

**A CASE REPORT OF DECOMPENSATED CHRONIC LIVER DISEASE WITH
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ABSTRACT

Decompensated liver disease also known as decompensated cirrhosis is the most common non haematological cause of Pancytopenia. Here we report a case of 43yr old man with hepatic cirrhosis & pancytopenia. He came to emergency with chief c/o yellowish discoloration of eyes, abdominal distension, bleeding per oral, Malena. Laboratory examination revealed pancytopenia, elevated total bilirubin, liver enzymes, moderate ascites, Splenomegaly & oesophageal varices on endoscopy. Patient was treated with beta-blockers, Diuretics, terlipressin and albumin. Endoscopic variceal ligation done. This case reveals multifactorial contribution in occurrence of pancytopenia in hepatic cirrhosis.

INTRODUCTION

Pancytopenia is characterised by decrease in all the 3 blood cell lineages RBC-<3.5 × 10¹²/L, WBC-<4.0X10⁹/L, PLT-<100 x10⁹/L.

Decompensated chronic liver disease is characterised by presence of jaundice, ascites, varices, encephalopathy & hepatorenal syndrome.

Liver plays an important role in homeostasis and metabolism of lipids, carbohydrates, proteins & has a role in endocrine & haematological manifestations. A breach in its function leads to subtle metabolic abnormalities, derangement in haematological parameters which ultimately lead to grave complications. Alcoholism, hepatitis B, C & NAFLD are most common insults which leads to breach in liver function. The liver constitutes a significant storage site for iron, produce erythropoietin and thrombopoietin. Lack of thrombopoietin and erythropoietin production, lack of iron storage in damaged hepatocyte, also splenic sequestration were the reason of pancytopenia in hepatic cirrhosis.

CASE PRESENTATION

We report a case of 43yr old man diagnosed with DCLD & pancytopenia. He came to emergency with yellowish discoloration of eyes, abdominal distension, loss of appetite, fatigue in the past 2 weeks there is increase in the girth of abdomen & from past 2 days he noticed blood drenched pillow & acknowledged bleeding per

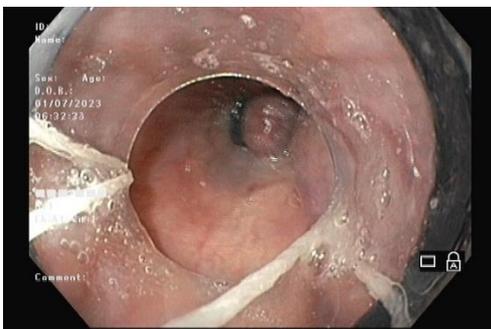
oral which alarmed him to seek medical assistance. He also gave history of regular alcohol consumption since 18yrs & an amber chewer, & Malena was present. On examining the pt has, PR-88/min, BP-130/80 mmHg, RR of 21/min, pale palpebral conjunctiva, yellow sclera, prominent abdomen with full flanks, & B/L pedal oedema is noted. Laboratory work up revealed Hb-8gm/dl, RBC- 2.8 x 10⁶/mm³, WBC-3200/mm³, PLT Count - 20 x 10³/mm³, Total protein -5.5, Total bilirubin - 6.2, Sr. Albumin-2.1 SGOT-236, ALP-249, INR -1.7 peripheral blood morphological examinations revealed - normocytic normochromic picture with ovalocytes & target cells, severe thrombocytopenia. An ultrasound abdomen revealed Altered echo texture of liver, splenomegaly, moderate ascites, & has a negative hepatitis Panel. Liver biopsy confirmed advanced cirrhosis with fibrosis and architectural distortion. Bone marrow aspiration and biopsy revealed hypocellular bone marrow with trilineage hypoplasia consistent with pancytopenia.

Given the h/o bleeding per oral & Malena, an additional workup of Upper GI endoscopy was done which disclosed grade II-III oesophageal varices, EVL banding with 5 bands are placed for further management after taking consent EVL was performed & he was also treated with beta blockers, diuretics, terlipressin, albumin & high protein diet was given. He was prescribed lactulose and rifaximin to manage hepatic encephalopathy. Supplementation with folic acid and vitamin b12 to support hematopoiesis. Further evaluation

of erythropoietin & thrombopoietin was done and are decreased. After the hospital course he was monitored regularly on OP visits for blood parameter workup & supportive management.



Image showing: Grade II-III oesophageal varices



- Endoscopic variceal ligation of esophageal varices. (A) Endoscopy reveals a red color sign on the esophageal varix through the transparent cap. (B) Variceal ligation is performed by placing the head of the endoscope with a rubber band ligation device over the varix to be ligated, suctioning the varix into the device, and tying the varix by discharging the rubber band.

DISCUSSION

Pancytopenia corresponds to multi lineage cytopenia is an outcome of multifactorial contribution. This case illustrates pancytopenia in hepatic cirrhosis due to lack of iron stores, lack of erythropoietin, thrombopoietin due to destruction of hepatocytes. Additionally, our patient has splenomegaly on imaging which assists retention of higher number of blood cells in spleen thus facilitating phagocytosis & destruction ultimately leading to peripheral cytopenia.

On top of that pt has variceal bleed which Loss which further contributed cytopenia's can be caused by bone marrow suppression mediated by toxins, i.e. alcohol here.

Moreover, thrombocytopenia's can lead to bleeding & bruising. The pt experienced prolonged bleeding & delayed wound healing & increased risk of infection, making it necessary to address the underlying cause & manage patients with pancytopenia & monitoring the blood counts.

The presence of jaundice, ascites, varices, elevated transaminases reveals that the liver injury has progressed to decompensated stage and associated with poor prognosis with pancytopenia.

CONCLUSION

- This case report reinforces the importance of inclusion of hepatic cirrhosis in differential diagnosis of Pancytopenia.
- Abnormal haematological indices are frequently encountered in cirrhosis, multiple factors contribute to its occurrence like destruction of hepatocytes, lack of iron stores, lack of erythropoietin, thrombopoietin, splenic sequestration, bleeding varices, portal HTN, bone marrow suppression.
- Timely intervention and comprehensive care can improve hematologic parameters and potentially

extend the window for liver transplantation, which remains the definitive treatment for advanced cirrhosis.

- Recent studies suggest presences of cytopenia with hepatic cirrhosis is associated with poor prognosis which may be due to clinical consequences like increased risk of bleeding & infection.

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