RESEARCH ARTICLE

Oesophageal varices, portal hypertensive gastropathy and spontaneous Sub-arachnoid haemorrhage in a case of liver cirrhosis diagnosed on autopsy.

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Abstract

Alcohol remains one of the most abused substances worldwide. Studies over the years have attributed chronic alcoholism as a major risk factor to liver cirrhosis. Patients with liver cirrhosis develop portal hypertension which put them at a higher risk of having esophageal varices and other associated complications. The authors present a case of a chronic alcoholic male individual who developed cirrhosis along with other less reported complications like sub-arachnoid hemorrhage and portal hypertensive gastropathy. The paper also profiles various changes associated with esophageal varices as observed during autopsy examination.

Keywords: Variceal bleeding; portal hypertension; sub arachnoid haemorrhage; portal hypertensive gastropathy.

INTRODUCTION:

Liver is an important organ of the body having functions of synthesis of plasma proteins and a range of blood clotting factors being produced. All of them are deranged in various liver disorders. Liver plays a predominant role in the regulation of haemostasis. Both cellular and plasmatic coagulation are defective, representing a hallmark of advanced liver disease[1]. High alcohol intake is an important risk factor for development of liver diseases and may further induce hypertension[2]. Patients with liver cirrhosis develop portal hypertension which put them at a higher risk of developing oesophageal varices. Esophageal varices are abnormal, enlarged veins in the tube that connects the throat and stomach (oesophagus). This condition occurs most often in people with serious liver diseases. Oesophageal variceal haemorrhage is a life-threatening complication of portal hypertension associated with a mortality rate significantly higher than that of other causes of gastrointestinal bleeding[3]. This study profiles morphologic changes of some internal organs associated with oesophageal varices as observed during autopsy examination. Liver cirrhosis is frequently associated with hematologic complications, especially thrombocytopenia and coagulation disorders[4]. In addition, alcohol intake is associated with changes in the coagulation system and may also affect the integrity of cerebral vessels[2]. The risk for development of ICH in patients with liver cirrhosis remains elusive[5]. Only a few studies have proposed that liver cirrhosis is a risk factor for ICH[5].It remains unresolved whether cirrhosis and other liver diseases are risk factors for ICH[6]. Esophageal varices usually don't cause signs and symptoms unless they bleed. Gastrointestinal haemorrhage is a well-recognized morbidity potentially occurring during the acute phase of stroke. Chronic abuse of alcohol has been attributed to 3.8% global deaths[8].

The deceased, a 42 years old man with history of chronic alcoholism for the past 15 years, was brought dead in Trauma Centre of Pt. B.D Sharma PGIMS, Rohtak. On history taken from the next to kin, patient was apparently healthy before two episodes of sudden hematemesis at home within 48 hours. The patient had purportedly stopped alcohol for the past 2 months. He was taken to the emergency department of the nearest government hospital (village Primary Health centre), from where the patient was immediately referred to our facility and he passed away during this transport.

CASE DISCUSSION:

Autopsy Findings

External Findings: The body was of a middle aged man who was markedly wasted, icteric and pale. There were no marks of violence on the body as well as no signs of decomposition. Blood stained secretions was present around angles of the mouth. Multiple pin head size to confluent ecchymosis were present over medial and posterior aspect of right arm situated 10 cm above to right elbow joint. (Figure1)

