Diffuse Pulmonary Haemorrhage and Crescentic Glomerulonephritis

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Dear Sir,

Boyce and Holdsworth [1] reported a case of fatal pulmonary haemorrhage associated with crescentic glomerulonephritis (GN) in the absence of immunereactant deposition (idiopathic Goodpasture's syndrome). We read this report with great interest because recently we have reviewed our experience with diffuse pulmonary haemorrhage (DPH) associated with crescentic GN. In 54 cases of crescentic GN, 12 (22.2%) showed DPH: 3 cases had a systemic vasculitis, 5 showed circulating antiglomerular basement membrane antibody (anti-GBM Ab) with linear deposits of IgG on glomerular capillary walls (anti-GBM Ab mediated Goodpasture's syndrome) and the other 4 patients had a crescentic GN with negative immunofluorescence (IF) and without clinical or histological data of vasculitis. The latter 4 cases are, then, similar to that reported by Boyce and Holds-

worth [1] (idiopathic Goodpasture's syndrome). When comparing patients with anti-GBM Ab Goodpasture's syndrome (group I) and those with idiopathic Goodpasture's syndrome (group II), several interesting findings arose: the bouts of DPH were accompanied by important decreases of haematocrit and pO₂ in every case, without differences between the two groups. The percent of glomeruli with crescents and the degree of renal failure were similar in both groups (table I), and no differences were observed in the radiological picture. Some patients showed localized lung consolidations like the case of Boyce and Holdsworth [1], although diffuse pulmonary infiltrates were more frequently seen. Every patient was treated with intravenous prednisolone pulses and cyclo-phosphamide, in addition with plasmapheresis in 4 patients of group I. Prednisolone pulses had a clear beneficial effect on DPH, although 1 patient from each group died as a consequence of massive DPH.

Table I.

Mean ± SD

45 ± 17 83 ± 13

6.3 ± 2.7

Diffuse Pulmonary Haemorrhage and Crescentic Glomerulonephritis

157

We obtained a lung biopsy in 2 cases of group I and in 3 cases of group II (table I). Alveolar haemorrhage was present in every case, but neither vasculitis nor deposition of immune reactants were detected in any of the cases; 2 of the 3 patients of group II in whom a lung biopsy had been performed died, and the necropsic study confirmed the absence of vasculitis in lung or other tissues, and the negativity of IF.

Several authors have pointed out that DPH can be a more frequent finding in non-anti-GBM Ab mediated Goodpasture's syndrome than has been reported so far [2–4]. Our experience agrees with these observations and with the comments of Boyce and Holdsworth [1]. The pathogenesis of DPH in the cases not mediated by anti-GBM Ab or not associated with vasculitis, remains completely unknown. Furthermore, in some cases of anti-GBM Ab Goodpasture's syndrome with DPH, the IF studies have not showed deposition of immunoglobulins in lung tissue [5], the same as our patient 1 (table I). These findings cast some doubt on the exclusive role of anti-GBM Ab in the genesis of DPH in such patients. It is possible that other mechanisms have a pathogenic role in pulmonary injury.

References

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