ACTA MEDICA IRANICA
vol. XVII. 1974, p.1-29

# THE SPECTRUM OF RENALDISEASEIN IRAN A POST-MORTEM STUDY

K. ARMIN M.D., N. PISHVA M.D., A. GHAFFAR-ADLY M.D.

The pathological and clinical study of renal disease has always been a great problem (41, 51, 2) because different anatomical lesions may cause similar clinical signs and symptoms. The end stage of all renal disease has a similar clinical and biochemical picture that is known as renal failure (21). Various terms such as nephritis, nephrosis, nephritis nephrosis, acut, sub-acute and chronic nephritis which clinicians used to use do not describe the underlying etiology and pathological lesions. On the other hand, the microscopic appearance of many renal diseases may become undistinguishable from one another at the end stage of the disease (27). Acute reaction of the kidney to injuries of various causes is like that of other tissues, an accumulation of polymorphonuclear leukocytes, and when the process becomes chronic, scarring and tissue destruction is prominent. We know very well-to-day, that urinary tract infections (48) with or without clinical symptoms are dangerous and may lead to renal failure and death (25). The problem may not be of such magnitude in countries where there are better facilities for medical care and where the disease can be diagnosed at an earlier stage (18, 53). Reliable statistics showing the incidence of renal disease in Iran are not available. We present here a report concerning fatal renal disease in Iran as seen in the autopsy material of the University of Tehran School of Medicine in the last 15 years, and a discussion of the related histopathological findings.

Department. Pathology. School of Medecin. Teheran. University.

## Material and Methods

In order to study the incidence and histopathological aspects of renal disease and its role in the mortality rate, the protocols of 4463 autopsies performed in the last 15 years at the University hospitals were studied. The majority of the patients admitted to these hospitals are farmers or workers from all parts of the country. Attempts have been made to complete the data by combining information from clinical and laboratory findings and the lesions found at autopsy.

The stains used for this report are:

1. Hematoxilin eosin.
2. Aniline blue for indentification of the glomerular basement membrane.
3. PAS (Periodic-acid-Schiff).

## Results and Comments

Our great problem in the diagnosis of renal disease is that we usually examine the kidney tissue in the end stage of disease, and that renal biopsy, the best tool for diagnosis, is not available for most of our physicians working in the provinces.

Table 1 shows that among 4463 autopsies performed in the last 15 years, there are 861 (20 %) cases of renal disease.

Table 2 shows the incidence of the different forms of renal disease.

**Disease of the renal interstitium:**

The most common renal disease in this report consists of 282 cases (33.2 %) of interstitial nephritis and pyelonephritis. The incidence of these diseases varies greatly in different reports, because the conception of this group of renal disease is not the same for all investigators. Chronic paelonephritis (39) is the end result of bacterial infection of the kidney, in which pathological changes are found in both parenchyma and the pelvic calyceal

system. At the present time chronic pyelonephritis has become a confusing term (20) because many pathologists concentrate purely on the parenchymal changes, forgetting that there is a pyelo-component. Table 3 shows the incidence of acute and chronic pyelonephritis according to sex and age.

Table. 1 **The incidence of renal disoders among 4463 autopsies**

| Age group | No of autopeies | Renal disorder | | Total |
|---|---|---|---|---|
| | | Mal | Female | |
| Over 14 years | 2298 | 467 | 249 | 716 |
| Under 14 years | 2165 | 92 | 53 | 145 |
| Total | 4463 | 559 | 302 | 861 |

Table 2 **The incidence of various renal diseases in 861 cases renal disorders among 4463 autopsies**

| Type of disease | No | % |
|---|---|---|
| Disease of the interstitium | 282 | 32,75 |
| Renal lesion in tuberculosis | 176 | 20,44 |
| Disease of the blood vessels | 112 | 13,01 |
| Disease of the glomeruli | 101 | 11,73 |
| Rheumatic carditis | 48 | 55,57 |
| Epithelial nephrosis | 30 | 3,48 |
| Non-tuberculous amyloidosis | 28 | 3,25 |
| Congenital malformation | 28 | 3,25 |
| Non-tuberculous diabetes | 20 | 2,32 |
| Tumor | 13 | 1,51 |
| Speticemia | 10 | 1,16 |
| Eclamoia | 8 | 0,93 |
| Myelomatous kidney | 5 | 0,58 |
| Total | 861 | 100 |

| Type of lesion | Age group | | | | Total |
|---|---|---|---|---|---|
| | Over 14 years | | Under 14 years | | |
| | Male | Female | Male | Female | |
| Chronic pyelonephritis | 93 | 47 | 15 | 5 | 160 |
| Acute pyelonephritis | 7 | 25 | 8 | 5 | 45 |
| Chronic interstitial nephritis | 10 | 29 | 2 | 1 | 42 |
| Hydronephrosis and calculus | 4 | 12 | 8 | 6 | 30 |
| Acute interstitial nephritis | 1 | 4 | — | — | 5 |
| Total | 163 | 69 | 17 | 33 | 282 |

Table 3

**The incidence of various diseases of the interstitium, according to age and sex**

**Renal lesions in tuberculosis:**

Tuberculosis may produce various types of renal lesion, of tuberculous or non-tuberculous nature. Table 4 shows that among 4463 autopsies there were 429 cases of tuberculosis (10 %), 253 cases of which did not show any renal alteration, but in 176 cases the renal lesions were striking. Table 5 shows the incidence of various types of renal lesion. The majority of patients, suffering from terminal pyelonephritis had preaortic tuberculous adenopathies. In the cases of non-tuberculous glomerulonephritis the glomeruli show hypercellularity and basement membrane thickening of various degrees. Thickening of the basement membrane can be diffuse or mottled (6), but the majority of capillaries are patent. The hypercellularity is usually caused by proliferation of monocytes and rarely polymorphonuclear leukocytes take part in the process.

| Age group | Number of autopsies | Number of tuberculosis cases | |
|---|---|---|---|
| | | With renal lesion | Without renal lesion |
| Over 14 years | 2298 | 144 | 159 |
| Under 14 years | 2165 | 32 | 34 |
| Total | 4463 | 176 | 253 |
| | | 429 | |

Table 4

**The incidence of tuberculosis among 4463 autopsies**

| | | Various renal diseases | | | | | |
|---|---|---|---|---|---|---|---|
| Age group | | Nephrotic syndrome | Amyloidosis | Pyelonephritis | Glomerulo-nephritis | Tuberculous follicles | Total |
| Oover 14 years | Male | 6 | 19 | 19 | 16 | 21 | 81 |
| | Female | 3 | 13 | 25 | 7 | 15 | 63 |
| Under 14 years | Male | — | 2 | 1 | — | 14 | 17 |
| | Female | 2 | 2 | 2 | 1 | 8 | 15 |
| Total | | 11 | 36 | 47 | 24 | 58 | 176 |

Table 5

**The incidence of various renal lesion in 176 tuberculous patients**

**Disease of the blood vessels:**

Various renal diseases (33) may cause secondary vascular alterations, but the term vascular disease is reserved for specific diseases with primary involvement of blood vessels. Table 6 shows the incidence of hypertension in various renal diseases according to age and sex. The most common causes of hyertension in this report were benign nephrosclerosis, malignant nephrosclerosis, renal infarction and cortical necrosis.

| Kind of disease | Age Group | | | | Total |
|---|---|---|---|---|---|
| | Over 14 years | | Under 14 years | | |
| | Male | Female | Male | Female | |
| Vascular disease | 30 | 30 | — | — | 60 |
| Pyelonephritis | 30 | 19 | 2 | — | 51 |
| Glomerulonephritis | 30 | 6 | 4 | — | 40 |
| Diabetes | — | 3 | — | — | 3 |
| Tuberculosis | — | 2 | — | — | 2 |
| Total | 90 | 60 | 6 | — | 156 |

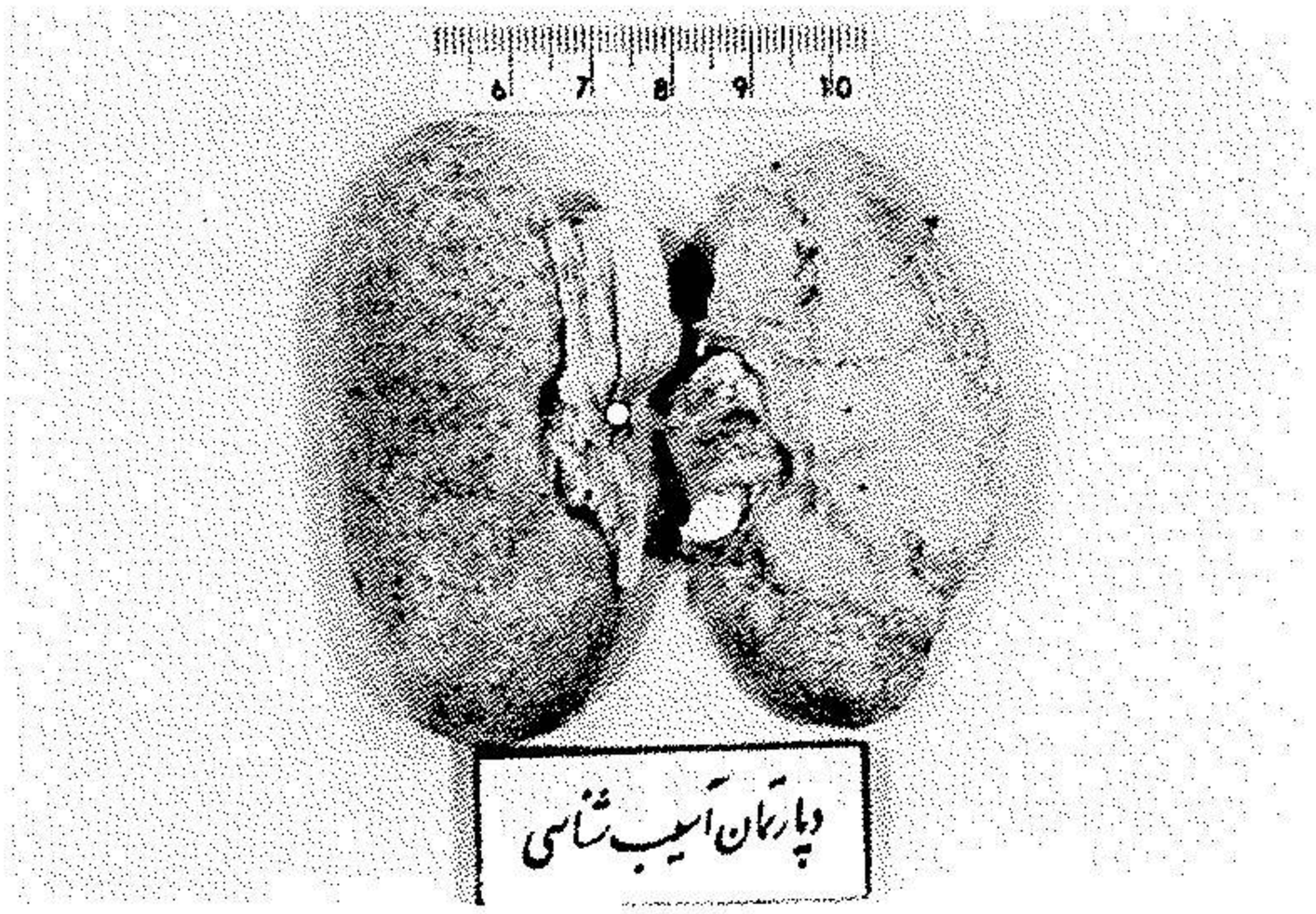
Table 6

**The incidence of hypertension in renal disease according to age group and sex**

**Malignant nephrosclerosis:**

Maliganat nephrosclerosis is a renal disease associated with malignant hypertension. This report report includes 8 cases of malignant nephrosclerosis, affecting younger individuals under the age 40 (40-24) years. It should be kept in mind (7) that in malignant hypertension, all vessels, particularly small renal arteries and arteriols, are involved.

The gross appearance (figs. 1, 2) of the kidney depends on the state of the kidney before the development of malignant hypertension. Grossly the kidney alteration may appear slight. Vascular congestion is usually present. In the majority of cases studied petechial hemorrhages were noted in the cortex as the result of rupture of arterioles or capillaries, and since the disease is rapidly fatal scarification of the kidney was absent. The most characteristic histopathological finding is the onionlike aspect of afferent arterioli and small artefies (figs. 3, 4 & 5), caused probably by proliferation of subendothelial fibroblastic cells narrowing the vascular lumen, and fatty or granular changes of the epithelial cells of the proximal convoluted tubules.




Chronic glomerulonephritis.

