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Case Report

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A Diagnostic Dilemma: Acute Myeloid Leukemia Masquerading as Surgical Abdomen Mimicking Abdominal Tuberculosis: A Case Report

Kritin Kondeti *[1], Nagineni Venkata Varun Kumar [1], R. Ponniah Iyyappan [2], Saravanan S [3]

[1]General Surgery Resident, Department of General Surgery, Sri Ramachandra Institute of Higher Education and Research, Chennai, India.
[2]Associate Professor, Department of General Surgery, Sri Ramachandra Institute of Higher Education and Research, Chennai, India.
[3]Professor, Department of General Surgery, Sri Ramachandra Institute of Higher Education and Research, Chennai, India.

*Corresponding Author: Kritin Kondeti; *kritin.kondeti@gmail.com*

Abstract

Acute myeloid leukemia (AML) is a hematopoietic malignancy that typically presents with constitutional symptoms, cytopenias, and bleeding manifestations. Its presentation as a surgical abdomen with ascites is exceedingly rare. Tuberculosis manifesting as peritoneal ascites is more commonly seen in the context of tuberculosis-endemic countries such as India. We report the case of a 19-year-old male who presented to our institute with abdominal pain and distension, unintentional weight loss, and easy fatigability, leading us to suspect peritoneal tuberculosis. Investigations were also in favor of tuberculosis, with elevated levels of adenosine deaminase (ADA). However, the presence of atypical lymphoid cells in the ascitic fluid led us to further evaluate the patient and come to a diagnosis of AML after extensive investigations. This case highlights a rare but critical diagnostic dilemma wherein AML presents with ascites and findings that closely mimic abdominal tuberculosis. This case is presented owing to its rare clinical presentation.

Keywords: ***Leukemia, Myeloid, Acute, Tuberculosis, Peritoneal, Ascites.***

Introduction

Acute myeloid leukemia (AML) is a hematological neoplasm characterized by the clonal proliferation of myeloid precursor cells, typically presenting with fatigue, anemia, bleeding manifestations, or opportunistic infections [1]. However, its presentation with abdominal symptoms severe enough to mimic a surgical abdomen is very rare and poses a diagnostic challenge. In tuberculosis endemic regions such as India, abdominal tuberculosis is a common differential diagnosis for patients presenting with ascites, abdominal pain, weight loss, and easy fatigability. A similar clinical picture of these two conditions may lead to misdiagnosis. Furthermore, peritoneal tuberculosis and leukemic ascites may even share overlapping features on imaging as well as ascitic fluid analysis [2]. Timely recognition of AML, even in its atypical presentations, is of utmost importance as it is an aggressive malignancy requiring suspicion to avoid misdiagnosis and prevent unnecessary surgical intervention.

Case Report

A 19-year-old male presented with a history of diffuse abdominal pain and distension for 1.5 months. The pain was insidious in onset, progressive in nature, of a dull aching type, and non-radiating. A history of breathlessness and oliguria was present. A history of cough with expectoration for 25 days and unintentional weight loss of 12 kg in 2 months, accompanied by easy fatigability, was noted. The patient had no history of altered bowel habits or loss of appetite. There was no history of any bleeding manifestations. On examination, the patient appeared poorly nourished, with an underweight body mass index. Pallor, bilateral inguinal and epitrochlear lymphadenopathy, and pitting pedal edema were noted.

The abdomen appeared grossly distended. On palpation, inspection findings were confirmed, and diffuse tenderness was noted. There was no guarding or rigidity. Bowel sounds were heard. Prior to admission, he was evaluated at an outside hospital where computed tomography (CT) showed diffuse peritoneal thickening with nodularity, which was suspected to be tuberculous peritonitis, and the patient was referred to our institute for further management.

On admission, the hemoglobin was 16.1 g/dL and total leucocyte counts (TLC) were 5980 cells/cu mm, with monocytes at 10.7%. Within 20 days the hemoglobin dropped to 7.6 g/dL and TLC

