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Hepatic encephalopathy: precipitating factors, clinical and evolutionary aspects at the university hospital campus of lome(Togo)

Abstract

Aim: To describe the precipitating factors, the clinical and evolutionary aspects of hepatic encephalopathy in the gastroenterology/liver unit of the Lomé University Hospital.

Material and methods: retrospective descriptive study on the case of in-patients at the gastroenterology/liver unit of Lomé University Hospital, from January 1st to December 31st, 2016, for acute or chronic hepatopathy complicated by hepatic encephalopathy.

Results: Thirty-nine patients were included in our study with a male predominance (sex ratio = 2.54) and a mean age of 48.9 years ± 12.98 years. The weak socioeconomic groups were the most affected (58.9%). Twenty-five patients (64.1%) had a pathological history, most often of upper digestive bleeding (33.3%). Most of the patients were taking herbal medications (76.9%). Ascites (74.4%) and jaundice (66.7%) were the most common reasons for hospitalization. On admission, 33.3% of patients had coma. The diagnoses retained were cirrhosis (69.2%); hepatocellular carcinoma (23.1%) and acute drug-induced hepatonephritis (7.7%). Triggering factors were dominated by the use of herbal medications (53.8%), infectious episodes (53.8%), upper gastrointestinal hemorrhage (33.3%) and ionic disorders (46.2%) in patients with cirrhosis. The evolution was marked by death in 48.7% of cases in all patients with hepatic encephalopathy, after an average of 11 days of hospitalization.

Conclusion: Hepatic encephalopathy is the consequence of liver failure, thereof it translates the seriousness; it can occur during acute or chronic hepatopathies, the mortality rate remains high, and emphasizes the need for specific hepatic resuscitation structures in our health structures.

Keywords: encephalopathy, liver disease, coma, liver failure, cirrhosis

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Abbreviations: PT, prothrombin rate; WHC, west haven criteria; HE, hepatic encephalopathy; SPSS, statiscal package for social science

Introduction

Hepatic encephalopathy (HE) corresponds to all the neurological and psychic manifestations determined by severe liver failure, whether acute or chronic. It appears in broad outline under two conditions: either a destruction of the liver by acute or chronic necrosis or a short circuit with shunt of the liver by abnormal shunt pathways (collateral circulation or portocaval surgical anastomosis). The term hepatic encephalopathy thus includes hepatic coma of viral or severe toxic hepatitis and portocaval encephalopathy of cirrhosis.[1]

In chronic diseases such as cirrhosis, episodes of hepatic encephalopathy are most often associated with an inter current complication such as gastrointestinal hemorrhage, infection, metabolic disorders, hypernatremia, or use of benzodiazepines.[2] It is one of the frequent complications (38.7%) in the cirrhotic patient in our department. Several studies, [3–5] have been carried out on cirrhosis in our department, but none has really examined this frequent and serious complication of acute or chronic liver disease. Hence this study on hepatic encephalopathy's, to evaluate the precipitating factors, the clinical and evolutionary aspects and to compare our results with the data of the literature.

Patients and methods

It was a retrospective and descriptive study, conducted on in-patient sat the gastroenterology department of the Lomé University Hospital, from January 1st to December 31st, 2016.Hepatic encephalopathy was diagnosed and classified into 4 stages according to the West Haven Criteria (WHC): Stage 1 was defined as impaired sleep-wake cycle, psychomotor retardation or paraxial coordination disorders. Stage 2 was defined by lethargy, confusion, moderate disorientation, abnormal behavior, the existence of asterixis. Stage 3 was defined by drowsiness but rousable, marked disorientation, aggressive behavior, asterixis and hyper-reflex. Stage 4 was defined by a non-rousable coma. Cirrhosis was defined by the presence of at least 3 of the following criteria: ascites, collateral circulation, prothrombin rate (PT), heterogeneous hepatic echo structure, irregular hepatic contours. Abdominal ultrasound was performed to look for signs of portal hypertension. Abdominal ultrasound began with a study of the hepatic morphology per subcostal, intercostal transverse and sagittal sections. All the recordings were made in the subject being in apnea in the middle of breathing. The operator identified the portal vein, including the segment between the spleno-portal junction and the intra hepatic bifurcation that was shaped as an anechoic tube within which he measured the largest diameter, as a caliber. The color doppler was used to detect the blood flow. Portal hypertension resulted in dilated portal vein (size over 14mm), splenomegaly (spleen size> 120 mm) associated with anechoic ascites. Portal thrombosis resulted in an

