INTRAVASCULAR COAGULATION IN RENAL DISEASE

by

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SUMMARY

The role of intravascular coagulation in the pathogenesis and natural history of many human renal diseases has been studied with the use of advanced techniques in coagulation and fibrinolysis and the examination of renal biopsy material. Whenever possible, opportunity was taken to study patients daily for long periods often far beyond any period of hospitalization. Information of most value was obtained from the use of Tanned Red Cell Haemagglutination Inhibition Immunoassay (T.R.C.H.I.I.) for the measurement of fibrin/fibrinogen degradation products (F.D.P.) in the serum and urine. Its routine application to urine samples in all long-term studies was particularly profitable.

In conditions such as acute ischaemic renal failure, the haemolytic uraemic syndrome, many forms of proliferative glomerulonephritis, and in rejecting renal homotransplants, coagulation data accurately reflected intra-renal events, which were evaluated by analysis of clinical details and histological, electron microscopical and in some cases immunofluorescent examination of renal biopsy material. Thus in acute ischaemic renal failure a novel hypothesis was proposed to explain the pathogenesis of the oliguria based on the findings, during the phase of oliguria, of significant abnormalities of coagulation and fibrinolysis, and electron microscopic evidence of glomerular coagulation. The

coagulation changes in the haemolytic uraemic syndrome were often more marked than in acute ischaemic renal failure and intrarenal thrombosis was more prominent and widespread. The prognosis in these cases seemed to depend on the degree of damage found in the kidney at the time of initial assessment, and whether the stimulus to coagulation was short-lived, persistent or recurrent. Anticoagulants did not influence the outcome.

The serial measurement of urinary F.D.P. excretion provided a reliable and sensitive index of disease activity, progression and natural history in proliferative forms of glomerulonephritis. Moreover, in certain instances of glomerulonephritis it was of diagnostic value. It also provided a useful monitor of the effects of certain anti-inflammatory drugs on some patients with glomerulonephritis as it was found that these drugs caused an acute reduction in urinary F.D.P. content. Similar observations were made on patients with renal homografts. In this situation elevated urinary F.D.P. concentrations were found in all episodes of clinical rejection. Furthermore, serial studies revealed other elevations which were considered to represent occult spontaneously reversible rejection.

The significance of these findings is discussed.