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Multiple Myeloma Associated with Gallbladder Dyskinesia

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Abstract: ***This article discusses a rare clinical case involving a patient with multiple myeloma who presented with symptomatic gallbladder dyskinesia. The report explores the underlying pathophysiological mechanisms, including autonomic neuropathy and amyloid deposition, which may impair gallbladder motility in the setting of plasma cell malignancy. A detailed account of the clinical presentation, imaging findings, and surgical intervention is provided, supported by current literature. The article underscores the importance of recognizing gastrointestinal manifestations in multiple myeloma, particularly those linked to paraneoplastic syndromes.***

Keywords: multiple myeloma, gallbladder dyskinesia, autonomic neuropathy, amyloidosis, biliary hypomotility

1. Introduction

Multiple myeloma (MM) is a malignant proliferation of plasma cells that typically affects the bone marrow and can present with systemic manifestations such as hypercalcemia, renal failure, anemia, and bone lesions [5], In addition, it can have neurological complications, including peripheral and autonomic neuropathy, particularly when associated with amyloidosis [1].

Although rare, autonomic nervous system involvement or amyloid deposition in visceral organs can disrupt gallbladder function, leading to dyskinesia or functional gallbladder disease. These gastrointestinal complications are underreported, making their recognition and timely management difficult. This case offers novel insights into the underreported gastrointestinal manifestations of multiple myeloma, specifically gallbladder dyskinesia associated with autonomic dysfunction or amyloid infiltration.

The purpose of this article is to present a rare clinical case linking multiple myeloma to gallbladder dyskinesia and to highlight its diagnostic and therapeutic considerations.

2. Case Presentation

A 73 - year - old female, recently diagnosed with stage ISS III IgG kappa multiple myeloma, undergoing treatment with carfilzomib, thalidomide, and dexamethasone. She came to the emergency room of our unit due to a 7 - day history of clinical symptoms, which began with asthenia, weakness, hypothermia measured at 35°C, and generalized tremor. Upon admission, her blood pressure was in the range of hypotension (77/55 mmHg). A vasopressor support approach was initiated with norepinephrine. During her diagnostic approach, paraclinical tests were requested, which reported:

Laboratories:
Complete blood count: Hemoglobin 9.7 g/dl, Hematocrit 29.58, Leukocytes 9.53/ ul, Neutrophils 65%, Bands 13%, Platelets 264, 000/ uL; Bleeding tendency tests: PT 12.9 seconds, INR 1.26, PTT 34.8 sec, D - dimer 1960 ng/ mL. Blood chemistry: Glucose 203 mg/dl, BUN 84.49 mg/dl, Urea 180.81 mg/dl, Creatinine 4.47 mg/dl, BUN/Crea ratio 18.9; Serum Electrolytes: Na 129 mEq/L, K 5.83 mEq/L, Cl 100 mEq/L, Ca 7.1 mg/dL, Mg 2.05 mg/dL, P 5.4 mg/dL. Liver Function Tests: Total Bilirubin 1.50 mg/dL, Direct Bilirubin 0.60 mg/dL, Indirect Bilirubin 0.90 mg/dL, AST 22 U/L, ALT 56 U/L, ALP 360 U/L, GGT 281 U/L, Total Protein T 5.6, Albumin 4.0, Globulins 2.7 g/dL, A/G ratio 1.1, LDH 292 U/L.

Contrast - enhanced computed tomography of the abdomen and pelvis, which reported a normal - sized liver, a beaver - tailed left hepatic lobe as an anatomical variant, heterogeneous parenchyma due to a cystic image in segment 8, no dilatation of the intra - or extrahepatic biliary tract was observed, the portal vein measured 13 mm and the common bile duct in its supraduodenal portion was up to 10 mm, the retroduodenal portion was 8 mm and the pancreatic portion was up to 7 mm, the gallbladder was enlarged to 375 cc, with homogeneous contents, a thickened and regular wall of 6.3 mm, and a dissection was present in its posterior wall.

HIDA scan: gallbladder ejection fraction of 18%, suggestive of biliary dyskinesia.

Autonomic dysfunction secondary to neuropathy due to amyloidosis associated with multiple myeloma was considered. Broad - spectrum antimicrobial treatment with carbapenems was initiated in addition to joint management with the intensive care unit until hemodynamic stabilization. Fasting, analgesics with nonsteroidal anti - inflammatory drugs (NSAIDs) were administered. Surgical resolution was decided by means of laparoscopic cholecystectomy. The following findings were found: a 15x15 cm tense gallbladder with 220 cc of bile drained, with a histopathological result of focal amyloid deposits confirmed with Congo red staining. During her postoperative period, with favorable postoperative evolution, she was discharged from the intermediate care unit 24 hours after surgery and hospital discharge 72 hours after the surgical event.

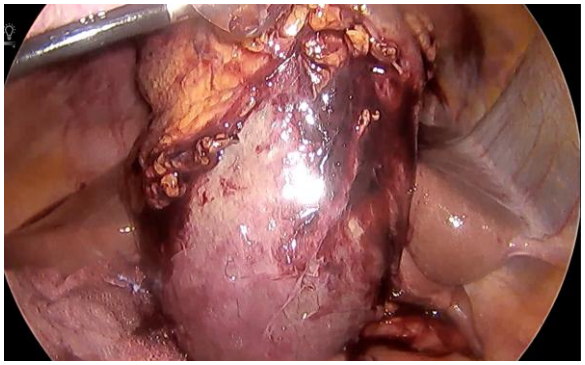

Figure 1: Transurgical image of the gallbladder

3. Pathophysiology

Gallbladder dysfunction in Multiple Myeloma may be due to:

Paraneoplastic autonomic neuropathy, especially in MM with light chain (AL) deposition, affecting the nerves that innervate the gallbladder.

Amyloid deposition in the gallbladder wall, which alters muscle contractility and sensitivity to cholecystokinin [4].

Hypercalcemia, common in MM, can induce gastrointestinal and biliary hypomotility[3].

Use of opioids, which also decrease gallbladder emptying.

4. Diagnosis

It is based on clinical features, ultrasound, and a functional study with a HIDA scan. Suspected amyloid involvement should be confirmed with biopsy and Congo red staining. Evaluation for autonomic neuropathy may also support the diagnosis.

5. Treatment

It includes symptomatic management, control of MM, and, in refractory or complicated cases, cholecystectomy. In patients with multiorgan amyloid involvement, the prognosis is more guarded.

6. Conclusion

Gallbladder dyskinesia may represent a rare but meaningful gastrointestinal complication in multiple myeloma, especially when autonomic dysfunction or amyloid deposition is present. Early diagnosis and surgical management, as demonstrated in this case, can lead to rapid recovery and improved patient outcomes. This case serves to broaden the differential diagnosis for atypical abdominal presentations in patients with hematologic malignancies.

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