Case Report

Hepatic encephalopathy in liver cirrhosis

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ABSTRACT

Liver cirrhosis is a worldwide gastroenterological condition, characterized by a slow, progressive and irreversible replacement of liver cells by fibrous tissue (scar) that prevents liver function. This condition often leads to the development of other syndromes. Cardiac complications can be indicated through abnormal QTc interval and arrhythmias, thereby their analysis aids in the prevention of cardiovascular events. Most cirrhotic cases have abnormal laboratory values (bilirubin, albumin, AST, ALT, AST/ALT, INR) indicating the presence of concomitant infection, inflammation and coagulopathy. In this case report, the usage Halstead-Reitan and Child-Pugh score helped in the assessment of the status of deterioration of brain. The knowledge of liver cirrhosis aetiologies help to determine the predisposition to development of hepatic encephalopathy and cardiomyopathy. The different values of liver enzymes and other blood laboratory analyses indicated the level of liver damage and poor prognosis.

Key words: alcoholic liver cirrhosis, Child-Pugh score, hepatic encephalopathy, liver cirrhosis, QTc interval, viral liver cirrhosis.

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Access this article online

Website: www.intern-med.com

DOI: 10.1515/jtim-2017-0013

Quick Response Code:

INTRODUCTION

Liver cirrhosis (LC) is a common gastroenterological pathology among adults. Its aetiological factors are: alcohol abuse, hepatitis B infection, hepatitis C infection, non-alcoholic fatty liver disease, non-alcoholic steatohepatitis and others.[1,2] Cirrhosis with multiple-aetiologies are more susceptible of developing multiple organ failures, predominantly kidney, brain, heart and others.[1,3] This disease can be classified using several methods and these cases will be focusing on Halstead-Reitan and Child-Pugh score classification. Furthermore, liver cirrhosis displays hemodynamic alterations revealed by the appearance of portal hypertension and hyper dynamic circulation.[4] Chronic liver disease displays numerous complications such as hepatic encephalopathy and cardiomyopathy (arrhythmias) resulting from extensive liver damage.[5]

Case 1

A 39-year-old male was referred to us by a local hospital, where he was followed up for liver cirrhosis induced by 4 years of chronic alcoholism. His condition was complicated by alcohol induced encephalopathy, delirium tremens, portal hypertension and arrhythmia. Anamnesis morbi: Patient had a history of frequent alcohol consumption which provoked a high tolerance intake (about 1 liter per day). The medical history included acute arterial hypertension, 3.5 months' recurrent abdominal ascites. Anamnesis Vitae: At the time of admission, the patient had reduced his Horilka (vodka) intake to 0.3 liter per day. The patient presented with symptoms and signs such as tremor, anxiety, white skin color of the head, loss of appetite, jaundice, bruises, fever, lack of energy, disorientation and orange urine color. The inspection revealed the existence of abdominal ascites, pedal oedema, clammy skin and the oesophageal varices were revealed on endoscopic examination. The neurological examination revealed that patient was presented with signs of depression, delirium, pathological reflexes (asterixis, hyperactive reflexes, extensor toe response later), short-term memory loss and displayed a moderate neurological impairment (Table 2). The sphygmomanometer detected hypertension of 142/91 mmHg and ultrasound showed 5 cm increase in the size of liver and dilated the oesophageal varices. The electrocardiography presented

an increased thickening of heart interventricular septum: 1.2 mm and slight increase of the left ventricular diastole: 1.1mm. Laboratory blood analysis confirmed the presence of anemia and also indicated a significant increase of hemoglobin and INR level (Table 3). The white blood cell count revealed leukocytosis with a shift to left (Table 4). Table 1 showed the elevation of some level of liver enzymes. Treatment: verospiron (spironolactone), furosemide (lasix), enterosgel, laktulosa (lactulose), heratan + glucose 5%, propranolol, recommendation of life style changes by complete alcohol withdrawal, low sodium and high protein intake.