The Kidney is small, comtracted, and firm, The outer surface is roughly and irregularly contracted and pitted

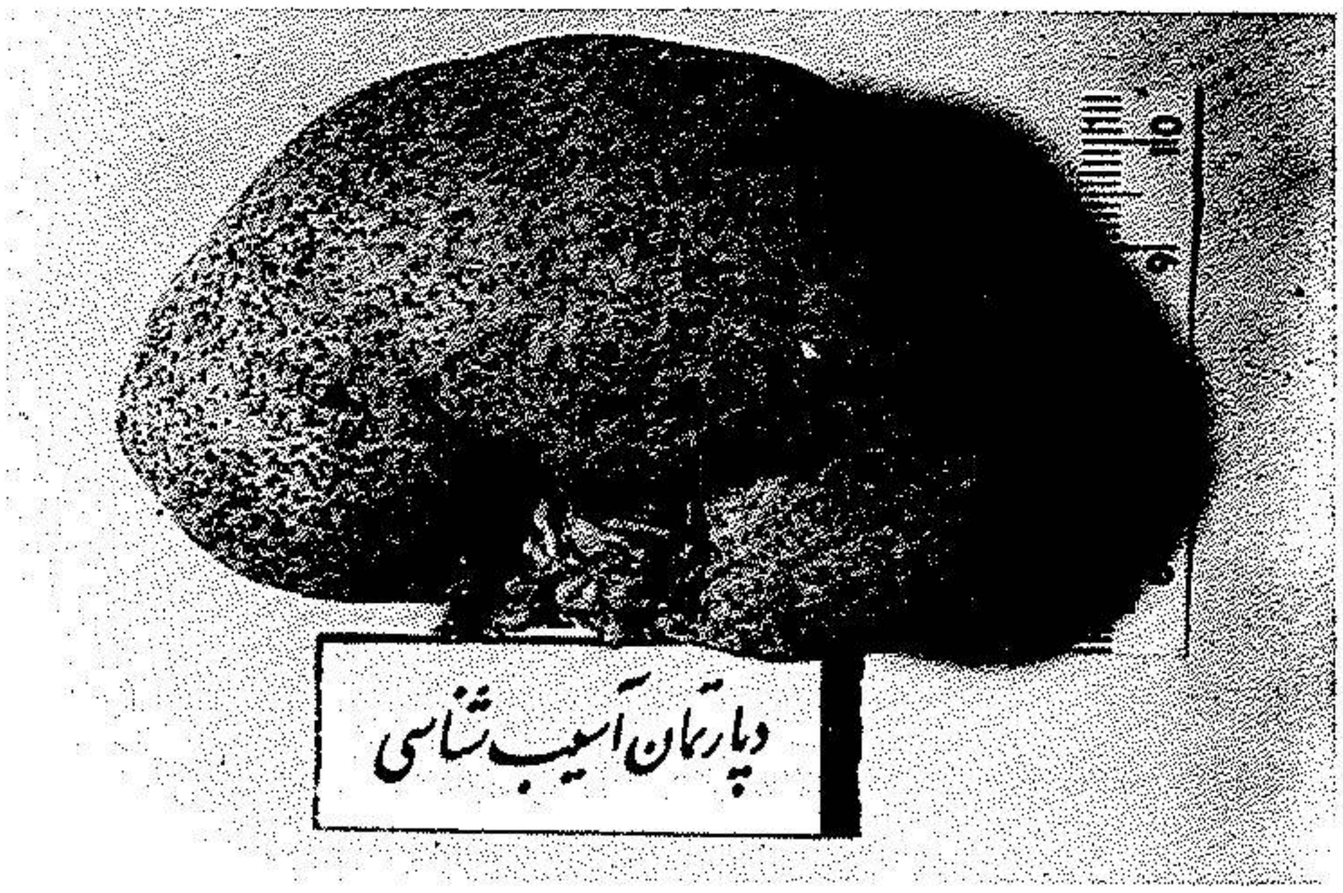



Fig. 1

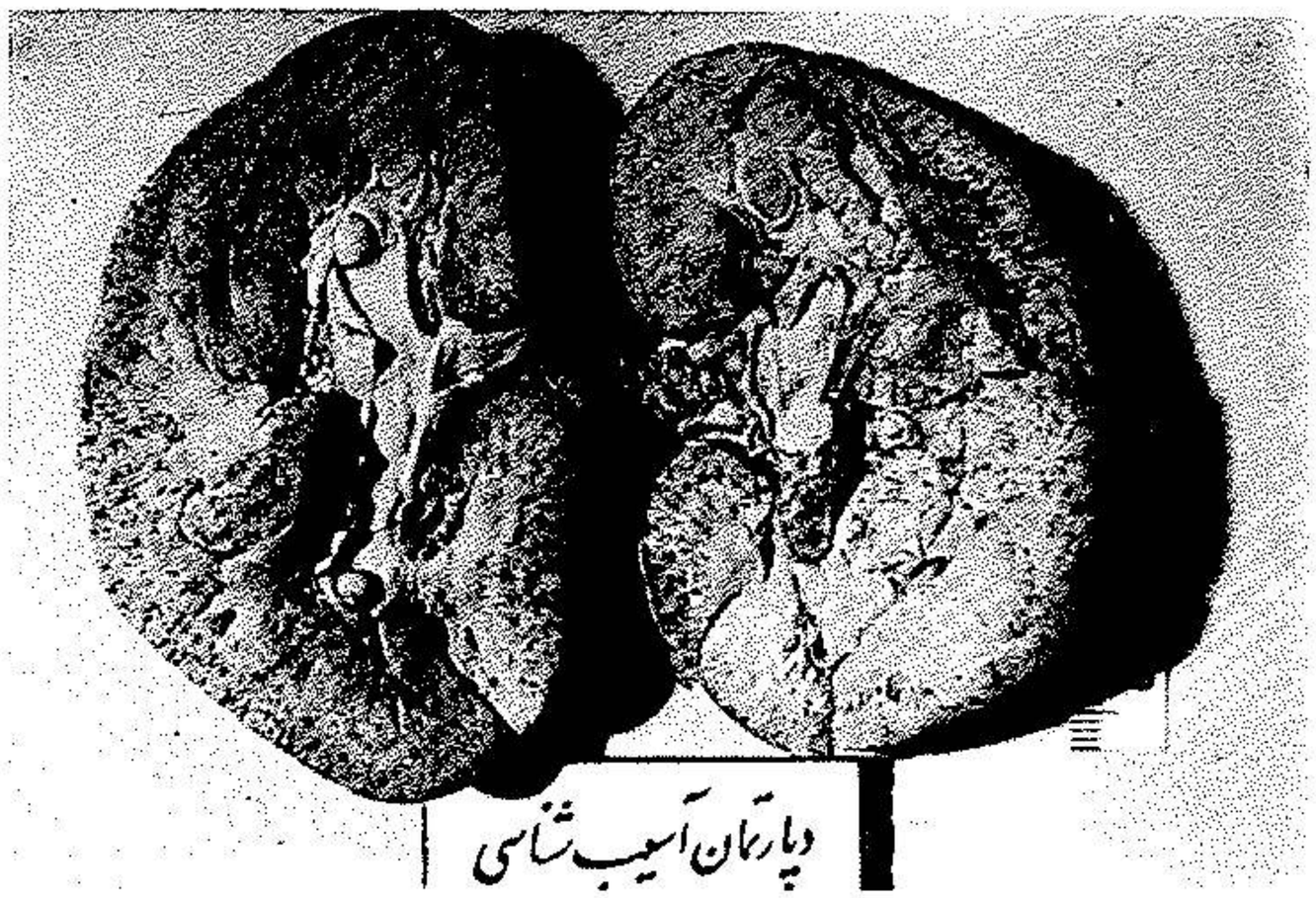



**Fig. 1,2.** Kidney of malignant hypertension, showing focal hemorrhages from arteriolonecrosis.
Autopsy 46/25 (A 30-year-old man, admitted for hypertension of 24/14, uremia, urea nitrogen 350 mg per 100 ml).

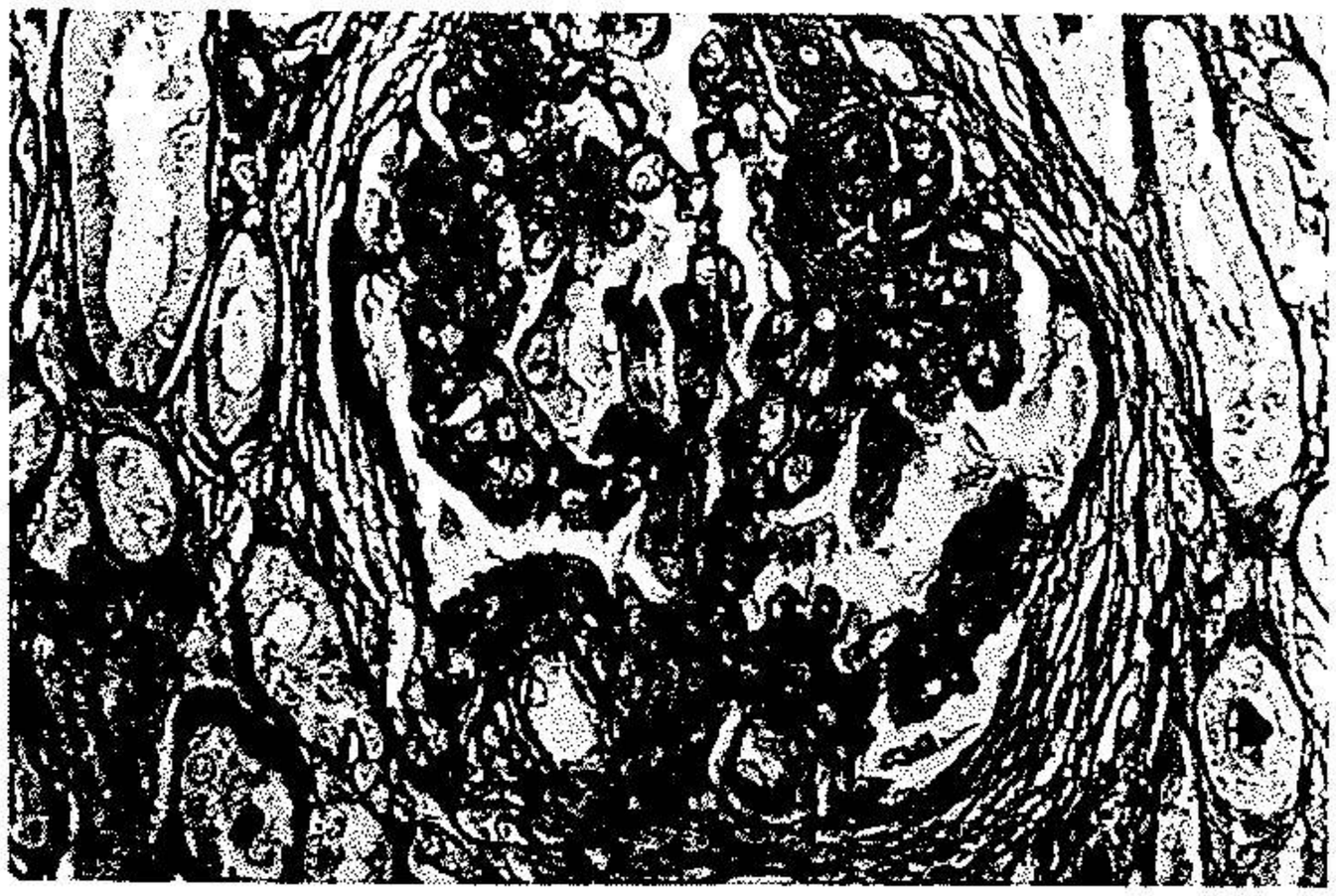


Fig. 3

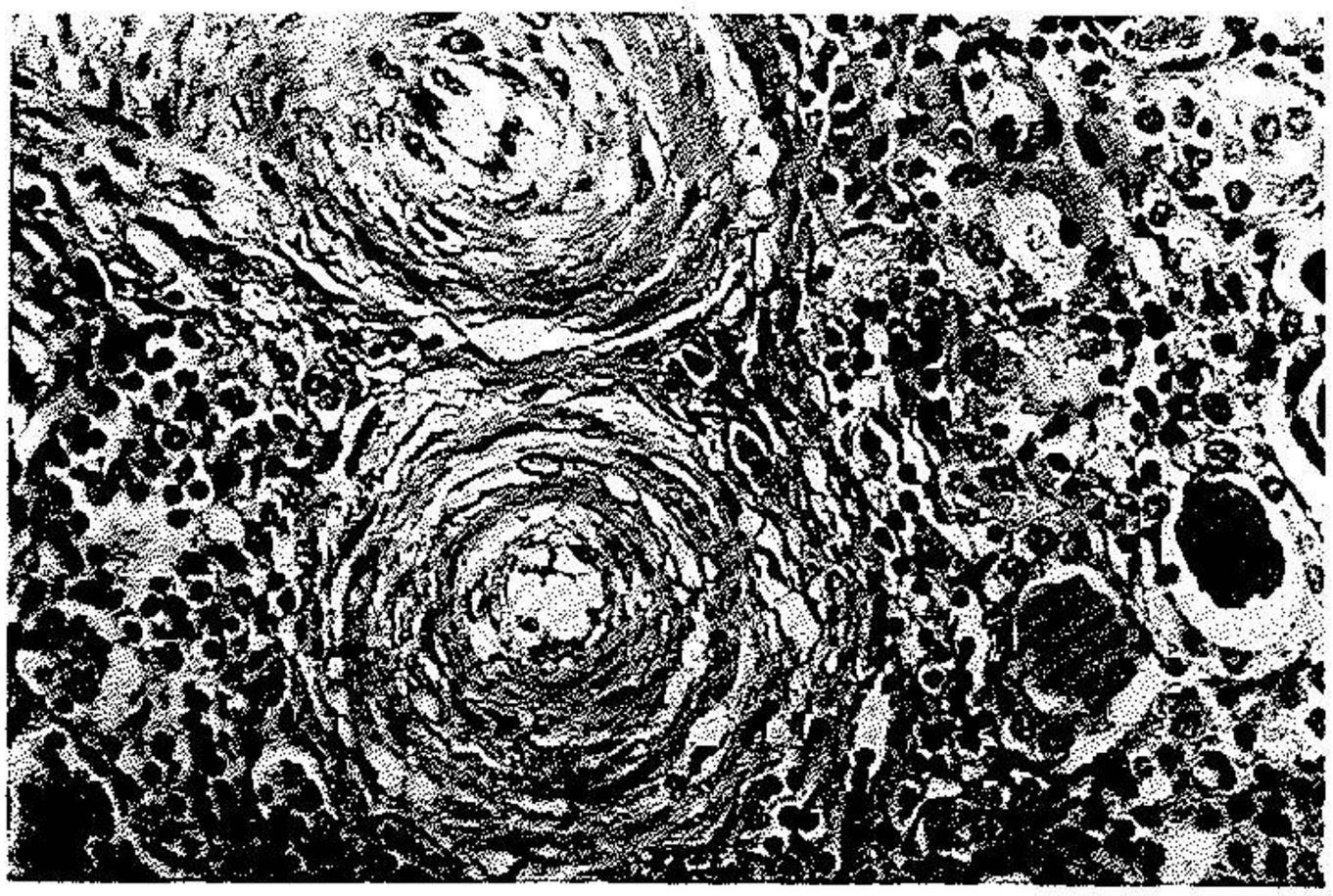


**Fig. 3,4,5.** The microscopic pattern of the kidnet from the same case, showing onion-like aspect of the afferent arterioli and small arteries.

