

Case Report

Acute liver failure secondary to chronic lymphocytic leukemia

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ABSTRACT

Chronic lymphocytic leukemia is an indolent malignancy of lymphocytes commonly seen in elderly. Dissemination of malignancy is a rare cause of acute liver failure and has rarely been reported. Here we describe the case of a sixty five year old male presenting with acute liver failure. He is a diabetic with no known history of seropositive hepatitis, drug intake or any addictions. Bone marrow and liver biopsy suggestive of chronic lymphocytic leukemia. He eventually succumbed to his illness.

Keywords: Acute liver failure, Chronic lymphocytic leukemia, Fulminant hepatic failure

INTRODUCTION

Acute liver failure is a condition in which there is rapid deterioration of liver function resulting in altered sensorium and deranged coagulopathy. Common causes of acute liver failure include drug induced injury, viral hepatitis, autoimmune hepatitis, shock and mostly idiopathic.¹ Acute liver failure secondary to lymphomatous infiltration of leukemia is quite uncommon.² Hematological malignancies frequently involve the liver during the course of dissemination but rarely do they present with acute liver failure.³ Mortality is higher in such presentations of malignant infiltration with diagnosis often being made on postmortem examinations. Only treatment modality in these cases is to identify cause of liver failure and proceed for palliation after ruling out necessary contraindications.⁴ Here we would like to discuss a case of 65 year old male who presented as a case of acute liver failure and was diagnosed with chronic lymphocytic leukemia with liver infiltrates. This case demonstrates the need for urgent liver biopsy in a patient with acute liver failure.

CASE REPORT

A 65-year-old male was admitted to emergency with complaints of yellowish discoloration of skin and sclera for 12 days along with progressive distension of abdomen, bilateral lower limb swelling for 10 days, hemoptysis (2-3 episodes) for 1 day and alteration of behavior since 1 day. He also complained of progressive difficulty in breathing in view of his rapid abdominal distension since the last 7 days and also complained of reversal of day-night sleep cycle. He had no history of fever, chest pain, abdominal pain, nausea, burning micturition, weight loss. The patient has no past known history of any previous blood transfusion, intake of herbal medicines, any tattoo or intravenous drug abuse. He was seronegative for hepatitis B and hepatitis C. He was a non-alcoholic and non-smoker. This patient had been on follow up for last 10 years seeking treatment for type 2 diabetes mellitus in our hospital. His past records were insignificant except for mild fatty changes in the ultrasound of the liver and type 2 diabetes for which he was taking insulin regular 8 units pre-meal and glargine insulin 14 units at bedtime. There is no significant history

of cancer, diabetes and hypertension in the family. He was a known alcoholic consuming three-four glasses of whiskey daily however had stopped after being diagnosed with diabetes and had been sober since then. (10 years).

On examination, his BP was 90/68mmHg supine position and was given bolus one liter IV fluids and repeat BP was 100/70 mmHg. He was afebrile at time of examination but had tachypnoea and tachycardia with SpO₂ of 93 percent. He had mild scleral icterus with dark yellowish discoloration of urine. He showed marked confusion, asterixis with constructional apraxia with slurring of speech. His Glasgow coma scale was-E3V4M5. He had mild to moderate ascites and splenomegaly 6cm below the left costal margin in left midclavicular line. Other systemic examination was within normal limits

Course and treatment

Patient was started on Inj. cefotaxime 2g IVI 8 hourly, Inj. Vitamin K 2cc ivi 8 hourly with 550mg of tablet rifaximin 12 hourly and syrup lactulose 45ml 8 hourly and injection insulin for sugars. He was shifted onto piperacillin tazobactam 4.5g IVI 8 hourly due to lack of improvement on cefotaxime keeping in mind pending ascitic fluid culture reports, was also transfused with 8 units of fresh frozen plasma and 3 units packed red blood cells. He developed complete alteration of sensorium on day 5 and GCS was E2V1M3. USG whole abdomen done revealed: moderate peritoneal collection with bilateral effusion with mildly coarse liver echo-texture with portal vein diameter 12mm and normal splenic vein diameter with moderate splenomegaly. He had persistent pancytopenia.

