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Case Report

Haemorrhagic shock leading to death due to ruptured esophageal varices: An autopsy based case report

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ABSTRACT

Introduction: and importance: Ruptured esophageal varices are fatal and cause haemorrhagic shock and consequently death if no immediate intervention is instituted to arrest bleeding. The role of forensic pathologists in investigating cases of sudden unexpected deaths is of paramount importance. Upon medico-legal work ups by forensic pathologists, autopsy would shed light to the exact cause of death as to whether it is natural or unnatural.

Case presentation: We are reporting an unusual case of an elderly man with alcoholic liver cirrhosis who succumbed to sudden death following ruptured esophageal varices which led to haemorrhagic shock.

Clinical discussion: Clinical autopsy was carried out to establish the cause of death. Tissue sections from the healed pathologically fractured bone, liver, prostate and esophagus were taken for histological evaluation. The bone tissue showed relative reparative changes and the prostate tissue showed hyperplasia of both stroma and epithelial components. The tissue from the esophagus showed proliferating distended, engorged vascular structures and some were thrombosed (photograph 3). Liver biopsy showed destruction of the normal liver parenchyma by both micro and macro nodular formation which was marked by the thick broad fibrous bands. A conclusive diagnosis of hemorrhagic shock secondary to ruptured esophageal varices caused by alcoholic liver cirrhosis was established as the cause of death.

Conclusion: Esophageal varices can be the likely cause of sudden death upon rupturing. Rupturing of esophageal varices in individuals with a known long-standing history of alcoholism must always be considered in order to prevent haemorrhagic shock from ensuing.

1. Introduction

Chronic abuse of alcohol is reported to be the common social indulgence worldwide. Regarding alcohol abuse, its estimated that about 2 billion people consume alcohol [1]. In the East African region, Uganda has been reported to be a country with the highest levels of alcohol consumption and the annual per capita alcohol consumption is reported as 23.7 L [2]. Over the past years, studies have been attributing chronic alcoholism as one of the major risk factors for liver cirrhosis [3,4]. Patients with liver cirrhosis develop portal hypertension which puts them at a higher risk of developing esophageal varices and it has been found that over 80% of patients with liver cirrhosis are at a risk of developing esophageal varices. Esophageal varices may culminate to sudden death via gastrointestinal bleeding [5,6]. There are two theories explaining the etiology of esophageal varices: One is that they are caused by a disturbance of the vascular regulating mechanism of the spleen and the other by an obstruction in the splenic or portal vein [7]. Besides, increased intra-abdominal pressure has also been reported as the cause of rupturing of esophageal varices [1]. There are three different types of esophageal varices which are classified based on the direction of venous flow and they have been named by their trivial names as "uphill," "downhill" and idiopathic [8]. Uphill," esophageal varices are the most common type of esophageal varices. The aim of the case report is to add emphasis on the importance of considering the possibility of rupturing of esophageal varices in individuals with a known longstanding history of alcoholism and this must always be considered in order to prevent haemorrhagic shock from ensuing which is virtually fatal. The work has been reported in line with the SCARE 2020 criteria [9].

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2. Case presentation

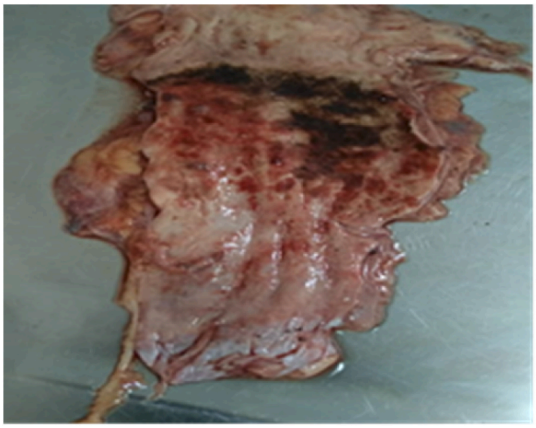
A 90-year old man, anaemic with pale and asymmetrical lower limbs due to swelling of the left lower limb. He had a known long standing history of diabetes mellitus (DM) and hypertension. A month before admission, he sustained fracture of the proximal left femur which was immobilized and healed and he could stand and walk alone. Later on he started to present with dry cough, fever and lower urinary tract symptoms. He was then diagnosed to have cystitis. He was brought to the hospital by an ambulance and three days post-admission, he developed haemoptysis. Upon admission he was reviewed and found to have aortic regurgitation and bilateral lower limb edema. Blood count indicated low white blood cells and platelet counts. Electrolyte assessment showed low serum sodium (Na^{+}) and potassium (K^{+}). He was suspected to have pulmonary embolism due to the previous one-month history of fracture of the femur. Two days after admission, his blood pressure dropped to 115/50 mmHg and Hemoglobin level was 30-g percent. He was transfused with 500 mL (mls) of whole blood but his blood pressure remained low with very unstable pulse rate.

