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Pulmonary Renal Syndrome in Multiple Myeloma

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Abstract: There are a variety of clinical circumstances in which the respiratory and renal systems are simultaneously involved. This association is known as pulmonary-renal Syndrome. We describe a case of a 38-year-old man who presented an acute renal failure associated to pulmonary hemorrhage at onset. Investigations led to multiple myeloma. This case reveals that MM can be responsible of pulmonary-renal Syndrome.
Keywords: Multiple myeloma, pulmonary hemorrhage, acute renal failure

INTRODUCTION

The pulmonary-renal syndrome (PRS) is characterized by the coexistence of life-threatening pulmonary hemorrhage and renal disease in individuals without any concomitant destructive pulmonary disease or coagulopathy [1]. It associates a rapidly progressive glomerulonephritis (RPGN) and alveolar hemorrhage due to an autoimmune etiology [2]. Numerous systemic diseases share this presentation, specifically, Goodpasture's syndrome, systemic lupus erythematosus, progressive systemic sclerosis, Granulomatosis with polyangitis (GPA),lymphomatoid granulomatosis, and Churg-Strauss syndrome [3]. Multiple myeloma (MM) is a very rare cause of renal-pulmonary syndrome described, to our knowledge, only in two cases in the literature. In this article, we describe a new case of MM complicated by a pulmonary hemorrhage and acute renal failure.

CASE REPORT

We report the observation of a 38-year-old Moroccan man without significant medical history except one brother died at the age of 34 years in a context of advanced renal disease not on dialysis. His mother, however, was on chronic hemodialysis due to an undetermined nephropathy. He was referred for an acute renal failure discovered during a blood test performed for abdominal pain.

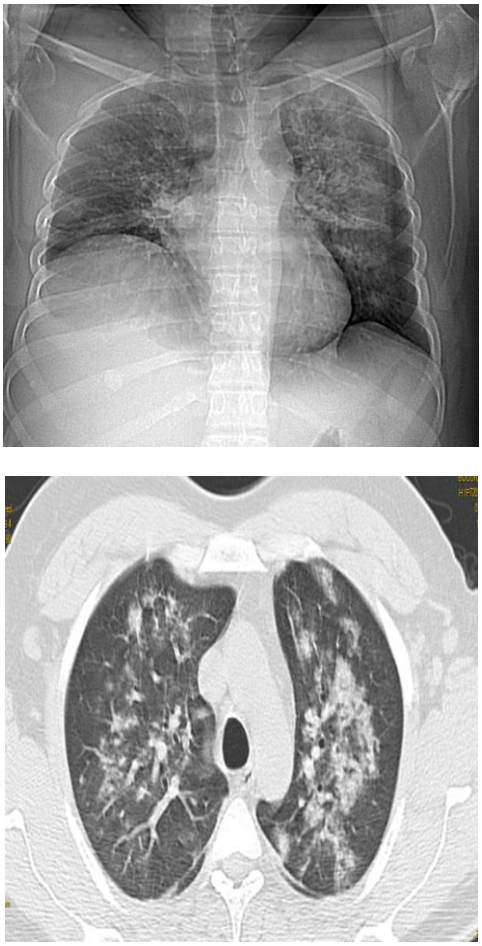
On admission, he had no hemodynamic or pulmonary disorder. His blood pressure was 120/70mmHg, pulse rate was 70/min, and his temperature was 37.2°C. Laboratory tests revealed these values: blood urea nitrogen 0,7g/l, serum creatinine 37 mg/l calcium 95 mg/l, white blood cells 6400/mm^3, hemoglobin 12,7 g/dL, platelets 106 000/mm^3, C-reactive protein (CRP) 9 mg/dL, Total protein 70 g/l, serum albumin 45 g/l. Serum protein electrophoresis showed: Albumin: 42g/l, α1 globulin=2,8g/l, α2 globulin=9,7g/l, β1globulin=4,5g/l, β2globulin=3,8g/l and aHypogammaglobulin emiagammaglobulin=4,2g/serum immunoelectrophoresis showed a kappa light chain band. Serum immunoglobulins test showed these values: Immunoglobulin M 0,25g/l, Immunoglobulin A 0,75g/l, Immuoglobulin G 3,54 g/l. Serum free light chain assay revealed 16085 mg/l of Kappa light chains. Bence-Jones proteinuria in a 24 h urine specimen was 2g of kappa light chains. The bone marrow aspirate showed 11% of plasma cells. Serum β2-microglobulin was 51mg/L. The diagnosis of kappa light chain multiple myeloma with Durie/Salmon stage IB and International Staging System (ISS) stage III was therefore established.