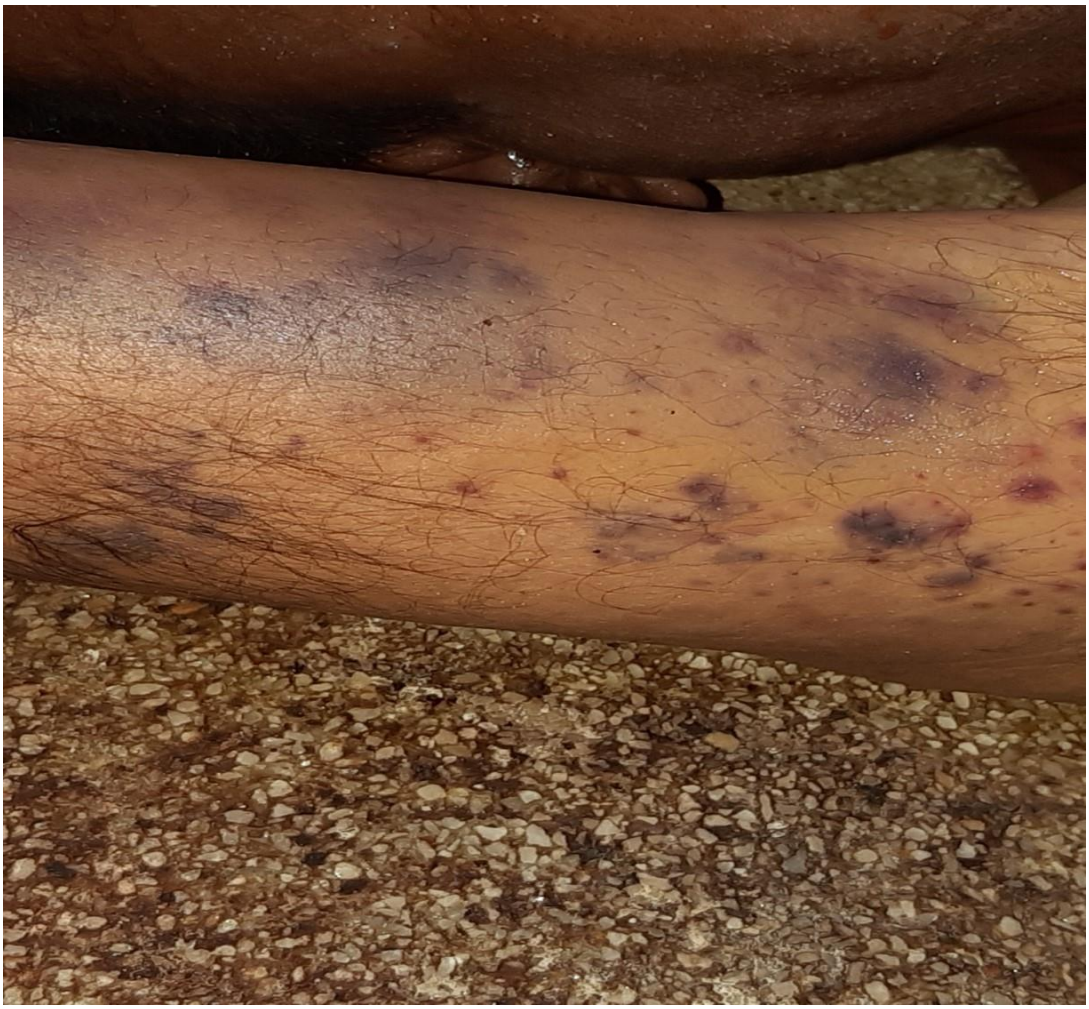

Figure 1: Multiple pin head size to confluent ecchymosis present over medial and posterior aspect of right arm

Internal Findings:

The oesophagus was congested. On dissection, mucosa of the oesophagus was congested and showed alternate area of reddish discolouration over the whole oesopahagus. The veins of lower end of oesophagus were found dilated (Figure 2). The oesophagus was sent for histopathological examination.

