Significance of Transaminitis in Guillian Barre Syndrome

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ABSTRACT

Guillian Barre Syndrome (GBS) is an immune-mediated disorder with a wide variety of predisposing factors and varied clinical manifestations. In this case, we report a 19-year-old male presenting with GBS of AIDP type associated with transiently elevated liver enzymes (AST/ALT) about 4-5 times above baseline that lasted for 1-2 weeks and start declining towards baseline after 2 weeks. We conclude that the majority of the time no cause can be attributable to such liver function disturbances (LFD). Therefore, treatment for GBS might be continued despite having LFD as this phenomenon is transient and does not interfere with treatment of GBS.

Introduction

Guillain-Barre syndrome (GBS) is an acute inflammatory immune-mediated polyradiculoneuropathy presenting typically with tingling, progressive weakness, pain and sometimes respiratory difficulties. According to recent epidemiology, the incidence of GBS ranges between 0.81 and 1.89 (median 1.11) cases per 100,000 person-years [1]. The clinical course, severity, and outcomes of GBS are highly variable.

Serum aspartate aminotransferase (AST) and serum alanine aminotransferase (ALT) are liver enzymes where increased levels usually indicate hepatocyte injury. Both the parameters are increased in a wide range of conditions that affect the liver including infections, malignancy, trauma and excess alcohol consumption. In this report, we discuss a unique case of GBS having elevated AST and ALT following admission to the hospital, its possible causes and influence on treatment of GBS.

Case

A 19-year-old gentleman was admitted to the hospital for bilateral lower limb weakness followed by left upper limb weakness. Electrophysiological studies showed early GBS of AIDP type. The patient was started on five doses of plasma exchange over 10 days. He denied any nausea, vomiting, coffee-ground emesis, jaundice, clay-colored stools, melena or hematochezia. He has no history of heavy alcohol abuse, IV drug intake, recent immunization or travel outside the country. He denied any family history of liver disease. On day 0 of admission his AST/ALT was 49/53 which was slightly above the upper limit (Normal 0-40/10-50) with an increasing trend until day 5 (154/240), which was about 4-5 times the upper limit and then started showing a decline (Figure 1A, 1B). His total bilirubin, PTT/PT/INR, total protein, albumin, ALP, fibrinogen, ceruloplasmin levels were in the normal range throughout. His viral panel (HAV IgM, HBcAb IgM, HBsAg, HCV A, CMV, EBV, autoimmune hepatitis workup), fluorescent anti-nuclear antibody (FANA), alpha 1 anti-trypsin, anti-smooth muscle Ab, and anti-mitochondrial Ab were negative. His abdominal ultrasound was unremarkable for any liver pathology. His liver function test was down trending and was completely normal when evaluated in his next clinic follow up after two months.

Discussion

As the pathophysiology of GBS would have started before hospital admission, this liver function disturbance can still be a part of GBS. There are a wide range of causes that can precede GBS and cause LFD like cytomegalovirus (CMV), Epstein–Barr virus (EBV), hepatitis (A, B, and E), and several other bacterial and viral infections. Vaccines

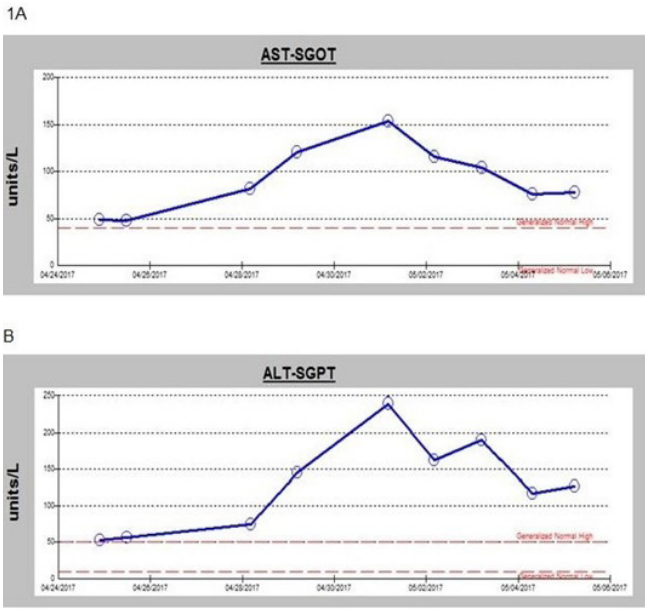

Figure 1. Trend of AST/ALT over the course of admission to discharge.

such as hepatitis A and B, yellow fever, influenza A, H1N1, and MMR have also been considered as a possible trigger of GBS [1] and LFD [2,3].

So far, there have been no extensive studies of LFD in GBS. The most likely explanation is immune-mediated liver dysfunction along with the peripheral nervous system dysfunction that occurs in GBS [4,5,6]. This could explain his slightly elevated ALT/AST at the time of admission and that the course of elevation of the enzymes is likely to be independent of the course of GBS. Another possible explanation for the presence of LFD would be that of infectious agents like hepatitis A and B. These infections occur more often than is reported because of high false-negative serologic tests. Though the level of transaminases was >2.5 times in this case, the LFD was of short duration; the peak was within one week after hospitalization and hence less likely to be hepatitis considering the duration [7]. Certain bacterial endotoxins can cause LFD with a common cause being Campylobacter jejuni. [6,8]. Although numerous factors might cause LFD in GBS, most of the time extensive investigations may not yield any reasonable cause. Oomes *et. al* [6] performed a prospective longitudinal study of measuring liver function in 100 patients. They found an increase in patients with LFD in the IVIG treatment group while no LFD was seen in the plasma exchange treatment group. After about one month, the significant difference in LFD had disappeared and by six months the percentage of patients with LFD was significantly lower in both IVIG and plasma exchange groups. It was determined that transient liver dysfunction was through an unknown mechanism. However, removal of plasma in plasma exchange may reduce the transient elevation of liver enzymes that might have been seen if the patients had not undergone this therapy and therefore LFD in IVIG as compared to plasma exchange could be artifactual. [9]

The course of the LFD seems to be benign and asymptomatic, increasing rapidly for about a week and declining thereafter. Most of the time this does not warrant stopping IVIG or plasma exchange. Other causes affecting LFD in GBS as mentioned above should be kept in mind and evaluated if there is symptomatic elevation of AST/ALT or if there is persistent elevation for >2 weeks.

Conclusion

GBS is a heterogeneous condition with numerous clinical associations and various inciting factors. LFD is one such association in which liver enzymes AST/ALT are transiently elevated that parallel the course of GBS without any cause attributed in the majority of cases. Therefore, treatment for GBS might be continued and the cause for these disturbances evaluated when it is persistently high or symptomatic.

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