echoicendoluminalecho image by the Doppler flow within the portal vein or in one of its main branches. A survey sheet recording the anthropometric data, antecedents, physical and general signs, factors precipitating HE, the main biological examinations in particular ALT and AST, albumin, bilirubin, blood creatinine, prothrombin rate (PR), the electrolyte abnormality, Hbs Ag and Anti-HCV, current medications as well as results of the ultrasound were used. After leaving the hospital, the patients were re-examined after one month by the medical doctor who had examined them to collect the news. Statistical analysis was performed using the Statiscal Package for Social Science (SPSS); the results were expressed as mean ± standard deviation.

Results

During our study period, 39 patients (21.7%) with hepatic encephalopathy were identified in 180 cases of liver disease admitted in the service. There were 28 males and 11 females (sex ratio=2.54). The average age of our patients was 48.9 years ±13 years, with a prevalence of 40 -59 years (53.8 %).The weakest socioeconomic groups (workers, craftsmen, farmers) were the most affected (58.9%). Twenty-five of our patients (64.1%) had a pathological history, which was dominated by upper gastrointestinal hemorrhage (33.3%) with hematemesis and/or melena and jaundice (28.2%).With regard to chronic diseases: 12.8% of patients were HIV-positive, 3% were diabetic and 5.1% had high blood pressure. The lifestyle of our patients was dominated by herbal medications (76.9%). (Table 1) The reasons for hospitalization were mainly ascites (74.4%), jaundice (66.7%) and lower extremity edema (56.4%); only 13 patients (33.3%) presented coma on admission (Table 2). The general signs were mainly, general condition deterioration (87.2%), jaundice (74.4%) and conjunctival pallor (58.9%). The physical examination of our patients revealed 79.5% of ascites, and hepatic encephalopathy in all patients at different stages: stage I (10.9%), stage II (38.5%), stage III (9.6%) and stage IV (41%). The diagnoses retained were cirrhosis (69.2%); hepatocellular carcinoma (23.1%) and acute drug-induced hepatonephritis (7.7%). The distribution of patients according to the Child-Pugh score: stage A (7.5%), stage B (29.6%) and stage C (62.9%).The paraclinical assessment for diagnostic purposes, noted a collapse of the prothrombin rate (PR) in all our patients. The electrophoresis of the proteins noted a lowering of albumin level in (64.1%), and a beta-gamma block (38.5%). Hundred patients (64.1%) had benefited from the study of ascites fluid which was transudative (test of Rivalta negative and protein content less than 25g/l in the ascites fluid) in 61.5% of cases. None of our patients presented a clinical and biological picture in favor of acute viral hepatitis. Abdominal ultrasound in 66.7% of cases had a heterogeneous micronodular liver in 53.8%; nodular macro in 15.4% with in all cases the ultrasound signs of portal hypertension, and a portal thrombosis in 20.5% of cases. Alpha fetoprotein was was significantly high in 20.5% of cases. Gastroscopy performed in all conscious patients with upper gastrointestinal hemorrhage and / or signs of portal hypertension, had found esophageal varices in 38.5% of cases with a red sign (23.1%); gastric varices (5.1%) and portal hypertensive gastropathy (43.6%); almost all our patients (82%) had anemia, which was normochromic normocytic (46%) or microcytic hypochromic (25.6%), we also found renal failure in 61.5% cases of patients; and electrolyte abnormality such as hyponatremia (46.1%) or hypokalemia (20.5%). The etiology of cirrhosis was hepatitis B virus (60%), alcohol (20%), hepatitis C virus (4%), hepatitis B and C viruses co-infection (2%); the cause of cirrhosis was unknown in 14 % of cases. The precipitating factors (Table 3) of hepatic encephalopathy were dominated by herbal medications (53.8%), upper gastrointestinal hemorrhage (33.3%) and electrolyte abnormality (46.2%). The evolution in our patients was marked by death in 48.7% of the cases after an average of 11 days of hospitalization. Mortality was correlated with the stage of encephalopathy: it was 97.6% for stage 4; 78.2% for stage 3; 38.1% for stage 2 and 15.4% for stage 1.

