PATTERNS OF FIBRIN DEPOSITION IN THE GLOMERULI
OF DISEASED KIDNEYS

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Immunofluorescent studies were performed on 217 percutaneous renal biopsies on patients with various renal diseases, which were examined in detail to assess the amount, character, and distribution of fibrin deposits in the glomeruli.

The fibrin deposits were classified into six different forms on immunohistologic grounds. These were adhesive, adhesive and occlusive, membranous, mesangial, crescent forming, and sclerotic types.

The sclerotic type was further subdivided into 2 groups: periglomerular fibrosing, and sclerosis with occlusion types. In many instances, immunofluorescence may reveal a pattern which is commonly associated with a characteristic abnormality on light microscopy.

Fibrin deposits were commonly correlated with the presence of glomerular immunoglobulin and βc/βλa-globulin. These findings interpreted as the immune reaction within glomeruli leads to an initiation of the coagulation process. The pathogenesis of each type of fibrin deposits and the sequences of glomerular hyalinization are briefly discussed.

THE ROLE played by fibrin deposits and platelet aggregates in renal microcirculation in the production and perpetuation of a variety glomerular disorders has been well documented.1–3

Fibrin has been found in the kidneys both when antibody is produced against glomerular basement membrane (GBM), as in experimental Masugi nephritis,5 and when antigen-antibody complexes lodge in the renal filters5–7

The administration of anticoagulants has been shown to modify the course of these diseases.6–7

Some of the coagulation processes in glomerular damage observed in hypersensitivity nephritis do not stem directly from the injurious effects of the immune reaction, as for example, in toxemia of pregnancy.8 Thus the fibrin deposits in the glomeruli depends not only on the presence of immunologic reactants, but also upon the injurious effects of the coagulation process itself.

An attempt is made herein to determine the patterns of intraglomerular fibrin deposits and to elucidate their relationship to the glomerular damage.

MATERIALS AND METHODS

The material studied was human cortical tissue of the kidney obtained by percutaneous needle biopsy for diagnostic purposes.

Key Words:
Fibrin deposits
Six different types
Coagulation process
Immune reaction

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patients included in the study ranged in age from 16 to 70 years. One hundred and six patients were male, and one hundred and eleven were female. Classification of the type of renal disease and the activity of the disease process was made on the basis of interval history, renal biopsy find-
Fig. 4. Malignant nephrosclerosis in the case U.K.: showing fibrinoid degeneration of arteriole (arrow). Hematoxylin and eosin stain. Original magnification ×200.

Fig. 5. Glomerulus stained for fibrinogen from a patient with membranous nephropathy. Diffuse granular depositions of fibrin are seen along the GBM. Original magnification × 160.

Fig. 6. Glomerulus stained for fibrinogen from a patient with chronic glomerulonephritis. It shows specific fluorescence confined mostly to the mesangial area. Original magnification × 160.

...ings, serial creatinine clearance, urinalysis, serum chemistry, and the erythrocyte sedimentation rate, according to the criteria outlined in recent publications.9-12

The renal biopsy specimen was divided into two portions. One was fixed in 10% formal saline and embedded in paraffin. Sections of 3–4 microns were cut from the paraffin block and stained with hematoxylin and eosin, and periodic acid-Schiff reagent.

The other portion used for immunofluores-

cent studies was embedded in paraffin.13 All sections were examined for the presence of immunoglobulins G(IgG), A(IgA), M(IgM), βc/βλ-globulin and Fibrinogen. Immunological reagents were purchased commercially (Behring-Werke, AG) and used diluted 1:6 in phosphate buffer saline.

The specificity of immunohistochemical reac-
tivity of the fluorescent conjugates was verified by immuno-electrophoresis in many, but not all, phases of this study. In those instances where labelled antibodies were absorbed with their specific antigens, subsequent immunofluorescent reaction with tissues was not seen. Alternatively, in those instances where sections of kidney were first treated with unlabelled antibodies, sub-
sequent immunofluorescent reaction with the corresponding labelled antibody was markedly inhibited.

For convenience, material stained with fluorescein-labelled antifibrinogen serum is referred to as fibrin, although the material stained includes other derivatives of fibrinogen.

RESULTS

Fibrin deposits in the glomerulus was classified into six forms on the basis of its immunofluorescent features: adhesive, adhesive and occlusive, membranous, mesangial, crescent forming, and sclerotic type.