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increased to 27930 cells/cu mm. The initial peripheral smear showed leukocytosis with neutrophilic predominance. Initial ultrasound of the abdomen revealed ascites with diffuse peritoneal thickening and nodularity, marked thickening of the mesenteric root, bilateral hydronephrosis and cystitis, mild bilateral pleural effusion, and bilateral hydrocele. A CT abdomen revealed large ascites with peritoneal nodularity and thickening in bilateral paracolic gutters, para-aortic and aortocaval lymphadenopathy, moderate bilateral pleural effusion, and anasarca. Diagnostic laparoscopy showed mucous flakes and straw-colored fluid, and the peritoneal biopsy was inconclusive. Cystoscopy and biopsy of the inflamed bladder wall were also inconclusive. Ascitic fluid analysis showed total nucleated cells of 23693 cells/cu mm, low serum-ascites albumin gradient (SAAG), high protein, and TLC of 39072 cells/cu mm (polymorphs-55%, mononuclear cells-45%). Adenosine deaminase (ADA) in pleural fluid was 119.3 U/L and in ascitic fluid, it was 310.7 U/L. Pleural fluid lactate dehydrogenase (LDH) was 5550 U/L, and ascitic fluid LDH was 9052 U/L. Ascitic fluid also showed atypical lymphoid cells, which created a suspicion of malignancy. A repeat peripheral smear following these atypical findings showed leucocytosis with 44% atypical cells with large convoluted nuclei and scant basophilic cytoplasm. It also showed normocytic normochromic anemia and thrombocytopenia serially. Serial peripheral smears within 1-day intervals further showed monocytic leucocytosis and finally revealed 60% blast cells, with increased nuclear cytoplasmic ratio, scant to moderate basophilic cytoplasm, and nuclei with prominent nucleoli. Flow cytometry showed 18% abnormal blast cells. Bone marrow biopsy showed monocytic blast cells with immunohistochemistry positive for myeloperoxidase (MPO) and CD45, which clinched the diagnosis of AML, and further karyotypic studies confirmed AML with inversion of chromosome 16 and trisomy of chromosome 8. Later during the course of chemotherapy, a repeat CT also suggested a soft tissue density lesion with peripherally calcified margins adjacent to the ascending colon on the lateral side-likely a chloroma.

The patient was treated with diuretics, analgesics, and repeated therapeutic paracentesis in view of abdominal distension. Empirical ATT started initially was thereafter discontinued following bone marrow results. Bilateral ureteroscopy with DJ stenting was done in view of anuria due to prerenal AKI and hydronephrosis. After confirmation of AML by cytogenetics, the patient was stabilized with rasburicase for tumor lysis and platelet transfusions for thrombocytopenia. He then received 2 cycles of standard 3+7 chemotherapy with daunorubicin and cytarabine and achieved remission. Following remission, he was started on high-dose cytarabine for two cycles as a consolidation. The patient was symptomatically better post chemotherapy. Peripheral blasts reduced to less than 3% within the induction phase. Morphological and molecular remissions were achieved following completion of chemotherapy.

This case poses an intriguing dilemma, as our primary diagnoses were abdominal tuberculosis and intraabdominal visceral organ malignancy, which were proven wrong with investigations. This case emphasizes the importance of investigating patients thoroughly, even in places endemic for tuberculosis with elevated ADA in ascitic fluid, for other diagnoses.

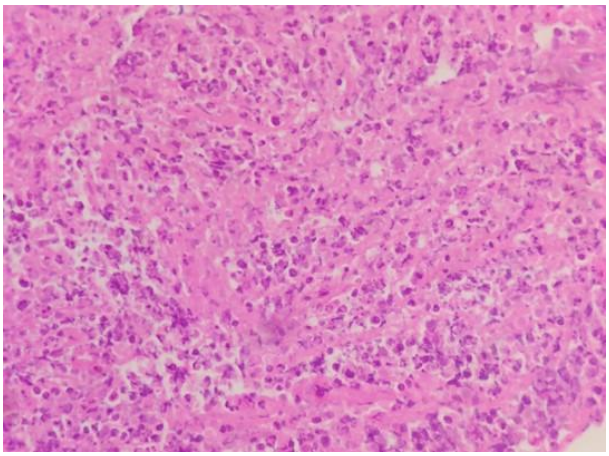

Fig 1: Mesenteric biopsy showing sheets of atypical lymphoid cells and apoptotic bodies]

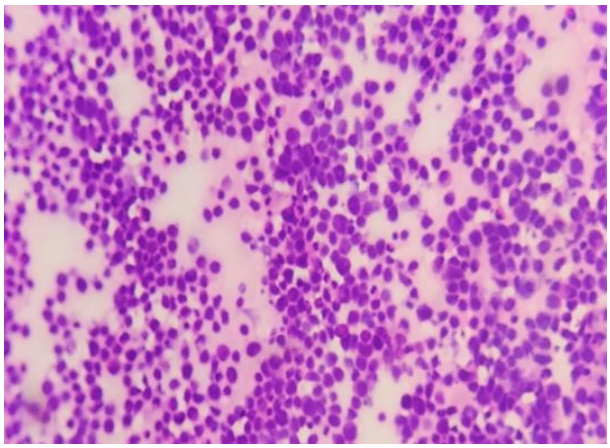

Fig 2: Pleural fluid cytology showing atypical lymphoid cells]

