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Chylous Ascites as a Presentation of Early T-Precursor Acute Lymphoblastic Leukaemia: A Case Report

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Abstract
Chylous ascites is an uncommon cause of abdominal distension resulting from the accumulation of triglyceride-rich lymphatic fluid within the peritoneal cavity. Malignancy, particularly lymphoma, is a major non-traumatic etiology; however, the presentation of lymphoblastic neoplasms with chylous ascites as an initial feature is rare and may delay diagnosis. We describe an 18-year-old previously healthy woman who presented with progressive abdominal distension and generalized lymphadenopathy. Paracentesis demonstrated milky ascitic fluid with elevated triglyceride levels, confirming chylous ascites. Cross-sectional imaging revealed extensive lymphadenopathy with hepatosplenomegaly, raising suspicion for a lymphoproliferative disorder. Excisional lymph node biopsy established T-lymphoblastic lymphoma with a high proliferative index. During follow-up, the patient developed pancytopenia with circulating blasts, and bone marrow examination demonstrated infiltration with immunophenotypic features consistent with early T-precursor acute lymphoblastic leukemia (ETP-ALL). This case highlights chylous ascites as an unusual initial manifestation of T-lymphoblastic lymphoma and emphasizes the continuum between lymphoblastic lymphoma and acute lymphoblastic leukemia.

Keywords: chylous ascites, diffuse lymphadenopathy, early t precursor all, hematologic malignancy, lymphoblastic neoplasm, t-lymphoblastic lymphoma

Introduction

Chylous ascites is an uncommon clinical entity characterized by the accumulation of triglyceride-rich lymphatic fluid within the peritoneal cavity, typically resulting from disruption or obstruction of lymphatic flow [1]. Although it may arise from a variety of etiologies, including trauma, cirrhosis, and infections, malignancy remains one of the most significant non-traumatic causes, particularly in adults [2]. Among malignancies, lymphomas are the most common cause, owing to their propensity to infiltrate and obstruct abdominal lymphatic channels [3].

Lymphoblastic neoplasms, encompassing T-lymphoblastic lymphoma (T-LBL) and T-cell acute lymphoblastic leukaemia (T-ALL), represent a spectrum of aggressive immature T-cell malignancies that

share overlapping biological and clinical features [4]. T-LBL typically presents with a mediastinal mass or lymphadenopathy, whereas T-ALL is defined by bone marrow involvement of ≥25% lymphoblasts [5]. A distinct subtype, early T-precursor acute lymphoblastic leukaemia (ETP-ALL), has been increasingly recognized for its unique immunophenotypic profile and poorer prognosis compared to conventional T-ALL [6].

The presentation of lymphoblastic neoplasms with chylous ascites is exceedingly rare and may obscure the underlying diagnosis, leading to potential delays in recognition and management [7]. The presence of chylous effusions in such cases reflects extensive lymphatic involvement and may indicate advanced or aggressive disease biology. Furthermore, the overlap between T-LBL and ETP-ALL highlights the continuum of disease rather than distinct pathological entities [6].

In this report, we describe a young patient who initially presented with chylous ascites and was subsequently diagnosed with T-lymphoblastic lymphoma, later demonstrating features consistent with ETP-ALL. This case underscores the importance of considering lymphoblastic malignancies in atypical presentations of chylous ascites and highlights the diagnostic challenges associated with this rare clinical scenario.

Case Presentation

An 18-year-old woman with no prior medical illness presented with a two-month history of progressive abdominal distension, associated with reduced exercise tolerance and bilateral lower limb edema. She also noted multiple painless swellings over the neck and axillary regions during the same period. She denied fever, weight loss, night sweats, bleeding manifestations, or changes in bowel habits. There was no significant family history of malignancy.

She was subsequently referred to the emergency department due to worsening abdominal distension and increasing respiratory discomfort. On presentation, she was alert but tachypneic. Physical examination revealed generalized lymphadenopathy involving the cervical, supraclavicular, axillary, and inguinal regions. The largest cervical lymph node measured approximately 3 × 4 cm, while the largest inguinal node measured 5 × 4 cm. Respiratory examination demonstrated reduced breath sounds with dullness to percussion over the right lower lung field, consistent with pleural effusion. Abdominal examination showed marked distension with clinical evidence of ascites.

Initial laboratory evaluation demonstrated anemia and thrombocytopenia. Peripheral blood smear showed microcytic hypochromic anemia with elliptocytes. The white blood cell differential revealed neutropenia with relative lymphocytosis and reactive lymphocytes, without identifiable abnormal lymphoid cells. A summary of the laboratory findings is provided in Table 1.

