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Case Report

Multiple myeloma masquerading as ascites: a rare case report

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ABSTRACT

A 45-year-old woman with progressive abdominal distension was diagnosed with multiple myeloma after presenting with unexplained ascites. Investigations revealed anemia, hypercalcemia, renal dysfunction, lytic bone lesions, and 62% atypical plasma cells in bone marrow. Liver biopsy showed amyloid deposition, suggesting amyloidosis-induced hepatic dysfunction as the cause of ascites. Other causes, including portal hypertension and malignancy, were ruled out. Myelomatous ascites is rare and requires high suspicion for diagnosis. This case highlights the importance of considering multiple myeloma in unexplained ascites, as early recognition and treatment can significantly improve patient outcomes.

Keywords: Multiple myeloma, Ascites, Amyloidosis

INTRODUCTION

Ascites, the abnormal accumulation of fluid in the peritoneal cavity, is typically linked to liver disease, malignancy, tuberculosis, or nephrotic syndrome. However, in rare cases, multiple myeloma can present with ascitic fluid, complicating the diagnosis.[1] Multiple myeloma, a plasma cell disorder, primarily affects the bone marrow and leads to systemic complications such as bone lesions, hypercalcemia, renal dysfunction, and immunosuppression.[2] Myelomatous ascites is rare and usually results from peritoneal involvement or liver infiltration by plasma cells. Diagnosing such cases requires a high level of suspicion. Here, we report a case of a 45-year-old woman with unexplained ascites later diagnosed as multiple myeloma.

CASE REPORT

A woman in her 40s presented in the Medicine Emergency on 06 February 2025, with progressive abdominal distension persisting for three months. The distension was associated with generalized weakness and lower back pain but without fever, jaundice, or urinary abnormalities. She had no history of tuberculosis, diabetes, thyroid disorders, blood transfusions, or high-risk behaviors. Initially treated at the nearby district hospital with an ascitic tap providing temporary relief, she was referred to tertiary healthcare centre for further evaluation after symptom recurrence.

Examination findings

The patient was hemodynamically stable, with pallor but no jaundice, cyanosis, or pedal edema. Abdominal examination confirmed ascites with flank fullness and shifting dullness. Systemic examination was otherwise unremarkable.

Investigations

Blood tests revealed anemia (Hb 5.5 g/dl), hypercalcemia (serum ionic calcium 5.88 mg/dl), renal dysfunction (serum creatinine 2.2 mg/dl), and a mildly elevated CA-125 (45.7 U/ml). Ascitic fluid analysis showed a high

serum-ascitic albumin gradient (SAAG), elevated protein content, and malignant cells. Ultrasound of the abdomen demonstrated mild fatty liver and hepatosplenomegaly. Non-contrast CT abdomen revealed hepatosplenomegaly, multiple lytic bone lesions, diffuse osteopenia, and vertebral collapse at L3, while an X-ray of the skull showed a classic "salt-and-pepper" appearance (Figure 1). Whole-body X-ray confirmed pathological fractures and lytic lesions (Figure 2 a-c). Cardiac evaluation via 2D echocardiography ruled out cardiac causes of ascites. Doppler ultrasound and upper gastrointestinal endoscopy excluded Budd-Chiari syndrome and portal hypertension. Bone marrow aspiration demonstrated 62% atypical plasma cells, confirming multiple myeloma. Serum protein electrophoresis showed an A/G ratio of 0.46 with an M-band peak (Figure 3).[3] Liver biopsy revealed eosinophilic hyaline material surrounding blood vessels, suggesting amyloidosis-related hepatic dysfunction.[4,5]

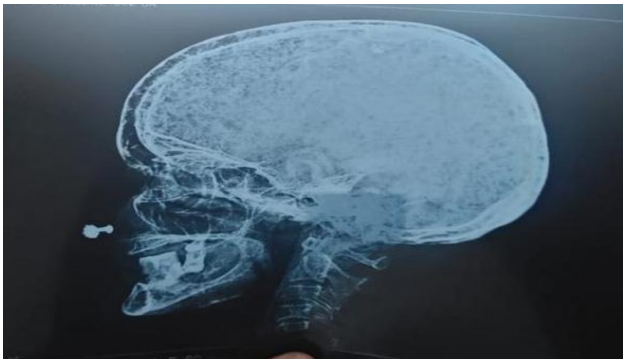

Figure 1: Digital X ray skull lateral view showing "salt and pepper appearance".

