

Destroyed Lung Secondary to Multiple Myeloma in a Previously Treated Tuberculosis Patient

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ABSTRACT

This case is presented with a pulmonary parenchymal destruction due to acquired multiple myeloma who has a history of pulmonary tuberculosis. A 55 year old male patient attempted to emergency department with complaints of shortness of breath. The final diagnose with the existing clinical and laboratory outputs were right pulmonary parenchymal destruction and pleural involvements secondary to multiple myeloma. Our prediagnose was confirmed by the overt plasma cell increase and Russell bodies in cytopathologic examination of the pleural fluid. Even though multiple myeloma leads to destruction in the pulmonary parenchyma rarely, patients should be scrutinized enough to elicit true diagnose.

Key words: Multiple myeloma, tuberculosis, destroyed lung

Daha Önce Tüberküloz Tedavisi Görmüş Bir Hastada Multipl Myelomaya Sekonder Harap Akciğer

ÖZET

Multipl myeloma bağlı, pulmoner parankimal destrüksiyon gelişen, daha önceden geçirilmiş pulmoner tüberküloz öyküsü olan vaka sunulmuştur. 55 yaşında erkek hasta acil servise nefes darlığı şikayeti ile başvurdu. Hasta klinik bulguları ve laboratuvar tetkikleri ile değerlendirildikten sonra, mutipl myeloma sekonder sağ pulmoner parankimal destrüksiyon ve plevral tutulum tanısı konuldu. Ön tanımız, plevral sıvının sitopatolojik incelemesinde artmış plazma hücresi ve Russell body hücreleri görülmesi ile doğrulandı. Multipl myelom nadiren pulmoner parankimal destrüksiyone yol açmasına rağmen, hastalarda ayırıcı tanı için ayrıntılı incelemek gerekmektedir.

Anahtar kelimeler: Mutipl myelom, tüberküloz, akciğer destrüksiyonu

INTRODUCTION

Multiple myeloma is a malignant disease running with proliferation of neoplastic plasmocytes, in consequence of which excessive production of immunoglobulin or light chain occupies the bone marrow (1). The entity arises with bone pains and tenderness, renal failure, anemia, hypercalcemia, however, pulmonary involvement is rare, which is much more associated with bad prognosis, and encountered in the terminal period or plasma cell leukemia (2,3) Occasions such as chronic heart failure, pneumonia, tuberculosis (TB), connective tissue diseases, carcinoma, AIDS and other viral causes, and pulmonary embolism should be eliminated before

the diagnosis of pulmonary or pleural involvement (4). Herein, a case is presented with a pulmonary parenchymal destruction due to acquired multiple myeloma who has a history of pulmonary TB.

CASE

A 55 year old male patient attempted to emergency department with complaints of shortness of breath, cough and hemoptysis. He was hospitalized in service of chest diseases with a pre-diagnosis of pneumonia for further investigation and treatment due to increased opacity and effusion in right lung areas on postero-anterior (PA)

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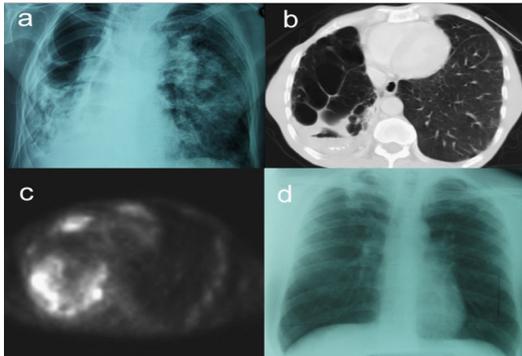


Figure 1. a: Postero-anterior chest X-ray at admission to our hospital revealed diffuse destruction in right lung areas. b: Pleural thickness, calcification and parenchymal destruction has been detected in thorax computed tomography. c: Increased right pleural and subpleural metabolic activity has been detected in PET. d: Postero-anterior chest X-ray one year after the second tuberculosis treatment revealed only a fibrotic lesion in right upper zone.

chest X-ray (Figure 1a). The patient had a history of 30 package/year smoke and diagnosed and treated for pulmonary TB twice 22 and 8 years ago. He had no characteristics in family history. In physical examination, body temperature was 35,5°C, pulse rate was 98 beat / minute, and blood pressure was 140/80 mm/Hg. The patient was cachectic, and there was no respiratory sound on the right hemithorax. Peripheral lymphadenopathy or organomegalia could not be detected.

In his medical history, he had been evaluated in chest diseases clinic with pre-diagnosis of recurrent TB and/or mesothelioma due to right pleura-based disseminated infiltrative nodular and calcified images in his PA chest-X ray. His sputum was negative for asidoresistant bacille (ARB) and mycobacterium culture for six times. His thorax computed tomography (CT) revealed chronic fibrotic changes filling almost the whole right hemithorax, and multiple calcifications and cavitary lesions (Figure 1b). Increased metabolic activity in right pleural and subpleural area and bones in his full body PET-CT scan performed in an external diagnostic center was interpreted as metastase (Figure 1c). Transbronchial and CT guided transthoracic biopsies were performed, with prediagnoses of lung cancer or mesothelioma; however, no sign of malignity was elicited in pathological examination. The patient had also been followed by internal medicine policlinic for his complaints of weight loss and anemia.