Table 1. Summary of Initial Laboratory Investigations at Presentation

Parameter	Value	Reference Range
Haemoglobin (g/L)	99	117—157
Total white blood cell count (x10^9)	4.47	4.50—12.50
Platelet count (x10^9)	115	150—410
Neutrophils (%)	20.8	40.0—80.0
Lymphocytes (%)	76.1	20.0—40.0
Serum albumin (g/L)	35	35—50
Lactate dehydrogenase (U/L)	194	125—220

Therapeutic thoracentesis drained approximately 1.7 L of straw-colored pleural fluid. Subsequent diagnostic paracentesis yielded approximately 6 L of turbid fluid with a milky component (Figure 1).

Figure 1. Gross Appearance of Turbid, Milky Ascitic Fluid Consistent with Chylous Ascites

Biochemical analysis of the ascitic fluid demonstrated an elevated triglyceride level of 1.9 mmol/L (168 mg/dL). The serum-ascitic albumin gradient (SAAG) was 7g/L, suggesting a non-portal hypertensive etiology. Additional parameters summarized in Table 2.

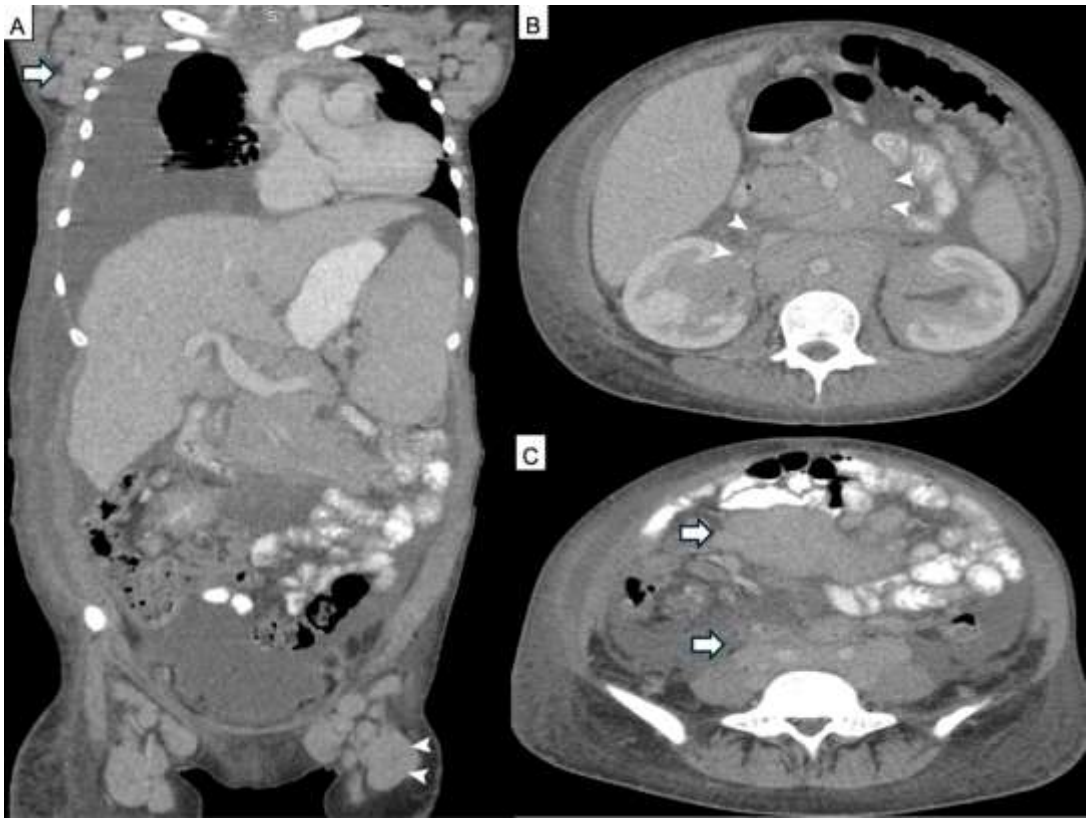
Table 2. Biochemical and Cytological Parameters of Ascitic Fluid

Parameter	Result	Unit
Appearance	Turbid with milky component, blood-tinged	—
Triglyceride	1.9	mmol/L
Protein	70	g/L
Albumin	28	g/L
Cytology	No malignant cells seen	—

Culture and sensitivity	No growth	—
AFB (acid-fast bacilli)	No acid-fast bacilli seen	—
SAAG (serum-ascitic albumin gradient)	7	g/L

Contrast-enhanced computed tomography of the neck, thorax, abdomen, and pelvis revealed widespread lymphadenopathy involving the cervical, mediastinal, axillary, mesenteric, para-aortic, iliac, and inguinal regions. Hepatosplenomegaly and moderate ascites were also present, raising strong suspicion for an underlying lymphoproliferative disorder (Figure 2).