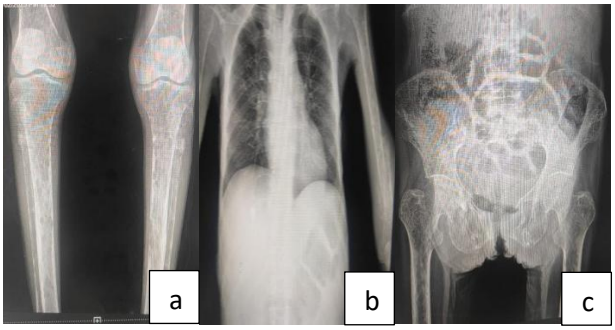

Figure 2: Whole body digital X ray showing pathologic fractures and lytic lesions, (a) multiple lytic lesions in bilateral knee joints, tibia and fibula, (b) multiple lytic lesions in bilateral shoulder joints and humerus with pathologic fracture of left humerus, and (c) multiple lytic lesions in pelvic bone and bilateral femur.

Treatment

Her paracentesis was done and she was managed conservatively with diuretics and calcium supplements. After her diagnosis of multiple myeloma was made, she was transferred to oncology department.

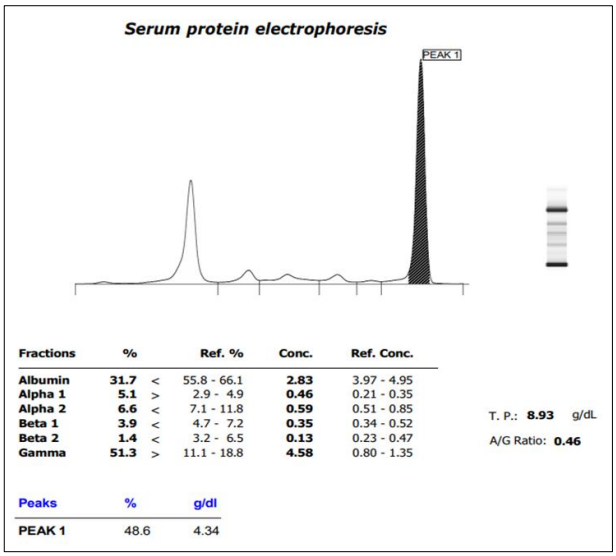

Figure 3: Serum protein electrophoresis showing an A/G ratio of 0.46 with an M-band peak.

DISCUSSION

Multiple myeloma primarily involves the bone marrow, leading to bone pain, anemia, renal dysfunction, and hypercalcemia. Rarely, it can present with extramedullary manifestations such as hepatosplenomegaly and ascites. Myelomatous ascites has been reported in a small percentage of cases and is often linked to plasma cell infiltration, amyloidosis, nephrotic syndrome, or thrombosis-related hepatic dysfunction.[6]

In our patient, liver biopsy revealed amyloid deposition rather than direct plasma cell infiltration, suggesting amyloidosis-induced hepatic dysfunction as the cause of ascites. Amyloidosis, a complication of plasma cell disorders, can impair organ function by causing vascular and tissue damage, ultimately leading to fluid accumulation. The eosinophilic hyaline material around blood vessels observed in this case is a hallmark of amyloid deposition, which compromised hepatic function and contributed to ascites formation, even in the absence of portal hypertension.[4,7]

Several mechanisms have been proposed for ascites in multiple myeloma, including peritoneal plasma cell infiltration, hepatic amyloidosis, nephrotic-range protein loss, and hypercoagulability-induced hepatic or mesenteric thrombosis.[8]

In our case, thrombosis and portal hypertension were excluded, making amyloidosis-induced hepatic dysfunction the most plausible cause.

A study by Kintzer et al found that fewer than 1% of multiple myeloma cases present with significant ascites.[1] Karahan et al reported a case of myelomatous ascites due to plasma cell infiltration of the liver, whereas our patient exhibited amyloid deposition.[9] These findings emphasize

the need to consider multiple myeloma in cases of unexplained ascites, particularly when systemic features like lytic bone lesions, anemia, hypercalcemia, and renal dysfunction are present. Early recognition of this rare presentation is crucial for timely diagnosis and management.[10]

Differential diagnoses considered

Malignancy-related ascites

It was initially suspected but ruled out based on ultrasound whole abdomen and non-contrast CT abdomen reports.

Liver cirrhosis with portal hypertension

It was unlikely due to normal upper GI endoscopy findings and imaging reports that did not suggest cirrhosis.

Tuberculous peritonitis

It was considered but unlikely due to the lack of fever, weight loss, or constitutional symptoms; ascitic fluid analysis did not support this diagnosis.

Cardiac ascites

It was ruled out based on normal 2D echocardiography.

Budd-Chiari syndrome and portal hypertension

It was excluded through normal Doppler studies and upper GI endoscopy.

CONCLUSION

This case underscores an unusual presentation of multiple myeloma with ascites and highlights the importance of thorough evaluation in cases of unexplained fluid accumulation. The presence of amyloid deposition rather than direct plasma cell infiltration in liver biopsy suggests hepatic dysfunction due to amyloidosis as the underlying mechanism of ascitic fluid formation. Given the rarity of myelomatous ascites, clinicians should maintain a high index of suspicion in patients with systemic signs of multiple myeloma. Early diagnosis and appropriate intervention can significantly improve patient outcomes.

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