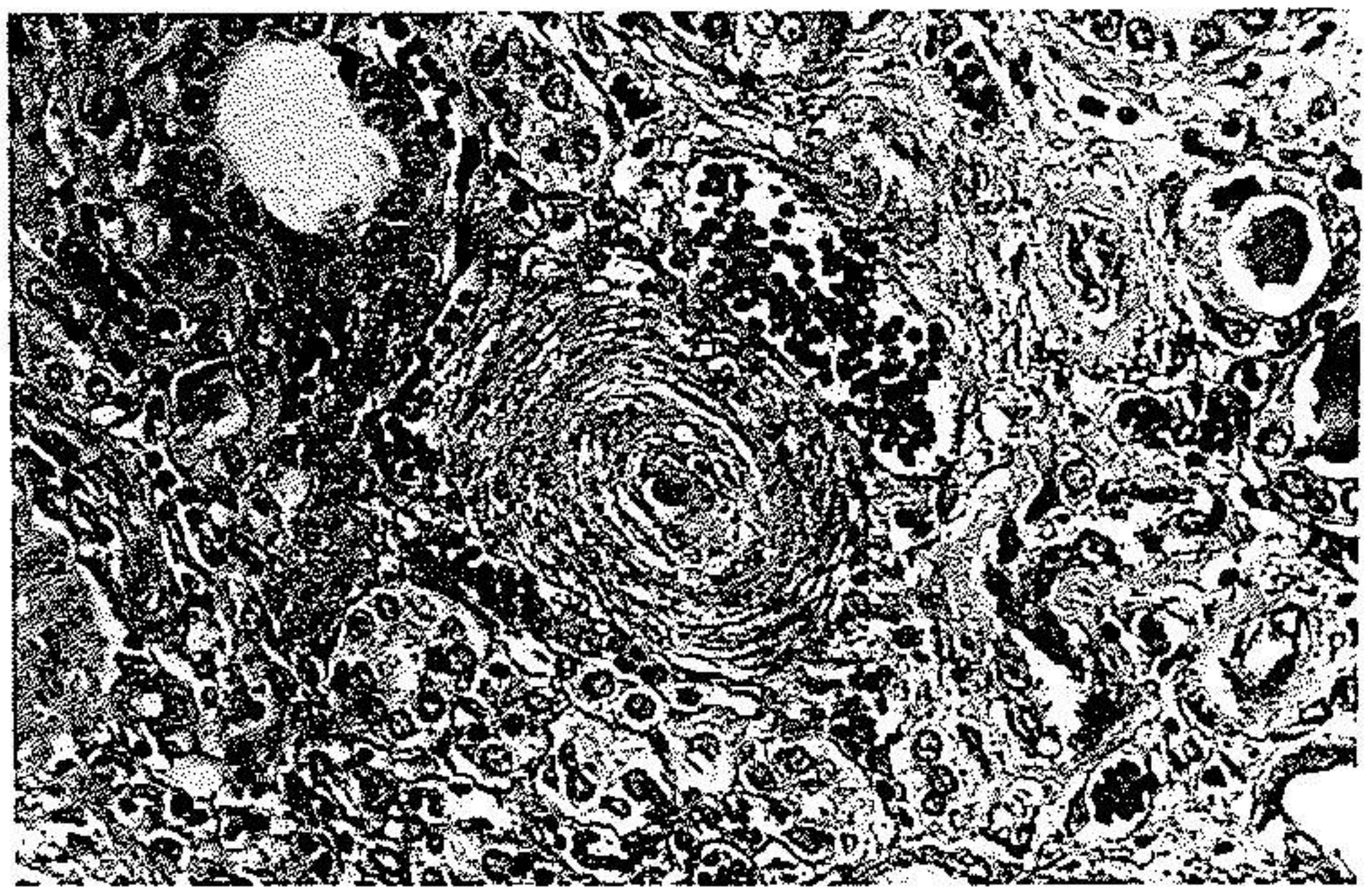


Fig. 5

**Diseases of the glomeruli:**

Glomerulonephritis: The term of glomerulonephritis has been used in this report for lesions involving primarily and diffusely the glomeruli. The etiology of glomerulonephritis is mostly unknown and obscure (17). It has been shown that some forms are due to an alteration in the immune response of susceptible individuals. In these individuals, following infection with certain bacteria, circulating antibodies are formed against the antigens released by the invading organism, thus giving rise to a hypersensitive state. Since in the majority of cases, acute glomerulonephritis occurs after streptococcal infections, the above-mentioned concept is generally accepted (16). In typical cases, the patient recovers from the causal infection in a short period of time, while there is still some reaction between his circulating antibodies and toxic products produced within his organism. This reaction finally leads to the renal lesion. The frequency and incidence of acute glomerulonephritis varies greatly. Among the factors causing this variability are:- 1) The different nephritogenic capacity of the various types of group a beta-hemolytic streptococci, 2) The type of population under study. 3) The possible influence of early antibiotic therapy. 4) The genetic predisposition for this form of disease. The onset and course of this renal disease seems to be different in children and adults (16, 26, 28).

**Proliferative Glomerulonephritis:**

Table 7 shows the incidence of various forms of glomerulonephritis according to age and sex.

Proliferative glomerulonephritis (17, 8, 26) usually occurs 2-3 weeks after a beta-hemolytic streptococcal upper respiratory infection, with or without treatment; it usually subsides promptly and the great majority of patients recovers completely, without any renal sequelae. A few of these patients later on develop chronic glomerulonephritis (41). The disease begins suddenly with fever, vomiting and asthenia. The urine is dark and blood-stained, containing a great amount of protein, red blood cells, epithelial cells, and numerous hyaline and granular casts. Visible edema is often initially limited to periorbital areas (17), and in some instances mild generalized edema develops later. Moreover, in the later stage, hypertension and urea retention may occur. After two to three weeks, the fever drops and the patient recovers, but the urine abnormality may continue for a long period of time. In some cases the disease lasts and becomes chronic (12). The prognostic evaluation is difficult, but it is generally accepted that 3 percent of affected children die and in 3 percent the renal lesion becomes chronic. Tables 8 and 9 show the incidence of acute and subacute glomerulonephritis in relation to preexisting illness in children and adults. The term of acute glomerulonephritis, idiopathic proteinuria (23, 49) and (14, 8) and nephrotic syndrome which are used so frequently, never correlate with the morphological aspect of renal lesion, and on the other hand the histopathological terms acute, subacute, focal and chronic glomerulonephritis, do not seem to be suitable diagnostic criteria, and can not explain all lesions correctly. Certainly the morphological diagnostic could never correlate to the nephrotic syndrome. In children the incidence decreases with increasing age (28, 53). The high incidence of the disease in pre-school children is possibly due to a lack of adequate defense mechanisms at this age against streptococcal infection (8). In the majority of our cases of glomerulonephritis in children previous streptococcal infection seems probable. As seen in tables 8 and 9 previous pharyngitis, cervical adenitis, otitis media, and

| Various forms of glomerulonephitis | Age group | | | | |
|---|---|---|---|---|---|
| | Over 14 years | | Under 14 years | | |
| | Female | Male | Female | Male | Total |
| Chronic glomerulonephritis | 2 | 3 | 8 | 45 | 58 |
| Acute glomerulonephritis | — | 2 | 3 | 6 | 11 |
| Subacte glomerulonephritis | 4 | 2 | 3 | 9 | 18 |
| Membranous glomerulonephritis | 2 | 3 | 1 | 2 | 11 |
| Rheumatoide purpure | — | — | — | 1 | 2 |
| Rupus erythematosus | — | — | — | 1 | 1 |
| Total | 8 | 10 | 15 | 68 | 101 |

Table 7

**The incidence of various from glomerulonephritis according to age and sex**

| Type of illness | Number |
|---|---|
| Suppurative acute in flammatory disease of the lung | 7 |
| Pharyngitis | 3 |
| Nephrotic syndrome | 1 |
| Kala-Azar | 1 |
| Acute leukemia | 1 |
| Total | 13 |

Table 8

**The incidence of acute and subacute glomerulonephritis According to preexisting illness in children**

| Type of Illness | Number |
|---|---|
| Unknown | 11 |
| Nephrotic syndrome | 7 |
| Suppurative acute inflammatory illness of the lung | 5 |
| Pharyngitis | 3 |
| Pheumatoid purrura | 2 |
| Acute leukemia | 1 |
| Lupus erythematosus | 1 |
| Total | 30 |

Table 9 **The incidence of acute and subacute glomerulonephritis according to preexisting illness in adults**

pulmonary infection were common. Our main problem in this report was the difficulty in estimating the latent period which follows the preceding infection and the time of clinical mainfestation of nephritis.

**Autopsy findings:**

In the majority of cases the anatomic diagnosis was acute glomerulonephritis plus other findings such as unresolved pneumonia, fibrous pericarditis and fibrous pleuritis. The kidneys were moderately enlarged weighing up to 180-200g each. In such cases the capsule is tense (47, 32, 52) stripped with ease, leaving a smooth, brownish-gray surface, stippled with punctuate hemorrhages. The kidneys are soft, and on section both cortex and pyramids

are swollen. In light microscopy, there are practically no glomeruli of normal appearance (figs. 6 & 7). The tufts are shrunken and bloodless, and there is adhesion between loops and capsule. In some cases the Bowmann spaces contain red cells and amorphous material (fig. 8), a finding which has been emphasised by others (5, 52, 47, 32, 55). In some cases polymorphonuclear leukocytes were seen in capsular spaces (fig 9). In adults, epithelial crescents are numerous, (figs 10 & 11) which is an outstanding feature of the glomerular lesion. Among the epithelial cells of crescents there are leukocytes and masses of fibrin (fig 12). In some cases (fig 13) the glomerular capillaries show necrosis and throbi, and the interstitial tissue is infiltrated by leukocytes and the proximal convoluted tubules contain red cells and casts. The collecting tubules and large blood vessels mostly appear normal. Nine cases of this group show a nephrotic syndrome (characteristic features of which are proteinuria exceeding 4g hypoprteinemia, edema and hyperlipemia). In these cases (fig 14) endo-epithelial proliferation and basement membrane thickening were present.