He died on the fourth day post admission. Relatives requested clinical autopsy and it was carried out to establish the cause of death. Externally, the body was pale with lower limbs asymmetry due to slight swelling of the left lower limb. The meatal urethra was hyperemic and swollen. Internally, remarkable findings included collection of ascitic fluid in the abdominal cavity measuring 1500 mls. The lungs were pale, congested and on sectioning, watery fluid was oozing. There was a pleural fluid measuring 320 mls which was straw coloured. The aorta had numerous atheromatous plaques. The kidneys were atrophic both with fine granules and they were covered with a thick fat pad. On opening the esophagus, there were ruptured dilated varices at the distal third (Photograph 1) and blood of 800 mls had collected in the stomach. The liver was nodular and shrunken with noticeable picture of cirrhosis (Photograph 2). Other organs were unremarkable.

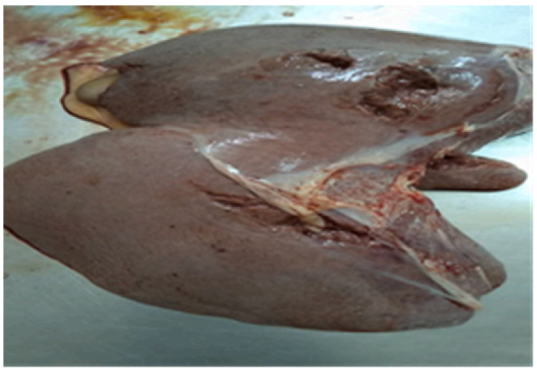
Tissue sections from the healed pathologically fractured bone, liver, prostate and esophagus were taken for histological evaluation. The bone tissue showed relative reparative changes and the prostate tissue showed hyperplasia of both stroma and epithelial components. The tissue from the esophagus showed proliferating distended, engorged vascular structures and some were thrombosed (Photograph 3).

Liver biopsy showed destruction of the normal liver parenchyma by both micro and macro nodular formation which was marked by the thick broad fibrous bands (Photograph 4).

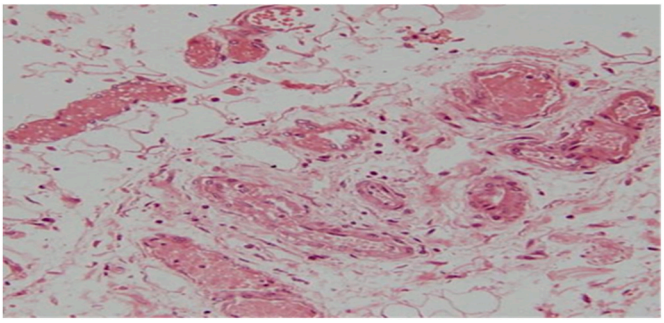
A conclusive diagnosis of hemorrhagic shock secondary to ruptured esophageal varices caused by alcoholic liver cirrhosis was established as the cause of death.



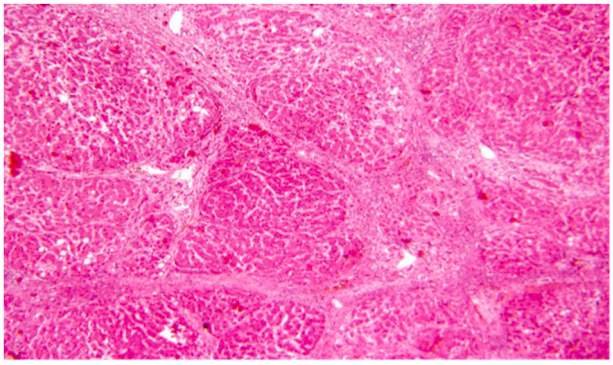
Photograph 1. Showing an area with ruptured varices. There is both fresh and old hemorrhage in the area with ulceration.



