Goodpasture's Syndrome: A Case Presentation

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Dear Sir,

Goodpasture's syndrome is a disease which is characterized by an increase in anti-GBM (glomerular basement membrane) antibodies, pulmonary hemorrhage, and glomerulonephritis [1]. We have reached the conclusion that our patient is suffering from this particular disease because he had a high concentration of anti-GBM antibodies in his serum, presence of hemoptysis and a pathology with crescentic glomerulonephritis features. We have used corticosteroids, immunosuppressives, plasmapheresis, anticoagulants, and hemodialysis, as derived from a recent study on this rare disease.

The 24-year-old male patient, who had no complaints at all up until 2 months before admission when he developed hemoptysis together with a color change of his urine, getting reddish. He was hospitalized immediately. Physical examination revealed blood pressure 120/80 mm Hg and pulse 80/min. During the clinical observation the blood pressure had been found to be 210/120 mm Hg, so nifedipine was administered orally. During the systematic examination of the patient no particular features had been found except for the following: facial edema, pretibial edema and painful hepatomegaly (4 cm). His complaints on hemoptysis had continued after he was hospitalized. Anti-GBM antibodies (1,280 U) normalized to 0-10 U after treatment. There were no special features on chest roentgenography. Crescentic glomerulonephritis and sclerosis (> 80% of the glomeruli) was found on kidney biopsy and light microscopy. The above-mentioned clinical and laboratory findings were indicative of Goodpasture's syndrome, and he was treated with corticosteroids, immunosuppressives, plasmapheresis, anticoagulants and hemodialysis.

Goodpasture's syndrome had been defined as a disease with presence of anti-GBM antibodies or an accumulation of immunoglobulin deposits in the glomeruli and/or alveoli [2].

Radioimmunoassay is the most sensitive and specific method in the exploration of anti-GBM antibodies in the serum [1]. Immunofluorescent microscopy may be used for detecting anti-GBM antibodies in the tissue and for the diagnosis of the disease it is important to prove the presence of linear immunoglobulins [3]. Since the 70 s considerable extension of the survival rate had been achieved with high doses of corticosteroids, pulse treatment with cyclophosphamide, and plasmapheresis [4-6]. Renal transplantation may be possible if the

disease does recur and if the production of anti-GBM antibodies ceases [4]. But there are high recurrence rates during the posttransplantation period [7]. Corticosteroids may be used to control pulmonary hemorrhage, and immunosuppressives may alleviate the renal damage and clear away the antibodies on plasmapheresis [5, 6, 8, 9]. If this fails, ciclosporin (6 mg/kg/day) [10], azathioprine (1 mg/kg/day) [3] and chlorambucil [11] may be used. Additionally, anticoagulants must also be administered. We have concluded that if the disease can be identified and treated timely, the mortality due to pulmonary complications may be reduced. Important are early diagnosis and adequate treatment, especially when the chest roentgenogram is normal.

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