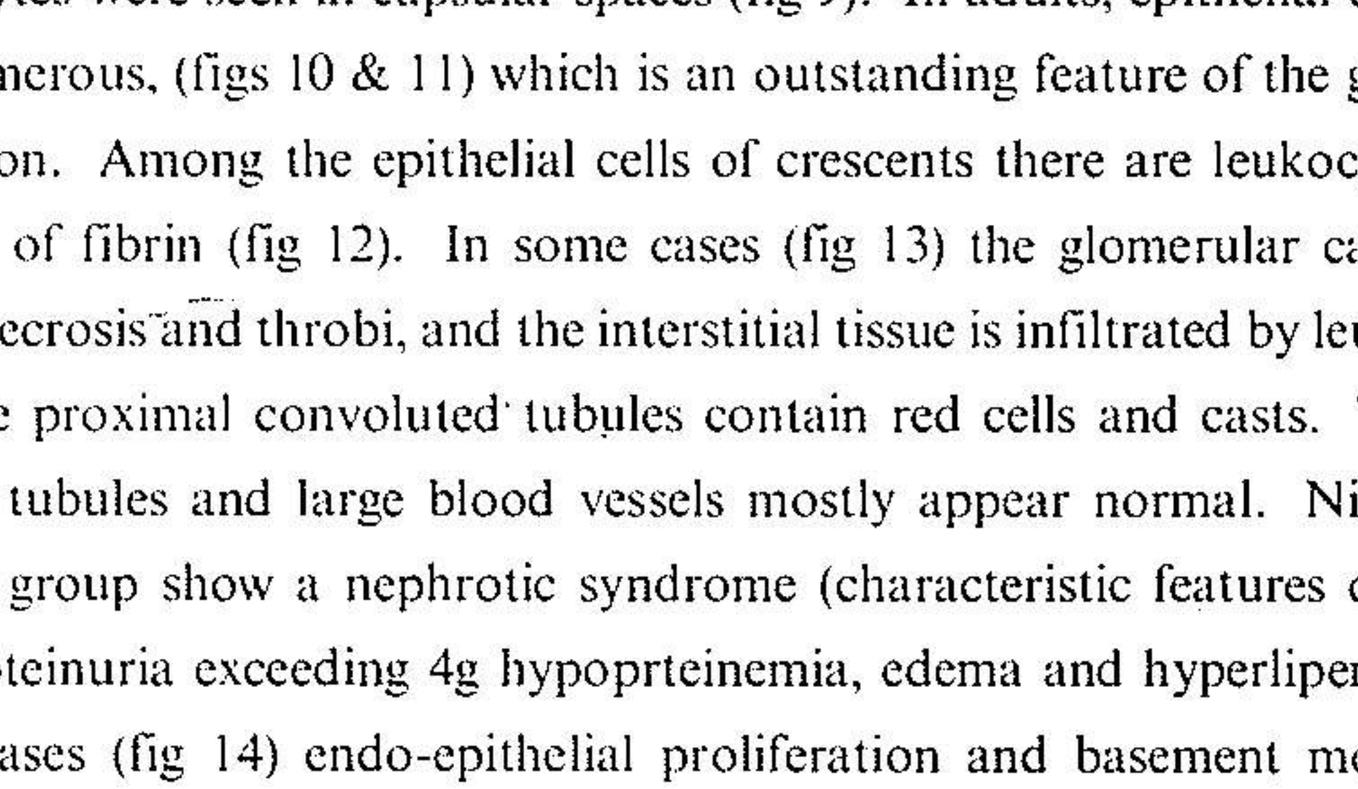


**Fig. 6** Autopsy 1/42 (A 60-year-old man admitted for previous pneumonia and acute exudative glomerulonephritis).
Exudative proliferative glomerulonephritis. No glomeruli of normal appearance are present (H and E).

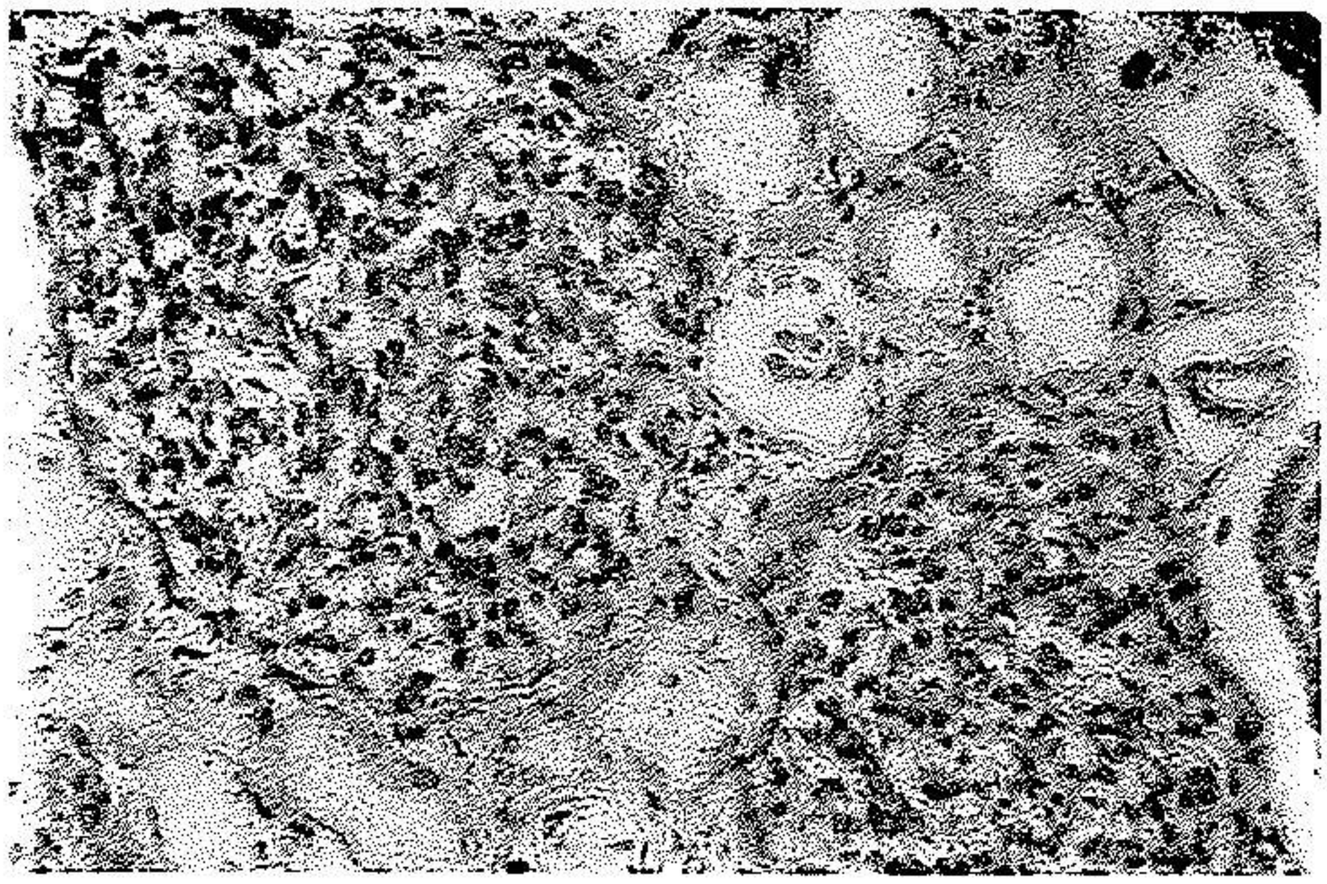


**Fig. 7** From the same case.
The tufts are shrunken and bloddless, and there is adhesion between the loops and capsule (anilin blue stain).

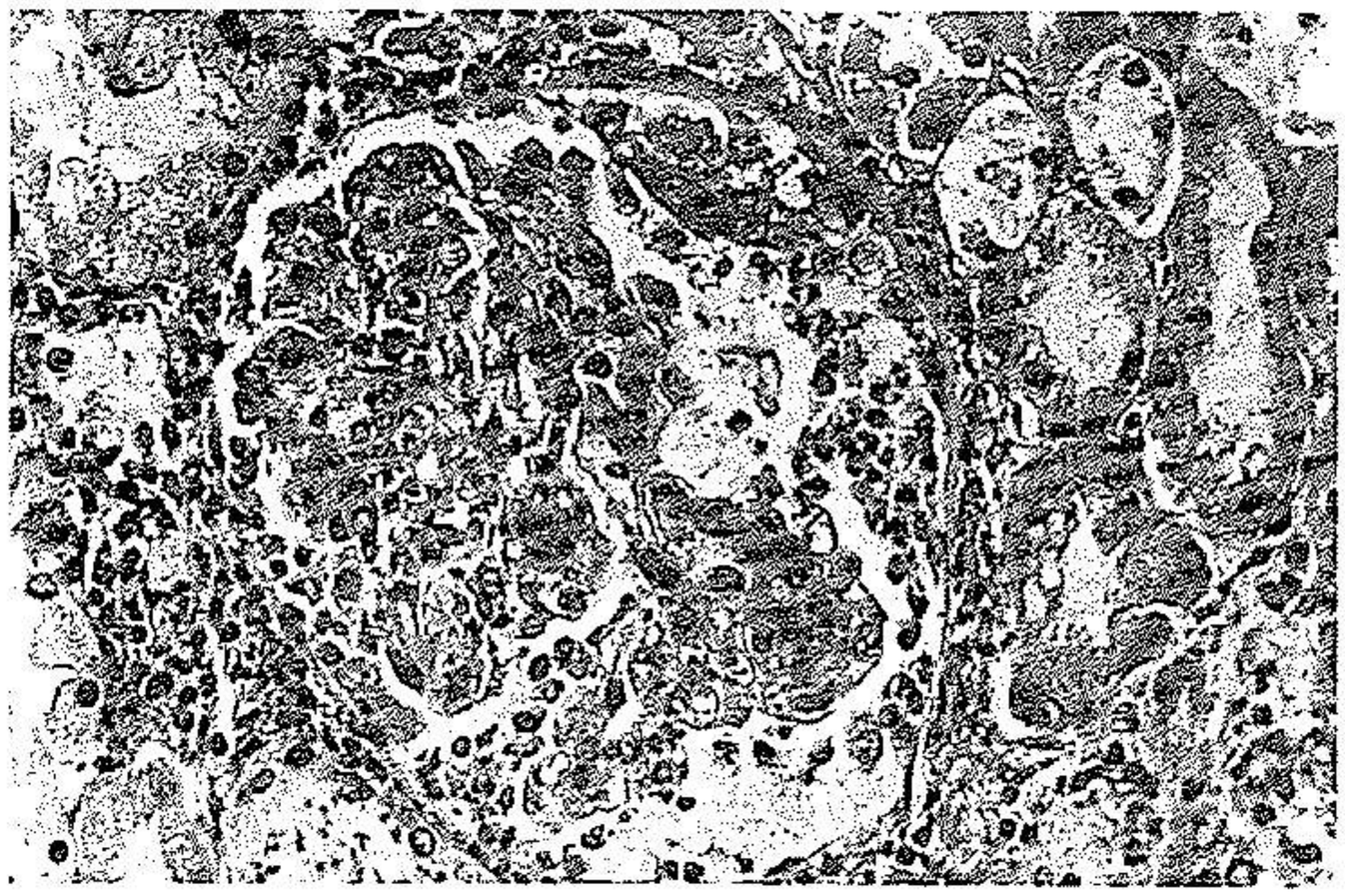


**Fig. 8** Autopsy 45/432 (A 40-year-old man, admitted for hypertension, generalized edema, ascites, and hepato-splenomegalia).
Proliferative glomerulonephritis.
The Bowmann spaces contain red cells and amorphous material.