Photograph 2. Showing a shrunken liver, reduced in size and with cirrhotic appearance.



Photograph 3. Photomicrograph of blood vessels in the submucosa of the esophagus (Hematoxylin and Eosin stain. X40).



Photograph 4. Photomicrograph of the liver tissue showing cirrhotic changes (Hematoxylin and Eosin stain. X40). The nodules marked by branching fibrous bands that effaced the liver parenchyma.

3. Discussion

Esophageal varices represent the most important complication of portal hypertension and arises from the opening of portosystemic collaterals as an adaptation to decompress the portal venous system [10]. It has been stated to be the commonest cause of upper gastrointestinal bleeding where studies suggest that ruptured esophageal varices are responsible for 50%–80% of upper gastrointestinal bleeding episodes [11].

Gastrointestinal (GI) bleeding is a common and serious complication of liver cirrhosis [10–12]. The most important source are esophageal varices, but bleeding from peptic ulcers is also common [7].

Available studies have ended up establishing alcoholic liver cirrhosis to be the primary factor in the pathophysiology of portal hypertension

[1,3,4]. Findings support the current clinical concepts that alcoholic liver cirrhosis is associated with a significantly higher risk of variceal bleeding than is cirrhosis arising from other causes and the prognosis for an individual patient depending on the severity of the bleeding episodes and underlying liver function is generally poor in patients with established cirrhosis and hepatocellular carcinoma [3,7]. The present case has a correlation of both variceal bleeding and alcoholism.

Reported clinical factors associated with an increased risk of bleeding from varices include poor liver function and continued alcohol abuse [8]. The mechanism by which these two clinical risk factors leads to bleeding from such varices are as follows; poor liver function contributes to abnormality in coagulation via chronic inadequate hepatic synthesis of coagulation factors whereas continuing alcohol abuse leads to progressive liver cirrhosis which consequently lead to increased portal hypertension and further deterioration of liver function status [12].

Fatal esophageal variceal bleeding accounts for a larger proportion of out of hospital deaths in the observed frequency of 0.75% of all deaths encountered in a medico-legal autopsy population [13]. The number of fatal GI hemorrhage in younger individuals occurring out of hospital is likely to be underestimated from the clinical viewpoint due to the under representation of such cases in the field of clinical pathology [13]. The mean ages of the outpatients depicted in one of the study is substantially younger than the inpatients presenting with acute esophageal variceal hemorrhage [8,11]. These results also explain that the deaths of younger individuals occurring out of hospitals are more often subjected to a medico-legal autopsy than are the deaths of older individuals that occur more often inside hospitals [13,14].

Such observation appears to be dissimilar to what has been observed in our case report where the old man died of ruptured variceal bleeding secondary to liver cirrhosis that consequently led to hemorrhagic shock. Future multicentric autopsy based studies may be designed to evaluate the actual burden of deaths ensuing from rupture esophageal varices so that the communities are well educated on the outcomes of alcoholism with vivid similar outcomes from our local communities. This case report has emphasized on the importance of fatal ruptured esophageal varices as the notable cause of sudden death in alcoholic patients following hemorrhagic shock.

4. Conclusion

Deaths from ruptured esophageal varices occurring as a result of complication of decompensated liver cirrhosis are more likely to occur due to the nature of causative mechanism that may be presenting with dull and unspecific symptoms. Rupturing of esophageal varices in individuals with a known long-standing history of alcoholism must always be considered in order to prevent haemorrhagic shock from ensuing that is almost always fatal.

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Ethical approval

Written informed consent was obtained from the deceased relative for publication of this case report and accompanying images, in line with local ethical approval requirements. No other requirements were stipulated.

Consent

Written informed consent was obtained from the deceased relative for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Author contributions

ZSA-Conceptualization, writing original draft of the manuscript, JJY-Conceptualization and reviewing the prepared original draft of the manuscript.

Registration of research studies

N/A.

Guarantor

The corresponding author is the guarantor of this manuscript.

Provenance and peer review

Not commissioned, externally-peer reviewed.

Declaration of competing interest

We declare there is no conflict of interest.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.amsu.2022.103932.

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