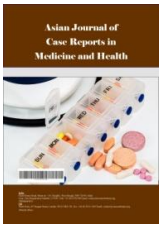

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Guillain-Barre Syndrome Associated with Acute Hepatitis A Infection: A Case Report

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Authors' contributions

This work was carried out in collaboration among all authors. Author MM designed the study, performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Authors RK and YF managed the analyses of the study. Author YF managed the literature searches. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Background and Aim: Guillain-Barré syndrome (GBS) is considered as an inflammatory postmanifestation of infectious, which acute ascending motor weakness, hyporeflexia or areflexia and sometimes sensory loss are hallmarks of this clinical syndrome. It is an acute polyradiculoneuropathy often provoked by an infectious agent. It is an acute polyradiculoneuropathy often provoked by an infectious agent, but its development following acute hepatitis A has rarely been reported.
Case Presentation: The authors report a 35 years old female of Guillain-Barre syndrome following acute hepatitis A in a young female, that developed symmetrically ascending motor weakness two weeks after the onset of initial symptoms of hepatitis infection. Our case presented with high-grade fever, vomiting and jaundice. The diagnosis of GBS was made according to clinical manifestations and albuminocytologic dissociation in cerebrospinal fluid. The patient was transferred in the ICU, NGT fixed, and plasmapheresis started. Patient's neurological condition improved after 8 times

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plasmapheresis every 48 h after seven day in ICU. Treatment outcome was satisfactory and her facial palsy had almost completely improved.

Conclusion: Although GBS due to hepatitis A is very rar, this complication should be kept in mind and the best way to prevent Hepatitis A through vaccination and personal hygiene especially in close contact with an infected household member, beside good environmental sanitation.

Keywords: Acute hepatitis; flaccid paralysis; hyporeflexia; infection; Guillain-Barré syndrome; weakness.

1. INTRODUCTION

Guillain-Barré syndrome (GBS) is an inflammatory manifestation of infectious and immune-mediated polyradiculoneuropathy, which often provoked by an infectious agent. However, its development following acute hepatitis A has rarely been reported.

Acute ascending motor weakness, hyporeflexia or areflexia and sometimes sensory loss are hallmarks of this clinical syndrome. GBS is considered as a neurology emergency and currently the most common cause of acute flaccid paralysis [1]. In a large number of cases 1-3 weeks after a viral infection or immunization, neurological symptoms appear [2]. Hepatitis B, C, and E are recognized to be triggering viruses for GBS in previous studies [3]. However, its accompaniment with hepatitis A wasn't proven yet. Here a rar case of GBS due to hepatitis A is presented.

2. CASE REPORT

A 35 years old female presented with high-grade fever, vomiting, and jaundice. One week before her admission she was evaluated at an outpatient clinic and diagnosed to have acute hepatitis with serum bilirubin of 8.9 mg/dl (0.1-1.2), aspartate transaminase of 1200 IU/L (up to 31 Iu/L), alanine transaminase of 1080 IU/L (>41 IU/l). There were no attributes of hepatic decompensation. From 15 days before her admission, she had presented with the flu-like syndrome, abdominal pain, light color stool, dark urine, loss of appetite, unexplained weight loss and jaundice. Her husband presented with acutehepatitis from one monthbefore without any complication.

On this admission physical examination revealed blood pressure: 120/80 mmHg, heart rate: 82/min rhythmic, respiratoryrate: 20/min, temperature 38.5°C, and the patient was icterus. Blood investigations revealed total bilirubin of 8.9 mg/dl (direct 7.9 mg/dl), aspartate transaminase of 200 IU/L, alanine transaminase of 560 IU/L; Creatine phosphokinase (CPK) level was normal; Renal functions and coagulation parameters werenormal. A routine blood test showed usual electrolytes, glucose, and hematological parameters; Hepatitis B surface antigen, antibodies to hepatitis C, IgM anti-HEV and anti-HIV were absent. IgM Hepatitis A Virus Antibody was positive. Her vasculitis markers and autoimmune hepatitis markers were negative. Ultrasonography of the abdomen revealed mild hepatomegaly. On presentation, she had jaundice and hepatomegaly without any features of hepatic encephalopathy.

