A Novel Therapeutic Approach: Managing Severe Alcoholic Hepatitis Complicated by Guillain-Barré Syndrome (AMSAN Subtype) using Multidisciplinary Precision Care

Chetan Kalal[1*]

[1]Department of Hepatology and Transplant Medicine Max Hospital, Mumbai

Correspondence:

Chetan Kalal

E-mail: chetan.kalal@nanavatimaxhospital.org

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Abstract:

We report the first documented case of a 41-year-old male diagnosed with severe alcoholic hepatitis (AH) complicated by Guillain-Barré syndrome (GBS), specifically the acute motor and sensory axonal neuropathy (AMSAN) variant. The combination of these two life-threatening conditions posed unique challenges, especially given the conflicting immunosuppressive and neuroinflammatory therapeutic demands. This case highlights a pioneering multidisciplinary approach, utilising corticosteroid therapy for AH alongside plasmapheresis (PLEX) for GBS. The patient showed significant improvement in both hepatic and neurological functions, underscoring the critical role of personalised, integrative care in managing complex comorbidities. This case sets a precedent for future clinical protocols in critical care hepatology and neurology.

Key words: Alcoholic Hepatitis, Guillain-Barré Syndrome (GBS), Plasmapheresis (PLEX), Dual Pathology Management.

Introduction

Severe alcoholic hepatitis (AH) is a serious and life-threatening condition often encountered in chronic alcohol abusers, characterised by jaundice, liver failure, and intense systemic inflammation. Prompt management with corticosteroids has been the standard approach to improving outcomes.[1] However, the situation becomes exponentially more challenging when severe AH occurs simultaneously with a rare autoimmune neuropathy like Guillain-Barré Syndrome (GBS), particularly the acute motor and sensory axonal neuropathy (AMSAN) subtype, which is known for its aggressive progression and poor prognosis.[2]

This report presents the first known case of a patient diagnosed with both severe AH and GBS (AMSAN subtype), demanding a novel therapeutic approach. A multidisciplinary team employed a strategy that integrated corticosteroids for AH management with plasmapheresis (PLEX) to address the autoimmune demands of GBS, resulting in favourable patient outcomes. This case emphasises the importance of innovative and integrative care in managing such complex cases in the critical care setting.

Case Report

A 41-year-old male with a history of heavy alcohol consumption (approximately 60 grams of alcohol daily for 10 years) presented with a two-week history of jaundice, nausea, vomiting, and abdominal pain. He had no prior medical history of liver disease.

Clinical findings and laboratory results:

On examination, the patient appeared jaundiced with hepatomegaly. His initial laboratory workup revealed: haemoglobin (Hb): 9.5g/dL, total leukocyte count (TLC): 4800/µL, total bilirubin: 9.2mg/dL (direct: 8.2mg/dL), aspartate aminotransferase (AST): 160U/L, alanine aminotransferase (ALT): 78U/L, international normalised ratio (INR): 4.7, Maddrey Discriminant Function (DF): 184, and Model for End-Stage Liver Disease-Sodium (MELD-Na) Score: 34.

Imaging findings:

Abdominal ultrasound showed hepatomegaly with fatty infiltration and coarse echotexture. Upper gastrointestinal (GI) endoscopy revealed oesophageal candidiasis, but no varices.

Based on these findings, the patient was diagnosed with severe AH. Corticosteroid therapy (40 mg of prednisolone daily) was initiated after ruling out sepsis. He was also started on empirical antibiotics and aggressive nutritional support. By Day 7, a Lille score of 0.01 indicated a favourable response to steroids.

Development of Guillain-Barré Syndrome (GBS)

By Day 10, despite improving liver function, the patient developed new neurological symptoms, including bilateral lower limb weakness, difficulty walking, ataxia, dysarthria with slurred speech, and paraesthesia in the hands and feet.

Neurological examination showed absent deep tendon reflexes, proximal muscle weakness, and coordination deficits. The workup confirmed GBS (AMSAN subtype) with the following findings:

- Cerebrospinal fluid (CSF) analysis showed albumino-cytological dissociation
- Nerve conduction studies revealed conduction block and absent reflexes.
- Brain magnetic resonance imaging (MRI) reported incidental non-haemorrhagic infarct.

The simultaneous onset of GBS in a patient on corticosteroids for AH presented a therapeutic dilemma, as immunosuppression for AH could exacerbate the autoimmune nature of GBS.

Management Strategy: Multidisciplinary Precision Care

The decision was made to initiate PLEX while continuing corticosteroid therapy for AH. The multidisciplinary team, involving hepatologists, neurologists, and critical care specialists, carefully monitored and balanced both conditions.

Plasmapheresis protocol:

The patient underwent seven sessions of PLEX over two weeks, resulting in significant improvement in neurological function, while steroid therapy for AH continued with close monitoring. Remarkable clinical outcomes included neurological improvement with resolution of ataxia, dysarthria, and proximal muscle weakness. Liver function also improved, with bilirubin levels dropping from 9.2mg/dL to 1.7mg/dL by the time of discharge.

Rehabilitation:

Early initiation of physiotherapy and speech therapy further supported the patient's recovery.

Outcome and follow-up:

After four weeks, the patient showed substantial improvement with a bilirubin of 1.7mg/dL, AST/ALT: 237/137 U/L and INR: 1.29. Full neurological recovery, including resolution of ataxia and dysphagia was seen.

The patient was discharged on a follow-up plan that included tapering steroids for AH and periodic neurological evaluations. By the latest follow-up, his liver function tests were normal, and he showed full recovery from the neurological deficits.

Discussion

This case represents a ground-breaking example of managing dual crises-severe and GBS (AMSAN subtype)—using an integrative and multidisciplinary therapeutic approach. The convergence of these two critical conditions posed unique challenges due to their overlapping yet conflicting treatment modalities. The strategic use of plasmapheresis, not only as a treatment for GBS but also as a modulator of systemic inflammation, highlights its potential role in managing severe AH[3,4].

The favourable outcome in this case demonstrates the effectiveness of innovative precision care and underscores the importance of timely intervention and coordinated multidisciplinary care in managing complex and rare medical presentations.

Conclusion

This case illustrates how innovative and multidisciplinary therapeutic approaches can successfully manage complex and overlapping conditions such as severe AH complicated by GBS. The combined use of corticosteroids and PLEX represents a novel therapeutic strategy, offering new insights for clinical management. This report also sets the stage for further research into integrative care models for treating intersecting pathologies in hepatology and neurology.

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