



# The Association of Hepatitis B and Polymyositis: Rare but Real

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## Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Study

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## ABSTRACT

**Aims:** Hepatitis B virus infection is a global health concern. Although Hepatitis B virus primarily affects hepatocytes, it sometimes develops disease manifestations outside the liver and can present as full fledged proximal myopathy.

**Presentation of Case:** We report a 68-year-old man presenting with progressive proximal bilateral lower limb weakness for the last 14 days. The weakness was associated with pain and one episode of fever. Examination revealed 3/5 power in the shoulder and hip joint without any sensory deficit. Biochemical investigation showed elevated CPK levels (1102 IU/L) and liver enzymes (AST – 625 IU/L, ALT – 356 IU/L). HBsAg and HBeAg came out to be positive with HBV DNA of 73000 copies/mL. MRI shoulder and hip joints revealed diffuse intramuscular and myofascial edema suggesting inflammatory myositis. Muscle biopsy was suggestive of Necrotizing Autoimmune Myopathy.

**Discussion:** Patient symptoms improved after starting IV Prednisolone and Entecavir therapy suggesting a close association between Hepatitis B infection and myopathy.

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**Conclusion:** Although Hepatitis B virus primarily affects hepatocytes, it sometimes develops disease manifestations outside the liver, such as Polymyositis. Therefore, it is imperative to screen for Viral markers in these patients.

*Keywords: Hepatitis B; polymyositis; CPK level.*

## 1. INTRODUCTION

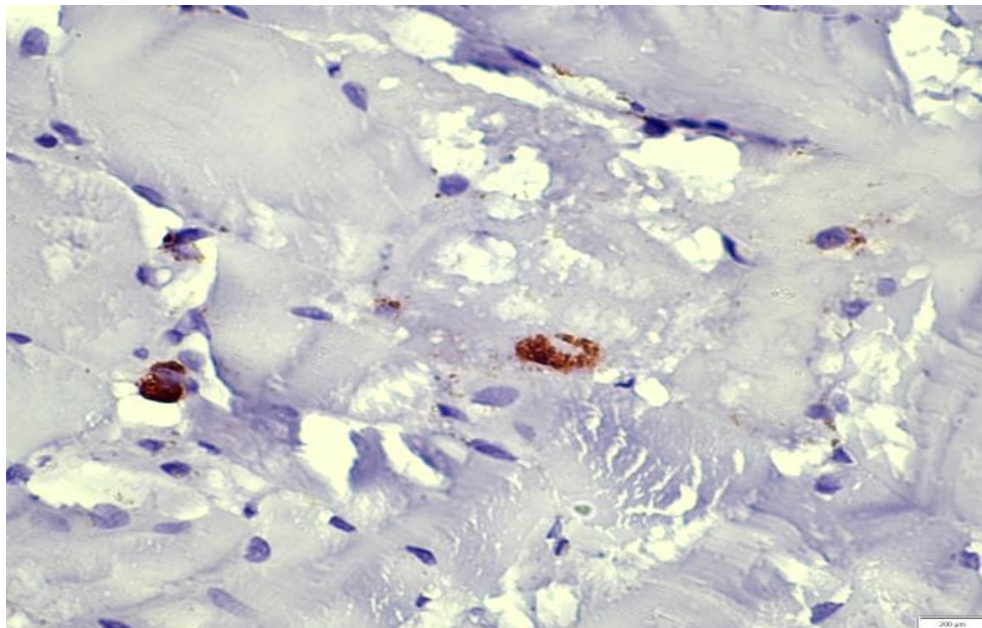
Polymyositis is a group of diseases characterized by inflammation of muscles. Primary symptoms may include muscle pain and soreness, fatigue and weakness in the affected parts. Weakness and pain in the muscles of the hips and shoulders is often a first sign, manifesting in the form of difficulty in getting up from a chair, combing your hair, or climbing stairs and may mimic a patient of paraparesis. Most common viral infections associated with Polymyositis include Influenza A and B, SARS-CoV-2 and Cocksackievirus but a few cases of Hepatitis B virus (HBV) have been reported [1-2].

## 2. PRESENTATION OF CASE

A 68 years old male, known case of Type 2 Diabetes Mellitus for 12 years, presented with complaint of Bilateral Lower limb weakness for 14 days, which gradually progressed from difficulty in getting up from squatting position to getting completely bed ridden in the next 6 days.

The weakness was associated with pain in Bilateral thighs. There was also history of one episode of fever on Day 1, recorded as 102°F, not associated with chills or rigors which got relieved with oral medications. Patient had inflammatory markers done from outside which revealed S. Ferritin -1459, LDH - 1100, IL6 - 53 and D-dimer - 2440.

On examination, the patient was conscious and alert. He was afebrile with BP of 120/70 mm Hg and PR of 84/min. Pallor was present and Bilateral pitting pedal edema was present, Chest: Bilateral normal vesicular breath sounds present, Abdomen: soft, non-tender, distended with shifting dullness. On neurological examination, GCS was E4 V5 M6 and MMSE 30. All cranial nerves were intact and there was no sensory deficit. Tone and bulk were normal in all 4 limbs. Motor examination revealed decreased power 3/5 in Bilateral proximal joints of upper limb and lower limb (shoulder and hip joint). Bilateral plantar reflex were flexor and cerebellar signs were absent.



