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Liver Cirrhosis as a Rare Manifestation of IgA Myeloma: Diagnostic Challenge and Clinical Insight

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Abstract | **Case Report**

Background: Liver involvement in multiple myeloma is rare and often underrecognized, typically presenting as hepatic infiltration by plasma cells or as amyloidosis. Cirrhosis secondary to multiple myeloma is an uncommon and poorly understood phenomenon. ***Case presentation:*** We present the case of an 84-year-old male with a known history of hypertension and no significant alcohol use, who was admitted with fatigue, weakness, lumbar pain, hypotension, diarrhea, and abdominal distension. Clinical evaluation and diagnostic imaging revealed signs of decompensated liver disease, including ascites and hepatomegaly with coarse echotexture. Extensive laboratory analyses and bone marrow findings were indicative of multiple myeloma. Other common causes of cirrhosis, such as alcohol, viral hepatitis, autoimmune hepatitis, cardiac liver disease, and metabolic liver disease, were ruled out. This case illustrates a rare hepatic manifestation of multiple myeloma that mimicked cryptogenic cirrhosis, underlining the diagnostic importance of considering hematologic malignancies. ***Conclusion:*** In elderly patients presenting with unexplained liver dysfunction and systemic symptoms, especially in the absence of conventional risk factors for liver disease, multiple myeloma should be considered in the differential diagnosis. This case emphasizes the importance of considering hematologic malignancies in the diagnostic workup of cryptogenic cirrhosis.

Keywords: Cirrhosis, Multiple Myeloma, Etiology, Plasma Cell Infiltration, Differential Diagnosis.

INTRODUCTION

Multiple myeloma (MM) is a malignant plasma cell disorder that primarily affects the bone marrow, leading to common systemic manifestations such as bone pain, anemia, renal failure, and hypercalcemia [1]. Extramedullary involvement, particularly in the liver, is rare and typically not associated with the classic signs of cirrhosis. However, liver involvement may occur due to plasma cell infiltration, amyloid deposition, or light chain-related hepatic injury [2]. Cirrhosis as a direct manifestation of MM is uncommon and not yet fully understood. Diagnosing it can be particularly challenging due to nonspecific clinical features and overlapping comorbidities. We present a rare case of liver cirrhosis in an elderly male, in whom MM was ultimately identified as the underlying cause after all common etiologies of liver disease had been excluded.

CASE DESCRIPTION

An 84-year-old man with a medical history limited to hypertension presented to the emergency department complaining of progressive fatigue, general weakness, and dull pain localized in the lumbar region. Over the previous few days, he experienced multiple episodes of diarrhea, persistent hypotension, and noticeable abdominal swelling. He denied alcohol consumption and reported no significant smoking history. He was admitted to the Internal Medicine department for further investigations.

During the initial physical examination, the patient looked pale and fatigued, exhibiting a distended abdomen suggestive of ascites. His blood pressure was low, and mild peripheral edema was observed. An abdominal ultrasound revealed a steatotic liver with coarse echotexture and scattered hyperechoic foci, raising concerns about possible acute hepatitis or

underlying chronic liver disease. (Fig.1) The right hepatic lobe measured 16.5 cm, indicating mild hepatomegaly. There was a significant amount of free fluid in the peritoneal, perihepatic, perisplenic, and pelvic cavities, consistent with ascites. The hepatic and portal venous systems appeared normal. An abdominal CT scan was performed, confirming the hepatic findings of the ultrasound and revealing no pathology in the bone structures.

Laboratory findings demonstrated hypoproteinemia (Total Protein 5.5 g/dL, Albumin 2.3 g/dL), elevated liver enzymes (AST 222 U/L, ALT 180 U/L, GGT 244 U/L), hyperbilirubinemia (total bilirubin 3.71 mg/dL), and elevated LDH (349 U/L). There was evidence of renal dysfunction (urea 200.8 mg/dL, creatinine 2.40 mg/dL) and mild normocytic anemia. Normal levels of cardiac enzymes (Troponin I level of 0.036 ng/mL, CK 92 U/L, CK-MB 2.9 ng/mL, and NTproBNP 420 pg/mL) and echocardiogram findings (Ejection Fraction 53%, normal valve function, normal heart chambers) ruled out heart failure as a possible cause of hepatic decompensation.

Given the ascites, a diagnostic paracentesis was performed. The ascitic fluid was transudative, featuring low protein content (0.6 g/dL), low LDH (67 U/L), and normal glucose and cholesterol levels, thereby ruling out spontaneous bacterial peritonitis or malignant ascites.