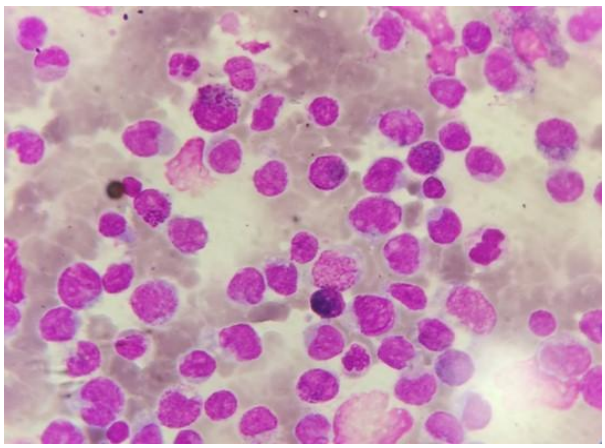

Fig. 3. Bone marrow aspirate showing myeloblasts]

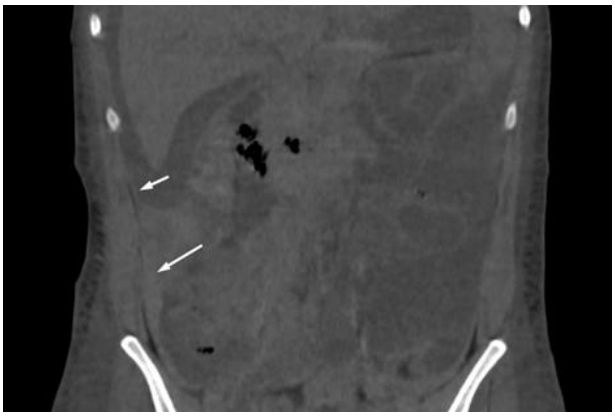

Fig 4: CT abdomen: coronal section with arrow showing peritoneal thickening]

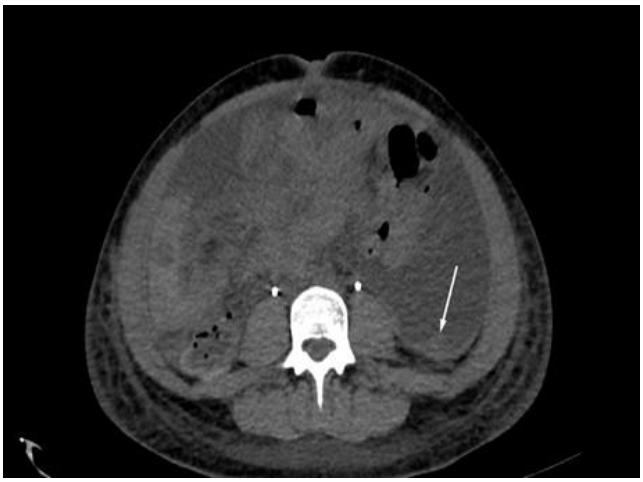

Fig 5: CT abdomen: axial section showing peritoneal thickening]

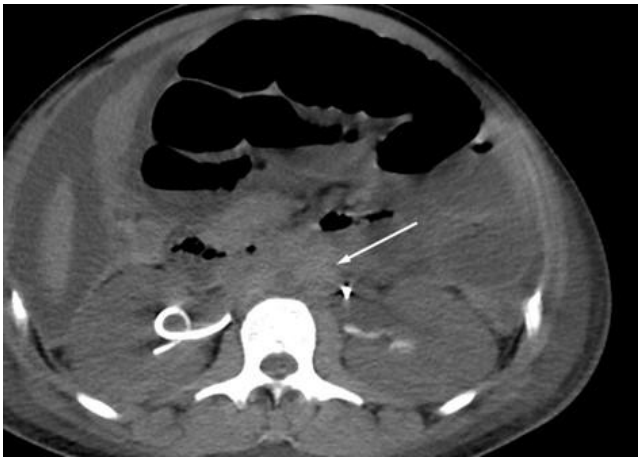

Fig 6: CT abdomen: axial section showing paraaortic and aortocaval lymph nodes]

Patient's perspective

I was initially informed at an outside hospital that I had tuberculosis and was advised to start treatment at a tertiary institute. At this institute, the doctors evaluated me thoroughly and made a diagnosis of blood cancer. Although it is a frightening diagnosis, I am thankful to the team for making it early, thereby preventing further spread and helping me through the treatment process. I am symptomatically better and regularly follow up with my doctors.