**Fig. 1. Muscle biopsy showing CD68+ macrophage consistent with immune mediated necrotising myositis**

Routine investigations revealed high liver enzymes (AST - 625, ALT - 356, ALP - 257) and manifold elevated CPK (1102), CK-MB (440), LDH (1160) and CRP (>10). Patient was managed with antibiotics and fluids and serial inflammatory markers were done. Patient incidentally came out to be Hepatitis B positive so a full Hepatitis profile and HBV DNA levels were sent which showed DNA copies of 73000 and positive HBeAg and Anti HBe IgM. Patient was started on Tablet Entecavir 0.5 mg OD. HIV antigen, Anti HCV antibodies and Cryoglobulin levels were negative.

MRI of Bilateral thighs and Bilateral shoulders revealed Diffuse intramuscular and myofascial edema with diffuse subcutaneous edema suggestive of inflammatory myositis. Muscle biopsy was taken from the left thigh which showed focal evidence of whorling and disorientation of myofibrils with isolated necrosis and CD68+ on Immunohistochemistry. This was suggestive of Necrotizing Autoimmune Myopathy (Fig. 1). To rule out other causes of myopathy, a full autoimmune profile and Myositis profile (anti Jo-1, anti Ku, anti Mi2, anti PM-Scl) was done which was negative. EMG and NCV were grossly normal.

Patient was then started on Prednisolone (1mg/kg IV) and showed symptomatic improvement in terms of increase in power on Day 3. Serial cardiac enzymes showed decreasing trends (CPK - 794, CK-MB - 81 and LDH - 327) and patient started regaining power in bilateral lower limbs. Repeat D-dimer and Ferritin levels showed decreased levels. On the fifth day of therapy, patient was able to walk with support.

### 3. DISCUSSION

The spectrum of clinical manifestations of HBV infection varies in both acute and chronic diseases and can have extra hepatic manifestations like Polyarteritis Nodosa, Dermatological conditions (Bullous Pemphigoid, Lichen Planus), Cryoglobulinemia and Neurological/Psychological conditions. On the other hand, Myalgia is a common musculoskeletal manifestation of Hepatitis B infection. Hepatitis B infection can trigger the immune system and cause immune-mediated syndromes such as peripheral neuropathy and myopathy or directly invade muscle fibers and cause myopathy [3]. The muscle involvement may vary from an asymptomatic elevation of CK

to severe Rhabdomyolysis secondary to antiviral therapy [4]. In acute viral Hepatitis, some patients develop myalgia and arthralgia, but muscle weakness rarely occurs. Concurrent elevation of liver enzymes with a background of myalgia and elevated CK levels should raise the suspicion of Acute Viral Hepatitis induced Polymyositis. Till date, only seven cases have been reported on this association. Deposition of Hepatitis B antigen antibody complexes in muscle suggests a possible etiology linking Hepatitis B to Polymyositis as the triggering antigen [5]. This is also supported by the study done by Ana Valle et al. which showed that Patients with HBV had a higher frequency of myositis-associated antibodies in comparison to patients without HBV [6]. Capasso et al. revealed that the pathogenesis of myopathy is an immune-mediated mechanism by demonstrating HBV DNA inside intact muscle fibers [7]. Newer MRI muscle imaging with Short Tau Inversion Recovery (STIR) sequence is a useful tool to demonstrate sub-clinical or overt viral myositis. These hyperintensities, although non specific, can be seen in muscle inflammation, edema or necrosis and need biopsy correlation [8]. Immunosuppressive therapy with Corticosteroid has been the backbone of treatment according to previous studies [2]. But the use of steroids before starting anti-viral therapy can result in significant immunosuppression and is considered harmful, even at low doses because of the risk of flare up of Hepatitis B infection. This was proven in a similar study where despite treatment with Entecavir for 10 weeks, the weakness persisted and improved only upon adding Prednisolone [9]. Therefore, patients with a combination of Hepatitis B presenting with Polymyositis should be treated with oral steroids as the cause is immune-related, But the treatment of Hepatitis B takes precedence over the treatment of Polymyositis.

### 4. CONCLUSION

Although Hepatitis B virus primarily affects hepatocytes, it sometimes develops disease manifestations outside the liver, such as myopathy. Our case adds on to these various extra-hepatic manifestations of Hepatitis B infection and focuses on the need of viral screening in a patient presenting with Polymyositis. Treatment for both Hepatitis B and Polymyositis is important as it has shown a favorable outcome, as demonstrated in our case. We declare no conflicts of interest.

## CONSENT

All authors declare that written informed consent was obtained from the patient for publication of this case report and accompanying images.

## ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

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We appreciate the patient who consented to the information reported in this article.

## COMPETING INTERESTS

Authors have declared that no competing interests exist.

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