The adhesive type. This is characterized by the localization of fibrin predominantly on the endothelial side of the basement membrane, with appearance of fibrin aggregates (Fig. 1).
This type of fibrin deposition often occurred in combination with the other five types.

Detachment of the endothelium of glomerular capillary loops and deposits of fibrin aggregates in this region has been reported in various stages of experimental nephritis^4,6^; it is interesting that this type of fibrin deposition is also in clinical cases.

Even in "minimal change" nephropathy this type of deposits was demonstrated to the (+) or (++) level, a finding which concurs with reports of fibrin seen electronmicroscopically to adhere to the endothelial side of glomerular capillaries^2^.

The adhesive and occlusive type. This is characterized by segmental fibrin occlusion in the glomerular capillary loops with the adhesive type of fibrin localized in the glomeruli (Fig. 2).

This type of fibrin deposits was seen clinically in the critical stage of acute glomerulonephritis and in the period of acute exacerbation in chronic glomerulonephritis, and was seen histologically in diffuse proliferative glomerulonephritis (Fig. 3). Also noteworthy is that this type of deposits was observed in the following case of glomerulonephritis associated with malignant nephrosclerosis.

Case U. K. A 23-year-old man was admitted to Kyoto University Hospital with renovascular hypertension. He had been well until 8 years previously, when gross hematuria occurred after excessive exercise. Seven years previously, a routine examination had disclosed hypertension (220/120) which had been treated for 2 years without success. He was seen at another hospital for further evaluation of hypertension, where he was considered to have renal arterial stenosis.

He was then referred to Kyoto University Hospital for examination and treatment. Blood-

Fig.12. Glomerulus stained for fibrinogen from a patient with diabetic nephropathy. Note the occlusion pattern of specific fluorescence in the sclerosing glomerulus. Original magnification × 160.

Fig.13. Stain for IgG in case I.S. Bright, granular staining is seen along the GBM. Original magnification × 160.

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\[^4^\] For specific references, please consult the original source.
pressure was 190/130. Urine protein level was 2.5 g/24 hr and GFR was 104 ml/min. Urine sediment was seen to contain 1 white cell, a few red cells and occasional casts per high-power field.

Renal biopsy showed chronic proliferative glomerulonephritis and malignant nephrosclerosis with fibrinoid degeneration of arterioles (Fig. 4).

Immunofluorescence revealed fibrin deposits of the adhesive and occlusive type (Fig. 2). IgG, \( \beta_{1c}/\beta_{1a} \)-globulin, IgA and IgM were also observed along the glomerular basement membrane and mesangium.

The membranous type: This is characterized by granular or linear fibrin deposits either sub-endothelially or within the basement membrane (Fig. 5). These deposits seem to occupy a similar place to the immune complex on the glomerular basement membrane (GBM).

Diffuse fibrin deposits found along the entire GBM were peculiar to membranous nephropathy. Segmental membranous type fibrin deposits were found in acute glomerulonephritis, chronic glomerulonephritis, SLE and diabetic nephropathy; immunoglobulin and \( \beta_{1c}/\beta_{1a} \)-globulin were frequently demonstrated in virtually identical sites.

The membranous type of fibrin deposits often occurred in combination with the adhesive type: a situation is thought to arise in which fibrin is prone to adhere to the endothelial side of the GBM due to an immunological mechanism (as in membranous nephropathy or SLE) or a non-immunological mechanism (as in diabetic nephropathy). Membranous nephropathy associated with renal vein thrombosis has also been reported, and the author has encountered a case of membranous nephropathy with extensive vascular thrombosis.

The mesangial type. This is characterized by patchy or granular fibrin scattered within the mesangium (Fig. 6). This type only was observed infrequently, but the following two cases were notable.

**Case S.M.** A 22-year-old female was admitted to the hospital with proteinuria and microscopic hematuria. Two months previously, routine examination had disclosed proteinuria and she came to the hospital for further evaluation.

Urinary protein level was 1 g/24 hr, and the sediment contained 20 red cells, 4 white cells and occasional granular casts per high-power field. GFR was 105 ml/min.

Renal biopsy showed slight mesangial proliferative changes (Fig. 7). Mesangial type fibrin deposits were observed by immunohistology (Fig. 6). IgG, IgA and FDP(E) deposits (Fig. 8) were also observed in the mesangial area.

The other case was a syphilitic patient in whom mild proteinuria was seen clinically at intervals, and in whom renal biopsy showed only slight mesangial proliferation. Hematuria was absent. Immunohistology revealed slight IgG, \( \beta_{1c}/\beta_{1a} \)-globulin and IgA deposits along the GBM.

