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A Multiple Myeloma Case with Ascites

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Case Report

History

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ABSTRACT

Ascite is a rare complication of multiple myeloma. It is a symptom of poor prognosis and can be noticed at any stage of the disease, not just at the initial diagnosis. The peritoneal involvement of a relapsed refractory multiple myeloma patient with Ig G lambda type is described in this case report. The patient has been prescribed two cycles of lenalidomide (15 mg) and dexamethasone (20 mg). The patient, however, did not respond to treatment.

Keywords: Multiple myeloma, ascites, plasma cell

Asitli Bir Multipl Miyelom Vakası

ÖZ

Asit, multipl miyelomun nadir bir komplikasyonudur. Kötü prognoz belirtisidir ve sadece ilk tanıda değil, hastalığın herhangi bir aşamasında fark edilebilir. Bu olgu sunumunda Ig G lambda tipi ile nüksetmiş refrakter multipl miyelom hastasının peritoneal tutulumu anlatılmaktadır. Hastaya 2 kür lenalidomid (15 mg) ve deksametazon (20 mg) reçete edildi. Ancak hasta tedaviye yanıt vermedi.

Anahtar sözcükler: Multipl miyelom, asit, plazma hücresi

Süreç

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Introduction

Multiple myeloma accounts for 10% of all hematologic malignancies. The disease is typically characterized by bone marrow infiltration as well as effects on the renal, cardiac, pulmonary, neurological, and bone structures. Ascites is a rare complication of multiple myeloma patients, and it is especially common in relapsed refractory multiple myeloma patients. There may be different mechanisms, such as hepatic infiltration of malignant plasma cells and myelomatous infiltration of the peritoneum in the pathogenesis of ascites of multiple myeloma. Multiple myeloma patients with ascites are more likely to have Ig G and Ig A monoclonal gammopathy. A case of multiple myeloma of the Ig G lambda type is presented here[1,2].

Case History

A 72-year-old patient was diagnosed with IgG lambda multiple myeloma. The patient's ECOG performance was 3. She was treated with seven cycles of melphalan-prednisolone therapy. Three cycles of bortezomib-melphalan-prednisolone were started due to recurrent disease. The patient gave up the bortezomib treatment voluntarily. However, the patient reached remission. She was unsuitable for stem cell transplantation at that time. Due to the disease's recurrence, lenalidomide and dexamethasone treatment were initiated. There was an excellent partial response. After that, the patient voluntarily gave up the medication. Then abdominal ultrasonography due to abdominal pain showed the free fluid measured 4 cm in depth between the bowel loops in the pelvic region.

Laboratory findings were: Hgb 9.86 g / dL, leucocyte 3.26x109 / L, platelet 136 x109 / L, alkaline phosphatase 80 U /L, aspartate aminotransferase 15 U / L, alanine aminotransferase 5 U / L, total bilirubin 0.55 mg/dL, direct bilirubin 0.5 mg / dL, lactic dehydrogenase 324 U / L, albumin 2.9 g / dL, total protein 10.3 g / dL, creatinine 0.86 mg / dL, calcium 9. 1 mg/ dL, CRP 0.6 mg/dL and INR [international normalized ratio]

100 cc fluid was obtained by paracentesis, and its color was yellowish. Total protein was found to be 7.81 g / dL, albumin 1.64 g / dL, LDH 1099, and glucose 73, respectively, in ascites. Flow cytometry could not be sent from the peritoneal fluid because the hospital could not work for technical reasons. After centrifugation, excentric nucleated plasma cells and atypic plasmacytoid cells are observed [Figure 2].