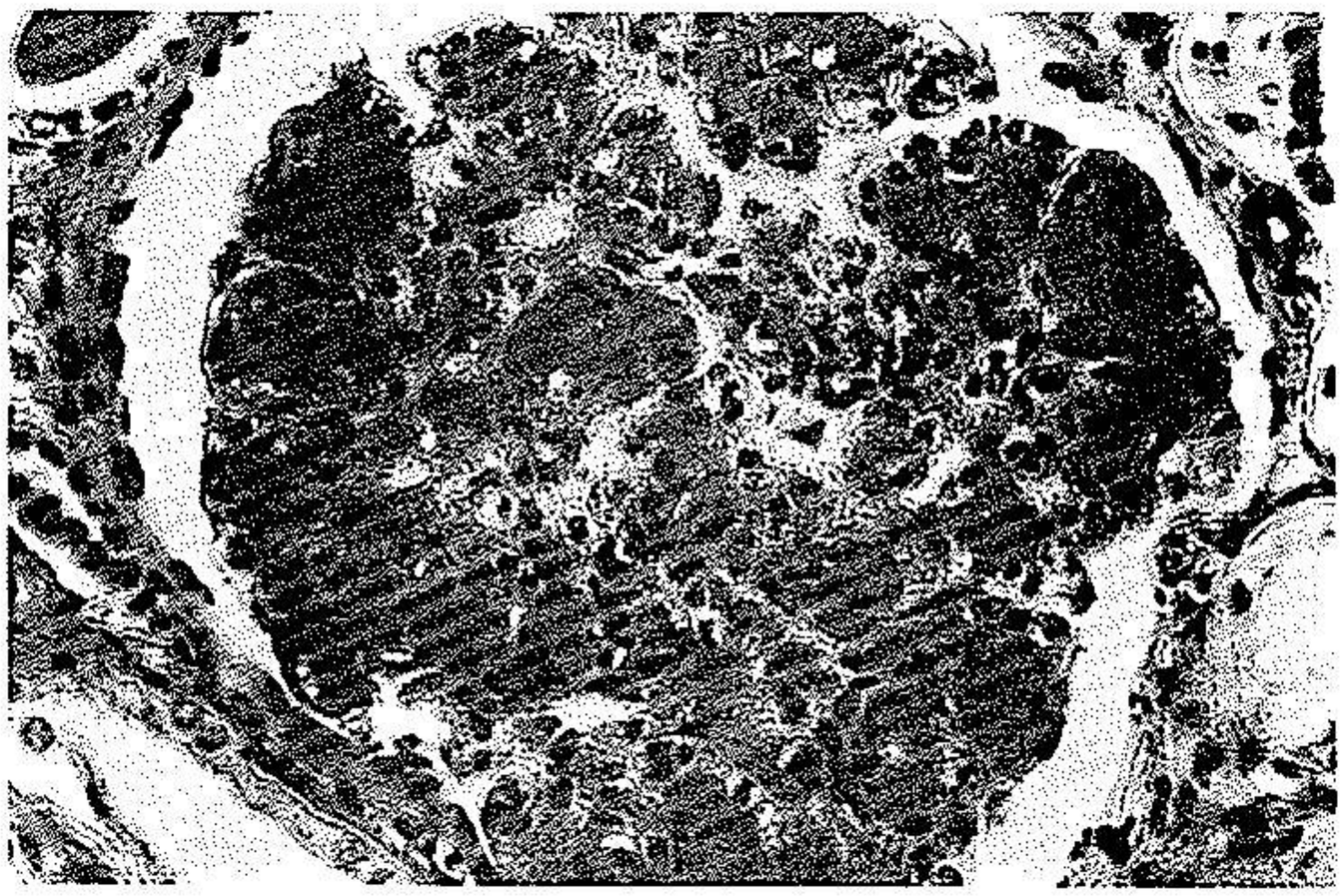


**Fig. 9** Autopsy 45/384
Proliferative glomerulonephritis. (The glomeruli contain polymorphonuclear lecukocytes.)

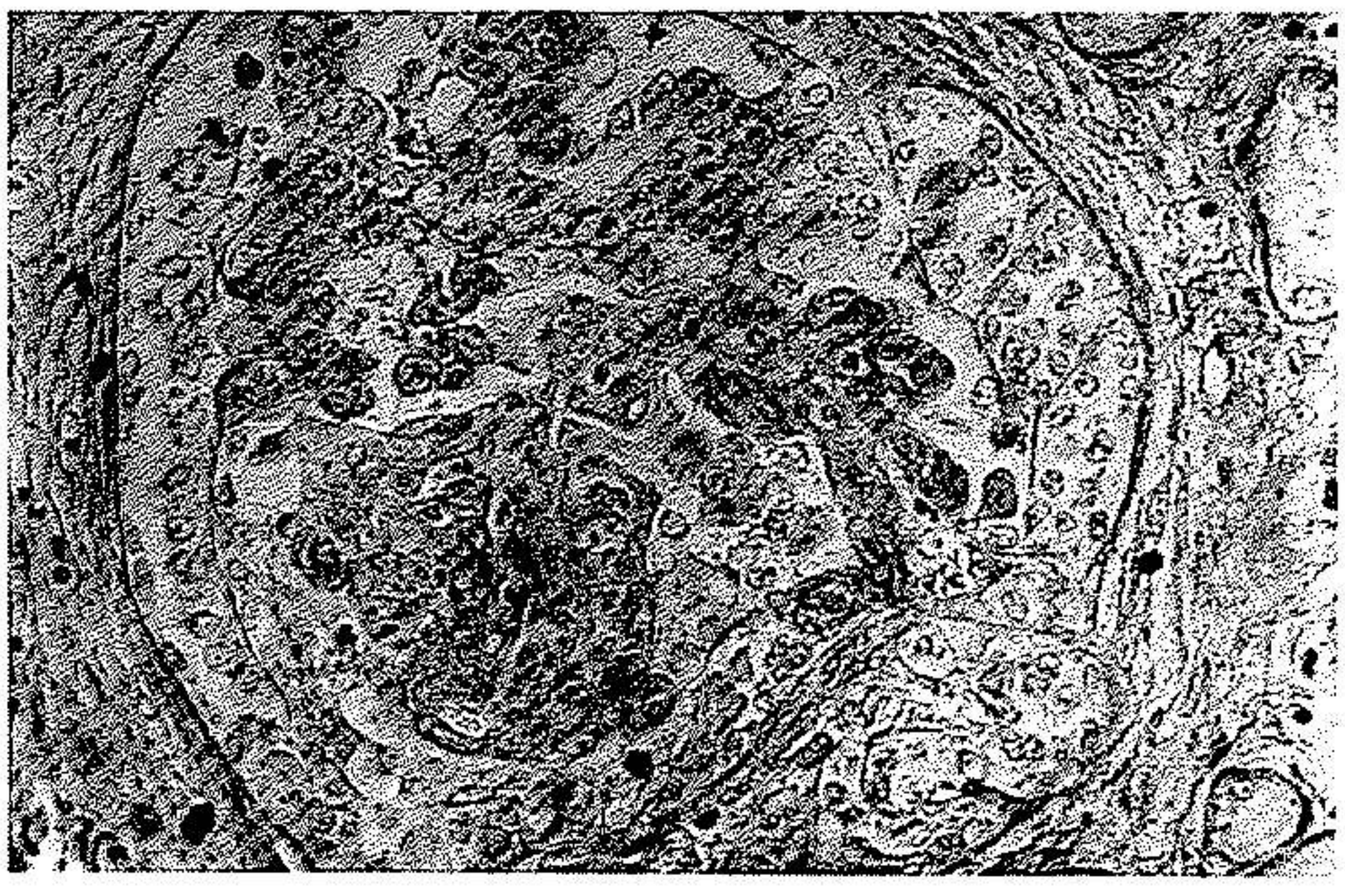


**Fig. 10**

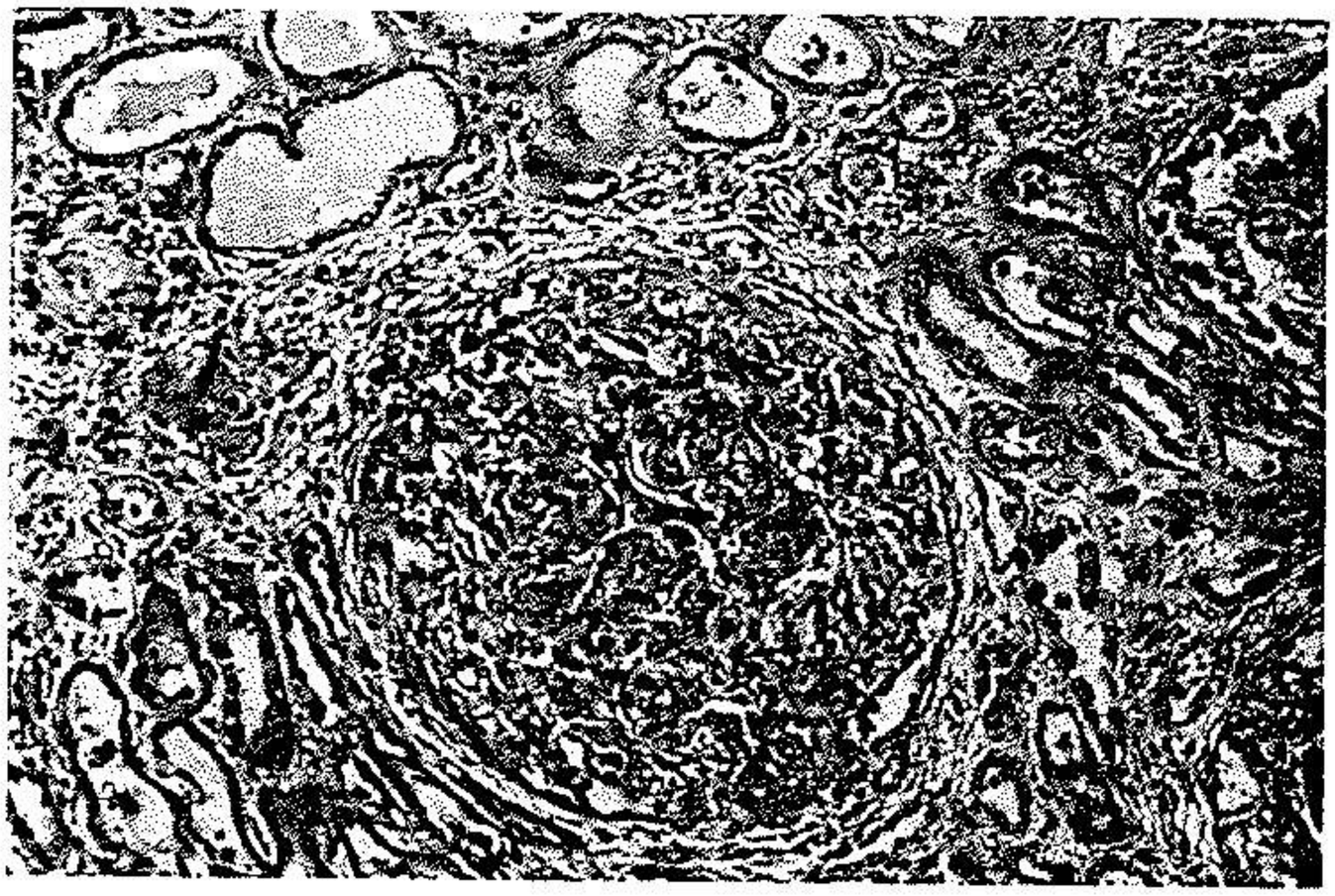


**Fig. 10,11** Autopsy 46/563 (A 40-year-old woman, admitted for sudden generalized edema, hematuria, and polyyuria).
Subacute glomerulonephritis. Very marked proliferation and crescent formation.

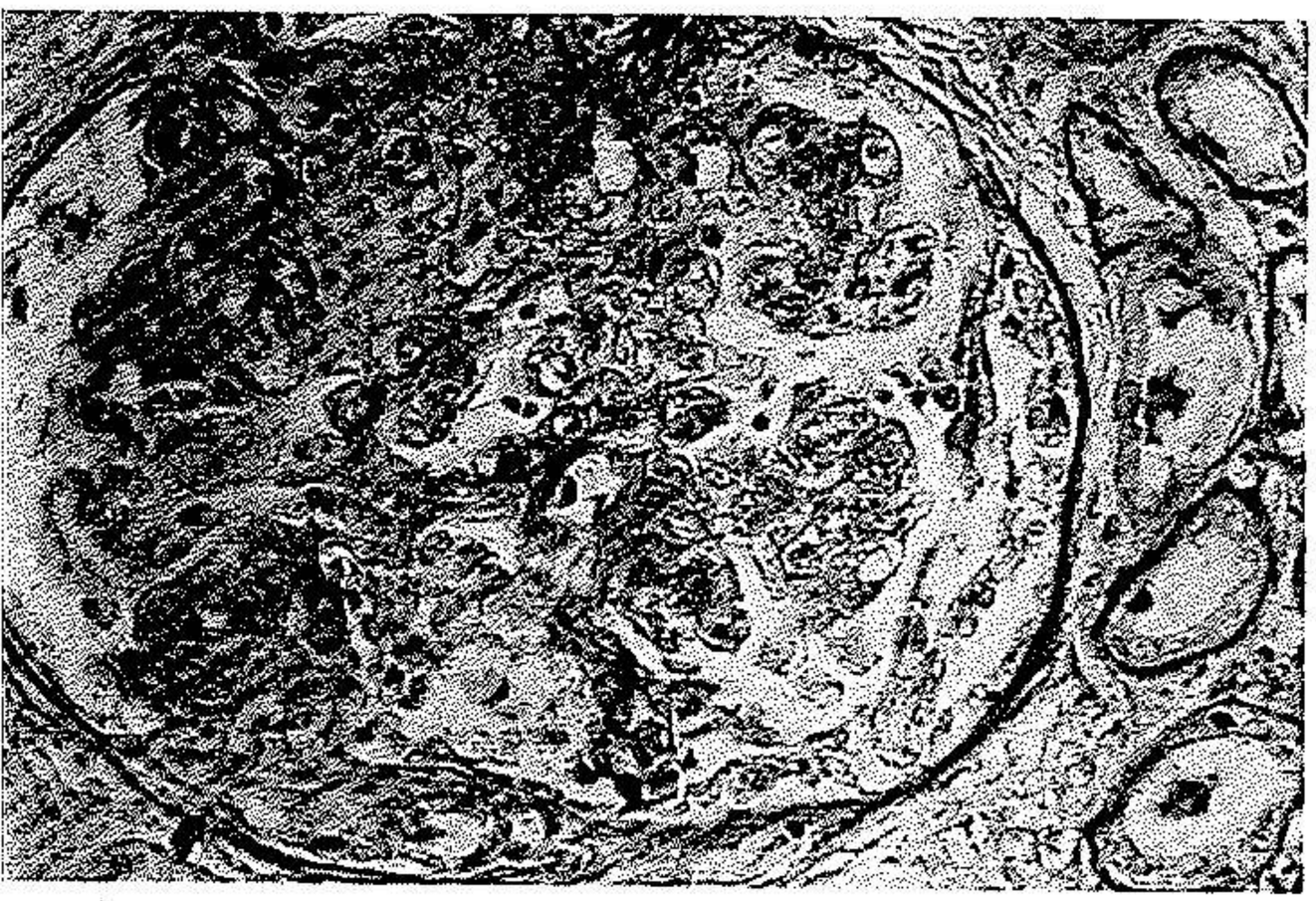


**Fig. 12**

Autopsy 46/563
Proliferative glomerulonephritis
(Among the epithelial cells there are masses of fibrine).

