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Multiple Myeloma Masquerading As ARDS: A Clinical Challenge

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ABSTRACT: Here we discuss a case of an elderly female presenting with progressive ARDS (acute respiratory distress syndrome) and renal derangement with altered sensorium. The electrolyte panel revealed incidental hypercalcemia. Multiple myeloma was diagnosed after bone marrow biopsy, which progressed to tumor lysis syndrome, reaching a fatal outcome. Myeloma presenting as progressive ARDS is infrequent and is challenging to delineate multiple myeloma causing ARDS, as compared to parenchymal and pleural disorders, without tissue diagnosis. A possible mechanism of this rarity is tumoral infiltration of lung parenchyma by tumor cells. In diagnostic evaluation, the possibility of multiple myeloma in non-infective progressive ARDS should be considered.

Keywords: progressive ARDS, tumoral infiltration, hypercalcemia, multiple myeloma,

I. INTRODUCTION

Multiple Myeloma is a clonal disorder of neoplastic origin manifesting as a proliferation of plasma cells producing a monoclonal immunoglobulin or a part of it. Clonal proliferation within the bone marrow manifests as bone pains, non-nutritional anaemia, hypercalcemia, and renal injury which ranges from acute kidney injury to chronic kidney disease and renal amyloidosis. MM accounts for 1% of all cancers and is the 2nd most common hematologic malignancy after lymphoma[1.] Less commonly, it is also known to present as extramedullary plasmacytomas[2]. Here we report a case of an elderly female hailing from Kerala presenting with multiple myeloma with pulmonary involvement, masquerading as progressive ARDS.

II. CASE REPORT

A 65-year-old female with a history of chronic fatigue and reduced appetite for 6 months, presented with recurrent vomiting for 2 weeks with disorientation and reduced responsiveness with fever for 1 week. Patient had a past history of depression for 4 years on regular treatment with SSRI with good adherence. Patient admitted into intensive care unit in view of altered sensorium. CT brain was normal with no clinical signs of meningitis.

UTI was suspected initially in view of pus cells, fever and acute kidney injury. Urine culture was negative. Chest radiograph showed bilateral perihilar and basal scanty reticulonodular opacities with Arterial blood gas showed a pH of 7.37, pCo2 33, Pao2 82, bicarbonate of 19.1, and Sa02 of 96 % with oxygen support of 4 L via facemask suggestive of mild ARDS (P/f 224), RTPCR for COVID was negative. Serology was negative. Routine bedside sonography revealed hepatomegaly with altered echotexture of the liver with multiple hyperechoic lesions with grade one renal parenchymal changes. Laboratory results showed ahaemoglobin of 11.5g/dl, ESR of 80mm/hr, Procalcitonin 16ng/ml. Blood culture showed no growth. Urea 69.9mg /dl, and serum creatinine of 2.64mg/dl, serum alkaline phosphatase of 546mg/dl, serum sodium 126meq/l, serum potassium 5.59meq/L. Serum albumin 2.82g/dl, globulin 3.29g/dl .

USG findings prompted tumour markers, in suspicion of liver secondaries. A complete electrolyte panel was ordered in view of non-improvement in sensorium with persistent fever spikes, revealed hypercalcemia of 19mmol/dl, hyperphosphatemia of 10mg/dl. CA-19-9 and Serum LDH were 346 and 365U/L respectively. CECT abdomen was deferred due to progressive renal failure requiring dialysis.

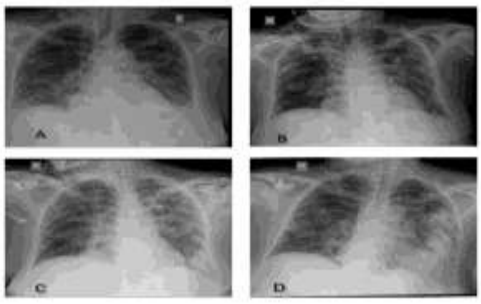

Figure 1: Sequential chest x-ray from admission (A-D) showing progressive semiconfluent patches of consolidation.

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In view of progressive worsening of respiratory failure with increasing reticulonodular patches on chest radiograph, a computerised tomogram of the chest (CT scan) was ordered which showed confluent air space opacities in both lung fields suggestive of pulmonary oedema. Not responding to diuretics.

She was ventilated with low tidal volume as per ARDS protocol in view of progressive hypoxic failure. Echocardiogram was normal.

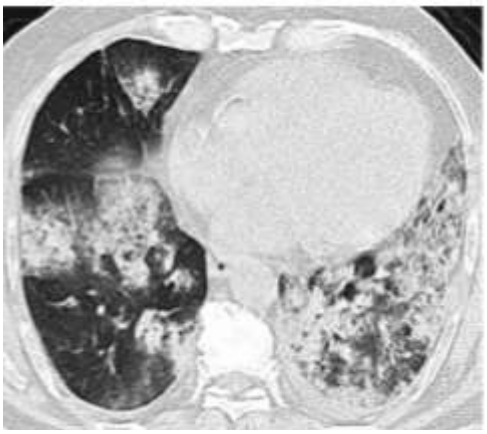

Figure 2: CT scan showing bilateral patchy consolidations

CT guided biopsy of lung lesion was performed. Due to laboratory profile of Multiple Myeloma, Serum protein electrophoresis was done which showed no evidence of M band. Bone marrow biopsy was performed which confirmed the diagnosis of multiple myeloma, with a bone marrow plasma cell percentage of 15.