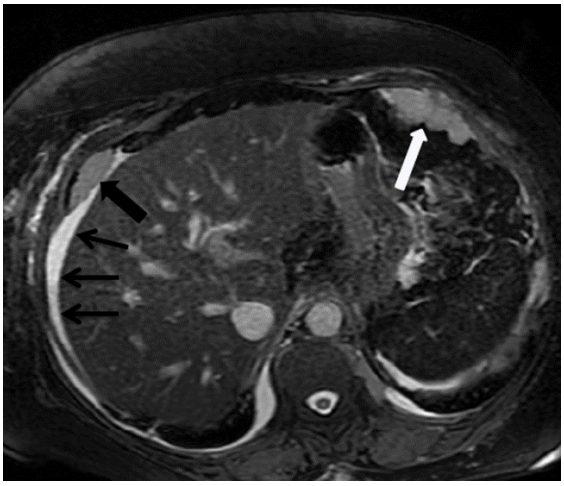

Figure 1: In T2-weighted lipid-printed axial cross-sections of the MR examination of the patient, hyperintense lesions with the largest 52x25 mm lobular contour (white arrow) are observed in the peritoneum of the epigastric region and in the right upper region of abdomen; adjacent to the free intra-abdominal fluid (thin black arrows) there is a hyperintense, 39x15 mm solid mass [solid black arrow] localized in parietal peritoneum.

A

B

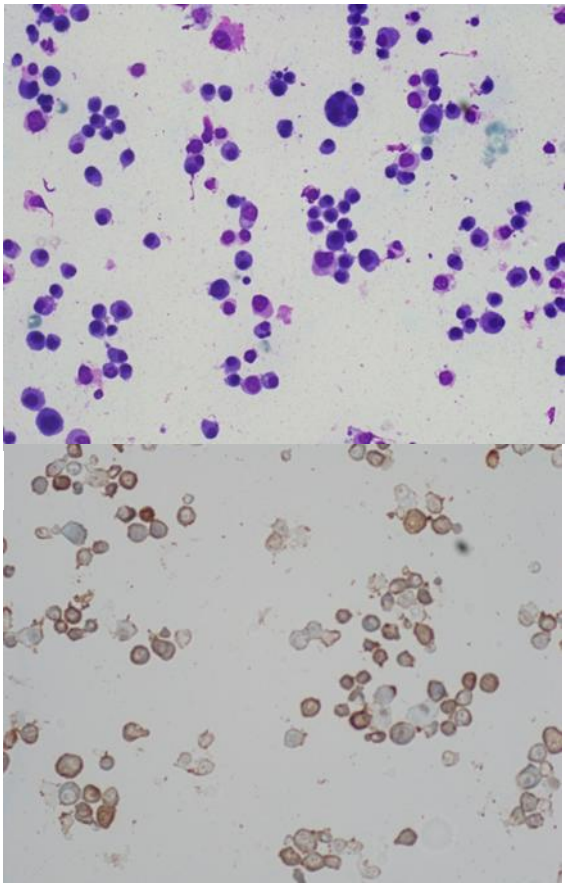

Figure 2: A) Ascites fluid cytology; May Grünwald-Giemsa [MGG] staining of atypical plasma cells (400x). B) Demonstration of plasma cells in ascites with liquid cytology CD138 (400x).

During this period, Ig G (7264 mg / dL) was found to be high; Ig A was 31 mg / dL, and Ig M was 34 mg / dL. Serum Ig G Lambda monoclonal band was also found, showing the recurrence of the disease.

Treatment with lenalidomide (15 mg) and dexamethasone (20 mg) was started. 2 cycles of therapy were given. However, the patient did not respond and was eventually exitus at the end.

Results and Discussion

Multiple myeloma can cause pleural effusion or serous cavity involvement in the peritoneum. It could be due to primary peritoneal infiltration of the disease, but it could also be due to hepatic involvement, renal amyloid accumulation, or cardiac failure [3].

In multiple myeloma, aspiration and cytological examination of ascites (with/ without biopsy) can be used for diagnosis or follow-up of patients. Malignant plasmacytic ascites is associated chiefly with implant formation in the peritoneum, and in most of these cases, liver involvement is either very small or absent [2,4]. In the secondary causes other than malignant plasmacytic ascites, the ascites is mostly severe, and the number of cells varies between 30-120 cells / mm^3. In plasmacytic ascites, plasma cells are present, and the cell number is usually between 8000 and 9000 cells/mm^3 [2].

Atypical, large-nucleated plasma cells can be seen, but so can more mature, excentric nucleated cells [4]. These cells can be distinguished from metastatic cancer cells, lymphocytes, and reactive mesothelial cells using immunohistochemistry, flow cytometry, immunofluorescence, or electron microscopy. In our case, the total cell count in ascites fluid was 11 000 cells / mm^3. Plasma cells were shown to be immunohistochemically positive for CD 138.

