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CASE REPORT

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Acute B-cell lymphoblastic leukaemia presenting as acute hepatitis

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Abstract

Acute lymphoblastic leukaemia (ALL) typically presents with symptoms of bone marrow failure. Acute hepatitis is a rare and atypical presentation. We report the case of a 16-years old healthy boy, who presented with elevated liver enzymes and jaundice. His Initial investigations ruled out common infectious aetiology of hepatitis. Despite symptomatic management, the patient had persistent bicytopenia, prompting further evaluation with bone marrow biopsy which confirmed CD10-positive B-cell ALL. This case report illustrates the importance of a pragmatic approach to a non-resolving hepatitis.

Keywords: acute B-cell lymphoblastic leukaemia, acute hepatitis

Introduction

Acute lymphoblastic leukaemia (ALL) occurs commonly in children aged 2-10 years and is rarely seen in adults.(1) It usually presents with symptoms and signs of bone marrow failure and hematopoietic infiltration like fever, recurrent infections, and excessive bruising. Though ALL is generally thought to originate in the bone marrow, leukaemic blasts are often seen systemically and it involves the reticuloendothelial system including bone marrow, thymus, liver, spleen, lymph nodes, testes, and central nervous system.(2)

ALL could present with hepatitis , more specifically with hyperbilirubinaemia due to leukaemic infiltration of the liver which is an extremely rare manifestation. (1) In rare instances ALL can also present with fulminant hepatic failure.(3) However altered liver biochemistry is observed frequently during therapeutic interventions in ALL.

Here we report the case of a 16-year-old boy who initially presented with acute cholestatic hepatitis and was subsequently diagnosed with CD 10 positive acute B-cell lymphoblastic leukemia.

Case presentation

A 16-year-old previously healthy boy presented with a 4 day history of intermittent right hypochondriac pain, yellowish discoloration of eyes and palms, pale stool and dark urine. There was no history of fever or loss of weight but he reported loss of appetite. He denied any travel history, consumption of undercooked food or unfiltered water, diarrhoea, substance abuse, alcohol consumption, night sweats, history of blood transfusion, needle prick injury, and history of tattooing or high risk sexual behaviours. There was no history of bleeding manifestation or family history of liver disease or leukaemia.

On arrival, his vital signs were stable with a heart rate of 90 bpm and blood pressure of 100/60 mmHg. Further, his physical examination revealed deep icterus with hepatomegaly, whilst his other systemic examinations remained unremarkable. His initial blood investigations are summarized in table 1 below.

Ultrasound scan of the abdomen revealed features consistent with hepatitis including hepatomegaly and gallbladder wall congestion. The initial blood

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Table 1 - Initial investigations

Blood investigation	Result	Reference range
WBC (x10^9 g/L)	1.09	(4-10)
Haemoglobin (g/dL)	10.5	(13-17)
MCV (fL)	87.1	(80-100)
Platelets (x10^9 g/L)	130	(150-400)
Neutrophil (%)	0.9	(37-72)
Lymphocyte (%)	88.1	(20-50)
Monocyte (%)	10.1	(0-14)
AST (U/L)	383	(15-37)
ALT (U/L)	2281	(16-63)
Alkaline phosphatase (IU/L)	444	(34-104)
GGT (U/L)	135	5-37
Total protein (g/dL)	56	(64-82)
Albumin (g/dL)	33	(34-50)
Total Bilirubin (micromol/L)	222	(0-17.1)
Direct Bilirubin (micromol/L)	142	(0-6.8)
LDH (U/L)	220	(120-246)
Uric acid (micromol/L)	199	(208-506)
INR	1.09	(0.8-1.2)
Serum creatinine (micromol/L)	40	(62-115)

investigations revealed moderate thrombocytopaenia and neutropaenia raising suspicion of an infective aetiology.

Further evaluation done to exclude the other causes of liver injury, yielded negative results for hepatitis A,B,C,D,E , cytomegalovirus (CMV) and Epstein-Barr virus (EBV)panel. The antinuclear antibody levels and urine toxicology test were also negative.
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In the following days his liver enzymes continued to rise, reached a peak and gradually declined. Though he showed clinical improvement in jaundice, his full blood count continued to show pancytopaenia. Repeat blood film examination revealed pancytopaenia with large cells which prompted a bone marrow biopsy and flow cytometry leading to the diagnosis of CD 10 positive acute B-cell lymphoblastic leukaemia as shown in figures.1 and 2 Despite specialized onco-haematology care, the patient succumbed to severe neutropaenic sepsis.