Figure 2. (A) Coronal contrast-enhanced CT showing hepatosplenomegaly and extensive lymphadenopathy (arrows). (B, C) Axial CT images demonstrating enlarged mesenteric and para-aortic lymph nodes (arrowheads) with associated ascites.

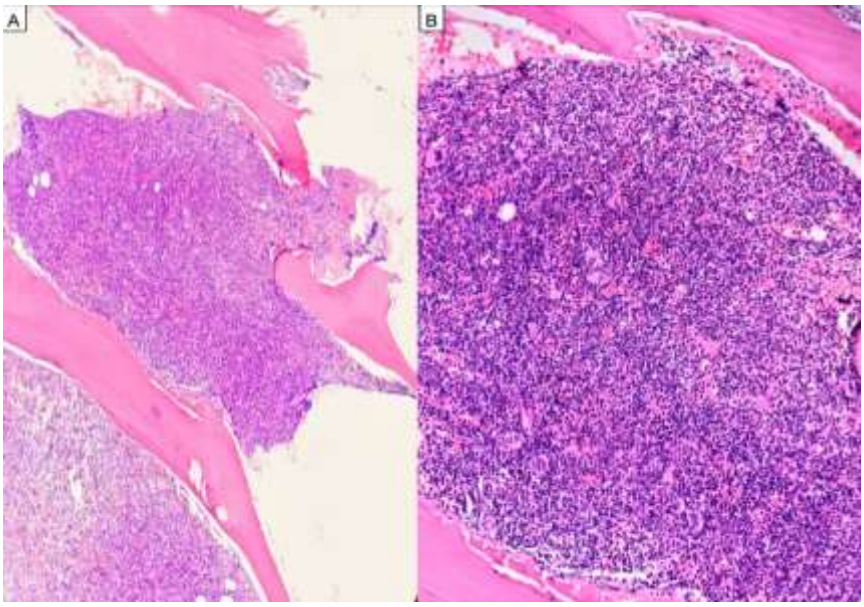


An excisional biopsy of the right inguinal lymph node demonstrated diffuse infiltration by intermediate-sized atypical lymphoid cells with a high nuclear-to-cytoplasmic ratio and frequent mitotic figures. Immunohistochemical analysis showed positivity for CD3, CD7, terminal deoxynucleotidyl transferase (TdT), and CD10, with co-expression of CD4 and CD8. The Ki-67 proliferation index was approximately 80%. These findings were diagnostic of T-lymphoblastic lymphoma.

Following histopathological confirmation, the patient was reviewed in the hematology clinic and noted to have recurrent symptomatic ascites. Repeat paracentesis drained approximately 2 L of milky fluid with a triglyceride level of 4.3 mmol/L (380.9 mg/dL). Concurrent laboratory evaluation demonstrated pancytopenia, and peripheral blood smear revealed approximately 8% circulating blast cells.

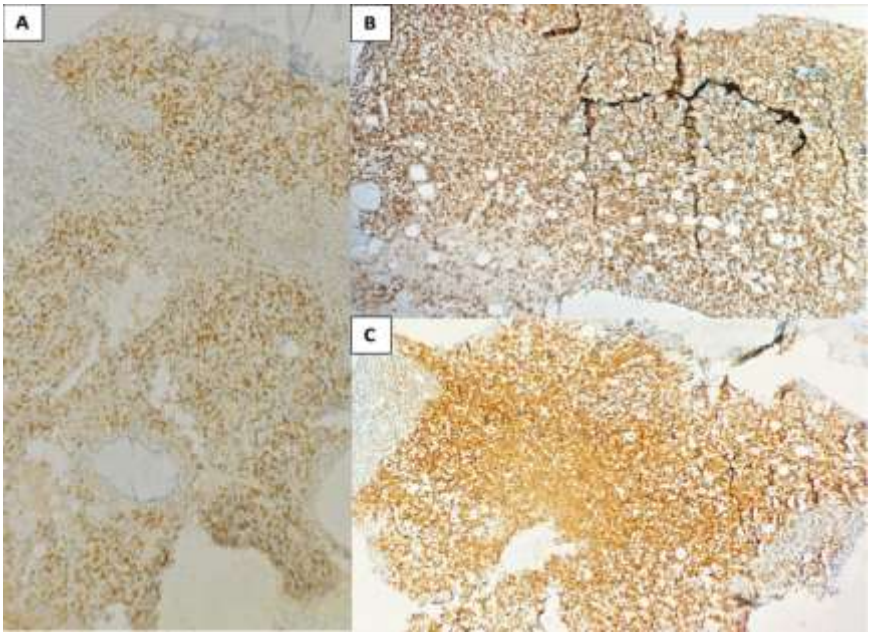
Bone marrow aspiration and trephine biopsy revealed a markedly hypercellular marrow with approximately 74% blast infiltration and suppression of normal hematopoiesis (Figure 3A–3B).