Case 2

A 53-year-old female, was hospitalized due to paroxysmal liver cirrhosis. Liver cirrhosis was due to hepatitis C and B, complicated by arrhythmia, encephalopathy and hepatosplenomegaly. Medical history indicated that the patient was diagnosed with HBV, 6 years prior to the diagnosis of liver cirrhosis and the laparoscopic removal of biliary vesicle 3 years ago. Anamnesis morbi: jaundice, hyperreflexia, extensor posturing associated with positive Babinski sign, loss of appetite, bruises and disorientation. The physical examination also revealed mild tenderness in the left upper abdomen with no rebound, oedema and swelling in both lower extremities, abdominal ascites and varicose veins on her left leg. Neurological examination score presented a moderate neurological impairment (Table 2). Liver ultrasound and Doppler images showed a 1.5 cm increase of the right lobe and right hepatic vein enlargement with pathological backflow and with 14 mm diameter vena porta. Spleen size: 18.9*7 cm showed a clear annual increase of its erogenous boundary. Laboratory blood analysis indicated a normal level of red blood cell count with a slight decrease of platelet count, an elevation of INR and an elevated white blood cell count showing a leukocytosis with a shift to left (Table 3 and 4). There was a significant elevation of liver enzymes levels (Table 1). Treatment: palatine regimen diet n'5, solution tiofriazolin (solution cefazolin), enterosgel, betapin, Hepa Merz, duphalac, gepadif.

Case 3

A 63-year-old female, with 2 years' history of liver cirrhosis induced by viral infection (HCV) and 17 years' addiction to alcohol (vodka). The patient contracted hepatitis C during a dental procedure and was simultaneously diagnosed with liver cirrhosis, due to the appearance of ascites. The disease expressed a minimal process of active decompensation stage and was complicated by portal hypertension, atrial extra systole, atrial fibrillation and hepatic encephalopathy. During physical examination, the presence of ascites, pedal oedema, jaundice, bruises, fever, disorientation, symptoms of encephalopathy and oesophageal varices was found. The patient was subjected to an episode of atrial fibrillation and had prolongation of QTc interval (Table 2). The neurological status indicated a moderate impairment (Table 2). Biopsy and histological examination of liver detected the replication phase of HCV, while laboratory blood analysis indicated a decrease in the level of components of complete blood count and biochemistry, and with an elevation of ESR. As for the liver function enzymes analysis, it indicated an elevation of its different values (Table 1, 3 and 4). Treatment: verospiron (spironolactone), asparcam, trifas, uregyt, livonorm, heparin, lasix, atoxil, heptral, lactulose.

Case 4

A 62-year-old female, suffering from an alcoholic liver cirrhosis complicated by stage 2 hypertension. During questioning, the patient revealed a history of 20 years' alcohol (vodka) addiction. On manual and endoscopy investigation, the presence of abdominal ascites and oesophageal varices were found. On the neurological view, severe neurological impairment was diagnosed (Table 2). Instrumental investigation: The electrocardiogram showed a critical shortening of QTc interval (Table 2). Laboratory blood analysis indicated critical low values of the complete blood count and biochemistry (Table 4). The white blood cell count results indicated leukocytosis with a shift to the left. Liver enzymes showed elevation of bilirubin level and lowering of albumin level. Treatment: palatine regimen diet n'5, verospiron (spironolactone), asparcam, trifas, uregyt, livonorm, heparin, lasix, heptral, lactulose.

DISCUSSION

The establishment of diagnosis of liver cirrhosis, is based on the combination of instrumental and objectives methods. In our cases, due to the non-availability of imaging equipment, the patients' diagnosis were based on the physicians' experiences, patients' symptoms and laboratory results. Thus, all the patients were not given the same systematic line of diagnostic analysis, due to the fact that physicians were required to make the decision about the most efficient, informative and cheapest method to be used. Therefore, the doctors were required to make the most efficient test due to patient's incomplete or lack of health insurance. This explains the missing data observed in the Tables. An efficient cirrhotic treatment depends on early stabilization of laboratory values and efficient diagnostic procedure. Therefore, the patients were symptomatologically treated depending on drug availability and patient's drug purchasing power.

Patients with INR>1.5 should be put in intensive care units and monitored, in order to prevent deterioration of their state by appearance coagulopathy.[6] The elevated level of

liver markers and/or PH were an indication of patients poor prognosis (Table 1).[10] Alcohol addiction during liver cirrhosis course resulted in generating an abnormal QTc interval (shortening or prolongation), arrhythmia (Table 2). QTc interval can be used to determine the chance of future harmful cardiovascular events. Moreover, the severe cases of liver cirrhosis seem to evolve into hepatic encephalopathy; that is why patients' neurological examination, intracranial pressure and coagulogram should be systematically monitored.