Discussion

ALL is rarely seen in young adults and is associated with poor prognosis demonstrating survival rates ranging from 20%–40%.(1) The extramedullary form of this disease is rare, and it commonly involves the bones, followed by soft tissue, skin and lymph nodes. (4) Common symptoms of ALL include fever, night sweats, weight loss and fatigue. Lymphadenopathy, hepatomegaly and splenomegaly can occur due to extra medullary involvement in up to 20% of patients.

Though liver involvement in ALL can be a frequent phenomenon, hyperbilirubinemia is not frequently observed.(5) The pathophysiology of obstructive jaundice in patients with ALL includes mechanisms, ranging from obstruction of the biliary duct, formation of bile duct strictures, and infiltration of hepatic sinusoids by tumour cells.(6) Acute liver failure is extremely rare and has been reported predominantly in the paediatric population and less often in the adult population.(7)

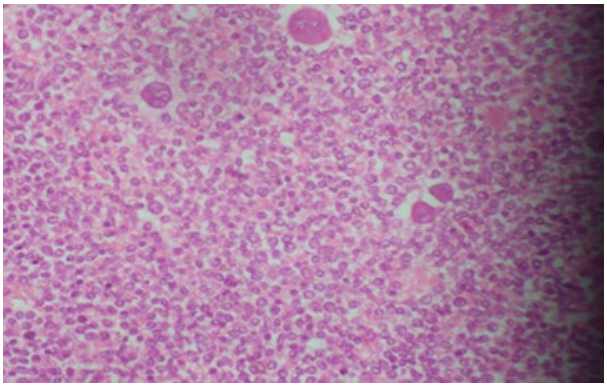

Figure 1 - Bone marrow trephine biopsy shows diffuse interstitial infiltration of blasts with suppression of normal haemopoiesis

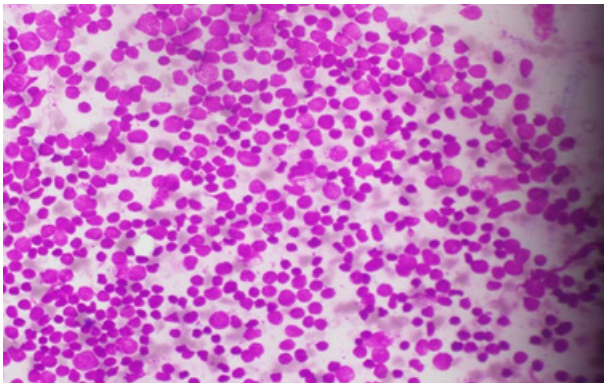

Figure 2 - Bone marrow aspirate shows more than 80% of marrow nucleated cells, medium sized blasts with high nuclear cytoplasmic ratio, diffuse nuclear chromatin pattern and indistinct nucleoli resembling lymphoblast

This case highlights an uncommon and challenging presentation where ALL manifested with features of acute cholestatic hepatitis, including elevated liver enzymes, jaundice, and hepatomegaly, leading to an initial suspicion of viral hepatitis, hindering the diagnosis of the underlying leukaemia. Neutropaenia, as seen in this patient, increases the suspicion of infective aetiology, including those that can involve the liver, but it is important to have a high index of suspicion of ALL. Although primary presentation with hepatitis is rare, we emphasize the need for considering haematologic malignancies in patients with unexplained cytopaenias and concurrent liver dysfunction.

Conclusion

Although acute lymphoblastic leukemia is more common in children than adults, this case illustrates the importance of clinical vigilance and thorough diagnostic evaluation in a young man presenting with hepatitis when the usual cases are ruled out. Acute lymphoblastic leukaemia, although primarily a hematological disorder, can present with atypical extramedullary signs, including acute cholestatic hepatitis. Early recognition and diagnosis are critical for prompt initiation of therapy and improving outcomes of such treatable potentially fatal conditions.

Declarations

Author contributions
All authors contributed to data interpretation and writing the manuscript. All authors read and approved the final manuscript.

Conflicts of interest
The authors declare that they have no conflicts of interest

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