Letters to the Editor

Acute crescentic glomerulonephritis developing during warfarin therapy

Sir,
Anticoagulants, together with immunosuppressive and antiplatelet therapy, have been advocated for the treatment of acute crescentic (rapidly progressive) glomerulonephritis (Kincaid-Smith *et al.*, 1970; Brown *et al.*, 1974). The use of anticoagulants is based on the histological demonstration of glomerular fibrin (Hoyer *et al.*, 1974), and evidence of consumption of coagulation factors in clinical and experimental rapidly progressive glomerulonephritis (Cameron, 1976). We have recently seen a case which calls into question the rationale for the use of anticoagulants in this situation.

A 71 year old man had undergone arterial bypass surgery for peripheral vascular disease 12 years previously, since which time he had been treated with warfarin. At a routine clinic visit, his prothrombin time, which until then had been consistently within the usual therapeutic range, was found to be greater than 120 seconds (control 13 seconds). Blood chemistry included urea 86 mmol/l, creatinine 1900 μ mol/l, potassium 8.3 mmol/l. Renal ultrasonography revealed normal kidney size and architecture. After correction of the clotting defect with vitamin K and fresh-frozen plasma, percutaneous renal biopsy was performed. This revealed an acute crescentic glomerulonephritis with 70% of glomeruli affected by circumferential crescents. Immunofluorescence showed intense fibrin deposition in glomeruli, mesangium, and capillaries, but no immunoglobulin deposition. Serum complement levels and immunoglobulins were normal, and tests for circulating immune-complexes were negative. Antibodies to glomerular basement membrane, nuclei, and double-stranded DNA were not detected. There was no clinical or histological evidence of a vasculitis or other systemic disease. The patient therefore had an idiopathic (nil immune deposit) crescentic glomerulonephritis (Stilmant *et al.*, 1979). He was treated with peritoneal dialysis, prednisolone, cyclophosphamide, and plasmapheresis, with eventual recovery of renal function and discharge from hospital.

Despite the fact that this patient was receiving anticoagulant therapy with a prothrombin-time consistently within or above the therapeutic range, he developed a diffuse crescentic glomerulonephritis, the predominant feature of which was intense fibrin deposition. We conclude from this case that intraglomerular fibrin deposition in rapidly progressive glomerulonephritis is not necessarily dependent upon

the activation of coagulation pathways leading to thrombin formation, but presumably derives from platelet-dependent coagulation. This is in keeping with the observation that glomerular fibrin deposition is not associated with demonstrable factor VIII antigen (Hoyer *et al.*, 1974), suggesting that the generation of fibrin is independent of thrombin formation by the classical coagulation cascade.

In the absence of any controlled trials demonstrating their efficacy in this situation, we would suggest that anticoagulation with warfarin or heparin, with their inherent risks, has no role in the treatment of idiopathic crescentic glomerulonephritis.

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Asymptomatic Graves' disease during lithium therapy.

Sir,
The patient reported by Drs Thompson & Baylis (1986) developed hyperthyroidism, while on lithium treatment. It was suggested that the drug had stimulated the thyroid antibody production. Eleven more cases of this syndrome had been described, and 5