She was conscious, well oriented, stabilization and obeyed verbal commands. She was given symptomatic treatment. (therapy generally supportive on the first day: Hydration and antiemetics was used as supportive treatment. Nausea and vomiting was treated with antiemetics. Dehydration managed with hospital admission and intravenous (IV) fluids and administered medications with care.

After oneday admission, paraesthesia in hands and feet developed, and therefore, Neurological consult wasdone. Brain and lumbosacral MRI and lumbar puncture were requested.The MRI was normal. Cerebrospinal fluid revealed protein: 80 mg/dl, sugar: 63 mg/dl, with no cells and normalpressure that was in concordance with albuminocytologic dissociation. Cytology of CSF andPCR for HSV (herpes simplex virus) and VZV (varicella zoster virus) were negative. Nerve conduction velocities in peripheral nerves were prolonged with slightly decreased axonal amplitudes in electromyography (EMG) Also, NMO (neuromyelitisoptica antibody) and OCB (oligoclonal bands) were negative.

The next day, rapidly progressive ascending paresis in all four limbs developed (ascending flaccid quadriparesis). Her neurological examination revealed bifacial lower motor neuron weakness, generalized hypotonia with areflexia, neck muscle weakness, and upper and lower limb power at Medical Research Council grade

(MRC) 2 with distal more than proximal weakness. Pathologic reflexes, such as the Babinski sign, were absent.

A diagnosis of Guillain-Barre syndrome was made based on progressive rapidly ascending flaccid are flexicquadriparesis. Intravenous immunoglobulins were given at a dose of 0.4 g/kg/day for five days. After twodays, she also developed difficulty in speaking and swallowing (bulbar dysfunction) andfacial palsy (more pronounced on the right side).

As such, the patient was transferred in the ICU, NGT fixed, and plasmapheresis started. Patient's neurological condition improved after 8 times plasmapheresis every 48 h after seven day in ICU. Her facial palsy had almost completely improved. Her quadriparesis also resolved and she was able to walk with support at the end of 3 weeks after initiation of GBS treatment and at the end of next week, she could walk without any support. AST, ALT, and bilirubin levels were normalized within 3 weeks (See Fig. 1).

3. DISCUSSION

The most commonly reported triggering pathogen for GBS are *Campylobacter jejuni*, cytomegalovirus, *Mycoplasma pneumoniae*, and Epstein-Barr virus. Other known infectious agents include HIV, shigella, clostridium and Haemophilus influenza [4,5]. Probability, GBS has been associated with acute viral hepatitis B, C, D, E and rarely with hepatitis [6]. It has been shown that the epitopes expressed by infected cells in infections such as CMV, EBV and *Campylobacter jejuni* can lead to the generation of anti-ganglioside antibodies. These antibodies cause severe axonal degeneration and development of GBS manifestations [3,7-10].

The pathogenesis of GBS with hepatitis is unknown, but it is considered that direct cytotoxicity of the virus could be a probable explanation [2,11]. Also, it is supposed that induced immune response and immune cross-reaction (because of sameness between hepatitis A epitopes and myelin structures of