Table 1: Lifestyle of patients

	Population(n)	Percentage (%)
Herbal medications	30	76.9
Alcohol	16	41
Tobacco	9	23.1
Drugs on the street	7	17.9

Table 2: Reason for hospitalization

	Population (n)	Percentage (%)
Abdominal distention	29	74.4
Jaundice	26	66.7
Edema of the lower limbs	22	56.4
Asthenia	20	51.3
Hepatomegaly	20	51.3
Hepatic encephalopathy	13	33.3
Pain of RHC*	11	28.2
Thinning	9	23.1
Asterixis	5	12.8
UGH**	5	12.8

*right hypochondrium, ** upper gastrointestinal hemorrhage

Table 3: Frequency of precipitating factors

	Population (n)	Percentage (%)
Herbal medications	21	53.8
Infectious episodes	21	53.8
Electrolyte abnormality	18	46.2
Constipation	15	38.5
Digestive bleeding	13	33.3
Diuretics	6	15.4
Sedatives	3	7.7

Discussion

This study highlights the high prevalence of hepatic encephalopathy (21.7%) in patients admitted for chronic or acute liver disease in our department. The male predominance was clear (sex ratio 2.5) with a mean age of 48.9 years; these figures are comparable to the results of Djibril[4] who also found a clear male predominance (sex ratio=5.2) with a mean age of 41.3 years in patients with cytolytic liver disease. Most of our patients had a history of upper gastrointestinal bleeding most often by variceal rupture, which is consistent with literature data.[2, 6-8] The clinical examination of our patients showed

just as in the literature, [2, 7, 9] that there was no specific sign for hepatic encephalopathy, despite the fact that 33,3% of patients presented at the outset an encephalopatic coma at admission. If the diagnosis of coma is easy, that of hepatic encephalopathy to which we must relate this coma in the context of our study is not always obvious and is based on the association of neurological signs and chronic liver disease.

Hepatic encephalopathy is a consequence of hepatocellular failure, of which it reflects the seriousness; thus it may occur during chronic liver diseases as was the case in our study where it was most frequently post-hepatitic and alcoholic cirrhosis and hepatocellular carcinoma. It can also occur during acute liver disease, and in our study it was mainly acute hepatitis due to herbal medications. We did not find any cases of acute fulminate viral hepatitis; these results are in line with literature data [2, 11, 12] and can be explained by the unfavorable socio-economic conditions in our countries with alcohol, hepatitis B and C viruses, HIV and self-medication. In Pakistan, the precipitating factors of HE most commonly found in their patients were infection (44%), GI bleeding (38%) and constipation (38%).[13] Onyekwere [14] in Nigeria, found that the identified precipitants of encephalopathy in the patients were sepsis 6 (29%), electrolyte in balance 3 (14%), gastrointestinal bleed 5 (24%), drug induced (5%), and possible malignant transformation 6 (29%). The triggering factors found in our study were herbal infusions, ionic disorders and the occurrence of upper gastrointestinal bleeding. These results are also consistent with data from the literature [2, 11, 12] and can be explained by the management of patients by traditional practitioners or in non-specialized structures before admission to our service. As for the evolution of our patients, it was marked by a high death rate (48.7%) which raises the problem of insufficient technical unit in the resuscitation of patients with acute or chronic liver disease; this is also explained by the late consultation of our patients, who most often go first to traditional practitioners, or first consult in non-specialized structures before admission to the gastroenterology/liver unit. The occurrence of a first episode of EH in a cirrhotic has a prognostic significance and is a turning point in chronic liver disease; survival is then estimated at 42% at 1 year and at 23% at 3 years in the absence of hepatic transplantation.[9] In our study, no patient had liver transplantation. In Bamako, the early mortality was 66.7%.[15] In our study, mortality was correlated with the stage of encephalopathy, which is consistent with the results of Ntagirabiri.[16] Saad Maqsood [13] found that out of all the 50 patients with hepatic encephalopathy in their study, 15 (30%) died including 4 patients (26.6%) in HE grade 3 and 9 patients (60%) in grade 4. Onyekwere[14] noted a death rate of 48% among the 21 patients included in their study; all patients in stages 3 and 4 had died. Higher fatality rates were recorded with increasing severity of encephalopathy.

Conclusion

Encephalopatic coma is the consequence of liver failure, thereof it translates the seriousness; it can occur during acute or chronic liver disease; the most common triggering factors were herbal medications, digestive bleeding, and electrolyte abnormality. The mortality rate remains high, and emphasizes the need for specific hepatic resuscitation structures in our health facilities.

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Conflict of interest

There is no conflict of Interest of any of the authors.

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