Hepatic infiltration was demonstrated histologically in 53% of the patients with multiple myeloma. Hepatic infiltration can be nodular or diffuse [2,5,6]. A review collected data between 1952 and 2014; the cases of multiple myeloma with ascites were examined. It was shown that only 7 of 65 cases presented with ascites at the diagnosis. 27 of the 65 cases have been identified as plasmacytic ascites [2].

No peritoneal involvement was detected in an autopsy series of 32 patients [6]. In an autopsy series consisting of 30 multiple myeloma patients with extraosseous participation, only one patient with peritoneal involvement was observed [7].

Exudative ascites was observed in 9 cases in 64 necropsy material of multiple myeloma patients, but peritoneal infiltration was not observed in these patients [4,5]. In another study, peritoneal involvement was demonstrated in 3 of 182 multiple myeloma cases with extramedullary involvement [8].

A published review showed that the mean age of myeloma patients with ascites was 60.6 (60.2 in males, 61 in females), 50% of cases had IgG, and 38% had IgA monoclonal gammopathy [2]. In our case, the patient's age at the time of diagnosis was 73 years.

Plasmacytic ascites is an indicator of increased tumor burden or widespread extramedullary involvement. The subsequent prognosis is poor, and the median survival after ascites formation is reported to be 1.5- 2 months [2,9]. In our case, the mean survival time after the ascites was approximately seven months.

Systemic chemotherapy, intraperitoneal chemotherapy, plasmapheresis, radiotherapy, and stem cell transplantation are the treatment options in cases of myelomatous ascites. Treatment options like bortezomib, melphalan, cyclophosphamide, and thalidomide have been reported [2]. One patient with multiple myeloma diagnosed with ascites was reported to have a complete response to autologous stem cell transplantation following one course of VAD chemotherapy. This response persisted over more than 14 months [10].

As a result, ascites can be seen at the diagnosis of multiple myeloma, or they can be seen at any stage of the disease. Ascites indicate that the prognosis is poor.

Conflict of Interest

The authors declared no conflict of interest.

Patient's Consent

The patient's son signed a document of informed consent.

References

1. Alacacioglu L, Turgut N, Piskin O, Ali Ozcan M, Hayri Ozsan G et al. Multiple myeloma presenting as massive ascites. J BUON 2009; 14(2): 307-8.
2. Mitra S, Mukherjee S, Chakraborty H, Bhattacharyya M. IgG Lambda Myeloma Presenting as Plasmacytic Ascites: Case Report and Review of Literature. Indian J Hematol Blood Transfus. 2015; 31(4): 472-9.

3. Sasser RL, Yam LT, Li CY. Myeloma with involvement of the serous cavities. Cytologic and immunochemical diagnosis and literature review. ActaCytol. 1990; 34(4): 479-85.
4. Karp SJ, Shareef D. Ascites as a presenting feature multiple myeloma. J R SocMed. 1987;80(3):182-4.
5. Thomas FB, Clausen KP, Greenberger NJ. Liver disease in multiple myeloma. Arch Intern Med. 1973; 132(2): 195-202.
6. Talerman A. Clinico-pathological study of multiple myeloma in Jamaica. Br J Cancer 1969; 23(2): 285-93.
7. Churg J, Gordon AJ. Multiple myeloma; lesions of the extra-osseous hematopoietic system. Am J Clin Pathol. 1950; 20(10): 934-45.
8. Hayes DW, Bennett WA, Heck FJ. Extramedullary lesions in multiple myeloma; review of literature and pathologic studies. AMA Arch Pathol 1952; 53(3): 262-72.
9. Görg C, Adler G, Görg K, Pflüger KH, Havemann K. Multiple myeloma of the serosa coat. J Cancer Res Clin Oncol 1988; 114(3): 312-6, 1988.
10. Alegre A, Martínez-Chamorro C, Fernández-Rañada JM. Massive myelomatous ascites responsive to VAD chemotherapy and autologous stem cell transplantation. Bone Marrow Transplant 1999; 24(3):343-4.