The crescent forming type. This is characterized by extensive fibrin deposits in the crescent and is observed in hyalinosing glomeruli in extracapillary glomerulonephritis (Fig. 9). The following case is typical.

**Case S. R.** A 25-year-old female was admitted to the hospital with gross hematuria. One year previously, she had a cold and sore throat, with frequently coughing and fever. At that time urinalysis was not performed. Three months before admission she came to the hospital with gastric upset, at which time proteinuria and microscopic hematuria were found. One month before admission, macroscopic hematuria occurred.

Urinary protein level was 2g/24 hr, and the urine sediment contained numerous red cells per high-power field. GFR was 80 ml/min. Renal biopsy disclosed extracapillary glomerulonephritis with crescent formation (Fig. 10). Immunohistologically, the crescent forming type of fibrin deposits was observed (Fig. 9). IgG, \( \beta_{1c}/\beta_{1a} \)-globulin, IgA and IgM were not observed in the crescent area.

The sclerotic type is characterized by fibrin deposits in the almost hyalinized glomerulus. This type was subdivided into 2 groups depending on the form of fibrin deposits in the glomerulus. These are the periglomerular fibrosing type (Fig. 11) and hyalinized glomerulus with fibrin thrombi (Fig. 12).

The periglomerular fibrosing type of deposits was observed by renal biopsy in glomeruli in case of chronic renal disturbance with a long course where renal function was relatively unimpaired. This occurred in membranous nephropathy, the latent stage of chronic glomerulonephritis and diabetic nephropathy. The following is one such case.

**Case I. S.** A 42-year-old man was admitted to
Kyoto University Hospital with heavy proteinuria and edema. At the age of 39, routine urinalysis had revealed proteinuria, but specific therapy was not administered.

Urine protein level was 5 g/24 hr and GFR was 133 ml/min. The urine sediment contained 7 red cells, 2 white cells and 2 coarsely granular casts per high-power field.

Renal biopsy revealed membranous nephropathy. IgG (Fig. 13), β_{1c}/β_{1a}-globulin, IgE, fibrin (Fig. 5) and, to a lesser extent, IgM were deposited in a granular pattern along the GBM. He received steroid treatment for one year with no improvement. A second renal biopsy was performed one year later. Immunofluorescence revealed the periglomerular fibrosing type of fibrin deposits (Fig. 11) in one of eight glomeruli. Other immunohistological findings were almost identical with those of the first renal biopsy except that a striking reduction in fluorescent staining of β_{1c}/β_{1a}-globulin was seen in the second biopsy specimen.

The second group of sclerotic type deposits, hyalinized glomeruli with fibrin thrombi, was observed in cases where blood flow had almost ceased in the almost completely hyalinized glomerulus.

A simplified scheme of fibrin deposits is shown in Fig. 14. The pattern of fibrin deposits and the frequency of IgG, IgA, IgM and β_{1c}/β_{1a}-globulin deposits in the glomeruli with various renal diseases are summarized in Table I A, B, C, D, E & F.

As can be seen in Table I, there is tendency to be a positive correlation between the intensity of fibrin deposits and the frequency of IgG, IgA, IgM and C'3 deposits in the glomeruli in various renal diseases. The results suggest that fibrin deposits are important relationship with immune complex deposits and development of renal disorders.

DISCUSSION
The presence of fibrinogen related antigens revealed by light microscopy immuno-fluo-

TABLE 1  GLOMERULAR PATHOLOGIC FEATURES BY IMMUNOFUORESCENT MICROSCOPY

<table>
<thead>
<tr>
<th>A) Acute Glomerulonephritis</th>
<th>Localized human proteins</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fibrin</td>
<td>Extent</td>
</tr>
<tr>
<td>No positive finding</td>
<td>2</td>
</tr>
<tr>
<td>Ad.</td>
<td>+</td>
</tr>
<tr>
<td>Ad.</td>
<td>++</td>
</tr>
<tr>
<td>Ad. &amp; Membr.</td>
<td>+</td>
</tr>
<tr>
<td>Ad. &amp; Membr.</td>
<td>++</td>
</tr>
<tr>
<td>Ad. &amp; Membr.</td>
<td>+++</td>
</tr>
<tr>
<td>Membr. &amp; Mes.</td>
<td>++</td>
</tr>
<tr>
<td>Ad. &amp; Occ.</td>
<td>++</td>
</tr>
<tr>
<td>Ad. &amp; Membr. &amp; Occ.</td>
<td>++</td>
</tr>
</tbody>
</table>