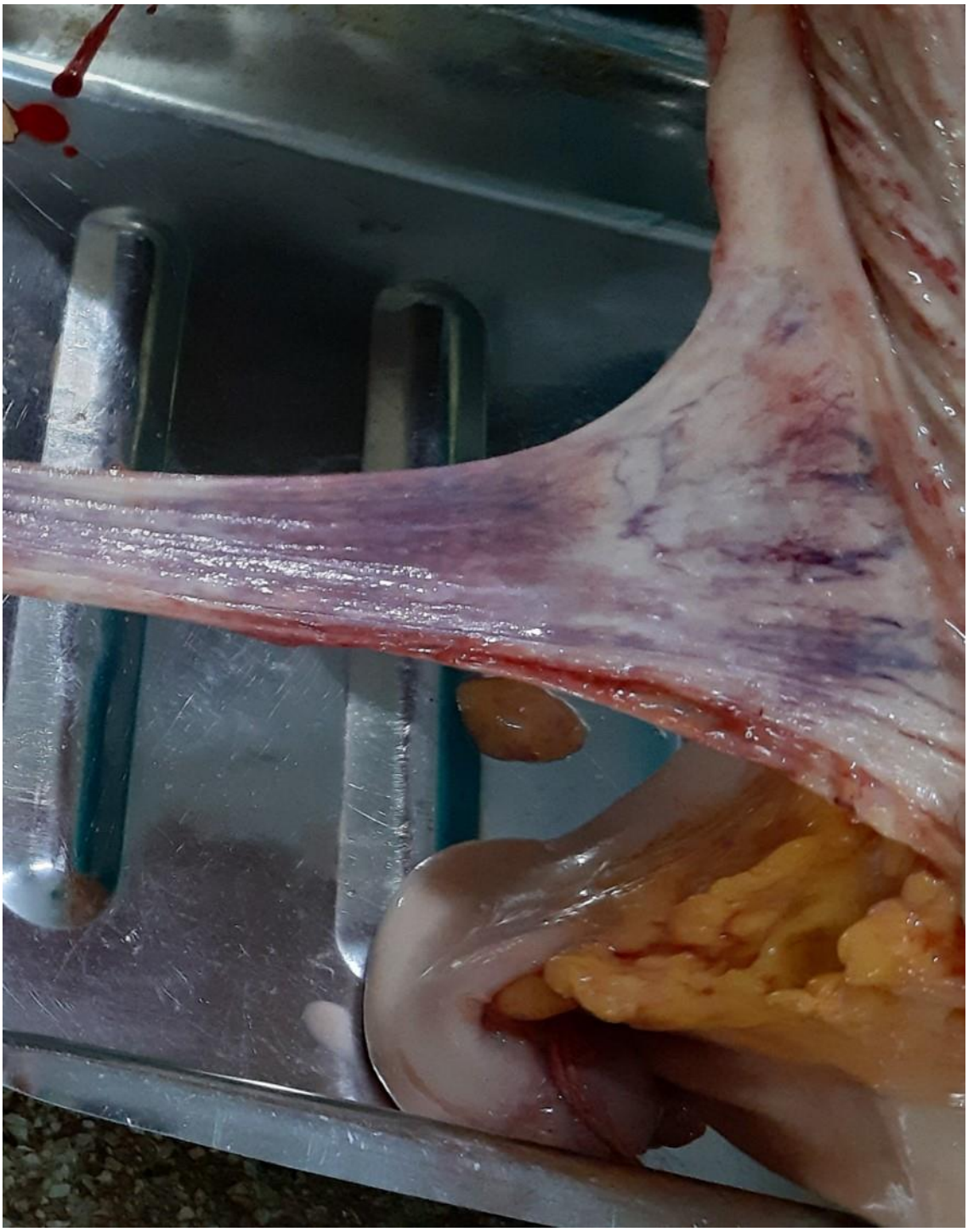

Figure 2: Mucosa of the oesophagus was congested and showed alternate area of reddish brown discolouration over the whole oesophagus. The veins of lower end of oesophagus were found dilated and tortious.

Lungs were congested and oedematous. On cut section frothy secretions were oozing out. A portion of the lungs was preserved for histopathological examination.
The peritoneal cavity contained about 1.2 litres of straw colour fluid. The stomach contained about 50 cc of greenish mucoid material. Mucosa was pale with haemorrhagic spots at places (Figure3). The stomach was preserved for chemical analysis along with its contents.

