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Ascites as the Presenting Symptom of Multiple Myeloma

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ABSTRACT

Multiple myeloma cases frequently present with complaints of infection and low back pain. Acid is a very rare finding in multiple myeloma. In this case report, we discussed a case of candida esophagitis and ascites, which was considered to be related with multiple myeloma.

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Introduction

Multiple myeloma is a clonal proliferation of malignant plasma cells bone marrow.[1] It is accounted for 13 % of hematological malignancies and 2 % of all malignancies worldwide.[2] It may present with many different manifestations such as anemia, bone tenderness, bone pain, weakness, bone fractures, kidney damage, hypercalcemia, nerve damage, skin lesions, enlarged tongue and infections. Ascites is a rare complication of multiple myeloma. It usually occurs during the course of a disease. It is rarely expected at presentation of the disease.[3] Ascites can occur in many different mechanisms. The most frequent reasons of ascites in multiple myeloma, are increasing permeability of the peritoneum and portal hypertension due to liver infiltration.[4] In this report, we studied a case of multiple myeloma that developed in an 80 years of male with ascites and candida esophagitis being the main presenting feature.

Case Presentation

An 80-year-old man without any chronic disease appealed to the hospital with weakness, weight loss (10 kilograms in four weeks) and difficulty during swallowing. The patient had progressive abdominal distention and peripheral edema just a month before the presentation. İn his first medical evaluation, he had normal vital signs. The heart exam was in normal limits with no murmurs, there was 2+ peripheral edemas. He had fluid in the abdomen but there were no profound lymph nodes and masses. In the lung examination there were Ralls at the basales segment of lungs. Firstly, upper gastrointestinal endoscopy was carried out because of dysphagia and apprehensive about gastric cancer. The only finding of upper gastrointestinal endoscopy was candida esophagitis. After the findings, 200 milligrams of fluconazole was prescribed daily. Following this, the patient was admitted to hospital for full diagnosis and the investigation of immunosuppressive condition and

possible malignancies.

Complete blood count revealed anemia with hemoglobin 9.8 g/dL and borderline white blood cell count (4.4 × 103 /μL) and platelet count (205000/μL). Lab tests had normal electrolytes, renal function and liver function with low albumin (3.1 mg/dL) and elevated globulin (4.5 mg/dL) levels. Thyroid function tests were also normal. Echocardiography was made to assess the cardiac function and rule out the heart disease. The ejection fraction was found as 60% and there were major valvular, pericardial and diastolic dysfunction. Abdominal ultrasonography was reported with normal hepatic and renal parenchyma, without organomegali, and masses, however, significant abdominal ascites were detected. Following this diagnosis, the patient underwent abdominal fluid tap. A sample of the ascetic fluid was sent for analysis. It revealed inflammatory cells with no suspicious malignant cells. Ascites sample were recurred in three times and all the results were detected similar. The level of adenosine deaminase was detected 23.4 U/L (5-20 U/L). After 2 weeks the control level of adenosine deaminase were present 39.7 U/L. PPD test and quantiferon test were completed. PPD resulted in 2 millimeters and the level of quantiferon were negative. Table 1 shows some laboratory results of the patient.

Table 1. Some laboratory findings of the case

Parameter	
Hemoglobin (g/dL)	9.8
White blood cell ($10^3/\mu$L)	4.4
Platelet ($10^3/\mu$L)	205
Electrolytes	within normal limits
Renal function tests	within normal limits
Liver function tests	within normal limits
Thyroid function tests	within normal limits
Albumin (mg/dL)	3.1
Globulin (mg/dL)	4.5
Free kappa light chains (g/dL)	5.79 (reference interval, 1.7-3.7)

Free lambda light chains (g/dL)	0.7 (reference interval, 0.9-2.1)
Free kappa/free lambda ratio	8.1
Beta-2-microglobulin (mg/L)	4.35
Adenosine deaminase	
At admission	23.4 U/L(reference interval 5-20 U/L)
After 2 weeks	39.7 U/L(reference interval 5-20 U/L)

Computed tomography of the chest, abdomen, and pelvis were performed. They revealed marked abdominal and pelvic ascites without masses and enlarged lymph nodes. Inguinal, cervical, axillary and supraclavicular ultrasonography were made. No enlarged lymph nodeswere detected.

Multiple myeloma was suspected based on anemia, and hyperglobulinemia, in addition to symptoms of weight loss and possible immunosuppressive condition. Protein electrophoresis were performed and monoclonal gammopathy were detected. Immunofixation electrophoresis revealed IgG kappa pattern. An assay for serum free light chain showed increased kappa light chains (5.79 g/dL; reference interval, 1.7-3.7 g/dL) and decreased lambda light chains (0.7 g/dL; reference interval, 0.9-2.1 g/dL); the kappa / lambda ratio was 8.1. The results of bone marrow biopsy revealed 20% plasma cells. Therefore, a diagnosis of multiple myeloma was demonstrated. Beta- 2-microglobulin level were detected 4.35 mg/L. X-ray skeletal series had no lytic lesions. Computed tomography of the abdomen was performed to assess the extramedullary hematopoiesis. There was no sign of extramedullary hematopoiesis.

After diagnosis chemotherapy was started (Bortezomib, dexamethasone, and zoledronic acid). After four cycles of treatment, there was a major improvement in hemoglobin levels and globulin levels. There was a marked decrease in the abdominal ascites.

Discussion

Multiple myeloma is a clonal proliferation of malignant plasma cells in the bone marrow. It can present with many systemic disorders and one of them is serous cavity involvement.[4] Involvement of serous cavity in multiple myeloma is unusual and it is only seen at 1% of patients with myeloma. The rate of involvement serous cavity is respectively firstly pleural,secondly peritoneal and lastly pericardial space.[1]

In 1990 Sasser et al. analyzed 56 cases of MM with serous cavities. In these group; pleural condition were demonstrated in 30 patients, peritoneal condition was demonstrated in 14 patients and pericardial condition were demonstrated in 2 patients.[2] Ascites are rarely seen as an initial symptom of multiple myeloma. It can develop in many different pathways. Primarily it can occur due to the peritoneal infiltration by plasma cells and it can also occur due to portal hypertension. The other rare causes are infectious peritonitis, heart failure secondary to amyloidosis, nephrotic syndrome, hemangioendothelial sarcoma, plasma cells and spontaneous rupture of the spleen.[1, 4]

In our patient malign cells were not detected in the aspirated fluid and all other etiologies for ascites were ruled out. Thus, a possible cause for ascites in this patient is the increased vascular permeability.

In this case, the presentation of multiple myeloma is unexpected. The reason being, the mainpresenting symptom were recurrent ascites without extramedullary hematopoiesis.

In conclusion, when a patient is admitted to the hospital for a newly onset abdominal ascites with suspicious signs of a malignancy (weight loss, opportunistic infectious etc.), should be investigated for solid organ malignancies. However, if there are some additional signs like anemia, low serum albumin and high serum globulin, the cause of ascites may be multiple myeloma and the patient should investigate and examine for multiple myeloma.

Declaration

The authors have no conflicts of interest to declare.

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