Guillain-Barre Syndrome Associated with Hepatocellular Carcinoma

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ABSTRACT:

This case study presents a rare association of Guillain-Barre Syndrome (GBS) Hepatocellular Carcinoma (HCC). The patient exhibited AFP-positive sensorimotor neuropathy, with negative lab findings for HIV, HCV, HBsAg, and VDRL. Despite initial treatment for GBS with IVIg, the patient showed limited improvement, prompting further investigation. Subsequent imaging revealed hepatocellular carcinoma. The patient was treated Lenvatinib, showing partial neurological recovery. This case highlights the importance of malignancy screening in atypical GBS presentations.

KEYWORDS:

Guillain-Barre Syndrome, Hepatocellular Carcinoma, AFP, Sensorimotor Neuropathy.

I. INTRODUCTION

Hepatocellular carcinoma (HCC) is the most common primary liver malignancy. While HCC often presents with hepatic symptoms, paraneoplastic neurological syndromes such as Guillain-Barre Syndrome (GBS) are rare. Chronic alcohol use is a major risk factor for HCC, contributing to liver damage and carcinogenesis. This case highlights a rare occurrence of AFP-positive sensorimotor neuropathy secondary to HCC.

II. CASE PRESENTATION

A 70-year-old male with a 20-year history of chronic alcohol use presented with sudden-onset bilateral lower limb weakness, numbness, and intentional tremors. Neurological examination revealed diminished deep tendon reflexes and mild

sensory loss. Nerve conduction studies confirmed sensorimotor mixed neuropathy, consistent with GBS. Despite treatment with IVIg, the patient's symptoms persisted.

Further evaluation revealed elevated alphafetoprotein (AFP) (520 ng/mL) and PIVKA-II (2,505 mAU/mL) levels. Imaging (CT and MRI) showed multiple heterogeneous enhancing lesions in the right lobe, consistent with hepatocellular carcinoma. Endoscopy findings were normal. Renal function tests (RFT) were within the normal range. The patient was started on Lenvatinib (4 mg/day). While mobility improved after treatment, intentional tremors persisted, suggesting ongoing neurological involvement.

III. DISCUSSION

This case underscores the diagnostic challenge of paraneoplastic neuropathy mimicking GBS. AFP-positive sensorimotor neuropathy has been documented in association with malignancies, particularly HCC. Elevated AFP and PIVKA-II levels supported the diagnosis. This case emphasizes the need for malignancy screening in patients with atypical or treatment-resistant GBS.

IV. CONCLUSION

In elderly patients with chronic alcohol use and atypical neurological presentations, malignancy screening should be considered. Early recognition and targeted treatment can improve outcomes in such cases.

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