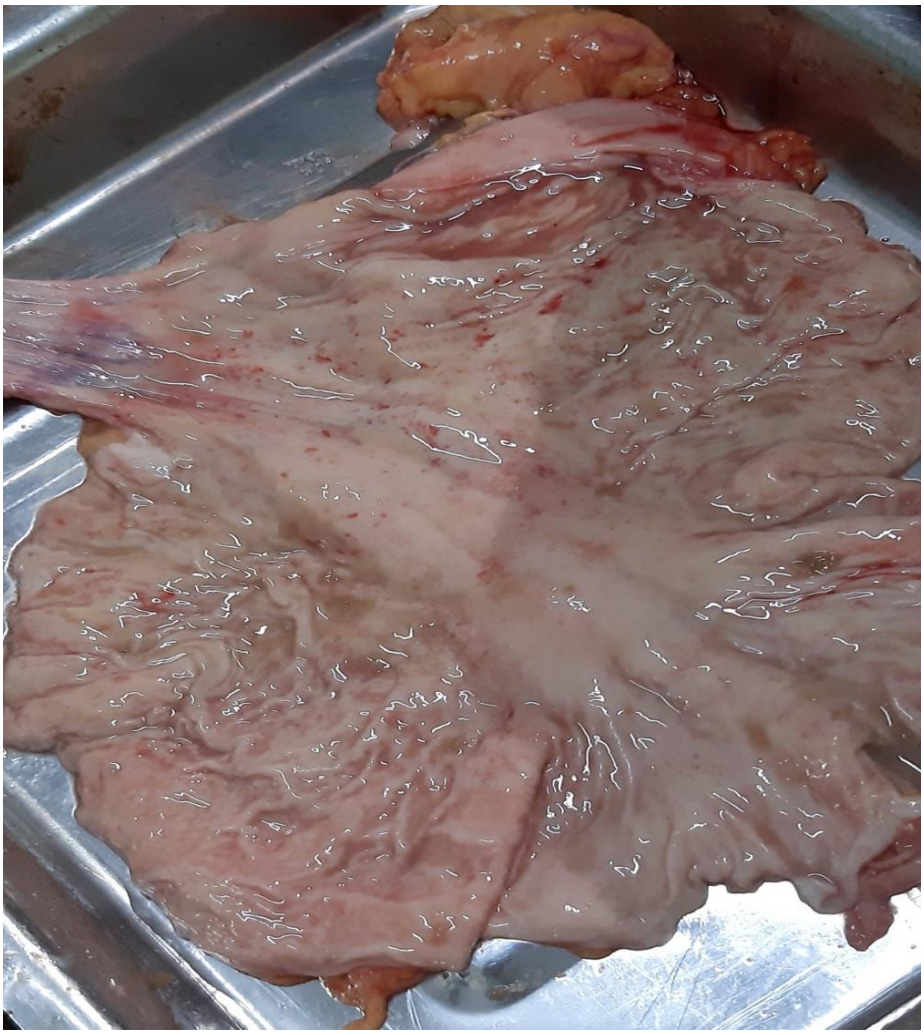

Figure 3: The stomach showing greenish mucoid material. Mucosa was pale with haemorrhagic spots at places.

The liver showed multiple nodules of size varying 0.25 x 0.25 cm were present over the surface of liver; congested on cut section (Figure 4). A portion of the liver was preserved for histopathological examination while a portion of the liver along with gall bladder was preserved for chemical analysis.