This was initially attributed to hypersplenism however a bone marrow biopsy done in view of pancytopenia and recurrent fever showed chronic lymphocytic leukemia. A transjugular biopsy of liver was done which revealed atypical lymphocytic infiltration in the liver parenchyma with altered nuclear to cytoplasmic ratio along with cholestasis. The patient's guardians were advised palliative chemotherapy however chemotherapy (fludarabine, cyclophosphamide) was deferred from the relative's side.

His progressive jaundice and hepatic encephalopathy showed no signs of improvement on treatment. The INR was deranged on admission and continued to deteriorate even after transfusion of 8 packs of fresh frozen plasma. His pancytopenia showed no improvement. He was diagnosed with chronic lymphocytic leukemia on day 7 of admission on bone marrow examination. His clinical and laboratory status deteriorated, He further developed 4-5 episodes of hematemesis. He aspirated on the same and was intubated in view of low oxygen saturation due to aspiration pneumonitis and shifted to ICU. He was started on terlipressin 2 g stat followed by 1g ivi 6 hourly and pantoprazole infusion. He eventually succumbed to aspiration pneumonitis on day 12.

Investigation

Hb-8g/dL TLC-1400/L DLC-N54L30 MCV-89 MCH-31 PLT-10x10⁹/L RBS-151 Na-141mmol/L K-5.5mmol/L Urea:68mmol/L-Creat-1.4mg/dl Total Bilirubin-10mg/dL Direct Bilirubin-7.1mg/dL, SGOT-115IU/L, SGPT-325IU/L, ALP-542IU/L, SPRO-7.2, SALB-2.1g/dL PT-25 INR-2.1 Viral markers: NON-REACTIVE. Transferrin saturation was 51%, Serum ceruloplasmin was within normal limits done revealed: pH:7.2 Lactate :4 Bicarbonate:22mmol/L Pao₂:68mmHg and Paco₂: 44mmHg. Patient was negative for antimitochondrial antibodies, anti-smooth muscle antibodies and antinuclear antibodies was 2+ with expanded nuclear antigen profile negative. Serum ammonia levels were 125micro mol/L. Negative serology for hepatitis A, hepatitis E, ebstein barr virus and cytomegalovirus. Screening for elevated levels of acetaminophen was negative. Peripheral blood smear showed reticulocyte count as 1% with sparsely distributed neutrophils and microcytic hypochromic red blood cells and elevated platelet count. Bone marrow (Figure 1) was done in view of persistent pancytopenia which revealed chronic lymphocytic leukemia. A transjugular biopsy of liver (Figure 2) (Figure 3) was done which revealed atypical lymphocytic infiltration in the liver parenchyma with altered nuclear to cytoplasmic ratio along with cholestasis. USG Whole abdomen done revealed: moderate peritoneal collection with bilateral effusion with coarse liver echo-texture with portal vein diameter 12mm and normal splenic vein diameter with moderate splenomegaly. Collaterals were not commented on. Diagnostic paracentesis in left quadrant revealed: ascitic fluid TLC:500 DLC: N10L90 PROTEIN:1.9 ALBUMIN:3.4 SAAG:1.5.

Differential diagnosis

- Drug induced liver failure
- Acute fulminant hepatitis
- Autoimmune hepatitis

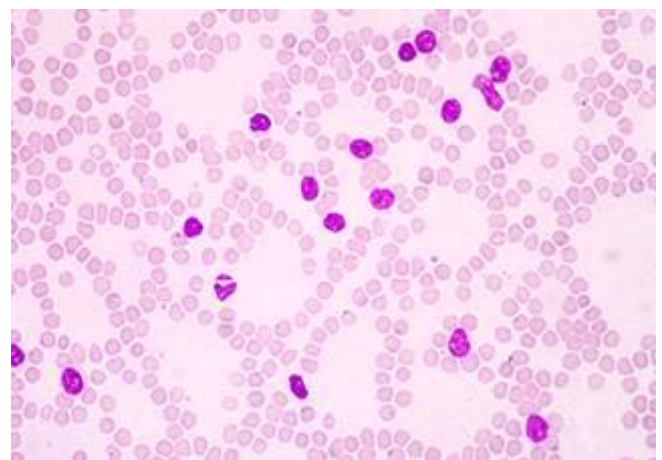


Figure 1: Peripheral blood smear of patient showing atypical lymphocytes suggestive of chronic lymphocytic leukemia

