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ACUTE GASTROINTESTINAL BLEEDING IN DECOMPANSATED CHRONIC LIVER DISEASE TRIGGERED BY SOFT TISSUE INFECTION: A CASE REPORT AND REVIEW OF LITERATURE

Shreyasi Maity[1], Mabuto Michelle Tanaka[2]

Abstract:

Decompensated chronic liver disease (DCLD) represents an advanced stage of cirrhosis characterized by life-threatening complications such as portal hypertension, variceal haemorrhage, ascites, infections, and hepatic insufficiency. We report the case of a 46-year-old male with a history of chronic alcohol use and poorly controlled type 2 diabetes mellitus who presented with hematemesis and haematochezia. Clinical examination revealed tachycardia with stable oxygen saturation. Laboratory investigations demonstrated anaemia, hyperbilirubinemia, hypoalbuminemia, and elevated blood urea, indicating impaired hepatic synthetic function and ongoing blood loss. Ultrasonography of the abdomen showed altered hepatic echotexture, surface irregularity, portal vein dilatation, and moderate ascites, findings consistent with cirrhosis and portal hypertension. Additionally, lower limb imaging revealed diffuse cellulitis with a subcutaneous collection, suggesting a superimposed infection that likely precipitated acute decompensation. Scrotal ultrasonography also demonstrated bilateral hydrocele. The patient was managed with broad-spectrum antibiotics, beta-blockers, proton pump inhibitors, insulin therapy, and supportive measures aimed at controlling bleeding, infection, and metabolic imbalance. This case highlights the complex and multifactorial nature of DCLD, where infection, metabolic comorbidities, and portal hypertension interact to accelerate clinical deterioration. Early recognition of precipitating factors, timely initiation of guideline-directed therapy, and appropriate use of imaging modalities are essential for improving patient outcomes. A multidisciplinary approach, including infection control, hemodynamic stabilization, and consideration for liver transplantation, remains critical in reducing morbidity and mortality in advanced liver disease.

Key words: Decompensated chronic liver disease (DCLD); Cirrhosis; Gastrointestinal bleeding; Portal hypertension; Cellulitis; Alcohol-related liver disease; Ascites; Ultrasonography

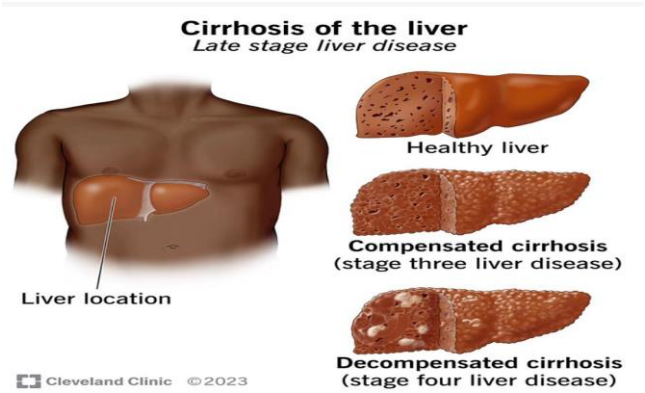

Figure 1: The stages to decompensated liver disease. As the liver is damaged in cirrhosis builds up, the liver function declines and symptoms progress.

Introduction: After a protracted period of inflammation, fibrotic tissue and regenerating nodules replace the healthy liver parenchyma, resulting in cirrhosis and portal hypertension [(1)]. The condition progresses from compensated cirrhosis, which is asymptomatic, to decompensated cirrhosis, which is symptomatic [(2)]. Decompensated chronic liver disease is a progressive condition that can cause portal hypertension, hepatorenal syndrome, aortopulmonary hypertension, ascites, spontaneous bacterial peritonitis (SBP), variceal hemorrhage, and cardiomyopathy [(3,4)]. If left untreated, these symptoms can lead to end-stage liver failure that necessitates liver transplantation [(5)].

Nearly 4.5 million persons in the US suffer from cirrhosis and chronic liver disease (CLD), which is the eleventh most prevalent cause of mortality.1 Between 2000 and 2015, the death rates associated with cirrhosis and CLD rose by 31% [(6)].

The most common causes of decompensated chronic liver disease include alcohol infection (Hepatitis C), non-alcoholic steatohepatitis [(7,8)]. If left untreated, acute decompensation can occur in patients with chronic liver disease for a variety of reasons. Any of the following issues could result from the decompensation which are, variceal bleeding in the esophagus, ascites, hepatic encephalopathy, hepatorenal syndrome, spontaneous bacterial peritonitis and Hepatocellular cancer [(9,10)].

Early detection and staging of liver disorders is essential for lowering healthcare expenses and Hepatocellular Carcinoma (HCC) related mortality [(11)]. Numerous computational techniques have been created for the radiological diagnosis of HCC and chronic liver disease [(12)]. Due to its exceptional performance on disease detection and prognosis, Machine Learning and Deep Learning techniques have drawn a lot of attention among the different possibilities because ultrasound (US) is easily accessible, non-invasive, well-tolerated, less expensive than its CT or MRI counterparts, allows for real-time image acquisition and display, and exempts patients from the negative effects of intravenous contrast or radiation, it is usually the first radiological study performed on patients suspected of having cirrhosis[(13)]. Grayscale US can show changes in the tissue composition of a cirrhotic liver [(14)].