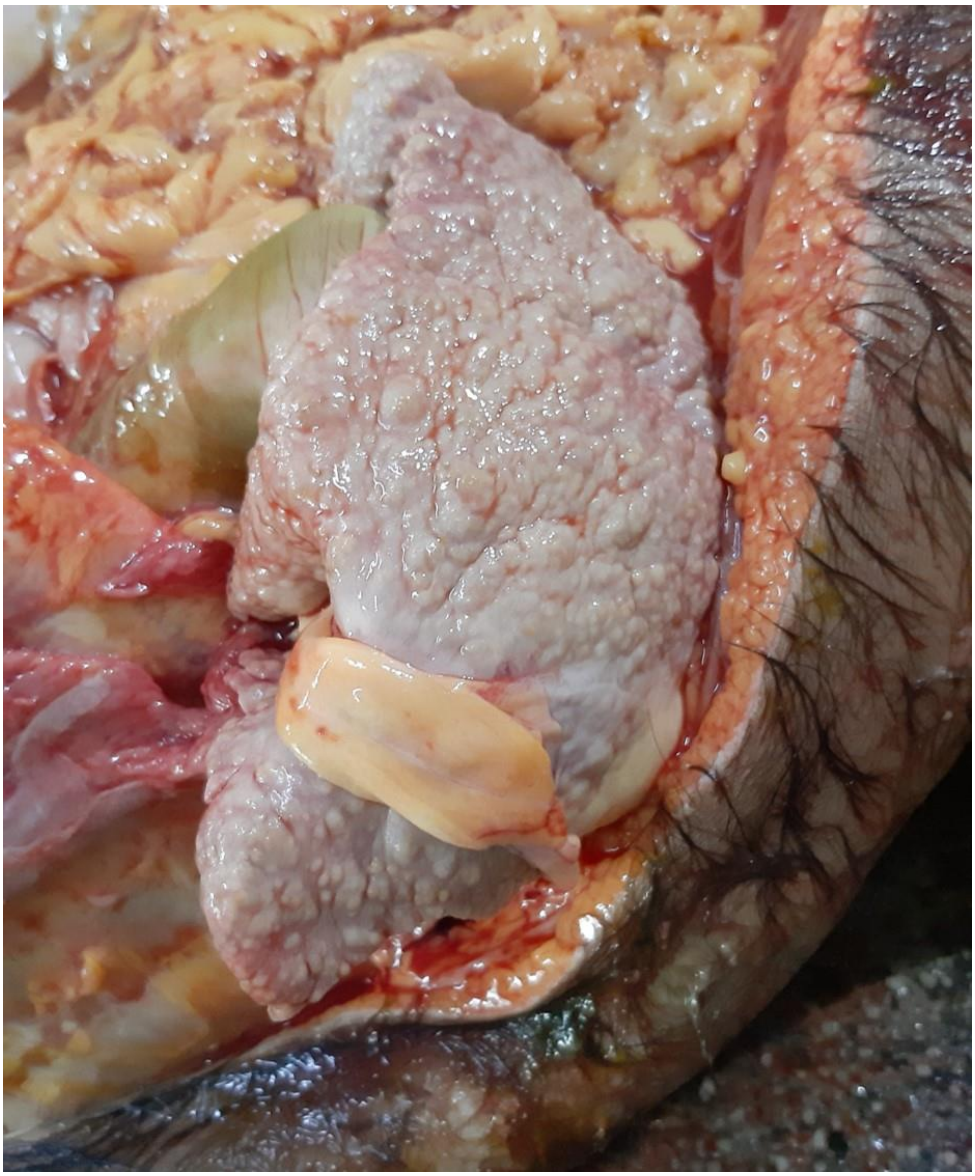

Figure 4: The liver showing multiple nodules of size varying 0.25 to 0.5 cm were present over the surface of liver

Results of exhibits sent for analysis:

1. Results of Chemical analysis of viscera: the chemical analysis report from the Forensic Science laboratory was negative for any common poison in all exhibits sent.
2. Results of Histopathological examination of viscera:
   a. Liver on gross examination: piece of liver measuring 13 x 7 x 5 cm with irregular surface. Microscopy: representative sections examined from piece of liver showed perivenular and pericellular fibrosis, regenerating parenchymal nodules separated by dense bands of fibrosis, mild periportal inflammation comprising of lymphocytes and plasma cells and hepatocytes.

b. Lungs: representative sections examined from piece of lungs showed oedema, congestion and leakage of blood into alveolar spaces.
c. Oesophagus: representative sections examined from portion of oesophagus showed elongation of the papillae touching close to the surface epithelium with proliferation and rupture of vessels in submucosa.
d. Brain: representative microsections examined from cerebrum showed subarachnoid haemorrhage while cerebellum and meninges showed congestion.
e. Heart: representative microsections examined from different portions of heart are unremarkable.
f. Spleen: showed congestion.
g. Kidney: both kidneys showed congestion.