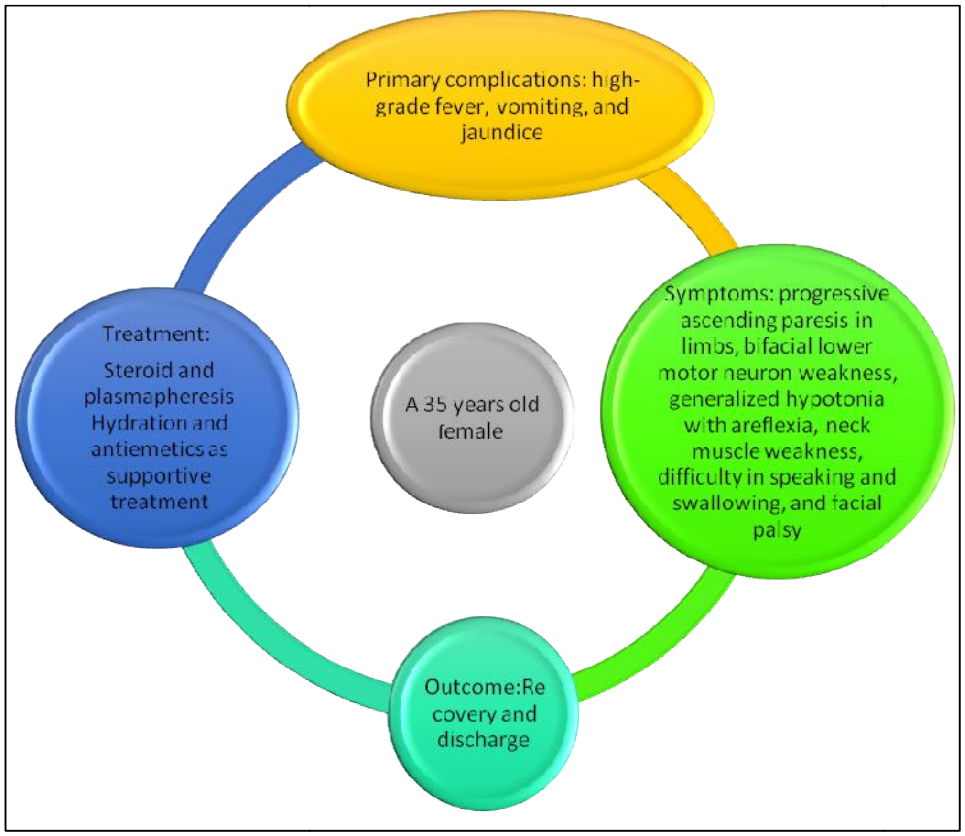

Fig. 1. Guillain-Barre syndrome associated with acute hepatitis

peripheral nerves) could be responsible for this entity [12].

Previous studies have shown that GBS symptoms usually develop about 3-14 days after the onset of acute hepatitis symptoms, which is the same as in our patient [11,13]. GBS following hepatitis A usually occurs in men more than women and facial nerve palsy, proprioception, and vibratory sense impairment, and decreased superficial skin-sensation is a common presentation in these patients [11].

Intravenous immunoglobulin (IVIg) and plasma exchange (PE) are two treatment choices in GBS patients and these therapy methods can be also used for GBS patients with hepatitis. On the other hand, it seems that steroids alone are not so much effective. IVIg has a lower risk for hepatic side effects and it seems to be more effective in GBS patients with hepatitis, so it is the preferred treatment in these patients [14]. GBS following hepatitis A (unlike *campylobacter jejuni*) often has a favorable prognosis and almost all patients are cured [6,11], as what we report in the present study.

Hepatitis A is a contagious liver infection that usually spread through close personal contact orby consuming contaminated food or water. In the past, due to the large population, low socioeconomic status, and inadequate clean water, high endemicity of HAV was seen in developing countries [15,16]. A person with Hepatitis A is most infectious during the 1 to 2 weeks before the onset of symptoms until at least 1 week after the onset of jaundice [2,8]. In the history of our patient, her husband presented with acutehepatitis Afrom one monthbefore her admission. Hence, hepatitis A vaccination could have been considered.

4. CONCLUSION

Although only a few cases of GBS due tohepatitis A have been reported, this complication should be kept in mind and the best way to prevent Hepatitis A is through vaccination and personal hygiene especially in close contact with an infected household member, beside good environmental sanitation.

CONSENT

As per international standard or university standard, patient's written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

All procedures were conducted in accordance with the ethical committee of Mashhad University of Medical Sciences.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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