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Case Report Open Access

Think Twice Before Labelling Hepatic Encephalopathy

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1. Abstract

1.1. Introduction

It is well said and proven in literature that important causes of cirrhosis of liver include alcohol intake, metabolic associated fatty liver disease and chronic hepatitis B & C. Less important causes include autoimmune & congenital liver disease, Wilson's and alpha1 antitrypsin deficiency disease. There can occur gradual progression from chronic hepatitis to cirrhosis and in certain cases can further reach stage of hepatocellular carcinoma. Hepatic encephalopathy is common complication of cirrhosis or end stage liver disease but there can be many other reasons of altered behaviour in these patients which have to be evaluated very carefully.

1.2. Case Report

We report a eighty-two-year-old female, a known case of hypertension and diabetes mellitus for last ten years and was controlled on oral hypoglycaemic and anti-hypertensive drugs. She presented with short duration fever which was associated with rigor & chills, along with anorexia, pain abdomen and diarrhoea. She gave history of fall fifteen days back and was on bed for last one week after development of fever episode. She was seen by some private practitioner who advised for ultrasonogram abdomen which revealed altered liver echotexture with abscess in right lobe. She was admitted in private hospital where complete hemogram revealed leucocytosis, thrombocytopenia and iron deficiency anemia. She developed hypotension and anuria, despite replenishment of intravenous fluids. Hence, she was referred to our center with probable diagnosis of hepatic encephalopathy or septic shock. She was started on low dose inotropic support, broad spectrum antibiotics, insulin for control of raised blood sugar and intravenous albumin for severe hypoalbuminemia. At this point of time, her ultrasonogram again showed slight altered echotexture of liver with abscess in right lobe, leucocytosis, thrombocytopenia, anemia, deranged renal function test, raised transaminases, hyperbilirubinemia, hypoproteinaemia and hypoalbunemia. She responded to treatment and inotropic support was removed within 36 hours and urinary output improved. Her sensorium still remained deranged and she had excessive sleepiness and even used to doze off while talking and taking food. She was even started on rifaximin thinking it to be hepatic encephalopathy. The detailed neurological examination, revealed right sided hemiparesis with right plantar being equivocal. Hence, in view of deranged renal function tests, she was subjected to MRI brain and abdomen which showed hepatomegaly with right lobe liver abscess in segment V1 and V11 with mild ascites and bilateral pleural effusion. The MRI brain showed subacute lacunar infarcts in bilateral semiovale, cortical aspect of bilateral frontoparietal and temporo-occipital lobes and posterior aspect of bilateral cerebellar hemispheres-likely in watershed territory of bilateral middle carotid and internal carotid artery, most likely embolic in nature. A chronic infarct was seen in left cerebellar hemisphere on posterior-inferior aspect. Age related mild diffuse cerebral cortical atrophy with chronic small vessel ischemia changes (Fazeka grade 11/111). Chronic microhaemorrhages in thalamoganglionic region of external capsule, right pons, cortical aspect of right frontal, bilateral parietal lobes- parasagittal aspect and right cerebellar hemisphere-likely hypertensive aetiology. She was shown to neurologist, who started her on low dose single antiplatelet ecosprin 75 mg, in view of thrombocytopenia. She partially responded to treatment and her sensorium improved within three days, accepted orally well, became haemodynamically stable with normal urinary output. Hence, she was discharged on OHA, insulin, antiplatelets and iron & multivitamins.

1.3. Conclusion

Cirrhosis diagnosis should not be made just on basis of ultrasonogram abdomen, as it is operator dependent. Moreover, every altered behaviour, even in cirrhotic patient should not be always labelled as hepatic encephalopathy and other causes, especially neurological, must be considered and ruled out.

2. Introduction

The data pertaining to India regarding various clinical aspects of chronic liver disease (CLD) like aetiology, natural history, clinical presentation, treatment recommendations and its effect of public health, is limited [1-3]. Moreover, in comparison to developed countries, the burden of morbidity and mortality has not been analysed in depth in developing countries like India [3-5] which is essential for determining the status of country's public health system [6-7]. The analysis of disease burden in particular geographical area helps in planning cost effective control measures [7]. The lack of exact data is detrimental in development of effective policies which can help in taming the menace of such deadly diseases and decrease the need of liver transplant [7]. The government bears huge expenditure on CLD patients which can be reduced substantially by investing on preventive strategies for developing chronic liver disease [8]. Patients with cirrhosis face an increased risk of hepatic encephalopathy but there can be many other reasons for altered sensorium like septicaemia, intracranial haemorrhage (ICH) or infarct. Clinically, it can be challenging to distinguish them from hepatic encephalopathy, necessitating careful evaluation for timely management to decrease morbidity and mortality.