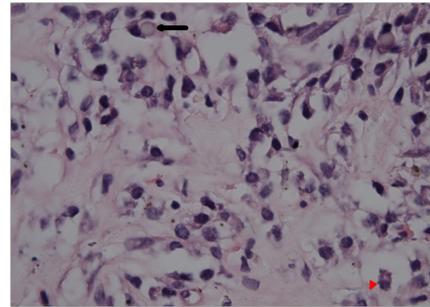


Figure 2. Cytopathologic examination of the pleural fluid revealed Russel body (black arrow) and plasmocytoid cells (red arrowhead).

During follow-up, upper GIS and colon endoscopies were performed to investigate his anemia. However, no pathology was determined. Although the specimen was reported to be insufficient to diagnose multiple myeloma; his bone marrow biopsy revealed increased number of plasmocytes. A serum protein electrophoresis performed in an external laboratory revealed monoclonal gammopathy [43,1% (11-21) gammaglobulin, 36.6% (52-68) albumin, 2.5% (2-5) alpha-1, 8.9% (6.6-13.5) alpha-2, 8.9% (8.5-14.5) beta]; a serum immunfixation electrophoresis revealed IgG:3440 (650-1600) mg/dL, IgA:550 (45-380) mg/dL, IgM 134 (50-300) mg/dL, kappa light chain : 786 (170-370) mg/dL, lambda light chain: 432 (90-210) mg/dL. In second bone marrow biopsy, signs concordant with reactive mature plasmocytosis were determined. Upon outpatient follow-ups as the patient did not refer for controls, no further diagnose or treatment plans could have been performed consequently.

During hospitalization in ward of chest disease, he developed dyspnea and massive hemoptysis. His respiration rate scaled up to 30/min, and his blood gas revealed pCO₂:64 mmHg, pO₂:62 mmHg, HCO₃:25, 7 mg/dL, O₂ saturation: 88%. Consequently he was taken to the intensive care unit, intubated and mechanically ventilated. In the further evaluation a chest x-ray which was taken seven years ago, after the second tuberculosis treatment was founded from local dispensary that had followed the patient for tuberculosis. The chest X-ray reported normal except apical fibrotic changes in right upper lung zone (Figure 1d). The final diagnose with the existing clinical and laboratory outputs were pulmonary parenchymal destruction and pleural involvements secondary to multiple myeloma. Our prediagnosis was

confirmed by the overt plasma cell increase and Russell bodies in cytopathologic examination of the pleural fluid (Figure 2). In spite of the given treatment regimen, patient died in the fourth intensive care day.

DISCUSSION

In multiple myeloma, pulmonary parenchymal and pleural involvements are rather rare, and presented as case reports. Nonetheless, to our knowledge, there is no case reported as pulmonary destruction by new developed multiple myeloma in a previously treated cure TB patient with mild sequela in Chest X-ray. Myeloma can affect the thorax by different ways. Thorax findings may be as skeletal infiltrations and anomalies, pleural effusion, and plasmocytoma. In a series of 958 myeloma cases, Kintzer et al. reported thoracic skeletal involvements as osteolytic lesions on costae and vertebrae in 267, detected pulmonary infiltrations in 95, and evaluated much of these as caused by infection. Of these, 58 had pleural effusion, however almost 50% had congestive heart disease. Only eight patients had pleural effusions due to myeloma (5). Schelle et al. reported a myeloma case with diffuse nodular involvement and left pleural effusion (6). Kamble et al. reported a myeloma case with diffuse parenchymal lesion (7).

In the case, the diagnosis was made by the findings of serum and urine protein immune fixation electrophoresis, increased plasma cells in bone marrow biopsy, detection of plasma cells and Russell bodies in pleural biopsy. This involvement was probably due to the spread from the adjacent bone structures since some malignancy findings were detected on axial and appendicular skeletal system by PET. The examinations for TB were all negative. There was not any positive culture for any infection, and any clinical and radiological response to the given empiric antibiotherapy. No pathology consistent with mesothelioma and/or lung cancer detected in biopsies. By the ruling out all these causes in differential diagnosis, the destructive pulmonary findings were considered secondary to multiple myeloma. Respiratory failure due to myeloma is too rare; the intraparenchymal causes of respiratory insufficiency are: infiltration by plasma cells, alveolar paraprotein accumulation, alveolar septal amyloidosis with metastatic calcification of alveolar walls and vessels (8). Two cases with pulmonary infiltrations in radiological examination and pathological evidence of neoplastic plasma cell infiltration of the lungs

without lung destruction were reported.⁸ Furthermore cases with lung calcifications that considered resulting from hypercalcaemia due to renal insufficiency caused by myeloma are reported in the literature (8).

In thorax radiography of multiple myeloma, multiple masses simulating solid tumor metastases, diffuse interstitial disease and consolidations may be detected (3,9,10). In the present case, destruction invaded the right lung entirely. Before he was taken to the ICU, since a TB history was present, examinations for TB, mesothelioma and lung cancer were performed. Culture materials were taken from the patient to detect a bacterial infection, and nevertheless, all were negative. By detailed evaluation of the patient's story, clinical and laboratory findings, a diagnose destroyed lung secondary to myeloma and pleural involvement of myeloma was considered.

In this report, to our knowledge; we present the first destroyed lung secondary to myeloma case. We suggest that, though patients have a history of a disease that can make sequale lesions in lung parenchyma like tuberculosis, clinicians should be careful about myeloma if consistent clinic and laboratory findings present. Even though multiple myeloma leads to destruction in the pulmonary parenchyma rarely, patients should be scrutinized enough to elicit true diagnose.

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