The combination of alcohol and viral hepatitis aetiologies catalyzed the appearance of conjoint encephalopathy and cardiomyopathy symptoms such as arrhythmias. Viral liver cirrhosis (HBV, HCV) cases triggered the appearance of hepatic encephalopathy and the development of cardiac complication, while alcoholic cirrhosis was associated with arrhythmia events.[8,9] Patients displayed cardiac arrhythmias and hypertrophy of the septum as symptoms of hepatic cardiomyopathy, while brain involvement was displayed by the symptomology of delirium, intellectual depletion, memory depletion, tremor, and epilepsy. The blood laboratory analysis detected markers of concomitant infection, inflammation and coagulopathy (Table 3 and 4). The data indicated that cirrhotic patients with prolonged alcoholism history are more likely to develop coagulopathy and/or infections. Therefore, the first concern while diagnosing a supposed case of liver cirrhosis must be the stabilization and reduction of abnormal values of the laboratory analyses.

Usage of neurological assessment test like Child-Pugh Grade and Halstead-Reitan showed the prevalence of moderate and severe neurological impairment as indication by the patient's poor prognosis (Table 2). The scores reflected the degree of neuro-muscular inabilities of cases: especially thinking difficulties, problems with speaking fluently, guiding movements, memory alteration and the

Table 1: Liver function tests and complete metabolic panel

	Case 1	Case 2	Case 3	Case 4
AST (U/L)	74.9	139	219	-
ALT (U/L)	32.9	116	79	-
AST/ALT	2.3	1.2	2,8	-
Total bilirubin (µmol/L)	151.2	35.5	79.8	138.1
Direct bilirubin (µmol/L)	119.7	14.1	26.2	103.7
Albumin (g/L)	-	76.4	44	-
Blood creatinine (µmol/L)	-	36	-	45
Ammonia (µmol/L)	76	82	80	89

ALT: alanine transaminase; AST: aspartate aminotransferase.

Table 2: Neuropsychological test, aetiology and QTc interval

	Case 1	Case 2	Case 3	Case 4
Halstead Reitan score	2	3	4	-
Child-Pugh score	9 (B)	9 (B)	9 (B)	10 (C)
QTc interval (ms)	-	-	660	353

QTc: corrected QT interval.

Table 3: Complete blood count and international normalized ratio

	Case 1	Case 2	Case 3	Case 4
Erythrocytes ($\times 10^{12}$/L)	2.7	3.9	3.2	2.7
Hb (g/dL)	92	12.4	10.2	74
Platelet count ($\times 10^{9}$/L)	-	195.5	128	162
INR	1.7	1.38	1.33	1.41

INR: international normalized ratio; Hb: hemoglobin.

Table 4: White blood cell count and erythrocyte sedimentation rate

	Case 1	Case 2	Case 3	Case 4
WBC ($\times 10^{6}$/L)	19.3	3.8	-	2.1
Segmented neutrophil	75%	67%	-	76%
ESR	-	34	18	-

ESR: erythrocyte sedimentation rate.

appearance of tremors in their distal extremities. The combination of both tests allowed to have a more accurate evaluation of the patient's cognition state and intellectual abilities.

To sum up, many issues remain unsolved regarding the diagnosis and treatment of liver cirrhosis and complications. Furthermore, the appearance of most severe syndromes within our study are because there was no respect of treatment plans and recommendations. The general findings indicated the recurrence of alcohol consumption during and after the treatments as one of the common triggers for further hospitalization, especially due to hypertension and cerebral deterioration. We hope to draw attention to this disease and syndromes by sharing experiences and lessons learnt with other professionals, in order to stimulate scholarly debate and provide suggestions for future research and studies about liver cirrhosis, hepatic encephalopathy, cardiomyopathy and portal hypertension. Hepatologist and surgeons should be aware of this entity for proper diagnostic and care, also by quickly establishing differential diagnosis with others pathologies.

Conflict of Interest

Author has no conflict of interest to declare.

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How to cite this article: Djiambou-Nganjeu H. Hepatic encephalopathy in liver cirrhosis. J Transl Intern Med 2017; 5: 64-67.