3. Case Report

We report a eighty-two-year-old female, a known case of hypertension and diabetes mellitus for last ten years and was controlled on oral hypoglycaemic and anti-hypertensive drugs. She presented with short duration fever which was associated with rigor & chills, along with anorexia, pain abdomen and diarrhoea. She gave history of fall fifteen days back and was on bed for last one week after development of fever episode. She was seen by some private practitioner who advised for ultrasonogram abdomen which revealed altered liver echotexture with abscess in right lobe. She was admitted in private hospital where complete hemogram revealed leucocytosis, thrombocytopenia and iron deficiency anemia. She developed hypotension and anuria, despite replenishment of intravenous fluids. Hence, she was referred to our center with probable diagnosis of hepatic encephalopathy or septic shock. She was started on low dose inotropic support, broad spectrum antibiotics, insulin for control of raised blood sugar and intravenous albumin for severe hypoalbuminemia. At this point of time, her ultrasonogram again showed slight altered echotexture of liver with abscess in right lobe, leucocytosis, thrombocytopenia, anemia, deranged renal function test, raised transaminases, hyperbilirubinemia, hypoproteinaemia and hemoglobinemia. She responded to treatment and inotropic support was removed within 36 hours and urinary output improved. Her sensorium still remained deranged and she had excessive sleepiness and even used to doze off while talking and taking food. She was even started on rifaximin thinking it to be hepatic encephalopathy. The detailed neurological examination, revealed right sided hemiparesis with right plantar being equivocal. Hence, in view of deranged renal function tests, she was subjected to MRI brain and abdomen which showed hepatomegaly with right lobe liver abscess but MRI brain showed which showed hepatomegaly with right lobe liver abscess in segment V1 and V11 with mild ascites and bilateral pleural effusion. The MRI brain showed subacute lacunar infarcts in bilateral semi vale, cortical aspect of bilateral frontoparietal and temporo-occipital lobes and posterior aspect of bilateral cerebellar hemispheres-likely in watershed territory of bilateral middle carotid and internal carotid artery, most likely embolic in nature. A chronic infarct was seen in left cerebellar hemisphere on posterior-inferior aspect. Age related mild diffuse cerebral cortical atrophy with chronic small vessel ischemia changes (Fazeka grade 11/111). Chronic microhaemorrhages in thalamoganglionic region of external capsule, right pons, cortical aspect of right frontal, bilateral parietal lobes- parasagittal aspect and right cerebellar hemisphere-likely hypertensive aetiology. She was shown to neurologist, who started her on low dose single antiplatelet ecosprin 75 mg, in view of thrombocytopenia. She partially responded to treatment and her sensorium improved within three days, accepted orally well, became haemodynamically stable with normal urinary output. Hence, she was discharged on OHA, insulin, antiplatelets and iron & multivitamins.

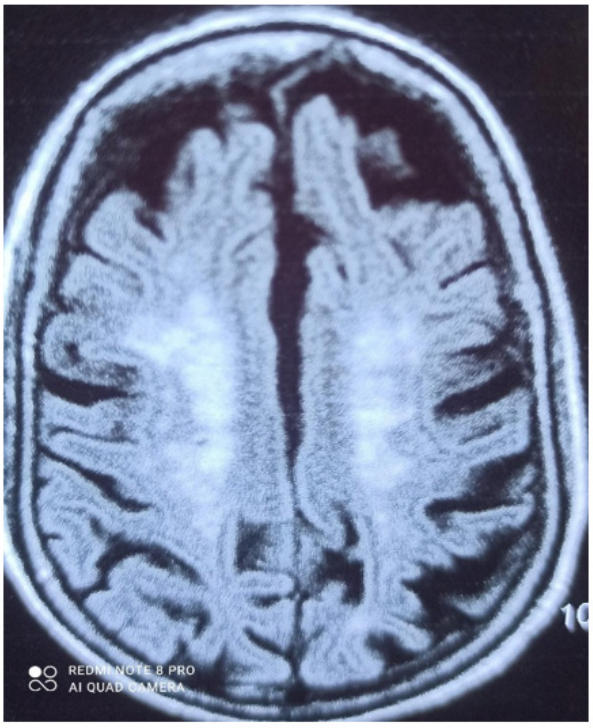

Figure 1: MRI Brain showing white matter bilateral changes.

