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Case Report

Guillain –Barrè Syndrome following Hepatitis E

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Abstract:

Guillain- Barrè Syndrome is characterized by acute progressive symmetric limb weakness and areflexia. A 32 year old female presented with progressive ascending areflexic muscular weakness and bilateral lower motor neuron type of facial palsy. She had anorexia, nausea and upper abdominal pain for 2 weeks. The findings of motor nerve conduction study are consistent with acute inflammatory demyelinating polyradiculoneuropathy. She had elevated liver enzyme and positive immunoglobulin M antibody against hepatitis E in blood. Based on clinical features, laboratory findings and electrophysiological study, she was diagnosed as Guillain- Barrè Syndrome following hepatitis E. She was treated with intravenous immunoglobulin and recovered fully.

Key words: *Guillain- Barrè Syndrome, acute inflammatory demyelinating polyradiculoneuropathy, Hepatitis E.*

Introduction:

Guillain- Barrè Syndrome (GBS) is a post infectious, immune mediated disease targeting peripheral nerves. The annual incidence of GBS is 0.6-4 cases per 100000 throughout the world.[1] Up to two third of cases, an infection precedes the onset of neuropathy by 1 to 3 weeks. Campylobacter jejuni is the commonest and cytomegalovirus is the second most common infection reported.[2] There are many reports linking hepatitis A, B and C with GBS.[3,4,5] Association of GBS with hepatitis E is rare. Here we report a case of acute inflammatory demyelinating polyradiculoneuropathy (AIDP) variety of GBS following hepatitis E.

Case report:

A 32 year old female presented with progressive weakness of both lower and upper limbs for 3days. She had anorexia, nausea and right upper abdominal pain for last 2 weeks. There was no history of blood transfusion, needle injury, vaccination or drug intake. On examination, she had pulse -72/min, blood pressure 120/80 mm of Hg, respiratory rate 18/min and single breath count of 24. She was afebrile and mild icteric. Nervous system examination on day 1 revealed normal higher psychic function with right lower motor neuron type (LMN) facial palsy, muscle power 3/5 in lower limbs, 4/5 in upper limbs and planter response were bilateral equivocal. Deep tendon reflexes were absent in both limbs. On day 2, she developed difficulty of swallowing to both liquid and solid food , her muscle power reduced to 2/5 in both limbs, single breath count reduced to 18 and bilateral LMN type facial palsy. All sensory functions were intact and there was no sign of meningeal irritation. Abdominal examination revealed mild tenderness in right hypochondrium and 3cm enlarged tender liver from right costal margin.

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Laboratory investigation revealed normal complete blood count , renal function test, coagulation profile and arterial blood gas analysis. Liver enzyme were elevated- ALT 310 U/L, AST 280 U/L, Alkaline phosphatase 130 U/L, Gamma GT 147 U/L, serum Bilirubin 4.2mg/dl. Immunoglobulin antibody against hepatitis E was positive and markers of hepatitis A, B, C were absent. Ultrasound abdomen revealed mild hepatomegaly with altered echotexture of liver parenchyma suggestive of acute hepatitis. The findings of motor nerve conduction study are consistent with Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP). Cerebrospinal fluid analysis showed increase in protein concentration of 130mg/dl without an increase number of leucocyte. Clinical, serological and electrophysiological study suggested a diagnosis of AIDP variety of GBS with acute hepatitis E virus infection.

She was managed with intravenous immunoglobulin in the dose of 0.4gm/kg/day for 5days. She recovered muscle power of 4/5 within 7days. Liver enzymes and muscle power returned to normal after 2 weeks of follow up.

Discussion:

GBS is considered to be an autoimmune disease triggered by a preceding bacterial or viral infection. The most common agents are Campylobacter jejuni followed by Cytomegalovirus, Epstein-Barr virus, Mycoplasma pneumoniae, Haemophylus influenza, HIV, Influenza A and B, Varicella zoster virus, Clostridium and Shigella. Vaccine like polio, tetanus toxoid, hepatitis B have also been reported to cause GBS.[2]

The exact pathogenesis of hepatitis causing GBS is not known. It is thought that the immune system mistakenly attacks myelin or axon by a molecular mimicry mechanism.[6] There are four common subtypes of GBS based on clinical and electrophysiological studies e.g. 1. Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP), 2.Acute Motor Axonal Neuropathy (AMAN), 3.Acute Motor Sensory Axonal neuropathy (AMSAN), 4. Millar Fisher's syndrome.[6] Nerve conduction study in this case was suggestive of AIDP subtype.

Hepatitis E is a frequent cause of acute hepatitis in Asia, Middle east, North Africa and South America and locally acquired hepatitis E has becoming an emerging problems.[7] The actual incidence of GBS with hepatitis E is still unknown because autochthonous hepatitis E is still underdiagnosed.[8]

Management of GBS is multidisciplinary. Both plasma exchange and intravenous immunoglobulin are equally effective in reducing disease severity and neurological deficits.[9]Our patient was managed with intravenous immunoglobulin and she recovered fully.

Our patient had the AIDP variety of GBS following acute hepatitis E infection. AIDP is characterized by segmental demyelination and subsequent remyelination associated with good recovery.[2]Determination of causative agent is important to assess prognosis of GBS subtype. Considering the GBS-HEV cases reported, it can be said that HEV testing should be done in patients of GBS with altered liver function.

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