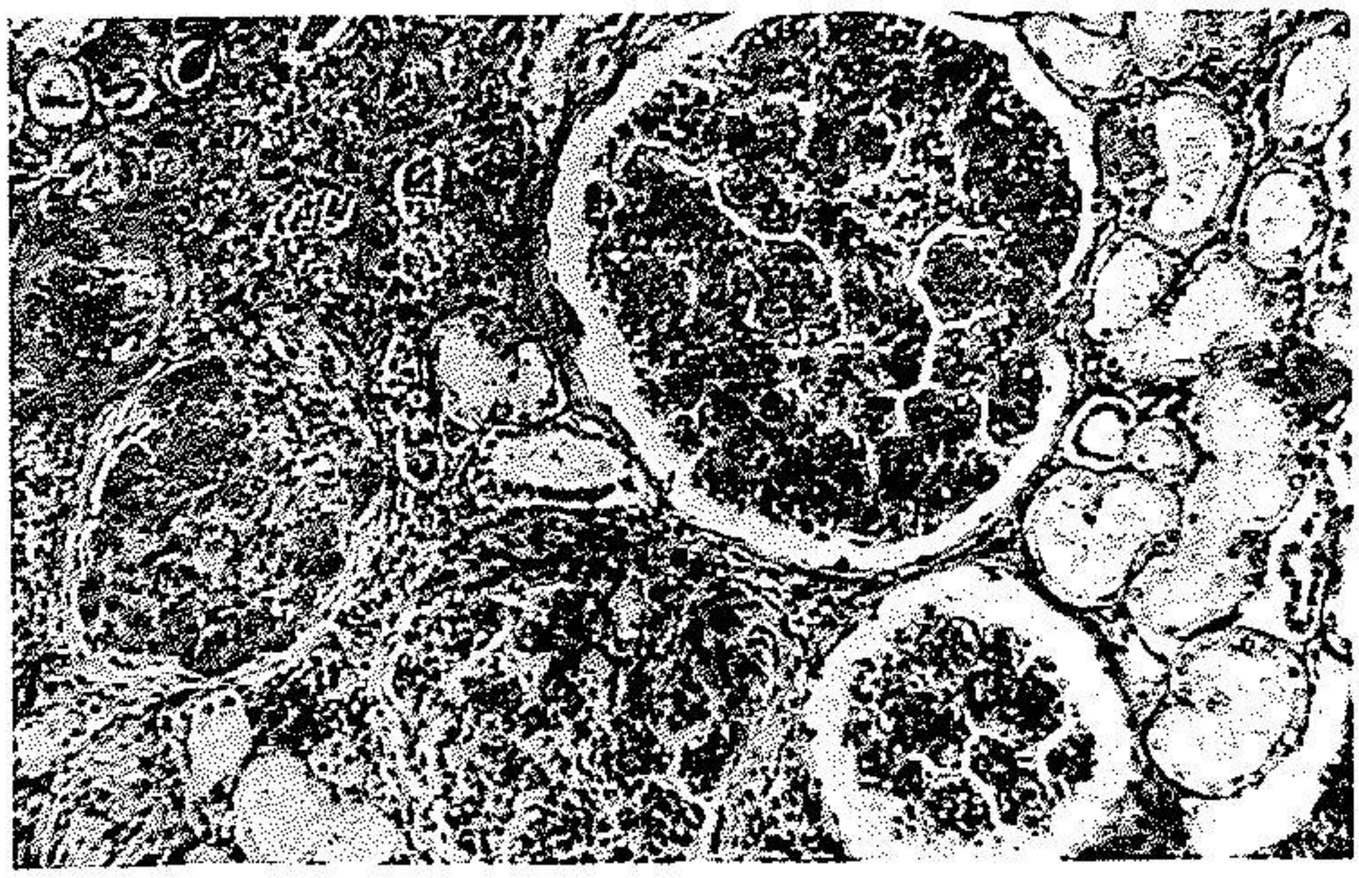


**Fig. 13** Autopsy 45/432 (A 40-year-old man, admitted for V. endocarditis) Proliferative glomerulonephritis. The glomerular capillaries show necrosis and thrombosis.

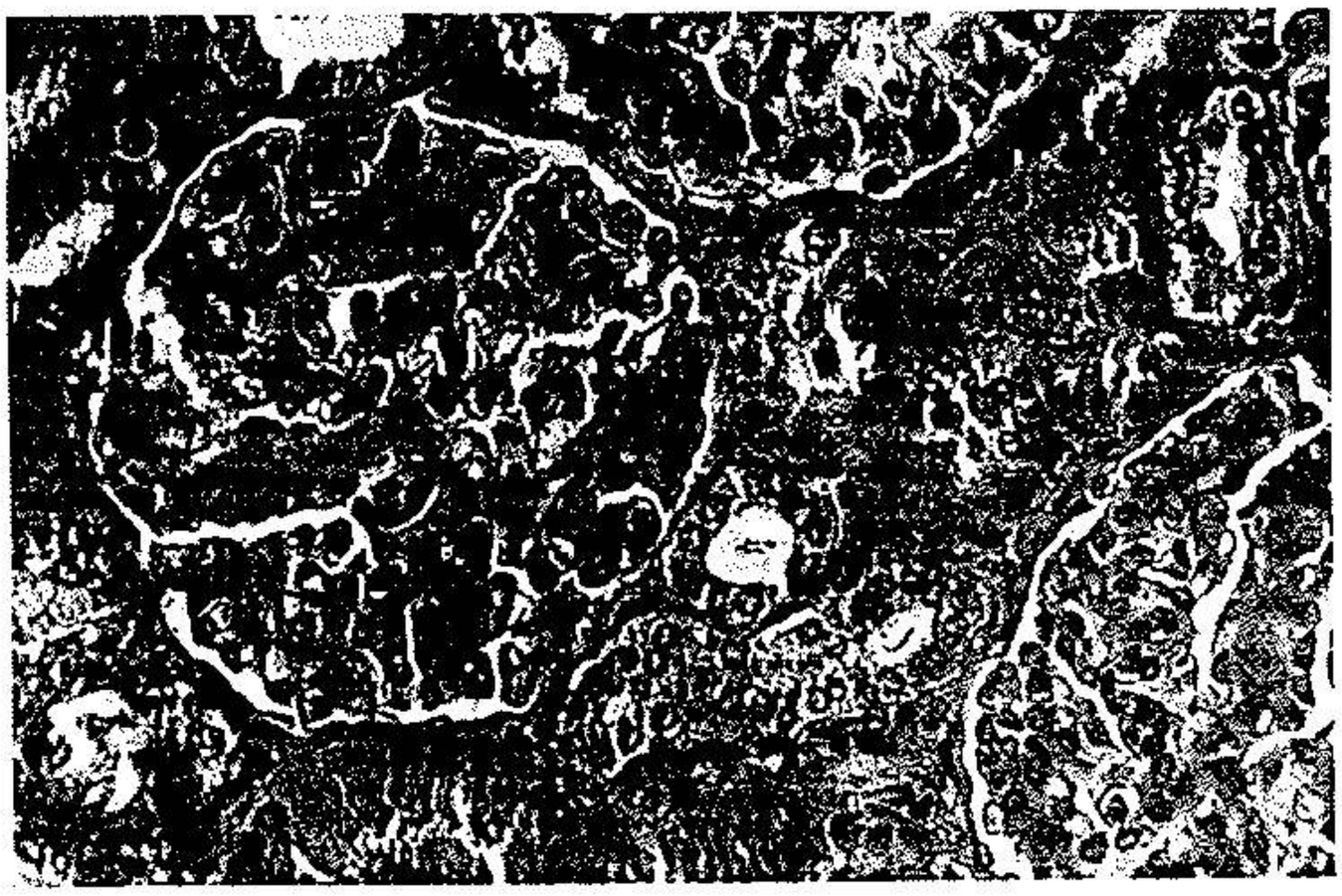


**Fig. 14** Autopsy 43/31 (A 18-year-old boy admitted for rheumatic pancarditis). Proliferative glomerulonephritis. (Endo-epithelial proliferation and basement membrane thickening is present).

**Membranous Glomerulonephritis:**

This type of glomerulonephritis is observed at any age, and it is a common cause of the nephrotic syndrome. Table 10 shows the incidence of various causes of the nephrotic syndrome. Membranous glomerulonephritis is a chronic recurring disease (29). With progressive swelling of epithelial and endothelial cells, and increasing density and irregularity of the basal membrane, ischemia of the glomeruli results, the glomeruli finally appearing similar to the advanced stage of proliferative glomerulonephritis. There is, however, no crescent formation in the membranous type (fig 15).

| Kind of disease | Age group | | | | Total |
|---|---|---|---|---|---|
| | Over 14 years | | Under 14 yeras | | |
| | Male | Female | Male | Female | |
| Tuberculosis | — | 2 | 6 | 3 | 11 |
| Acute and subacute glomerulonephritis | — | 2 | 6 | 1 | 9 |
| Non-T. B. amyloidosis | — | — | 2 | 4 | 6 |
| Chronic glomerulonephritis | — | 1 | 4 | — | 5 |
| Chronic pyelonephritis | — | — | 2 | 1 | 3 |
| Acute pyelonephritis | — | — | 1 | — | 1 |
| Total | — | 5 | 21 | 9 | 35 |

Table 10

**The incidence of nephrotic syndrome caused by various renal diseases**

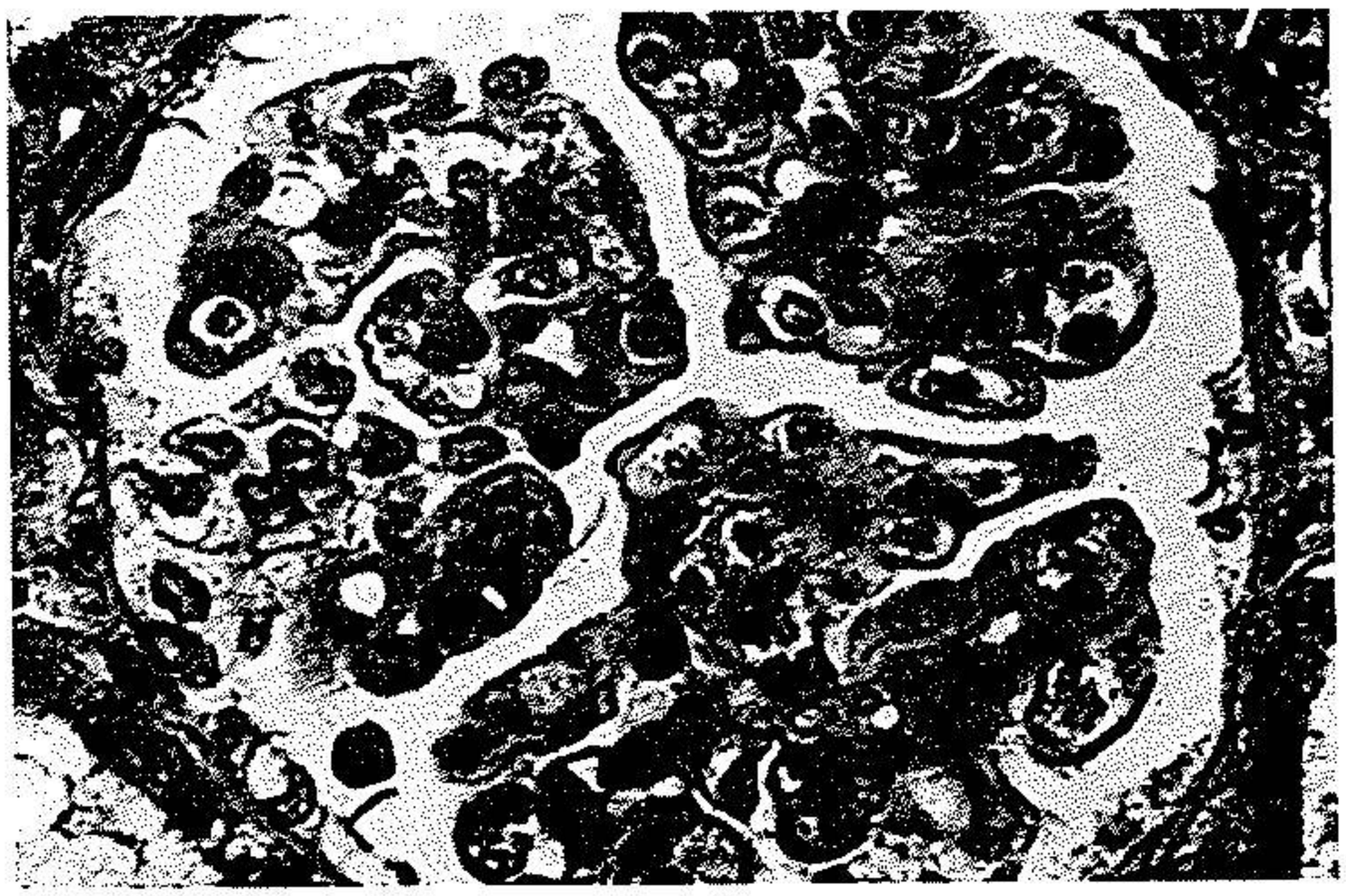


**Fig. 15** Autopsy 45/466 (A 13-year-old boy; admitted for acute leukemia). Membranous glomerulonephritis. Swelling of the epithelial and endo-thelial cells; thickening of the basement membrane is present.

**Chronic Glomerulonephritis:**

Chronic glomerulonephritis (21) should be separated from other types of glomerulonephritis. Although many early investigators believed that most if not all cases of chronic glomerulonephritis developed from acute forms, we feel that the question of how many cases of acute glomerulonephritis end up as chronic glomerulonephritis, has remained unanswered. In our material (861 cases of different renal diseases) there were 58 cases of chronic glomerulonephritis (adults and children). The onset in 10 cases (21 %) was after the age of 50. The incidence before the age of 30 years was 50 percent (29 cases) and from age 30-50, 19 cases (32 %). The gross appearance of the kidney in chronic glomerulonephritis depends on the stage of the disease. In the early stages, the kidney appears granular (weighing about 110g), and in advanced stages, the kidneys are small and granular,

weighing 80-90g (figs 16, 17). Among 58 cases of chronic glomerulonephritis found at necropsy, 14 % had edema, 32 % had hypertension.

