



DEATH DUE TO HEMORRAGIC SHOCK DUE TO RUPTURE ESOPHAGEAL VARICES: CLINICAL AND AUTOPSY BASED CASE REPORT

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ABSTRACT

This is a case report of A 27 years old female was brought to the emergency department with the complaint of progressive abdominal distension with pain in abdomen and early satiety for last few months. On further clinical, radiological and laboratory evaluation, patient was diagnosed with portal hypertension with splenomegaly and got collapsed during the course of treatment. On autopsy examination, the cause of death was hemorrhagic shock due to rupture of esophageal varices.

KEYWORDS :

INTRODUCTION

Portal hypertension is typically due to the presence of underlying liver cirrhosis, though it can develop from other pathological conditions also. Non-cirrhotic portal hypertension (NCPH) can arise from idiopathic non cirrhotic portal hypertension, extra hepatic portal venous thrombosis, schistosomiasis, veno-occlusive disease, and congenital hepatic fibrosis. Most common affected age group is younger population in Asia while in western countries; median age of diagnosis is 40 years with male predominance.

Case Report

A 27 years old female was brought to the emergency department with the complaint of pain in abdomen, progressive abdominal distension and early satiety for last few months.

ON examination, she was conscious and well oriented. She was mildly febrile having high pulse, low BP with pallor and edema over the face and feet. On auscultation chest was clear with normal vesicular breaths and heart sounds were normal with no murmur. Abdominal examination showed a protuberant abdomen with fluid wave, shifting dullness, and palpable spleen.

Laboratory investigation showed hemoglobin 7gm/dl with leucocytosis 23200 with neutrophilia. Liver function parameters were normal except albumin, which was slightly raised. International normalized ratio (INR) was normal. Ultrasonography reported moderate amount of fluid in the peritoneum confirming ascites with mild pleural effusion, splenomegaly, dilated portal veins indicating portal hypertension. X ray examination revealed blunt costochondral angles indicating pleural effusion.

Ascitic tap was performed to relieve abdominal distension. Acid-fast bacilli culture was negative though the culture showed the growth of E Coli and Streptococcus viridians. Serological examination for hepatitis was negative and no parasitic infection was detected in stool examination

Upper GI endoscopy revealed the development of varices of grade I, II and III in esophagus and bleeding points were detected in body and fundus of stomach. Patient was stabilized and kept under observation but after 12 hours patients suddenly collapsed. Patient's attendants charged the treating doctor for medical negligence. Body was sent for autopsy for confirming the cause of death.

On autopsy

External Findings:

The dead body was of a poor built young adult female individual. Both eyes lids were closed, pupils were dilated and fixed. Rigor Mortis was present all over the body. The body was wasted, icteric and pale. Dried blood clothes were present around the oral cavity. There was no evidence of struggle or violence marks on the body as well as no sign of decomposition.

Internal Findings:

The inner aspect of the lower one third of esophagus showed multiple varices of varying size; few of them were ruptured (Fig 1). Tortuous blood vessels were present over the lower half of the esophagus.

The stomach contained about reddish-brown liquid blood along with few clots with hyperaemic mucosa. Externally brownish-black hue was present over the small intestine and contained brownish-black colored blood admixed with liquid chyme. Liver was enlarged, firm with hemorrhagic areas (Fig. 2). Spleen was enlarged with congestion. Brain and lungs were edematous and congested.

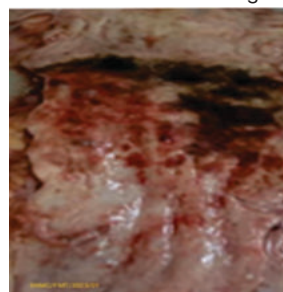


Figure 1. Esophageal varices



Figure 2. Enlarged liver with hemorrhages

DISCUSSION

Globally, cirrhosis accounts for 85% of cases of ascites.¹ Remaining 15 % constitutes multiple etiologies like heart failure, peritoneal carcinomatosis, nephrotic syndrome, tuberculosis peritonitis, and Budd-Chiari syndrome. Other risk factors and etiologies of ascites are history of recurrent blood clots, presence of IVC filter, and history of HIV. NCPH is confirmed if one must have clinical signs of portal hypertension with patent hepatic vasculature on imaging, biopsy should exclude cirrhosis, and exclusion of other causes of liver disease on laboratory work-up.² Acute bleeding or re-bleeding from ruptured esophago-gastric varices is one of the

most severe complications of cirrhosis which culminate to death³. Shetty et al⁴ reported a case in which a seventy years old, male, a manual labourer by occupation suddenly had episodes of vomiting of blood, autopsy confirmed that oesophageal varices was the cause of death in this case. Ossei et al⁵, described a case of a 53 years old male individual have been found dead in front of his room at the lawn. The cause of death was massive hemorrhage due to rupture of esophageal varices. Similarly this case report showed that the death occurred due to rupture of esophageal varices that were developed due to portal hypertension of idiopathic non cirrhotic type.

CONCLUSION

The cause of death in case of idiopathic portal hypertension may be progressive hepatic failure, massive hemorrhage from ruptured gastroesophageal varices, and hemorrhagic intestinal infarction due to mesenteric venous thrombosis.

Variceal hemorrhage is a medical emergency in which many patients die due to hemorrhage refractory to standard treatment, either because of failed bleeding management or as a result of early re-bleeding. In this case, a female presented with the abdominal pain, later on diagnosed with portal hypertension and died during the course of treatment, autopsy revealed cause of death was hemorrhagic shock due to rupture of esophageal varices. It is advised that such cases may deteriorate even in recovery phase also must be managed thoroughly to avoid mortality.

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