotropism of HCV, two studies analyzed systematically HCV RNA in the brain and in the CSF (13,14). Radkowski et al. (13) detected HCV replication in the central nervous system, in two patients with a different genotype compared to the one detected in the serum. Laskus et al. (14) found HCV sequence in CSF on 8 of 13 HCV positive patients, CSF- derived virus was similar to them found in peripheral blood mononuclear cells (PBMC), which suggests that PBMC could carry HCV into the brain.
In others cases HCV RNA was not detected in the CSF, suggesting immune- mediated mechanisms (5,6).Our patient presents a transverse myelitis revealing a chronic hepatitis genotype 2. He was treated by pegelated bitherapy with a sustained virological response and stabilization of neurological signs. Mestre (2) treated his patient with pegelated bitherapy. Eight weeks later, HCV RNA was undetectable. Evolution was marked by delirium remitted, a slight paresis but the flaccid paraplegia persisted. Therefore, it seems that, antiviral treatment induces a partial improvement of neurological signs. This treatment should be prescribed under a strict screening because interferon is associated with uncommon side effects of neuropathy demyelinating (15,16,17).

In conclusion, screening of HCV infection must be done in patients with transverse myelitis and no clear aetiology. Treatment of HCV infection seems to be associated with no neurological complications in case of this association.

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