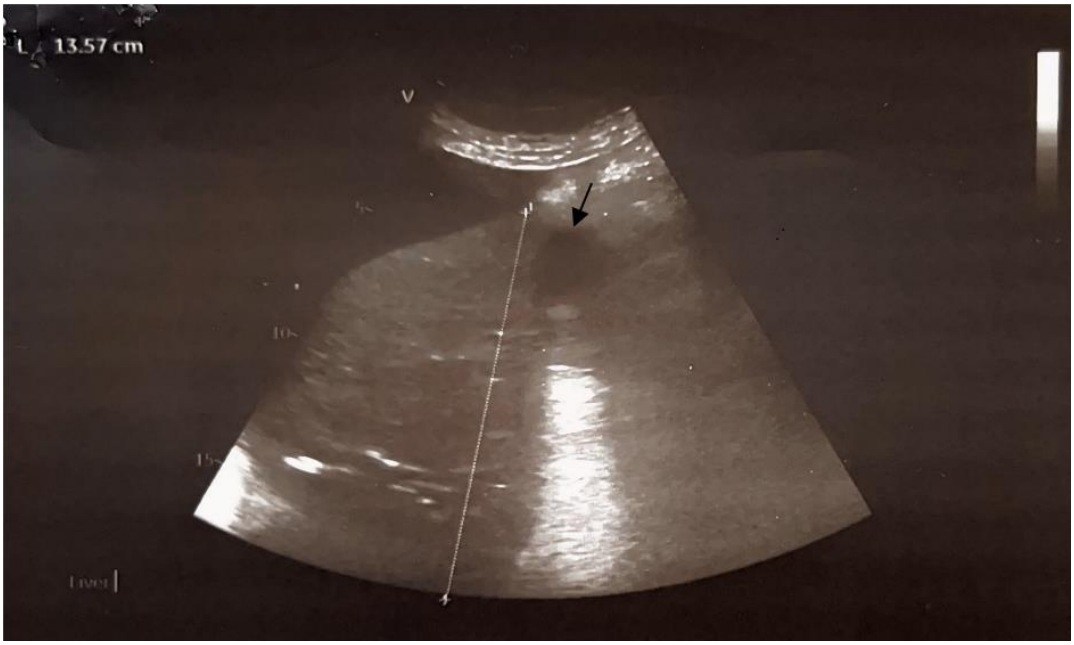

Figure 1. Abdominal ultrasound showing hyperechoic foci within the liver parenchyma (black arrow), suggestive of steatosis and coarse echotexture

Further investigation into the etiology of liver disease was conducted. Serologic testing excluded viral hepatitis, with HBsAg, HCV, HBV-DNA, and all other tests returning negative. The patient showed evidence of past exposure to hepatitis A and B (positive HAV IgG and anti-HBc, positive anti-HBs) but had no active infection. Notably, C-reactive protein levels were not elevated, decreasing the likelihood of active infection or inflammation. Autoimmune markers, including ANA, AMA, and anti-dsDNA, were all negative, thereby ruling out autoimmune hepatitis. The patient was also tested for HIV, which resulted in a negative test. Tumor markers (CEA, Ca 19-9, PSA) were assessed to rule out an abdominopelvic malignancy, and they all returned negative as well. There was no history of hepatotoxic drug use.

On the 7th day of hospitalization, the patient reported black-colored stool. A fecal occult blood test was performed, which resulted positive. The patient underwent a fibrogastroscopy procedure, revealing diffuse gastritis accompanied by grade II esophageal varices, suggestive of portal hypertension. The diagnosis of diffuse gastritis clinically explains the presence of blood in the stool, while the presence of esophageal varices indicates an advanced stage of hepatic cirrhosis.

Despite the lack of a typical etiology, the patient's condition resembled decompensated cirrhosis. Given the presence of systemic symptoms such as fatigue, renal dysfunction, and lumbar pain, a hematologic malignancy was suspected. Serum protein electrophoresis revealed a significant reduction in gamma globulins (5.4%) and a pronounced monoclonal peak in the beta-2 globulin fraction. Immunofixation further indicated an IgA elevation (1996.60 mg/dL), with suppressed IgG and IgM levels, and a kappa/lambda light chain ratio of 0.37, suggesting lambda predominance. A bone marrow examination confirmed the suspicion of plasma cell dyscrasia, showing 11% plasma cells in the

myelogram, alongside the presence of normoblasts and a preserved megakaryocytic lineage. These findings established the diagnosis of multiple myeloma.

With other causes excluded, the hepatic manifestations were attributed to multiple myeloma. Possible mechanisms included hepatic infiltration by plasma cells or light chain-induced hepatic injury; however, a liver biopsy was not performed because of the patient's clinical status. The presence of transudative ascites, hypoalbuminemia, and imaging findings supported the diagnosis of cirrhosis as a rare but serious complication of MM.

DISCUSSION

Hepatic involvement in MM is well documented in the autopsy series, with up to 45 % of patients showing plasma cell infiltration of the liver; however, clinically overt liver disease and decompensation are notably rare [3]. The most frequent hepatic findings include hepatomegaly, abnormalities in liver tests, and cholestatic patterns, while acute liver failure, cirrhosis, and transudative ascites occur infrequently.