Acute glomerulonephritis with crescent formation*

| Solitary crescent forming type** | 1 | 1/1 | 1/1 | 1/1 | 0/1 |
| Ad. | + | 3 | 3/3 | 1/3 | 3/3 | 1/3 |
| Ad. | ++ | 2 | 2/2 | 2/2 | 2/2 | 2/2 |
| Ad. & Membr. | + | 2 | 2/2 | 0/2 | 2/2 | 1/2 |
| Ad. & Membr. | ++ | 2 | 2/2 | 1/2 | 2/2 | 2/2 |
| Ad. & Membr. & Occ. | ++ | 1 | 1/1 | 1/1 | 1/1 | 1/1 |

* Crescent forming type was observed in some of glomeruli in the biopsy specimen  
** Crescent forming type was observed in some of glomeruli, but the other glomeruli were no  
positive finding of fibrin deposits  
Note: Ad.=adhesive type; Occ.=occlusive type; Membr.=mesangial type; Membr.=membranous type;  
Sclero.=sclerotic type  
Numerator=Number of patients showing deposition  
Denominator=Number of patients in the test  
+, ++, +++: Grading of lesions signifies a combined estimate of  
The amount of immunofluorescence  
The size of the individual deposits  
The intensity of fluorescence of deposit in the glomerulus

Rescent studies15-17 and electron microscopic studies8,18 suggest that intravascular coagulation may play a role in the pathogenesis and progression of certain renal diseases.

These observations and studies in experimental animals have led to the use of anticoagulants in human renal disease19,20. The suggestion that coagulation might be of importance came from the observation in some cases of severe and rapidly progressive acute glomerulonephritis.

It is generally agreed that crescent formation appear to represent organization of fibrin deposited within Bowman's space. That fibrin formation plays a major role in the process of crescent formation and of glomerular sclerosis is in accord with recent reports21 and current concept on their pathogenesis.

Rabbit Muraghi nephritides resembled human rapidly progressive glomerulonephritis in its clinicopathological features including fibrin precipitates in the crescents4,6.

It also would appear that in the early lesion of glomerulonephritis the endothelial cells were detached from the basement membrane2. Using immunofluorescence methods, the author demonstrated adhesive type fibrin deposits in

### B) Chronic Glomerulonephritis

<table>
<thead>
<tr>
<th>Localized human proteins</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fibrin Pattern</strong></td>
</tr>
<tr>
<td>----------------------</td>
</tr>
<tr>
<td>No positive finding</td>
</tr>
<tr>
<td>Ad.</td>
</tr>
<tr>
<td>Ad.</td>
</tr>
<tr>
<td>Ad.</td>
</tr>
<tr>
<td>Memb.</td>
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<tr>
<td>Memb.</td>
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<td>Memb.</td>
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<tr>
<td>Memb.</td>
</tr>
<tr>
<td>Memb.</td>
</tr>
<tr>
<td>Mes.</td>
</tr>
<tr>
<td>Ad. &amp; Occ.</td>
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**Chronic glomerulonephritis with crescent formation**

<table>
<thead>
<tr>
<th></th>
<th><strong>Solitary crescent forming type</strong></th>
<th><strong>Ad.</strong></th>
<th><strong>Ad.</strong></th>
<th><strong>Ad. &amp; Mem</strong></th>
<th><strong>Ad. &amp; Mem</strong></th>
<th><strong>Ad. &amp; Mem</strong></th>
</tr>
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<td></td>
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<td>2/2</td>
<td>0/2</td>
<td>1/2</td>
<td>1/2</td>
<td>1/2</td>
</tr>
<tr>
<td></td>
<td>++5</td>
<td>4/5</td>
<td>3/5</td>
<td>5/5</td>
<td>5/5</td>
<td></td>
</tr>
<tr>
<td></td>
<td>++2</td>
<td>2/2</td>
<td>2/2</td>
<td>2/2</td>
<td>1/2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>++2</td>
<td>2/2</td>
<td>0/2</td>
<td>2/2</td>
<td>2/2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>+++1</td>
<td>1/1</td>
<td>1/1</td>
<td>1/1</td>
<td>0/1</td>
<td></td>
</tr>
</tbody>
</table>