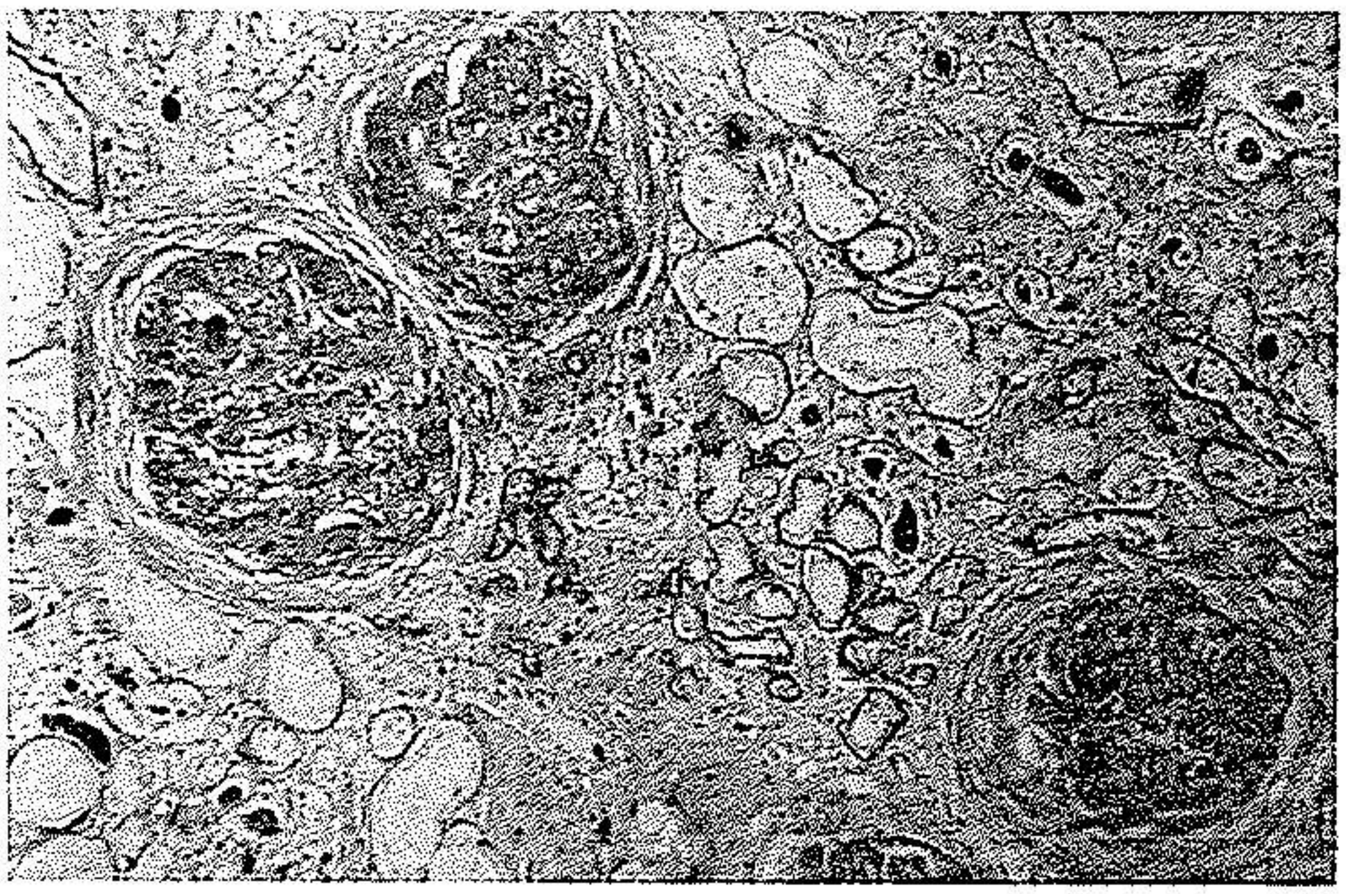


**Fig. 16** Autopsy 45/382 (A 20-year-old boy, admitted for glottic edema and uremia, two months after scarlatina).
A large proportion of glomeruli are partially changed into hyaline masses.

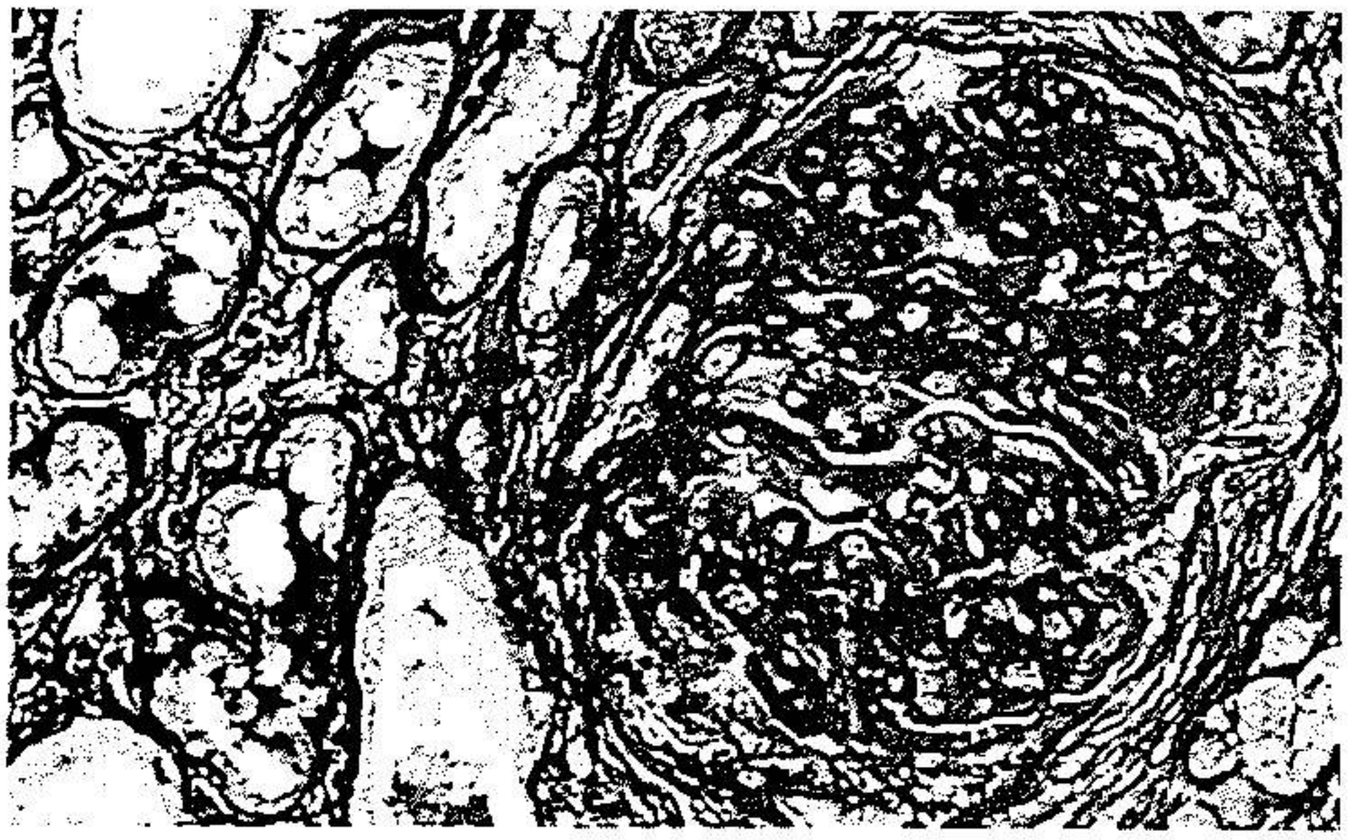


**Fig. 17** Autopsy 43/477 (A 30-year-old man, admitted for generalized edema). Subacute glomerulonephritis about to change into chronicity). Note the glomerular fibrosis, tubular dilatation and arterial thickening.

**Nodular Glomerulosclerosis:**

This disease, also known as diabetic glomerulosclerosis, is found in approximately 50 % of autopsied diabetic patients, and it is generally believed that the histological feature is specific for the disease (22, 41). The characteristic altheration is the deposition of spherical hyaline masses within the peripheral capillary tufts of the glimeruli. Since the hyaline masses appear to be located between the capillaries (11, 9, 24, 30, 50), the term of intercapillary glomerulosclerosis has been adopted (fig 18).

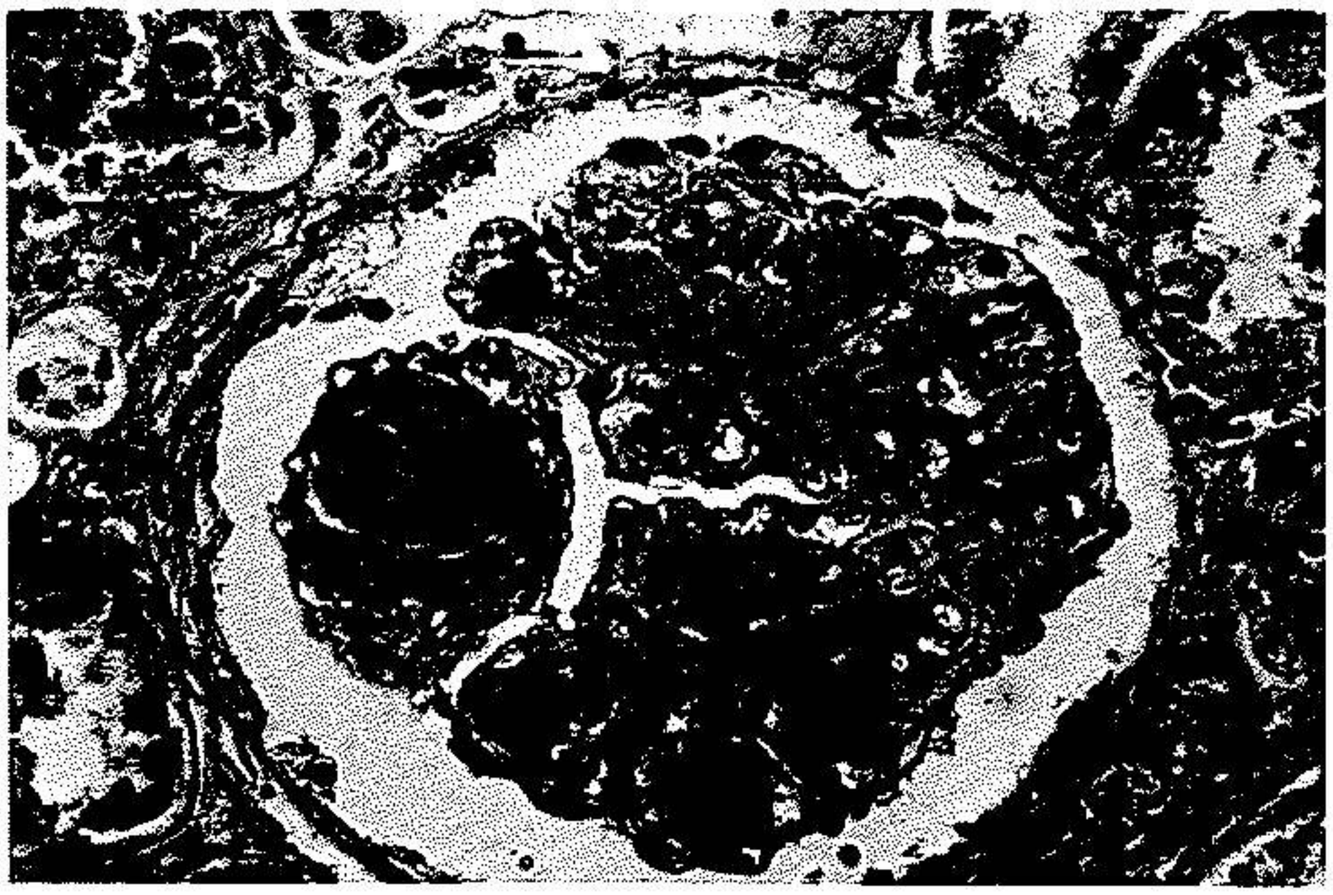


**Fig. 18** Autopsy 44/258 (A 65-year-old woman diabetic during a 15 year period). Deposition of spherical hyaline masses within the peripheral capillary tufts.

**Amyloidosis of the kidney:**

The kidney was severely affected in systemic amyloidosis in 64 cases. Table 11 shows the incidence of various causes of amyloidosis. The deposits occur in the glomeruli, blood vessels and interstitial tissue as well as within

the tubular membrane (41). As summarized in table 11, the most common causes of amyloidosis are tuberculosis of the lung, nontuberculous chronic lung disease, lung cancer, osteomyelitis multiple myeloma, ulcerative colitis, amebiasis, leprosy, and syphilis. Our findings indicate that in addition to tuberculosis, less common disease such as amebiasis, ulcerative colitis (15) and leprosy should be considered in cases of amyloidosis.

| Age group | | cause | | | | | | | | | Total |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | tuberculosis | non-T. B. disease of the lung | lung cancer | osteomyelitis | myeloma | ulcerative colitis | amebiasis | leprosy | syphilis | |
| Over 14 years | Male | 19 | 14 | 1 | 2 | 2 | 1 | 1 | 1 | 1 | 24 |
| | Female | 13 | 3 | — | — | — | — | — | — | — | 17 |
| Under 14 years | Male | 2 | 1 | — | — | — | — | — | — | | 3 |
| | Female | 2 | — | — | — | — | — | — | — | — | 2 |
| Total | | 26 | 18 | 1 | 2 | 2 | 1 | 1 | 1 | 1 | 64 |

Table 11

**The incidence of amyloidosis among 4463 autopsies according to age group, sex, cause**

**Miscellaneous Glomerular Lesions:**

Glomerular lesions are encountered in a number of systemic diseases (51, 34, 31, 1) such as rheumatoid arthritis (two cases) and disseminated lupus erythematosus (one case). In some of these cases death may be due to renal involvement. In disseminated lupus erythematosus, the glomeruli show the following features:

1. Endothelial proliferation and basement membrane thickening.
2. Deposition of fibrinoid material between basement membrane and vascular endothelial cells (10, 13, 42, 49).