In our case, the patient presented with a constellation of findings, including hepatomegaly, significantly elevated liver enzymes and bilirubin, hypoalbuminemia, transudative ascites, and esophageal varices, that collectively mimicked decompensated cirrhosis. Despite exhaustive evaluation, no common etiology for liver disease was identified. The discovery of IgA type MM with lambda light chain predominance, along with suppressed immunoglobulin subclasses and clonal plasma cells in bone marrow, pointed toward a hematologic origin as the underlying cause of hepatic dysfunction.

While a liver biopsy could have clarified the presence of plasma cells, light-chain deposition, or amyloid, it was deferred due to the patient's frailty. Nonetheless, the clinical presentation aligns with other documented cases where MM alone precipitated cirrhosis-like decompensation. There is evidence of the coexistence of MM and liver cirrhosis. Hindosh *et al.,* [4] reported the presence of liver cirrhosis in a 66-year-old Caucasian female, who was admitted with acute kidney injury and a recent history of SARS-CoV-2 infection, with a bone marrow biopsy confirming kappa-light chain myeloma. Bellouhou *et al.,* [5] published the case of a 73-year-old patient with MM and liver involvement, who had no abnormalities of the osteo-articular system, like our patient. Evidence of liver cirrhosis in a patient with MM without a history of alcohol use was also reported by Christopher *et al.,* [6]. In our patient, clinical hepatic involvement was observed in the early stages of the diagnosis of MM, which is also a rare finding. A similar case was reported by Cull *et al.,* [7], highlighting the importance of considering hematologic pathology in the presence of unexplained liver cirrhosis. A study from Talamo *et al.,* [8] revealed that gastrointestinal involvement was present in 24 out of 2584 myeloma patients, often presenting in nodular, sinusoidal, diffuse, or mixed patterns during autopsies.

The pathophysiology of liver involvement in MM typically involves the extramedullary spread of clonal plasma cells through alterations in adhesion molecules, chemokine receptors, and overexpression of heparinase-1, along with angiogenesis and mutations in the factor-kB pathways [9]. Liver infiltration occurs when malignant plasma cells disseminate from the bone marrow into the peripheral blood, facilitated by the loss of adhesion molecules such as CXCR4 and CD44. These cells migrate to the liver, guided by chemokine gradients like CXCL12, and adhere to hepatic tissue through interactions with endothelial adhesion molecules [10]. The liver's microenvironment, particularly its rich supply of interleukin-6 (IL-6), supports plasma cell survival and proliferation [11]. Infiltration may be diffuse or nodular and can involve portal tracts, sinusoids, or hepatic parenchyma. In some cases, infiltration is accompanied by light chain deposition or amyloidosis, contributing to liver dysfunction and, rarely, cirrhosis. Light chain deposition disease (LCDD) involves the accumulation of immunoglobulin light chains in various organs, leading to gradual tissue damage. The deposited light chains are predominantly of the κ subtype and tend to bind strongly to basement membranes due to somatic mutations that increase their hydrophobic properties [12]. In MM, although genetic mutations initiate the disease, the bone marrow (BM) microenvironment plays a critical role in supporting the growth, survival, and drug resistance of malignant plasma cells [11]. A major contributor is interleukin-6 (IL-6), produced mainly by bone marrow stromal cells, but also by macrophages, osteoblasts, osteoclasts, eosinophils, and megakaryocytes. Some myeloma cells can also produce IL-6 themselves.

Our patient's profile, including IgA-dominant disease, renal dysfunction, normocytic anemia, and low gammaglobulins, matches the pattern seen in aggressive and extramedullary MM, which is linked to a poorer prognosis. In such cases, early detection of hepatic involvement is essential because systemic chemotherapy (e.g., bortezomib-based regimens) may reduce liver damage caused by the lytic activity of plasma cell-mediated injury [13].

This case highlights that unexplained hepatic decompensation, especially with hepatomegaly, ascites, and elevated enzymes, should lead to considering hematologic malignancies. MM and amyloidosis must be part of the differential diagnoses, even if there are no classic bone lesions. When a tissue biopsy isn't possible, supporting data from electrophoresis/immunofixation, bone marrow evaluation, and ruling out other causes can be enough for diagnosis and prompt treatment.

CONCLUSIONS

Liver cirrhosis as a manifestation of multiple myeloma is an uncommon but important diagnostic consideration, particularly in elderly patients with unexplained liver dysfunction after common etiologies have been excluded. This case underscores the need for heightened clinical suspicion toward underlying hematologic malignancies in instances of cryptogenic cirrhosis. Given the aggressive nature and systemic impact of IgA-predominant MM, hepatic involvement may be underrecognized and warrants earlier investigation in the setting of cryptogenic cirrhosis. Early identification can enable timely and targeted treatment, potentially improving patient outcomes. A multidisciplinary approach, engaging internists, hematologists, and hepatologists, is essential in the diagnostic evaluation and management of such complex cases.

COMPETING INTERESTS DISCLAIMER:

Authors have declared that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper

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