Acute Hepatitis B Associated Neuromyopathy

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Research Article

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Abstract

A 67 year old man presented with abdominal discomfort and jaundice for 1 month with difficulty in walking with severe pain in both thighs of 3 days duration. He was a diabetic and hypertensive on medications. There was no history of HMG CoA reductase inhibitor use. On examination he had icterus and grade 2 power in the proximal upper and lower limbs. Deep tendon reflexes were inelicitable. On day 1, CRP was 37mg/L and liver function tests were deranged [Total Bilirubin 16 mg%, direct 14.3mg%, SGOT [920 U/L], SGPT [590 U/L], Alkaline Phosphatase 276.5 U/L. Serum CPK levels [9768 U/L], LDH [979 U/L] and Ferritin [7264 ng/ml] were elevated on day 2. ANA profile was negative. Leptospiral antibody, dengue serology and SARS-CoV2 RT-PCR were negative. Hepatitis B serology was compatible with an acute infection. On day 3, nerve conduction studies showed an axonal sensory-motor polyneuropathy predominantly involving the lower limbs. F waves were absent. Fibrillations and positive waves were picked up from the Tibialis anterior muscles bilaterally. He was started on IVIG 2gm/kg x 5 days. On day 4, his CPK levels increased to >42,000 U/L and he was shifted to the ICU and started on forced alkaline diuresis. Urine myoglobin was positive.. On day 6, MRI whole body muscle STIR imaging showed patchy ill-defined STIR hyperintensities involving the muscles of the gluteal, pelvic girdle muscles, both thighs and leg muscles with fascial edema. The muscles of the upper limbs and shoulder girdles also showed patchy STIR hyperintensities. Diffuse subcutaneous oedema was noted in the soft tissue of the thighs, legs and abdominal wall. Over the next few days, the weakness in the upper limbs worsened and he developed a weak cough. He did not consent to a lumbar puncture or muscle biopsy. Over the next 9 days, his liver function tests and CPK levels gradually normalised. He was also started on Entacavir. By day 10, his upper limb and distal lower limb had improved to grade 4/5 power and he was able to stand with support and he was discharged.

Main Text

A 67 year old man presented with abdominal discomfort and jaundice for 1 month with difficulty in walking with severe pain in both thighs of 3 days duration. He was a diabetic and hypertensive on medications. There was no history of HMG CoA reductase inhibitor use. On examination he had icterus and grade 2 power in the proximal upper and lower limbs. Deep tendon reflexes were inelicitable. On day 1, CRP was 37mg/L and liver function tests were deranged [Total Bilirubin 16 mg%, direct 14.3mg%, SGOT [920 U/L], SGPT [590 U/L], Alkaline Phosphatase 276.5 U/L. Serum CPK levels [9768 U/L], LDH [979 U/L] and Ferritin [7264 ng/ml] were elevated on day 2. ANA profile was negative. Leptospiral antibody, dengue serology and SARS-CoV2 RT-PCR were negative. Hepatitis B serology was compatible with an acute infection. On day 3, nerve conduction studies showed an axonal sensory-motor polyneuropathy predominantly involving the lower limbs. F waves were absent. Fibrillations and positive waves were picked up from the Tibialis anterior muscles bilaterally. He was started on IVIG 2gm/kg x 5 days. On day 4, his CPK levels increased to >42,000 U/L and he was shifted to the ICU and started on forced alkaline diuresis. Urine myoglobin was positive.. On day 6, MRI whole body muscle STIR imaging showed patchy ill-defined STIR hyperintensities involving the muscles of the gluteal, pelvic girdle

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Acute myositis is commonly associated with influenza. Acute viral myositis is often a self-limiting illness but rare cases can be complicated by rhabdomyolysis or compartment syndrome.

Table. Causes of viral myositis

SARS-CoV2 virus [Covid-19]
Influenza A & B viruses
HIV infection
HTLV-1 infection
Hepatitis A, B, C viruses
Epstein-Barr virus
Coxsackieviruses type A and B
ECHO viruses
Adenoviruses
Respiratory syncytial virus
Cytomegalovirus
Herpes simplex virus
Varicella-zoster virus
Dengue virus
Herpes simplex virus 2
Mumps virus
Parainfluenza virus
Parvovirus B19
West Nile virus

In contrast, Hepatitis viruses are uncommonly associated with myositis.^{1, 2, 3, 4} A search of the literature with the terms “Hepatitis B”, “polymyositis”, “dermatomyositis” and “myositis” revealed only 10 articles. Polymyositis with hepatitis B was thought to be an autoimmune response due to its response to oral corticosteroids.⁵ Chronic myositis is more often with Hepatitis and retroviruses. An acute presentation as in our case with a neuro-myopathy has not been reported in literature. Far more common is the reverse situation, whereby patients with rheumatological diseases or inflammatory myopathies undergo a reactivation of Hepatitis B infection after treatment with immunosuppressive medications. Viral myositis can occur due to a direct viral muscle invasion or the ensuing host immune response.

As our patient developed acute myositis with rhabdomyolysis, we cannot rule out a direct viral muscle invasion. This phenomenon is evident even in the current COVID-19 pandemic, where patients have developed acute myositis during the acute viral illness. We have also previously reported viral myositis with other viral illnesses such as Herpes zoster and Dengue. However the concurrent development of an acute neuropathy, with the rhabdomyolysis and myopathy as well as the prompt response to IVIG suggest the likelihood of an immune mediated neuro-myopathy associated with Hepatitis B infection.

MRI is a useful tool to demonstrate subclinical or overt viral myositis. Newer MRI muscle imaging protocols include T1, T2, T2 with fat suppression, diffusion tensor imaging and T2-relaxation incorporating Whole body or regional sequences. A short TI inversion recovery (STIR) sequence is capable of demonstrating hyperintense STIR muscle lesions. MRI muscle hyperintensities can be seen in muscle inflammation, edema or necrosis. Although they are non-specific and need biopsy correlation, the occurrence of rhabdomyolysis makes muscle inflammation or necrosis more likely. Although our patient did not undergo a muscle biopsy, we have demonstrated florid MRI changes compatible with an acute myositis in the setting of a neuromyopathy associated with acute hepatitis B infection. This is also the first report of an acute neuro-myopathy in the setting of acute Hepatitis B infection.

Declarations

Competing interests: The authors declare the following competing interests: Patient consent was obtained for the article

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Figures

Figure 1

STIR muscle MRI image sequences Panel A; Coronal whole body image showing subcutaneous fluid. [Orange arrow] Panel B; Coronal whole body image showing levels at which the axial images [following] were obtained Panel C- Axial image through mid arm level showing long head of triceps hyperintensity [blue arrow] Panel D- Axial image through mid thigh showing adductor muscle hyperintensity Panel E- Axial image through mid leg showing Tibialis posterior muscle hyperintensity. Panel F- Patchy STIR hyperintensity involving the vastus lateralis.[Blue arrow]. Fascial hyperintensity [green arrow].