Acta Scientific Neurology (ISSN: 2582-1121)

Volume 4 Issue 5 May 2021

Case Report

A Rare Presentation of Liver Cirrhosis

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Received: February 04, 2021

Published: April 12, 2021

Abstract

A 56 year old male patient presented with slowness of activities, gait disturbance and altered behaviour for four weeks. Patient had been diagnosed as a case of alcoholic liver disease for two years. Neurologically, he had rigidity, severe bradykinesia with short stepping gait while walking. Following investigations, patient was diagnosed as a case of alcoholic liver disease with hepatic encephalopathy. He was treated with appropriate diet, laxatives and low dose levodopa therapy. He improved over the next few days as his blood ammonia levels also fell. Parkinsonism is a rare presentation of liver cirrhosis with hepatic encephalopathy.

Keywords: Hepatic Encephalopathy; Cirrhosis; Parkinsonism

Introduction

Hepatic encephalopathy is a known complication of liver cirrhosis. It generally presents with altered sleep wake cycle and behavioural changes in early stage.

Case Report

A 56 year old male patient presented with slowness of activities, gait disturbance and altered behaviour for four weeks. The altered behaviour was in the form irritability and agitation. Patient had been diagnosed as a case of alcoholic liver disease for two years. He had not been consuming alcohol since then. On physical examination, he had mild icterus in eyes, and leukonychia. There were no corneal deposits. Neurologically, his higher mental functions were normal on detailed formal testing. He had hypomimia, hypophonia and micrographia. He had lead pipe rigidity in all four limbs without any involuntary movements. He had severe bradykinesia with short stepping gait while walking. Rest of the neurological examination was unremarkable. His routine blood investigations showed haemoglobin - 13.2 gm/dl, total bilirubin - 1.5 mg/dl, direct bilirubin - 0.8 mg/dl, aspartate aminotransferase - 68 U/L, alanine aminotransferase - 56 U/L, alkaline phosphatase - 74 U/L, serum albumin - 2.4 gm/dl, prothrombin time - 16 seconds, International Normalised Ratio (INR) - 1.4 and blood ammonia - 184 micromol/L (normal range - 18-40). Serum ceruloplasmin and urinary copper levels were normal. HBsAg and anti HCV antibody were negative. The abdominal ultrasonogram showed a shrunken liver with coarse echotexture, mild splenomegaly with mild free intraperitoneal fluid. The Magnetic Resonance Imaging (MRI) of brain and electroencephalogram were unremarkable. His cerebrospinal fluid examination was also normal. Patient was diagnosed as a case of alcoholic liver disease with hepatic encephalopathy. Patient was treated with appropriate diet, laxatives and low dose levodopa therapy. He improved over the next few days as his blood ammonia levels also fell. He became less irritable and rigidity and bradykinesia also partly improved. He was later discharged in stable condition to follow up in out-patient department.

Discussion

Parkinsonism is a rare presentation of liver cirrhosis with hepatic encephalopathy. Burkhard *et al* had reported parkinsonism

in patients with cirrhosis, recruited from a hepatic transplantation unit [1]. All patients had progressive symmetric parkinsonism without apparent cognitive impairment like in our case. Though, unlike our case, these patients had basal ganglia hyperintensities in MRI brain. Such changes had been attributed to manganese deposition [2,3].

Conclusion

We conclude that liver cirrhosis should be suspected in a patient presenting with rapidly progressive symmetric parkinsonism with or without cognitive impairment. Patients benefit with anti hepatic coma measures and levo-dopa therapy. Patients, who are eligible, should be offered liver transplantation as definite therapy.

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