Case Presentation: A 46-year-old male patient complained of having fresh blood in his stool since the afternoon, as well as one morning bout of vomiting that happened right after eating. Three haematochezia episodes with roughly 75–100 millilitres of fresh blood each were recorded by him. The bleeding was painless and happened while defecating normally. There were no related anal growths. The patient denied experiencing palpitations, dizziness, exhaustion, shortness of breath, chest pain, or trouble passing faeces. No traumatic history existed. He also mentioned a single instance of hematemesis that went away on its own as well as bilateral oedema on his left lower limb. He has a noteworthy medical history of type 2 diabetes mellitus, for which he is not now taking medication, and decompensated chronic liver disease (DCLD), for which he was previously hospitalized. The patient is an alcoholic.

On physical examination the patient showed the following results on admission:

Vital Investigation	Results
B. P	150/90mmHg
Pulse Rate	140bpm
Temperature	98.4°F
Respiratory Rate	30cpm
SpaO2	99%@RA

Table 1: physical examination values.

Following this, laboratory investigations were carried out to assess the patient's clinical abnormalities, which revealed the following findings:

Investigation	Results	Normal Range
Hemoglobin	10.2gm/dl	12-15 gm/dl
PCV	29.9%	40-50%
RBC	2.87Million/Cu.mm	4.5-5.5Million/Cu.mm
Lympocytes	08%	20-40%
Total bilirubin	4.8mg/dl	0.2-1.2mg/dl
Direct bilirubin	2.8mg/dl	0-0.3mg/dl
SGOT	58IU/L	5-40IU/L
Albumin	1.7g/dl	3.5-5.2g/dl
Blood urea	54mg/dl	12.6-42.6mg/dl

Table 2: abnormal laboratory investigations observed.

Upon radiological evaluation, ultrasonography (USG) of the left lower limb revealed diffuse skin thickening with subcutaneous oedema involving the entire lower leg, demonstrating a characteristic cobblestone appearance, predominantly over the anterior aspect. Additionally, an ill-defined subcutaneous heterogeneous collection with internal moving echoes was noted in the anterolateral region of the left leg, with associated skin thickening measuring approximately 1.5 cm, suggestive of an infective collection.

Ultrasonography of the scrotum demonstrated the presence of a right-sided funicular hydrocele along with a left-sided hydrocele. A bilateral lower limb arterial and venous Doppler study showed diffuse subcutaneous oedema consistent with cellulitis, along with bilateral inguinal lymphadenopathy. There was also evidence of mild incompetence of the right saphenofemoral junction.

Ultrasound examination of the abdomen revealed an altered hepatic echo pattern with surface irregularity, a dilated portal vein, and moderate ascites, findings suggestive of chronic liver disease.

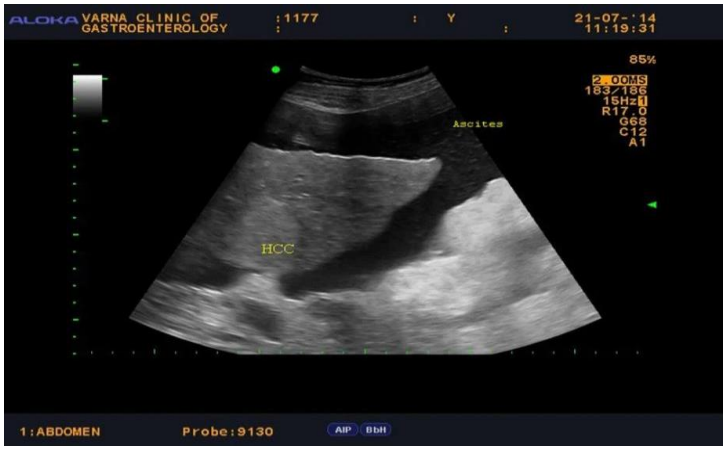

Figure 2: Results of an ultrasonography of the abdomen in a patient with decompensated viral cirrhosis

Based on the clinical presentation, laboratory investigations, and imaging findings, the patient was diagnosed with decompensated chronic liver disease complicated by hematemesis.

The patient was prescribed with the following treatment:

DRUG	DOSE	ROUTE OF ADMINISTRATION	FREQUENCY	INDICATIONS
Inj Ceftriaxone	1gm	IV	BD	Antibiotic
Tab Carvedilol	3.125mg	PO	OD	Anti-hypertensive
Tab diltiazem	90mg	PO	BD	Anti-hypertensive
Hepamez Sachets	3gm	PO	BD	Treat liver cirrhosis
Tab Rifaximin	550mg	PO	BD	Antibiotic
Tab Thiamine	100mg	PO	BD	Treat heart failure and metabolic disorders
Tab Pantoprazole	40mg	PO	OD	PPI
Inj Human Actrapid		S/C	According sliding scale	Antidiabetic
Syp Sucralfate	10ml	PO	TID	Treat gastric ulcer
Inj Tranexamic Acid	500mg	IV	BD	Prevent excessive bleeding

Table 3: patient prescribed treatment plan.

