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Case Report Open Access

Acute Respiratory Distress Syndrome Related to an Anti-glomerular Basement Membrane Disease without Evidence of Lung Hemorrhage

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Abstract

A 72-years-old woman was admitted for severe Acute respiratory distress syndrome (ARDS) with acute renal failure and proteinuria. Anti-glomerular basement membrane (ABMA) disease was confirmed with anti-GBM antibodies and renal biopsy but no lung hemorrhage was found. To our knowledge, the association between ARDS and ABMA disease has not been described so far in the literature. An effective treatment of the renal disease didn't improve the respiratory injury.

Keywords: Good pasture syndrome; Anti-glomerular basement membrane disease; Acute respiratory distress syndrome; Lung hemorrhage

Introduction

Antiglomerular basement membrane antibody (ABMA) disease is manifested by progressive glomerulonephritis, lung hemorrhage and antiglomerular basement-membrane antibodies whose target is the alpha 3 chain of type IV collagen (found both in glomerulus and alveolar membrane) [1]. When not obvious with hemoptysis, intra-alveolar hemorrhage can be shown with bronchoalveolar lavage through Golde score [2].

Case Report

A 72-years-old woman was admitted to the intensive care unit of Reunion Island University Hospital for febrile dyspnea. She had a previous diagnosis of Sjogren's syndrome complicated with pulmonary fibrosis (NYHA grade 2 dyspnea), had an immune thrombocytopenic purpura, hypertension, gout and transient ischemic attack. Her usual treatment was prednisone 5 mg per day, furosemide and inhaled corticosteroids. She never smoked and drunk alcohol occasionally. At medical examination, she presented with fever, declive edema, tachypnea, diffuses pulmonary crackles and oliguria. Laboratory examination revealed acute renal failure (serum creatinine 1700 µmol/l), anemia (9 g/dl), hyper leukocytosis, severe hypoxemia and proteinuria (10 g/g proteinuria/creatinine urine ratio). The eosinophil count was normal (0.2 G/L at admission and 0.3 G/L at the peak). Chest radiograph revealed bilateral patchy alveolar opacities (Figure 1) that were not present in an anterior radiography (Figure 2) and echocardiography excluded left ventricular failure. ABMA disease was confirmed with anti-GBM antibodies and renal biopsy showing cellular crescents in all glomeruli. She was also positive for p-AntiNeutrophil Cytoplasmic Antibody (p-ANCA).

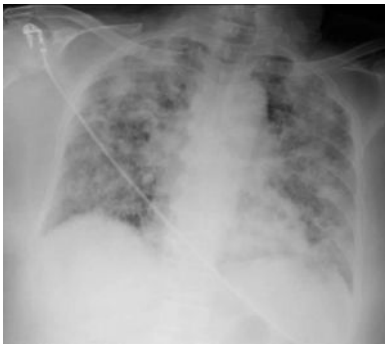

Figure 1: Admission chest radiography showing characteristic bilateral infiltrates.

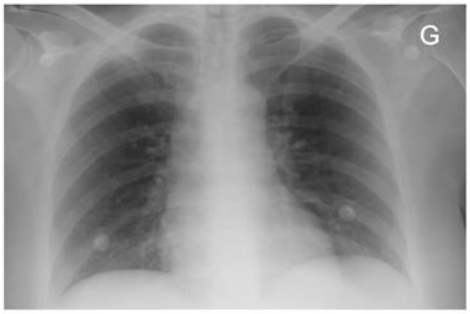

Figure 2: Anterior chest radiography (one month before).

Bronchial endoscopy and four bronchoalveolar lavages failed to show lung hemorrhage with a Golde score<20. The patient presented with a severe acute respiratory distress syndrome (ARDS) according to

the Berlin definition [3] justifying her intubation, the setup in prone position and the administration of atracurium. She was treated empirically with tazocillin, rovamycin and trimethoprim sulfamethoxazole. At the admission and during the hospitalization, haemocultures and bronchoalveolar lavage cultures were negatives. Legionella and pneumococcal antigen urine test were negatives, as well as many pulmonary pathogens (Table 1). Bronchoalveolar lavage cytology showed only macrophages and a few neutrophils but no eosinophils. The patient required renal replacement therapy from day one, corticosteroid therapy was started at day 5 (500 mg/kg/day for 3 days than 1 mg/kg/day) and plasmapheresis at day 9 after the results of the antibodies. We observed no improvement of her acute respiratory distress syndrome although the rate of antibodies decreased significantly and became negative at day 11. She died at day 15 from hypoxemia and we didn't sample any lung biopsy.

Variable	Serum	BALF
Serology	*Leptospira, Dengue virus*	-
PCR	*Aspergillus*	*P. jirovecii, Aspergillus*
	Leptospira, Dengue virus[*]	*Legionella, C. pneumoniae, M. pneumoniae*
	Adenovirus, CMV, EBV	*H. influenzae B, K. pneumoniae, S.aureus*, HSV-1 and 2, Influenzae virus A, B and H1N1, Parainfluenzae virus 1, 2, 3, 4, Rhinovirus, Coronavirus, Metapneumovirus, Bocavirus, Parechovirus, VRS[*]
[*]PCR multiplex		

Table 1: Negative microbiology.

Discussion

Intra-alveolar hemorrhage is known to occur more often with smokers [4] which was not the case here. Bronchoalveolar lavage is able, when not obviously red or pink, to indicate the lung hemorrhage by counting the deposition of hemosiderin in macrophage cells. An intra-alveolar hemorrhage is confirmed when the Golde score>100 [2]. In the literature, ABMA disease cases with lung hemorrhage are often easy to prove as the patients have hemoptysis or blood visible in endoscopy and in bronchoalveolar lavage [5]. Lung hemorrhage is an unusual cause of ARDS [6] but it is well-known in ABMA disease [5]. In this case, we failed to prove its responsibility to explain the ARDS. Moreover, we failed to find any usual cause of ARDS. Obviously, pneumonia was the second hypothesis in an immunocompromised patient (corticosteroids treatment) but no pathogen was recovered on different bronchoalveolar lavage cultures. The p-ANCA positivity may suggest an association with a microscopic polyangiitis (MPA) that also causes intra-alveolar hemorrhage. Diagnosis of eosinophilic granulomatosis polyangiitis is excluded in the absence of eosinophil in bronchoalveolar lavage and blood. The previous pulmonary fibrosis diagnosis may affect the severity of the ARDS but is not one of the recognized etiologies. Our hypothesis is that lung hemorrhage is possibly just a symptom of autoimmune lung injuries and is not correlated with the severity of the respiratory distress. This hypothesis is underlined by the fact that in a series of 28 cases with lung hemorrhage, there is no correlation between anemia and hypoxemia. On the other hand, in this series, no patient died and no ARDS were described [5] Furthermore, anti-GBM antibodies have affinity to the type IV collagen present in the alveolar basal membrane and induce alveolar-capillary barrier injuries [6]. Endothelial damages are also described in ARDS [7] similarly, in both pathologies, there is a cell-mediated inflammatory reaction with neutrophil activation and macrophages which are found in our samples [8]. To explain the fatal issue despite a right treatment, we know that ARDS progress independently from the treatment of its cause [7] so even if the rate of antibodies decreased significantly, the pulmonary lesion may have continued to advance on their own.

This case questions the obvious link between ABMA disease and ARDS as neither the evident cause that is lung hemorrhage nor the usual explanations were objectified. Moreover, it reminds us that acute or subacute kidney injury with respiratory distress, even without lung hemorrhage, should impose anti-GBM antibodies research.

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