Case Report

Diffuse peritoneal lymphomatosis or abdominal tuberculosis: a clinico-radiological conundrum

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ABSTRACT
Primary extra nodal lymphoma occurs in approximately 25% to 40% of patients and is more common in patients with NHL. Diffuse large B-cell lymphoma (DLBCL) is the dominant histological subtype. A 60-year-old gentleman presented to our hospital with pain abdomen associated with abdominal distension since a month, few constitutional symptoms with bilateral axillary and inguinal lymphadenopathy. Blood investigations showed high total count with significantly high LDH levels. Provisional diagnosis of tubercular Abdomen/Malignant Ascites with Unknown primary was made. CT Abdomen showed large bowel wall thickening with parietal peritoneal lesions with omental cake. Excision biopsy of inguinal lymph node showed lymphoid follicles with poorly defined attenuated mantle zones having centrocytes and centroblasts. Immunohistochemistry was positive for markers, which confirmed the diagnosis of Stage 4B DLBCL, germinal center B-cell type. Sepsis and acute kidney injury occurred and the patient expired, 6 days after diagnosis. To conclude, Peritoneal lymphomatosis is an extremely rare condition. This case highlights the need to have a high index of suspicion for malignancy, when a given case is clinically diagnosed with tuberculosis as and when new findings appear during investigation.

Keywords: Peritoneum, Non-Hodgkin Lymphoma, Tuberculosis

INTRODUCTION
Primary extra nodal lymphoma occurs in approximately 25% to 40% of patients and is more common in patients with NHL. Diffuse large B-cell lymphoma (DLBCL) is the dominant histological subtype.1 It is often intermediate to high grade and indicates a poor prognosis.2,3 The gastrointestinal tract (stomach) is the most frequent extra nodal site of lymphoma, accounting for about 40% of cases.

CASE REPORT
A 60-year-old gentleman presented to our hospital with pain abdomen associated with abdominal distension for 1 month. Additional symptoms included low grade fever, fatigue, anorexia, significant weight loss (12 kg) and night sweats for 1 month. He was a chronic smoker and alcoholic. Diabetic and hypertensive since 4yrs on oral medications. On general physical examination patient was cachexic, anaemic, with ECOG status of 3. Systemic examination revealed generalised gross abdominal distension, mild tenderness in lower quadrants with tense ascites. No other palpable mass. Respiratory examination revealed crepitations with reduced breath sound on the bilateral basal lung fields. All other systems were normal. Lymph node examination revealed bilateral axillary and inguinal lymphadenopathy with no matting. Provisional diagnosis of tubercular abdomen/malignant ascites with unknown primary was made. Routine investigations...
revealed total count of 12370/mL (N-84, L-8.0, M-9), ESR-30 mm/hr, Sr. Alb-3.2 g/dL; LDH-3094 U/L. Tumour markers CA-125-425.8 U/mL; CEA, CA-19.9 were normal. The serology for HIV, hep-B and hep-C was negative. Chest radiograph showed bilateral basal pleural effusion. Paracentesis showed few malignant mesothelial cells with WBCs-58000/mm³ (N-1, L-76, M-23), LDH-3200 U/L, protein-2.8 g/dL; alb-1.8 g/dL; SAAG-1.5, ADA-30 U/mL. Acid-fast stain of ascites was negative. CT Abdomen showed bowel wall thickening involving ileum, caecum, ascending and descending colon. Massive ascites with thickened mesentery, parietal peritoneal lesions with omental cake, along with metastatic celiac, preaortic, paraaortic and aortocaval group of lymph nodes with splenomegaly. Excision biopsy of inguinal lymph node showed lymphoid follicles with poorly defined attenuated mantle zones having centrocytes and centroblasts. Massive infiltration of capsule and pericapsular fat by lymphoid cells. Immunohistochemistry was positive for CD20, Bcl-2, CD10, MUM1, Bcl-6, and c-MYC, and negative for CD3, which confirmed the diagnosis of stage 4B DLBCL, germinal center B-cell type. Sepsis and acute kidney injury occurred and the patient expired, 6 days after diagnosis.
DISCUSSION

CT findings of omental caking with homogeneous bulky masses, rather than a nodular pattern, diffuse lymph node involvement can be seen in both TB and PL, but splenomegaly with the presence of tumours in the gastrointestinal tract, attribute to PL. SAAG greater than 1.1 g/dL is favouring PL, this may be because peritoneal lymphoma changes the permeability of peritoneal vessels and leak into cavity. Low SAAG and high ADA levels favours TB, whereas High LDH in ascitic fluid has high sensitivity and low specificity for malignant ascites. Extensive peritoneal or omental lymphomatosis might lead to elevated serum CA-125. But in case of TB, there is no role for markers. Ascites cytology can elicit lymphocyte predominance with low protein levels in TB and florid mesothelial hyperplasia in PL. Histology is still the gold standard for diagnosis for PL as to CBNAAT for TB. R-CHOP regimen used to treat PL and ATT for TB.

CONCLUSION

In conclusion, peritoneal lymphomatosis is an extremely rare condition. The ascitic ADA level could help differentiate this disease from abdominal tuberculosis. However, histopathology is gold standard for diagnosis of peritoneal lymphomatosis. This case highlights the need to have a high index of suspicion for malignancy, when a given case is clinically diagnosed with tuberculosis and when new findings appear during investigation.

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