Discussion:

An advanced stage of cirrhosis known as decompensated chronic liver disease (DCLD) is marked by problems such ascites, variceal haemorrhage, hepatic encephalopathy, and infections. The patient in this instance had ascites, hypoalbuminemia, hyperbilirubinemia, gastrointestinal bleeding, and other classic signs of decompensation. These results align with the natural course of cirrhosis, which is characterized by fibrosis, architectural distortion, and portal hypertension as a result of prolonged liver damage [15].

One of the most dangerous side effects of portal hypertension is gastrointestinal bleeding, especially hematemesis, which is typically brought on by gastric or oesophageal varices. About 30% of cirrhosis patients experience variceal bleeding, which has a high morbidity and death rate [16]. An acute upper gastrointestinal bleed, most likely caused by portal hypertension, is suggested by the patient's presentation of hematemesis, haematochezia, tachycardia, and anaemia.

One of the main causes of cirrhosis in this patient is chronic alcohol use. One of the main causes of cirrhosis worldwide is still alcohol-related liver disease, which is closely linked to the development of decompensated illness, especially in middle-aged men [17]. Furthermore, poorly managed type 2 diabetes may have accelerated hepatic injury by contributing to the disease's progression through inflammatory and metabolic pathways.

The increased bilirubin, low albumin, anaemia, and increased blood urea are among the laboratory results that are consistent with decreased hepatic synthetic function and potential continuous blood loss [18]. This patient's hypoalbuminemia, which is a known indicator of a bad prognosis, demonstrates both systemic inflammation and impaired hepatic production.

Ascites, surface nodularity, portal vein dilatation, and changed hepatic echotexture are radiological signs of portal hypertension and cirrhosis. Due to its affordability, accessibility, and safety, ultrasonography continues to be the primary imaging modality; nevertheless, in the early stages of a disease, its sensitivity may be limited [19].

The occurrence of cellulitis with subcutaneous collection, which probably served as a trigger for rapid decompensation, is a noteworthy feature of this case. Immune failure linked to cirrhosis puts patients at risk for bacterial infections, which can affect up to 30–50% of hospitalized cirrhotic patients and greatly raise mortality. Acute decompensation and acute-on-chronic liver failure are also known to be triggered by infections [20].

Along with these conclusions, this case illustrates a number of crucial DCLD care factors that may enhance patient outcomes. Current guidelines recommend early initiation of vasoactive therapy, such as Octreotide, along with prophylactic antibiotics, such as Ceftriaxone, and urgent endoscopic evaluation, all of which have been shown to reduce mortality and rebleeding rates. The presentation with hematemesis and haematochezia strongly suggests variceal bleeding.

Decisions on intensive care and early referral for transplantation would have been guided by early prognostic stratification using proven scoring methods like the Model for End-Stage Liver Disease, which would have given important insight into the severity of the disease. Additionally, as infections greatly impair outcomes in cirrhotic patients, the appearance of cellulitis requires the early introduction of broad-spectrum antibiotics and proper source control.

Recent developments in imaging, such as the use of deep learning and artificial intelligence in ultrasonography, have demonstrated promise in improving the early detection and staging of hepatocellular carcinoma and chronic liver disease, possibly improving patient outcomes through earlier intervention [21].

The intricate interactions between portal hypertension, infection, metabolic comorbidities, and persistent liver damage that led to decompensation are highlighted by this case. Improving the short-term and long-term outcomes of patients with decompensated cirrhosis requires a thorough, multidisciplinary, and guideline-directed strategy that emphasizes early detection, timely treatment of complications, and management of underlying risk factors.

Conclusion:

This case demonstrates the complicated and multifaceted character of decompensated chronic liver disease, where abrupt clinical deterioration is caused by a combination of metabolic comorbidities, infection, and portal hypertension. The patient's presentation of superimposed soft tissue infection, ascites, and gastrointestinal bleeding highlights the significance of early identification of triggering events that can hasten decompensation. Reducing morbidity and mortality requires fast application of guideline-directed management, which includes aggressive treatment of infections, timely control of variceal haemorrhage, and adequate management of ascites.

The importance of early prognostic evaluation, organized monitoring, and prompt referral for liver transplantation in patients with advanced liver disease is further highlighted by this instance. In order to stop the course of the disease, it is still crucial to address modifiable risk factors, including alcohol consumption and poor glycaemic control. Patient outcomes may be further improved by earlier detection and better risk stratification, given the expanding importance of advanced imaging and new technologies. In general, early intervention and comprehensive care can greatly impact both short-term stabilization and long-term survival, making a multidisciplinary and proactive approach essential in the management of decompensated cirrhosis.

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