Discussion

Ascites is defined as an pathological accumulation of fluid in the peritoneal cavity [3]. It is commonly encountered in clinical practice secondary to cirrhosis and portal hypertension (85-90% of cases). Malignant causes are relatively rare, accounting for around 10% of ascites cases [4]. Tuberculosis, which is endemic to India, is also an important cause of ascites. Peritoneal tuberculosis accounts for around 7.7% of extrapulmonary tuberculosis cases in the Indian population and hence is considered an important differential diagnosis in the context of ascites [5].

Paracentesis plays a central role in diagnosing the cause of ascites. Traditionally, emphasis was made on differentiation between exudate and transudate; however, this outlook is outdated [2]. Parameters evaluated from an ascitic fluid sample are usually its gross appearance, ascitic fluid total protein and SAAG ratio, LDH levels, glucose levels, ADA level, cell counts, and cultures.

Tuberculous ascites typically shows an elevated TLC, high protein (>2.5 g/dL), SAAG <1.1 g/dL and importantly, an elevated ADA level. ADA levels ≥30 U/L are highly sensitive for tuberculosis [6]. However, our case report demonstrates that leukemic ascites also exhibits ADA elevation. CT findings in the case of peritoneal tuberculosis include omental caking (diffuse omental thickening forming a mass that may get displaced from the abdominal wall), mesenteric lymph node enlargement, and smooth peritoneal thickening with pronounced enhancement. However, these are non-specific and cannot be relied upon to diagnose peritoneal tuberculosis [7].

Leukemic ascites is very rarely encountered in practice; leukemic ascites with elevated ADA is even rarer, with only a few cases reported in literature [8]. In leukemic ascites, the ascitic fluid may appear either clear or hemorrhagic and typically shows elevated TLC with lymphocytic predominance, increased protein levels, low SAAG, elevated LDH, and the presence of blast cells. Flow cytometry is done for confirmation.

AML is a hematopoietic malignancy characterized by the neoplastic proliferation of stem cell precursors of the myeloid lineage. It is a rare malignancy but accounts for a third of leukemia cases. It has a diverse clinical presentation, ranging from incidental detection on routine blood investigations to complications such as infections, ascites, and disseminated intravascular coagulation. The mutations noted in our case, inversion of chromosome 16 and trisomy of chromosome 8, generally offer a favorable to intermediate prognosis. Treatment is with chemotherapy, through an intensive induction phase to achieve complete remission with no measurable disease, followed by post-remission therapy to prevent relapse [1].

In contrast to the predominantly surgical entity of advanced abdominal tuberculosis, leukemias are a group of rapidly progressive disorders that necessitate early diagnosis and chemotherapy and palliative surgery as required.

Abdominal tuberculosis and AML are two entities poles apart, each of which requires a completely different spectrum of diagnostic workup and management. Clinical presentation can be deceiving, and abdominal tuberculosis may even occur as an opportunistic infection due to granulocytopenia in AML [9].

Moreover, it should always be noted that all the peritoneal thickenings and ascites with miliary tubercles and abdominal lymph node involvement need not be tuberculosis, they can also arise due to leukemic cell infiltrations, though rare.

Though elevated ADA has good sensitivity and specificity for tuberculosis, and tuberculosis is endemic to our region, it is important to evaluate for other non-infectious causes such as malignancy as well before labelling a patient as a case of tuberculosis. This case report emphasizes the need for in-depth evaluation of all non-cirrhotic ascites cases and the importance of correlating clinical and investigative findings to avoid diagnostic oversight.

List of abbreviations

AML- acute myeloid leukemia

CT- computed tomography

TLC- total leucocyte count

SAAG- serum-ascites albumin gradient

ADA- adenosine deaminase

LDH- lactate dehydrogenase

MPO- myeloperoxidase

ATT- antitubercular therapy

Declarations

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Conflict of interest

The authors declare no conflict of interest regarding the publication of this case report.

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Authors' contributions

All authors contributed equally to the conception, drafting, and revision of the manuscript. All authors approved the final version and agree to be accountable for all aspects of the work.

Data availability

All relevant data supporting the findings of this case report are included within the article.

Consent to participate

Written informed consent was obtained from the patient for participation in this case report.

Ethical Clearance

NA

Trial details

NA

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