Oncology was involved, initiated on Bortezomib and steroids.

Intravenous Rasburicase and hydration was given as she was in tumour lysis with hypercalcemia, hyperphosphatemia and hyperuricemia(9.37mg/dl).

She also received Injectable Meropenem, fungal prophylaxis (Fluconazole) and vasopressive support due to distributive shock. Due to extensive disease burden patient ultimately succumbed.

Post mortem biopsy revealed direct nodular tumoral invasion of lung parenchyma manifesting as non-cardiogenic pulmonary oedema.

III. DISCUSSION

Patient presented with fever, altered sensorium and patchy consolidation on chest radiograph with hemodynamic instability which led to a provisional diagnosis of sepsis.However due to negative blood cultures, hypercalcemia, multiple hyperechoic lesions in liver, a bone marrow revealed a diagnosis of multiple myeloma presenting as progressive ARDS. Infection is least likely to be a contributing factor to the lung picture due to failure to respond to antibiotics, negative blood cultures and post mortem evidence of direct tumoral invasion of lung by malignant cells.

Multiple myeloma presenting as progressive ARDS due to direct tumoral invasion is an extreme rarity.

Multiple myeloma may involve the thorax in a variety of ways but pulmonary parenchymal involvement is uncommon. Thoracic manifestations may include bony abnormalities, infiltration, pleural effusions, plasmacytomas and diaphragmatic dysfunction due to peripheral neuropathy. In a review of 958 patients with multiple myeloma at the Mayo Clinic, Kintzer et al reviewed the pleuropulmonary abnormalities in multiple myeloma.[1]

The abnormalities were chest roentgenograms. The most common finding was thoracic skeletal abnormality (28%), osteolytic lesions, localised or diffuse pulmonary infiltrates. Four patients had a diffuse infiltrate thought to be caused by a plasma cell infiltrate (proven in one). In 113 patients, plasmacytomas were seen (intramedullary 102, extramedullary 11). Pleural effusions were present in 58 patients (6%) and extramedullary plasmacytomas in 11 (1%).

Harinder et al[2] reported a patient of Multiple Myeloma since 5 years presenting with type 1 respiratory failure, due to infiltration of lung by malignant plasma cells secondary to orbital plasmacytoma.

Rare involvement of lung in myeloma patients can be due to intraparenchymal or mechanical dysfunction of thoracic cage and diaphragm. The intraparenchymal causes of respiratory failure from multiple myeloma include alveolar septal amyloidosis, plasma cell infiltration of lung parenchyma, accumulation of alveolar paraproteins, and metastatic calcification of the alveolar walls and blood vessels. [2–5]

Two other patients without acute lung injury had bilateral lung densities on radiographic imaging and pathological evidence of neoplastic plasma cell infiltration of the lungs. Chejfec et al described these pulmonary infiltrates as neoplastic plasma cells with a crystalline casts as “myeloma lung”[6-7.]

Poe et al[3] reported a patient with multiple myeloma with hypercalcemia presenting as progressive adult respiratory distress syndrome(ARDS). Post mortem analysis revealed diffuse metastatic calcification of alveoli and hyaline membrane formation.Respiratory involvement without parenchymal involvement has

been described through two main mechanisms: spontaneous flail chest and diaphragmatic dysfunction.

Spontaneous flail chest arises from multiple osteolytic rib cage lesions leading to pathological fractures, destabilisation and paradoxical breathing ensuing to respiratory failure.

Diaphragmatic dysfunction due to M protein associated sensory demyelinating polyneuropathy with phrenic nerve involvement.[8]

Multiple myeloma may produce varied patterns on chest radiographs including multiple masses mimicking solid tumour metastasis, diffuse interstitial disease from alveolar septal amyloidosis, or consolidation.[9-10]

In our observation, the patient presented with a chest radiographic pattern that was consistent with the diagnosis of ARDS, as defined by the American European Consensus Conference on ARDS.[11]

Since our patient did not respond to antibiotics, with a negative blood culture and striking hypercalcemia, hyperphosphatemia, with liver lesions. A prompt bone marrow biopsy was done to diagnose multiple myeloma with malignant infiltration of the lung presenting as non-infective adult respiratory distress syndrome.

IV. CONCLUSION

Multiple Myeloma presenting as ARDS is a rarity but should be kept in mind in a background of plasma cell dyscrasia and treatment unresponsive ARDS. High index of clinical suspicion with complete laboratory profile and early tissue diagnosis or bone marrow biopsy should be sought for the betterment of the patient.

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