



INTRAVASCULAR COAGULATION IN RENAL DISEASE

by

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Thesis presented for the degree of
Doctor of Medicine of the University of Adelaide

Biological January, 1972 P.D.P.

Biological implications of P.D.P.

Biological implications of products of fibrinolysis

CONTENTS

1. <u>Historical development of the concept of intravascular coagulation in renal diseases.</u>	1
1.1 Intravascular coagulation	2
1.2 Intravascular coagulation and renal disease	6
1.3 Haemostasis	16
1.4 Platelet function in haemostasis	18
1.4.1 Platelet adhesion	19
1.4.2 Platelet aggregation	21
1.4.3 The platelet release reaction	24
1.5 The coagulation system	26
1.5.1 Contact activation	29
1.5.2 Tissue activation	32
1.5.3 The prothrombinase concept	32
1.5.4 Fibrinogen to fibrin conversion	33
1.5.5 Fibrin monomer complexes	35
1.6 Paracoagulation	37
1.6.1 Electron microscopy of fibrinogen to fibrin conversion	38
1.7 Fibrinolysis	38
1.7.1 Components of the fibrinolytic system	39
1.8 The proteolysis of fibrinogen and fibrin by plasmin	49
1.8.1 Characterization of F.D.P.	49
1.8.2 Biological properties of F.D.P.	52
1.8.3 Quantitation of F.D.P.	53
1.8.4 Clearance of products of fibrinolysis	54

1.9	Aims of project	54
2.	<u>Materials and Methods</u>	57
2.1	Materials	58
2.1.1	Buffers	58
2.1.2	Reagents	59
2.2	Methods	62
2.2.1	Plasma fibrinogen	62
2.2.2	Soluble fibrin monomer complexes	64
2.2.3	Plasma plasminogen activator	65
2.2.4	Urokinase	67
2.2.5	Fibrin/fibrinogen degradation products (F.D.P.)	67
2.2.6	Platelet factor 4	74
2.2.7	Column chromatography	75
2.2.8	Immune-electrophoresis	76
2.2.9	Urinary protein	77
2.2.10	Protein selectivity	77
2.2.11	Histological studies	79
3.	<u>Acute Renal Disease</u>	81
3.1	Acute ischaemic renal failure	83
3.1.1	Patients and Methods	86
3.1.2	Results	88
3.1.3	Coagulation: fibrinolytic studies	89
3.1.4	Histological and electron microscopic studies	94
3.1.5	Discussion	97

3.2	The haemolytic uraemic syndrome	105
3.2.1	Case selection	107
3.2.2	Clinical features	107
3.2.3	Laboratory features	109
3.2.4	Coagulation studies	110
3.2.5	Histopathology	112
3.2.6	Progress and management	116
3.2.7	Discussion	117
4.	<u>Glomerulonephritis</u>	123
4.1	Introduction	124
4.1.1	Glomerulonephritis caused by anti-glomerular basement membrane (anti-G.B.M.) antibodies	124
4.1.2	Glomerulonephritis caused by non-glomerular antigen-antibody complexes	125
4.1.3	Mediators of the inflammatory reaction	126
4.1.4	Classification of glomerulonephritis	130
4.2	Patients and methods	132
4.3	Results	133
4.3.1	Activity of disease	134
4.3.2	Relation between serum F.D.P. concentration and disease activity	136
4.3.3	Relation between urine F.D.P. concentration and disease activity	137
4.3.4	The significance of urinary fibrin excretion	139
4.3.5	Proteinuria and urinary F.D.P. excretion	143
4.3.6	Urokinase and urinary F.D.P. excretion	144
4.3.7	Column chromatographic analysis of urinary F.D.P. in glomerulonephritis	145

4.4	Discussion	149
5.	<u>Treatment of glomerulonephritis based on the urinary excretion of fibrin/fibrinogen degradation products (F.D.P.)</u>	154
5.1	Patients and methods	155
5.2	Results	157
5.2.1	F.D.P.	157
5.2.2	Chromatography	159
5.2.3	Renal function	159
5.2.4	Control studies	160
5.3	Discussion	161
6.	<u>Fibrin/fibrinogen degradation products following renal homotransplantation</u>	167
6.1	Introduction	168
6.2	Patients and methods	172
6.2.1	Patients	172
6.2.2	Methods	173
6.3	Results	173
6.3.1	Serum F.D.P.	173
6.3.2	Urinary F.D.P.	174
6.4	Discussion	177
7.	<u>Conclusion</u>	184
	Bibliography	191

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.