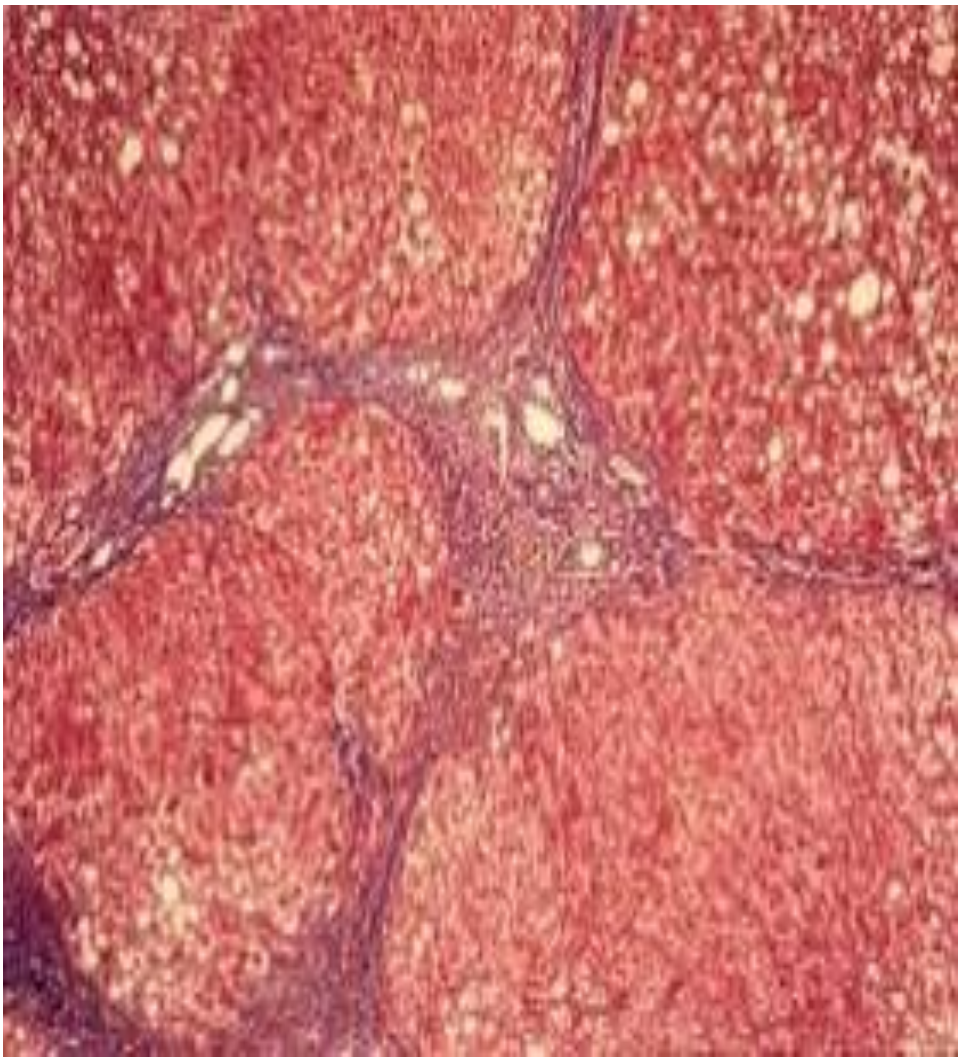

Figure 5: Microscopy: representative sections examined from piece of liver showed perivenular and pericellular fibrosis, regenerating parenchymal nodules separated by dense bands of fibrosis, mild periportal inflammation comprising of lymphocytes and plasma cells and hepatocytes.