**Advanced glomerulosclerosis due to glomerulonephritis**

<table>
<thead>
<tr>
<th></th>
<th><strong>Ad.</strong></th>
<th><strong>Ad. &amp; Occ.</strong></th>
<th><strong>Ad. &amp; Occ.</strong></th>
<th><strong>Ad. &amp; Mem</strong></th>
</tr>
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<tbody>
<tr>
<td></td>
<td>++1</td>
<td>1/1</td>
<td>0/1</td>
<td>1/1</td>
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<tr>
<td></td>
<td>+1</td>
<td>1/1</td>
<td>1/1</td>
<td>1/1</td>
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<tr>
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<td>++1</td>
<td>1/1</td>
<td>0/1</td>
<td>0/1</td>
</tr>
<tr>
<td></td>
<td>++3</td>
<td>3/3</td>
<td>2/3</td>
<td>3/3</td>
</tr>
</tbody>
</table>

***The sclerotic type was observed in many of glomeruli, but the glomeruli that were less severely involved had various patterns of fibrin deposits.***

The adhesive type of fibrin deposits in glomeruli has been found to be the most common in various renal diseases. This observation led to the suggestion that the endothelium of the glomerular vessels is damaged by deposits of complement and by the consequent inflammatory reaction, which is followed by aggregation of platelets and the occurrence of local intravascular coagulation resulting in formation of fibrin deposits in the glomeruli. It is also known that during coagulation various kinds of incompletely polymerized fibrin...
Patterns of Fibrin Deposition in the Glomeruli of Diseased Kidneys

C) Membranous Nephropathy

<table>
<thead>
<tr>
<th>Fibrin</th>
<th>Extent</th>
<th>No.</th>
<th>IgG</th>
<th>C'3</th>
<th>IgA</th>
<th>IgM</th>
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<tbody>
<tr>
<td>No positive finding</td>
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<td>3/3</td>
<td>1/3</td>
<td>1/3</td>
<td>1/3</td>
<td></td>
</tr>
<tr>
<td>Ad.</td>
<td>+</td>
<td>2</td>
<td>2/2</td>
<td>1/2</td>
<td>1/2</td>
<td>1/2</td>
</tr>
<tr>
<td>Ad. &amp; Memb.</td>
<td>+</td>
<td>2</td>
<td>2/2</td>
<td>2/2</td>
<td>2/2</td>
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</tr>
<tr>
<td>Ad. &amp; Memb.</td>
<td>++</td>
<td>4</td>
<td>4/4</td>
<td>3/4</td>
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<td>2/4</td>
</tr>
<tr>
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<td>+++</td>
<td>1</td>
<td>1/1</td>
<td>1/1</td>
<td>1/2</td>
<td>1/1</td>
</tr>
<tr>
<td>Memb.</td>
<td>+</td>
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<td>3/3</td>
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</tr>
<tr>
<td>Memb.</td>
<td>++</td>
<td>2</td>
<td>2/2</td>
<td>2/2</td>
<td>2/2</td>
<td>2/2</td>
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<td>+++</td>
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<td>7/7</td>
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**** The periglomerular fibrosing type was observed in some of glomeruli.

D) Minimal Change

<table>
<thead>
<tr>
<th>Fibrin</th>
<th>Extent</th>
<th>No.</th>
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<th>C'3</th>
<th>IgA</th>
<th>IgM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ad.</td>
<td>+</td>
<td>3</td>
<td>0/3</td>
<td>0/3</td>
<td>1/3</td>
<td>0/3</td>
</tr>
<tr>
<td>Ad. &amp; Memb.</td>
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<td>1</td>
<td>0/1</td>
<td>0/1</td>
<td>0/1</td>
<td>0/1</td>
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<tr>
<td>Ad. &amp; Memb.</td>
<td>++</td>
<td>1</td>
<td>1/1</td>
<td>0/1</td>
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E) Diabetic Nephropathy

<table>
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<th>Fibrin</th>
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<th>No.</th>
<th>IgG</th>
<th>C'3</th>
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<th>IgM</th>
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<tbody>
<tr>
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<tr>
<td>Ad.</td>
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<td>5</td>
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<td>1/5</td>
<td>3/5</td>
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</tr>
<tr>
<td>Ad.</td>
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<td>1</td>
<td>1/1</td>
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</tr>
<tr>
<td>Ad. &amp; Memb.</td>
<td>+</td>
<td>3</td>
<td>3/3</td>
<td>2/3</td>
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<tr>
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<td>++</td>
<td>1</td>
<td>1/1</td>
<td>1/1</td>
<td>1/1</td>
<td>1/1</td>
</tr>
<tr>
<td>Ad. &amp; Memb. &amp; Occ.****</td>
<td>++</td>
<td>2</td>
<td>2/2</td>
<td>2/2</td>
<td>2/2</td>
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<tr>
<td>Sclerot.</td>
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</table>

In minimal change and some cases of diabetic nephropathy in Table I: D, E, the staining pattern of glomeruli for IgG, IgA and IgM showed ultralinear distribution.


