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EDİTÖRE MEKTUP / LETTER TO THE EDITOR

Pleural involvement in plasma cell neoplasm

Plevral tutulumu olan plazma hücre neoplazisi

Aykut Bahceci[1], Semra Paydaş[1], Emine Kılıç Bağır[2], Aysun Uğuz[2]

[1]Cukurova University Faculty of Medicine, Department of Medical Oncology, [2]Department of Patology, Adana, Turkey

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Dear Editor,

Myelomatous pleural effusion (MPE) is very rare and has a very poor prognosis. The effusion is thought to be a late manifestation of the disease and MPE is a sign of aggressive behavior of the disease.

A 57 year old male admitted with back pain, fatigue and weight loss and he diagnosed as multiple myeloma (MM). He had back pain due to T11-12 vertebral involvement. The patient was given zoledronic acid plus 3 cycles of VAD chemotherapy (vincristine, adriamycin and dexamethasone) and received palliative radiotherapy to T11-12 vertebrae. Response to VAD was insufficient. Due to the reimbersment problems patient received 4 cycles of DCEP (dexamethasone, cyclophosphamide, etoposide, cisplatin) chemotherapy an autologous stem cell transplantation (ASCT). Massive pleural effusion, dyspnea, hyperglobulinemia, hypoalbuminemia, M-peak and high β2 microglobulin levels were detected. Adenosine deaminase (ADA) in pleural fluid was within normal limits. Pleural cytology and pleural biopsy showed malignant plasma cells and plasmacytoma infiltration, respectively. In pleural biopsy sample cytokeratin and lambda were negative while kappa was positive. Bortezomib was started, then patient lost to follow-up.

Plasma cell neoplasms are typically characterized by the abnormal and excessive production of a monoclonal immunoglobulin or light chain and may be present as a solitary (solitary plasmacytoma) or multiple bone or soft tissue lesions (MM). Solitary plasmacytomas occur most commonly in the bones, but may also occur in soft tissues as extramedullary plasmacytoma (EMP). EMPs have been reported in 15-20% of the patients at the time of diagnosis and 15% during the course of the disease[1]. Lymph nodes, skin, liver, gastrointestinal tract, kidney and meningeal involvements have been reported. These patients often have poor prognosis, even with aggressive treatment[2].

Table 1. Etiological factors of pleural effusion in multiple myeloma.

Amyloidosis secondary to congestive heart failure
Chronic renal failure
Renal tubular infiltration with paraprotein and glomerular injury secondary to Nephrotic syndrome
Direct infiltration from neighboring tissues
Hypoalbuminemia
Pulmonary embolism
Secondary neoplasm
Lymphatic obstruction due to tumor infiltration
Infections
Pleural myelomatous effusion

Pleural effusion in MM is seen in about 6% of the patients with MM and is usually due to nephrotic syndrome, pulmonary embolism, amyloidosis, congestive heart failure, secondary malignancies, tuberculosis and specific or nonspecific infections[3-4] (Table 1). Myelomatous pleural effusion occurs in less than 1% of cases and is usually associated with a poor outcome[3].

The diagnosis of myelomatous pleural effusion is based on pleural biopsy, the presence of abnormal-clonal plasma cells in pleural fluid cytology, and also demonstration of monoclonal proteins in pleural fluid with protein electrophoresis. Kintzer et al. found pleural effusion in 58 cases among 958

Yazışma Adresi/Address for Correspondence: Dr. Aykut Bahçeci, Cukurova University Faculty of Medicine, Department of Medical Oncology, Adana, Turkey E-mail:aykutbahceci@gmail.com
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patients with MM, and only 8 of these were associated with infiltration with plasma cells[3]. Myelomatous effusion has been found to be asssociated with very poor prognosis and very short survival, median 4 months[5]. Cho Y-U et al. found pleural effusion in 54 cases among 734 patients with MM, and nineteen of these were associated with MM[6]. It is very well known that the most common isotypes in MM are IgG (50%) and is followed by IgA (20%), light chain (20%) and much less frequently Ig D, Ig E, Ig M[4]. The most notable finding in Chou's study is the high incidence of myelomatous pleural effusion due to IgD (6 of 19 patients, 31.6%). Another important and/or interesting finding in this study is high ADA activity in patients with myelomatous pleural effusion. It is clear that high ADA activity is usually associated with tuberculous pleural effusion. However, active pulmonary tuberculosis was not shown in this study. This is important in cases with myleomatous pleural effsion due to the high prevalence of effusions with infectious etiology in cases with myeloma and clinicians must be aware of this point. Another interesting finding in Chou's study is the high frequency of chromosome 13 abnormalities (monosomy 13 or 13q deletion) in patients with myelomatous pleural effusion.

In conclusion it should be considered that myelomatous pleural effusion may be present in patients with MM, pleural effusion and/or biopsy are important tools for diagnosis, ADA activity may be higher in pleural fluid and chromosome 13 abnormalities are more frequent in these cases.

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