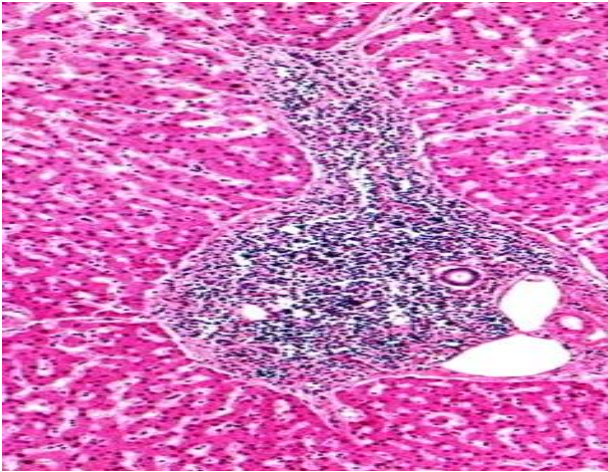


Figure 2: Liver biopsy imaging showing histopathology of infiltration of mature malignant lymphocyte in portal tracts and liver parenchyma.

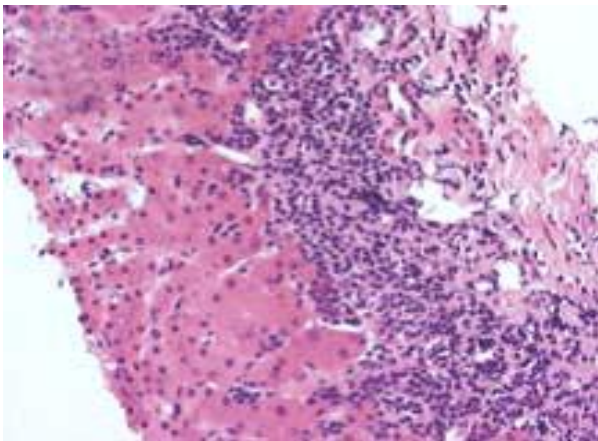


Figure 3: Liver biopsy imaging showing histopathology of infiltration of mature malignant lymphocyte liver parenchyma.

DISCUSSION

Acute liver failure is associated with high mortality and requires treatment in the form of liver transplantation in most cases unless contraindications for same exist. Common causes include autoimmune and infectious and hepatotoxic drugs and rarely malignancies. Here we discuss a case of acute liver failure caused by a malignant lymphocytic infiltration of the liver by a hematological malignancy.⁵ Hematological malignancies tend to involve the lymph nodes and subsequently liver and spleen in their course of dissemination.⁶ In a study done by D rowbotham et al, 18 cases of hepatic infiltration of lymphoma were identified, 17 of them succumbed to acute liver failure thereby proving that only with accurate histological diagnosis from liver biopsy the management of such patients can be improved and an attempt at best chance of recovery be made.⁷ Chronic lymphoid leukemia is an indolent malignancy of unknown etiology and is associated with prolonged survival and is rarely associated with infiltration of liver and hepatic dysfunction. It accounts for 7 percent of non-

Hodgkin lymphoma and is mostly diagnosed incidentally during investigations.⁸ It commonly presents with fatigue which is also the most common presentation in chronic liver disease.⁹ CLL tends to be silent in a patient and present as harmless lymphadenopathy. CLL cells are positive for CD 5 and CD 23. Despite being popular for its indolent course, these cancers can transform into high grade non-Hodgkin Lymphoma described classically as Richter syndrome.¹⁰ In this paper we highlight how acute liver failure can occur due to hematological malignancy and apart from discussing the rarity of this phenomenon warranting its reporting we also wish to highlight the use of biopsy in acute liver failure in finding this diagnosis. Given the patients history of diabetes and alcoholism patient could have had alcoholic hepatitis or non-alcoholic steato-hepatitis as it being in common occurrence; we also would like to press on the need to keep an eye out for the occasional rarity of an indolent hematological malignancy and use biopsy as a method to aid the diagnosis.

CONCLUSION

The unusual presentation of an indolent haematological malignancy as aggressive liver disease. Recommendation of the use of liver biopsy to aid diagnosis in acute liver failure when the cause is uncertain. Not all pancytopenia associated in chronic liver disease patients are secondary to hypersplenism thus peripheral smear must be done in all cases of chronic liver diseases.

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