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are formed, such as cryoprotfibrin, and that they behave in the circulation like colloidal material in that they are phagocytosed by cells of the reticuloendothelial system. Although circulating colloidal material in general is largely cleared by the reticuloendothelial system, some is also arrested in the glomeruli, probably due to trapping along the filtering bed, as well as phagocytosis. It would thus appear reasonable to assume that some of this trapped colloidal material in the glomeruli is also present as adhesive fibrin deposits along the GBM.

In any event, coagulation in the glomeruli may occur by both immunological and non-immunological mechanisms. In the former, it has been shown that immune complexes can accelerate clotting in vitro through platelet damage and activation of the Hageman factor. In addition, when large amounts of immune complexes are formed in or introduced into the circulation, coagulation occurs rapidly within the blood stream, leading to widespread deposits in capillaries, especially in the glomeruli. This would accentuate the profuse local clot formation which occurs on the damaged endothelial surface and could be an important fundamental etiologic factor resulting in platelet sticking and localized fibrin thrombi, which are responsible for the occlusive type of fibrin deposits.

The mesangial and membranous type of fibrin deposits in the glomeruli showed a distribution pattern comparable to that of immunoglobulins and complement.

Immune complexes can produce considerable deposits of fibrin in glomeruli in vivo, notably when fibrinolytic mechanisms are impaired by renal damage. In the case of hypersensitivity reactions, the formation of fibrin could result from the action of antigen-antibody complexes on platelets and the fibrinoid in such cases might contain immune complexes.

Hence it can be understood how the membranous and mesangial types of fibrin deposits can occur in virtually the same location as the immune complexes. Moreover, in the mesangial type, it is also possible that the mesangium may phagocyte the fibrin aggregates as foreign bodies.

In malignant nephrosclerosis, the participation of an immunological mechanism in the fibrinoid necrosis of renal arterioles has been reported. It is conceivable that in combination with glomerulonephritis the coagulation process would be further accelerated leading to the appearance of more severe fibrin deposits of the occlusive type.

Further, when one considers the process of transition from fibrin deposits to hyalinosis of the glomerulus, in the early stages of the disease there is damage or detachment of the endothelium by either immunological or non-immunological mechanism and in this region the adhesive type of fibrin deposits occurs.

As described previously, under conditions where the reaction occurs more strongly, the occlusive type of deposits which causes total or partial occlusion of the glomerular capillary loop may occur. The body reacts to reverse this trend by the fibrinolysis process, but depending on the
Fig. 15. Glomerulus stained for fibrinogen from a patient with extracapillary glomerulonephritis. Note the fibrin leakage (arrow). Original magnification ×160.

From the standpoint of fibrin deposits in glomeruli, the sequences of glomerular hyalinization are postulated as follows:

1. Coagulation
2. Fibrinolysis

Adhesive type
Mesangial type
Membranous type

Occlusion type

Release of vasoactive amines

Increased glomerular permeability

Leakage of fibrin

Hyalinization

Fibrosis

Periglomerular fibrosing type

Occlusion type

condition of the disease, the coagulation process which accentuates the situation may also act. When fibrinolytic activity decreases due to renal damage, the latter action tends to predominate. If the coagulation processes, vasoactive amines are released by the reaction of fibrin and aggregated platelets, and these enhance glomerular permeability and raise further the deposits of immune complexes.

Linked with this, attraction of polymorphs, C' fixation etc., may further intensify the reaction and bring about disruption of the basement membrane. Leakage of fibrin into Bowman's space (Fig. 15) will cause proliferation of epithelial cells in reaction.21 and development of the glomerular crescent will be accelerated. In extracapillary glomerulonephritis, hyalinosis of the glomerulus occurs by this type of reaction which is rapidly progressive.

However in renal diseases of protracted course, for example in membranous nephropathy or in the latent stage of chronic glomerulonephritis, the periglomerular fibrosing type of deposits is seen and fibrosing is thought to occur slowly.

Nevertheless, for either process to lead finally to fibrosing, complete hyalinosis due to arrested blood flow through some degree of occlusive type deposits with sclerosis is thought to occur.

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