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Gullian Barre Syndrome and Acute Hepatitis E: A Rare Association

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Abstract: ***Hepatitis E is a serious public health problem in developing countries. Most of the patients with Hepatitis E virus (HEV) infection present with typical acute hepatitis symptoms. However, in few patients it may lead to complications such as liver failure and extrahepatic symptoms. One of the rare extrahepatic presentations of this infection is neurological complications such as Guillain-Barré syndrome (GBS) which is observed in 5.5% of HEV infected patients (mainly in developed countries). Moreover, only genotype (gt) 3 HEV was found in association with GBS among patients in developed countries whereas molecular characterization of HEV cases detected from developing countries have not been reported till now.***

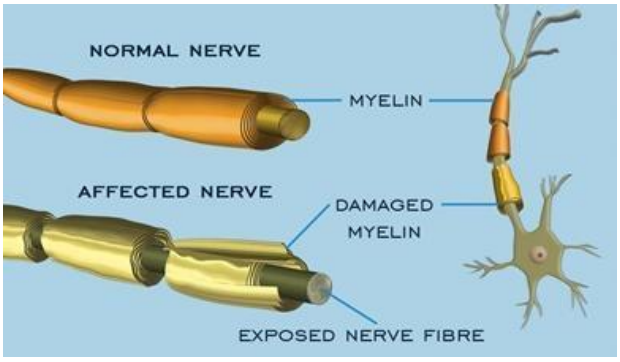
Keywords: Hepatitis E, public health, Guillain-Barre Syndrome

1.Introduction

GBS is rapid onset muscle weakness caused by the immune system damaging peripheral nervous system. Initial symptoms being muscle weakness in limbs, were developing over hours. Plasmapheresis and Intravenous Immunoglobulins are main Tx modalities. Hep A, B, C, and D may have GBS as occasional neurological complication, but is rare with HepE infection.

2.Case History

A 30 years old female developed jaundice since 5 days and had c/o anorexia and abdominal pain. After 3days, she developed bilateral sym, metrixal lower muscle weakness with normal upper limbs, cranial nerves, autonomic and higher functions. Gradually, power worsened in upper limbs and cranial nerves were also involved resulting in weak gag reflex. No history of fever, diarrhoea, blood transfusion, vaccination or medication therapy.



Clinical Examination

On examination, patient was icterus with anemia.

On abdominal examination, there was no ascities or organomegaly, with normal cardiorespiratory and respiratory system. All higher functions and cranial nerves were normal, upper limbs had 5/5 power but lower limbs power decreased to 1/5. Gradually, poer of upper limbs decreased, and by day-3 it became 3/5 with weak GAG reflex. Single breath count test was near normal (18) which gradually decreased to 12 by day-3.

Investigations, Tables, Charts, Radiological Image

CBC was normal. SGPT - 420 U/L

Total illirubin being 7.4mg/dl of which 5.8mg/dl was direct. antiHEVigM was REACTIVE by ELISA method.

HIV, HBsAg, HCV Non-Reactive.

CSF suggestive of 41.7mg% protein, 64% glucose, total cells 13 with 30% polymorph and 70% lymphocytes.

NERVE CONDUCTION STUDY suggestive of acute motor sensory generalized demyelinating and axonal axonal polyradiculopathy. Liver function improved by day-3, with SGPT 71 U/L and bilirubin, total being 3mg/dl and direct was 2.7mg/dl.

Diagnosis

From the above clinical features and laboratory diagnosis, we suspected Guillian-Barrè Syndrome which was conferred by Nerve Conduction Study (NCS) which was a complication of Hepatitis E.

Treatment

Patient was managed by 7 plasmapheresis cycles (40mg/kg wt) after 6th cycle, power of lower limbs improved to 3/5. Liver function test also returned to normal value by 10th day.

3.Discussion

Acute Viral Hepatitis is uncommonly associated with neurological conditions like: meningitis, encephalitis, GBS, myelitis, rediculitis, etc But, GBS has been reported with Hep A, B, C and D, but its association with HepE is rare. There is possibility of molecular mimicry between antigens of hepatotropic virus and components of mylein sheath of peripheral nerves.

4.Conclusion

GBS may follow unusual infections like HepE. Clinical symptoms are confirmed by NCS.

References

Walgraad C et al: prediction of respiratory insufficiency in Gillan Barrè Syndrome.67: 781, 2010. Kuif ML et al: J immunol 185: 748, 2010