Figure 3. (A) Low-power view of bone marrow trephine biopsy showing a markedly hypercellular marrow with diffuse infiltration by atypical lymphoid cells. (B) High-power view demonstrating lymphoblasts with high nuclear-to-cytoplasmic ratio and fine chromatin.



Flow cytometric immunophenotyping demonstrated blasts expressing cytoplasmic CD3, CD7, CD2, dim CD5, dim CD99, partial TdT, and partial CD33. Histological examination of the trephine biopsy showed diffuse infiltration by blast cells, with immunohistochemistry demonstrating strong positivity for CD3 and CD99 and partial positivity for TdT (Figure 4A–4C). These findings were consistent with early T-precursor acute lymphoblastic leukemia.

Figure 4. Immunophenotypic characterisation of bone marrow infiltrate by immunohistochemistry. (A) TdT showing partial nuclear positivity. (B) CD99 showing dim membranous and cytoplasmic positivity. (C) CD3 showing diffuse membranous positivity.



Following confirmation of ETP-ALL, the patient was commenced on systemic chemotherapy using the MASPORE protocol, a pediatric-inspired regimen for acute lymphoblastic leukemia. She remains under hematology follow-up and has not experienced recurrence of chylous ascites since initiation of treatment.

Discussion

Chylous ascites arises from disruption or obstruction of abdominal lymphatic flow, leading to the accumulation of triglyceride-rich lymphatic fluid within the peritoneal cavity, typically characterized by a milky, opaque appearance and an ascitic fluid triglyceride level exceeding 200 mg/dL (2.3 mmol/L) [8]. It is an uncommon condition with a broad differential diagnosis, including trauma, congenital abnormalities, cirrhosis, infections, and malignancy. Among non-traumatic etiologies, malignancy-particularly lymphoma- accounts for a significant proportion of cases in adults [2,3]. In this setting, infiltration of lymphatic channels by malignant cells results in impaired lymphatic drainage and subsequent leakage of chyle into the peritoneal cavity [9,10].

In lymphoma, chylous ascites is most commonly attributed to direct lymphatic obstruction or increased lymphatic pressure secondary to extensive nodal disease [3]. Although it has been more frequently reported in indolent lymphomas, aggressive lymphoid malignancies may also present with chylous effusions, particularly in advanced disease [7]. Chylous ascites has been described in association with various lymphoma subtypes, including diffuse large B-cell lymphoma (DLBCL). However, its occurrence as a presenting feature of lymphoblastic neoplasms, particularly early T-precursor acute lymphoblastic leukemia (ETP-ALL), remains exceedingly rare.

T-lymphoblastic lymphoma (T-LBL) and T-cell acute lymphoblastic leukemia (T-ALL) are closely related entities that exist along a spectrum of immature T-cell malignancies [4,5]. The distinction between these entities is primarily based on the extent of bone marrow involvement, with ≥25% lymphoblasts defining T- ALL [5]. Early T-precursor acute lymphoblastic leukemia (ETP-ALL) is a distinct subtype characterized by a unique immunophenotypic profile and is associated with inferior clinical outcomes compared to conventional T-ALL [6].

This case highlights several important clinical considerations. First, chylous ascites may represent an atypical presenting feature of aggressive lymphoid malignancies, including T-LBL, potentially leading to misdiagnosis or delayed recognition, particularly in young patients without classical B symptoms. Second, the progression from T-LBL to ETP-ALL observed in this patient underscores the biological continuum between lymphoblastic lymphoma and leukemia, rather than representing entirely distinct disease entities. This evolution emphasizes the importance of close hematologic monitoring in patients with lymphoblastic malignancies.

Furthermore, the presence of chylous ascites in this context may reflect extensive lymphatic involvement and a high disease burden. Early recognition and prompt tissue diagnosis are essential to facilitate timely initiation of appropriate chemotherapy. In this patient, treatment with a pediatric-inspired regimen resulted in clinical improvement and resolution of chylous ascites, highlighting the importance of early intervention in altering disease trajectory.

Conclusion

This case highlights chylous ascites as an uncommon and atypical presentation of aggressive lymphoid malignancies. While it has been reported in association with various lymphoma subtypes, its occurrence as an initial manifestation of T-lymphoblastic lymphoma with subsequent progression to early T-precursor

acute lymphoblastic leukemia is exceedingly rare. This case underscores the importance of considering underlying hematologic malignancy in patients presenting with unexplained chylous ascites, particularly in the presence of lymphadenopathy. Early recognition and prompt diagnostic evaluation are essential to facilitate timely initiation of appropriate therapy and improve clinical outcomes.

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