Two weeks later, his clinical condition rapidly deteriorated. He developed a severe dyspnea with signs of respiratory distress. He also developed hemoptysis, hypoxia (arterial oxygen saturation at room air was 80%) and oliguria. Urinalysis showed 2+ proteins and 1+ blood. He was rapidly transferred to intensive care unit where he received non invasive ventilation. He was also treated with continuous hemodialysis for oliguria. Chest X-ray and computed tomography (CT) scan indicated diffuse pulmonary infiltrates in the lung (Fig.1). Bronchoscopic examination and broncho-alveolar lavage (BAL) revealed a severe pulmonary hemorrhage.

Serum tests revealed a rapid deterioration of renal function (serum creatinine 130 mg/l, blood urea nitrogen 2,5 g/l, anemia (hemoglobin 6g/dl) and thrombopenia (platelets 40 000/mm^3).

Immunological tests were negative ANA, DNA antibody, p-ANCA and c-ANCA, anti-GBM. Complement levels were normal. Viral serologies HIV, HVB, HVC were all negative.

Immediate treatment concerned pulmonary-renal syndrome. He received a 3-day course of intravenous methylprednisolone (1g/day) followed by oral prednisolone and intravenous cyclophosphamide bolus. A few days later, the evolution was marked by improvement in clinical condition. He had no more dyspnea or pulmonary hemorrhage, his arterial oxygen saturation at room air was 95%. However, he remained dialysis dependent. This treatment also allowed improvement of anemia and a small improvement of thrombocytopenia. Renal biopsy has never been possible because of thrombocytopenia. Subsequent treatement concerned multiple myeloma. CDT protocolwasthenstarted (Cyclophosphamide-Dexamethasone-Thalidomide).



Fig−1: Chest X-ray and CT scan showing diffuse bilateral lung infiltrates

DISCUSSION

Alveolar hemorrhage in pulmonary-renal syndrome (PRS) occurs largely as a result of small vessel vasculitis of the lungs. ANCA-associated vasculitis and Goodpasture's disease are the most common causes of diffuse alveolar hemorrhage, while other pathologies including systemic lupus erythematosus and antiphospholipid syndrome, are rare causes of this syndrome [4]. These diagnoses have been excluded in our patient because autoantibodies were all negative and no other evidence supported these diseases.

Multiple myeloma is a malignant disease characterised by proliferation of clonal plasma cells in the bone marrow typically accompanied by the secretion of monoclonal immunoglobulins that are detectable in the serum or urine[5,6]. It is a very rare cause of pulmonary-renal syndrome, described to our knowledge, only twice in the literature [7,8]. We have presented, in this article, a new case of MM complicated by a PRS.

In fact, pulmonary hemorrhage can occur rarely within MM [9] and different mechanisms are involved including tracheobronchial amyloidosis, anoxia and thrombosis in capillary circulation, perivascular amyloid, and/or an acquired coagulopathy, such as coagulation factor X deficiency in primary amyloidosis [10] or pulmonary infection [7]. No evidence existed for these entities.

Renal failure is not rare in multiple myeloma. Approximately, 20% myeloma patients develop

progressive renal failure caused in most cases by free light chains deposit [11]. Acute kidney injury during MM could be secondary to an acute tubular necrosis, iatrogenic effects or an acute tubule interstitial nephropathy [12]. Our patient presented actually an acute renal failure, but wasn't secondary to these situations. Indeed, he had a rapidly progressive glomerulonephritis consecutive to the pulmonary renal syndrome.

One of the two cases of PRS in MM described already in the literature benefited of plasmapheresis[7]. However, further large-scale study is needed to clarify its usefulness [9,13]. Our patient was treated by Solumedrol and Cyclophasphamide bolus before MM diagnosis. His multiple myeloma was treated by CDT protocol with a very successful evolution.

CONCLUSION

Finally, this observation supports further the causality of multiple myeloma in pulmonary-renal syndrome.

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