DISCUSSION:

Venous blood from portal vein moves to the liver and to vena cava. Pathologic conditions such as liver cirrhosis block this movement of blood from portal vein to the liver. The blockage leads to development of portal hypertension. Portal hypertension triggers collateral channels to develop and this shunt blood directly to the vena cava without passing through the liver. Oesophagus is one of the few organs where collateral channels are formed. Portal hypertension dilates collateral or anastomotic channels in the submucosa of oesophagus while culminate to rupture and cause massive gastrointestinal bleed. Other morphological changes of portal hypertension include expanding parenchymal nodules and peri venular fibrosis. The nodules may present as micro nodules or macro nodules depending on the chronicity of the condition. Persistent alcohol abuse for a long period of time has a devastating effect on the liver. Major complications include hepatic steatosis, alcohol hepatitis and cirrhosis.

The onset of liver cirrhosis is dependent on the type of alcoholic drink abused, gender, genetic factors and the quantity of daily alcohol intake[8]. Patients with alcohol-induced liver cirrhosis may present clinical symptoms such as distended abdomen, wasted extremities, malaise, weakness, weight loss and ascites. A major life-threatening complication of liver cirrhosis is esophageal varices. It is a fatal complication to portal hypertension among cirrhotic patients[9].This is usually asymptomatic and can diagnosed only during autopsy. Other clinical consequences of portal hypertension include splenomegaly, ascites and hepatic encephalopathy [10-13].

The autopsy showed massive intra gastric and intestinal bleed from esophageal varices. This was due to the liver cirrhosis. Though liver cirrhosis has diverse risk factors, the clinical, police and family reports confirm chronic alcohol abuse as the major cause of the liver cirrhosis in the deceased. In cirrhosis, the Clotting time in both bleeders and non-bleeders is within normal range, as shown by previous studies. But the mean value for bleeders is quite higher than non-bleeders as shown by Rastogi et al[9] in 18 cases. In cirrhosis of liver, bleeding time and clotting time show normal range but mean value for bleeders is higher than non bleeders. In viral hepatitis and obstructive jaundice, bleeding time and clotting time shows normal range..[17] A connection between liver disease and gastrointestinal haemorrhage is noted in populations with either ischemic stroke or SAH. Rumalla et al. analysed acute ischemic stroke in the largest database of the United States, but information on the subgroup with liver disease is scanty[12]. Interestingly, a study from Taiwan reported that abnormal liver function is related to gastrointestinal haemorrhage after ischemic stroke[13]. The pathophysiology of gastrointestinal haemorrhage after acute strokes is not completely realized, and several theories exist. Intracranial lesions or elevated intracranial pressure affect brainstem or hypothalamic nuclei with hyperactivity of vagal tone and lead to haemorrhagic peptic ulcerations because of hypersecretion of gastric acid[14]. Second, acute stroke predisposes to gastroparesis resulting from interruption of the axis between the central

nervous and digestive systems and seems to increase the risk of gastrointestinal bleeding[15]. In addition, a humoral surge in catecholamine's or cortisol is usual in the acute phase of stroke[16] and possibly causes vasoconstriction or mucosal damage of the gastrointestinal tract.

Apart from aspects of forensic pathology, the demographics of studied population are also noteworthy from the viewpoint of social medicine, since at least 31 individuals had a history of chronic alcohol consumption confirmed by autopsy findings. In bringing such deaths and the social setting to light, the field of legal medicine reflects the society of a special region. By nature, medico legal autopsy studies analysing sudden death in outpatients often lack detailed information about the clinical picture preceding death; therefore, conclusions of studies are based mainly on autopsy findings and police reports. Nonetheless, the data presented here stress the importance of fatal esophageal variceal haemorrhage as a relevant cause of sudden death occurring outside the hospital in socially isolated, alcohol-addicted individuals.

Conclusion

Alcohol abuse has devastating effect on human lives especially in developing countries. It is evident that deceased died from haemorrhagic shock which began with alcohol induced liver cirrhosis. Attention to nutrition and long-term rehabilitation are particularly important in those alcoholic cirrhotic patients who survive with a urgent need to better critical health facilities in first response medical units such a primary health centres in developing countries where such patients can be stabilised before they are referred to apex medical institutes.

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