Acute Renal Failure in Elderly due to Goodpasture’s Syndrome

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Sir,

Goodpasture’s syndrome is a disorder defined by anti-GBM antibodies with crescentic glomerulonephritis and hemoptysis. This syndrome usually occurs in the third decade, although no age is exempt and cases have been recorded in elderly patients [1]; respiratory failure may be a critical factor and a patent cause of death; the outcome of the severe form of glomerulonephritis is similar to that of rapidly progressive glomerulonephritis and spontaneous recovery from renal disease is uncommon [2].

An 81-year-old man was admitted to our hospital because of a 1-week history of dyspnea, cough, fever (38 °C) and oliguria. He was not a smoker. On admission serum creatinine was 6.6 mg/dl, BUN 117 mg/dl, total serum protein 7.3 g/dl, albumin 2.2 g/dl. WBC count was 12.3, hemoglobin 7 g/dl, hematocrit 30%, platelet count 317,000. Cryoimmunoglobulins and anti-DNA antibodies were negative; ASO titer and complement components were within the normal range. High titers of anti-GBM antibodies (1,000 EU) and ANCA (1,267 EU) were detected in the serum. Urinalysis showed glomerular proteinuria 2 g/l and 100 RBC/high power field. A chest X-ray revealed the presence of bilateral alveolar-interstitial infiltrates. Renal sizes were normal at sonography. Renal biopsy showed cellular crescents in 95% of the glomeruli with areas of capillary necrosis, widespread loss of tubular epithelial cells, hyaline and RBC casts; there was evidence of arteriolosclerosis; edema, fibrosis and infiltration by inflammatory cells were present in the interstitium. A continuous linear pattern of IgG and C3 outlining the glomerular capillary loops, and fibrinogen in the crescents, were found by immunohistology (fig. 1). During the following 10 days the patient developed progressive renal failure, anuria (BUN 119 mg/dl, creatinine 10 mg/dl) and conspicuous, relapsing hemoptysis. He was treated by hemodialysis, methylprednisolone pulses (15 mg/kg/day for 3 days), followed by oral prednisone (2 mg/kg every other day); plasma exchange, with removal of 2 liters at each session, was performed on the 2 days before the death of the patient. The patient died after 35
days of disease because of respiratory failure. Hemoptysis and renal failure were not improved by the treatment. Autopsy showed extensive hemorrhages in both lungs. Goodpasture’s syndrome is rather rare in Europe and in Italy too, and we have never observed anti-GBM glomerulonephritis as a cause of acute renal failure in our elderly patients. Two large series of Goodpasture’s syndrome have been reported in the literature: (i) in the Wilson and Dixon [3] study most patients were young males, and only 1 patient was 63 years old; (ii) among the 29 patients examined by Teague et al. [4] only 4 were older than 60 years.

In our 81-year-old patient we excluded the presence of cryoglobulinemia, systemic lupus erythematosus, polyarteritis nodosa, or Wegener’s granulomatosis. We did not recognize any events that could trigger the production of anti-GBM antibodies such as influenza A2 virus or hydrocarbon solvents inhalation. The clinical and histological diagnosis of Goodpasture’s syndrome was confirmed by the plasmatic determination of anti-GBM antibodies. Moreover, in our patient a high titer of ANCA was detected; the clinical significance of the association of anti-GBM antibodies with anti-neutrophil cytoplasm antibodies is still not well defined [5].

The treatment with steroids and plasma exchange did not improve the pulmonary picture and renal failure in our patient; however, poor results can be expected in oliguric patients or in those with renal failure severe enough to require dialysis. In the literature we did not find the occurrence of Goodpasture’s syndrome in patients over 80 years; in aged patients it is not surprising that the immunological responsiveness could give rise to such a disease [6]. The systematic execution of renal biopsy in old patients with acute renal failure might reveal a higher proportion of similar cases.

References