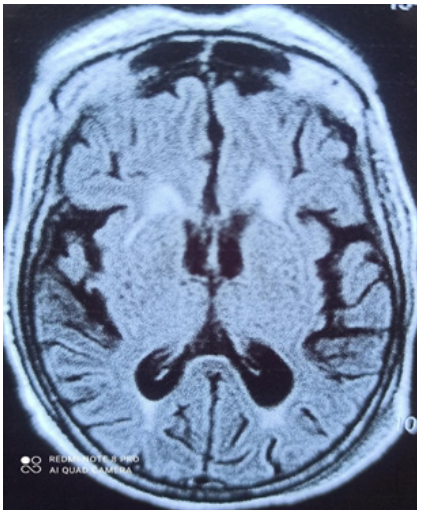

Figure 2: MRI brain showing multiple lacunar infarcts.

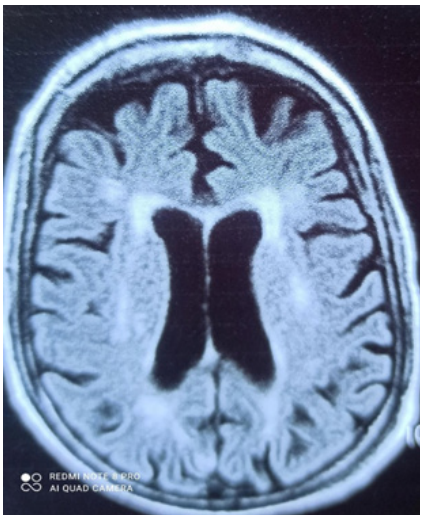

Figure 3: MRI Brain showing multiple Bilateral lacunar infarcts.

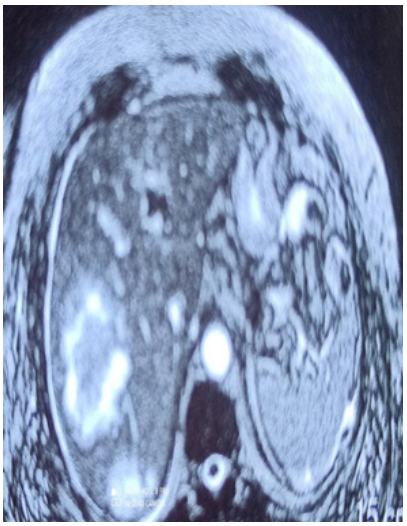

Figure 4: MRI Abdomen showing liver abscess in right lobe.

4. Discussion

Liver disease is common and associated with clinical and laboratory evidence of hypoproteinaemia, hypoalbuminemia, thrombocytopenia, anemia, raised transaminitis, hyperbilirubinemia, coagulopathy and ultrasound abdomen showing altered echotexture will make everybody consider diagnosis of cirrhosis of liver. The same scenario occurred in initial stages of our case. Moreover, background diabetes mellitus pointed towards metabolic associate steatotic liver disease as the probable reason of developing cirrhotic changes. Raised transaminases and hyperbilirubinemia was due to liver abscess and mimicked cirrhosis. The history of fall followed by being bed ridden was attributed to generalized unwellness, along with poor dietary intake. The detailed neurological examination was turning point which showed hemiparesis and equivocal right plantar which prompted for radiological evaluation of brain and abdomen. The MRI abdomen confirmed hepatomegaly which was due to diabetes mellitus along with liver abscess without any changes of cirrhosis. The MRI abdomen clinched the diagnosis of generalized multiple lacunar infarcts in brain with white matter changes which explained the findings on neurological examination. The authors have already reported a case report in which intracranial bleed mimicked hepatic encephalopathy [9].

5. Conclusion

Cirrhosis diagnosis should not be made just on basis of ultrasonogram abdomen, as it is operator dependent. Moreover, every altered behaviour, even in cirrhotic patient should not be always labelled as hepatic encephalopathy and other causes, especially neurological, must be considered and ruled out.

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