**Eclampsia:**

Glomerular lesions of significant importance were encountered in our study in 8 cases of toxemia of pregnancy. The kidney in eclampsia may be normal or may exhibit some alteration. It should be emphasized that kidney in gravidic toxemia has characteristic pathological changes which are used as criteria for the classification of gravidic nephropathy (35, 36, 37, 38). Proteinuria, hypertension, and edema alone or together are seen in these patients. The observed pathological findings are:

1. Proliferation of glomerular epithelial cells and crescent formation in the space, resulting in hypertrophy of the glomeruli.
2. Focal thickening of vacular walls and also thickening of intercapillary zones.
3. Fibrinoid necrosis. The glomeruli become bloodless, and fibrinoid necrosis of the glomerular tuft seem to be striking.
4. Acute necrosis of the arterioli, degenerative lesions of the tubal epithelium, and occasionally diffuse congestion and interstitial edema.

**The Incidence of renal lesions in rheumatic carditis:**

Glomerulonephritis of rheumatic carditis (45, 1, 19) does not seem to be a rare complication, its incidence is reported as 39 % in some statistics, and 26 % in our statistics (3). Sometimes the existing nephritis seems to be

advanced and the kidney is scarred but the most common is proliferative glomerulonephritis, which we have found in 32 of our total of 48 cases (fig 19). It should be pointed out that in 36 % of patients presenting glomerulonephritis at necropsy, active lesions (Ashoff Bodies) were present in the heart. While in 29 % of patients without renal disease, the active lesion was present.

**Disease of the tubules:**

Nephrosis is the morphologic term used to refer to all types of renal disease in which tubules are initially affected. The cases of nephrosis in this report consist of 3 cases of toxic nephrosis (two cases of mercurial intoxication (44) and one case of intoxication by ingestion of depilatory paste), 27 cases pf cholemic nephrosis and 5 cases of myelomatous nephrosis. In the case of toxic nephrosis the renal tubular epithelium (proximal convoluted tubules) shows vacuolar degeneration. (fig 20).

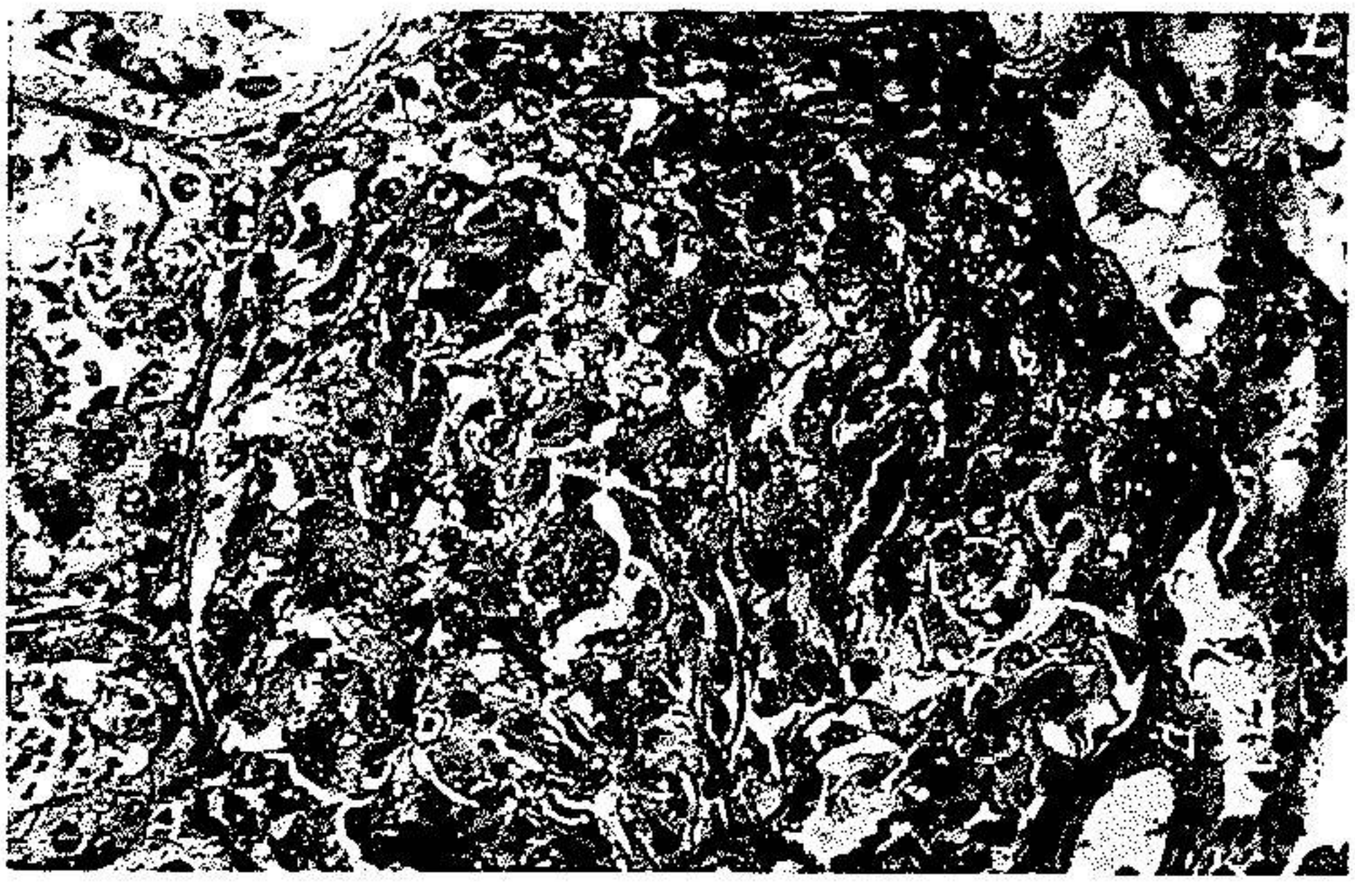


**Fig. 19**

Autopsy 45/219 (A 20-year-old man, admitted for rheumatic pancarditis). Proliferative glomerulonephritis.

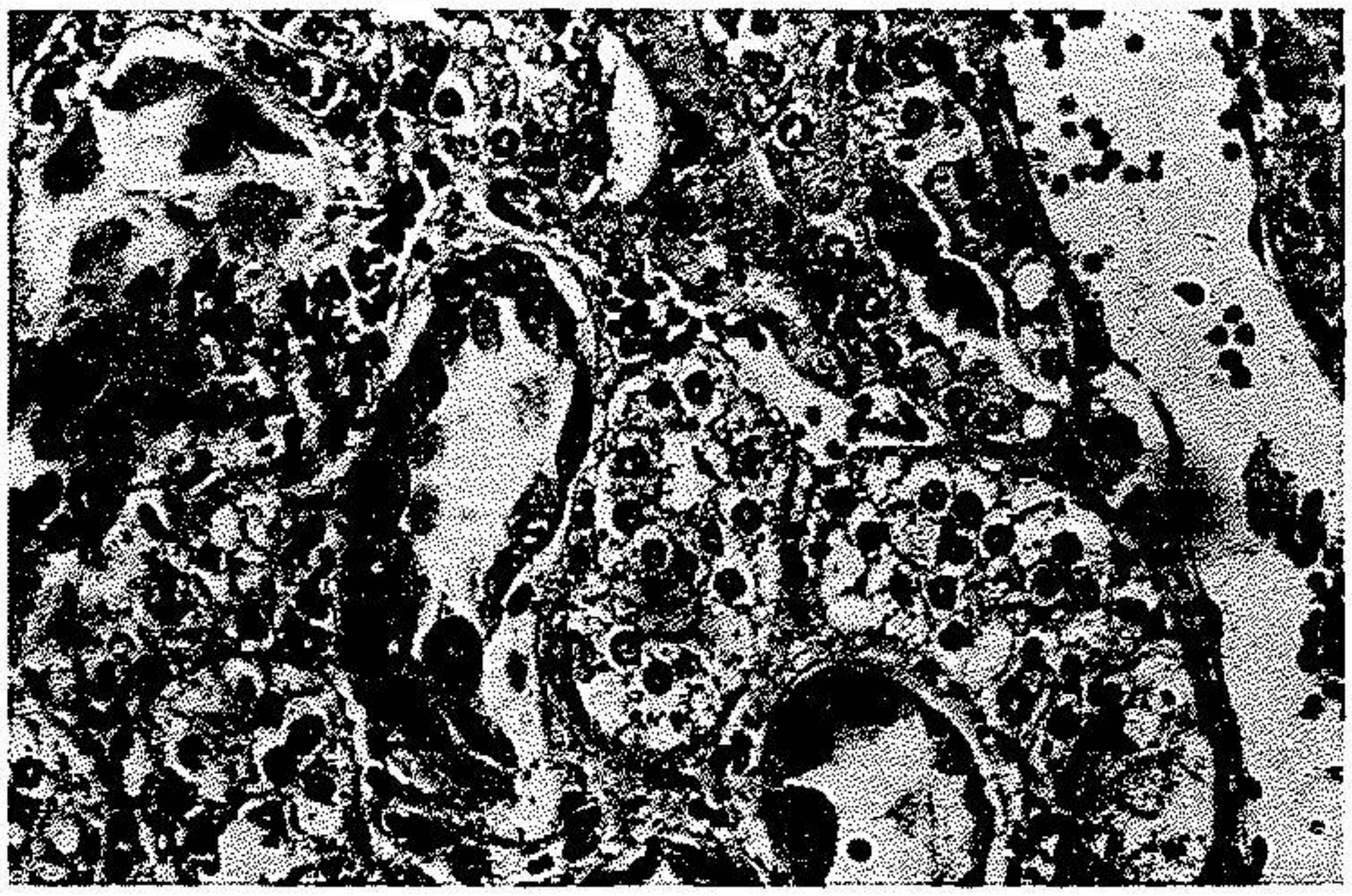


**Fig. 20** Autopsy 43/43 (A 18-year-old woman, admitted for mercurial intoxication).
Renal tubal epithelium (proxymal convoluted tubules) show vascuolar degeneration.


## Summary

In order to study the incidence and histopathological aspects of renal disease and its role in the mortality rate in Iran, the protocols of 4463 autopsies, performed in the last 15 years at the Tehran University Hospitals were reviewed. Among 4463 autopsies there were 861 cases (20 %) of various renal diseases including 282 cases (32.75 %) of disease of the interstitium, 176 cases (20.44 %) of renal lesions in tuberculous patients, 112 cases (13.01 %) of vascular disease, 101 cases (11.73 %) of diseases of the glomeruli, 48 cases (5.57 %) of rheumatic carditis, 30 cases (3.48 %) of epithelial nephrosis, 28 cases (3.25 %) of non tuberculous amyloidosis, 28 cases (3.25 %) of congenital malformation, 20 cases (2.32 %) of non tuberculous diabetes, 13 cases (1.51 %) of neoplastic disease, 10 cases (1.16 %) os septicemia, 8 cases (0.93 %) of eclampsia and 5 cases (0.58 %) of myelomatous kidney.

The underlying diseases in 35 cases of nephrotic syndrome were: tuberculosis, 11 cases (31 %); Non-tuberculous amyloidosis, 6 cases (17 %); chronic glomerulonephritis, 5 cases (14 %); chronic pyelonephritis, 3 cases (10 %); and acute pyelonephritis, one case. The kidney was severely affected in systemic amyloidosis in 64 cases.

Although this report confirms the high incidence of underlying T.B. in amyloidosis, less common diseases such as amebiasis, ulcerative colitis and leprosy should be considered in the etiology of amyloidosis. In 185 cases of rheumatic carditis, 48 cases with renal lesions, including 32 cases of proliferative glomerulonephritis were observed. Among 4463 autopsies, there were 429 cases of tuberculosis, 176 cases of which presented tuberculous- or non-tuberculous renal lesion (nephrotic syndrome, amyloidosis, pyelonephritis, glomerulonephritis).

## References


1- Anonymous (1966). Renal disease and rheumatoid arthritis. Lancet II: 1451-1452.
2- Anonymous (1970). Renal disease in old people. Lancet II: 644.
3- Armin, K. (1971). The Spectrum of rheumatic carditis in Iran. J. or Medical Faculty of Tehran, 29, 1.
4- Ashworth, C. T., Erdmann, R. R. and Arnold, N. J. (1960) Age changes in the renal basement membrane in rats. Amer. J. Pathol. 36: 165-180.
5- Berger, M. J. and Gall, P. (1963). Nephropathic associante une glomerulite proliferative et depots au sein de basales. J. Urol. Neph. 59: 90-96.
6- Berman, L. B. and Antonovysh, T. T. (1969). Glomerular abnormalities in tuberculosis. Arch. Pathol. 69: 52/278-58/284.
7- Biana, C. G., Dyrda, I., Sergio, J. G. and Bencasme, A. (1964). Reneal byalin arteriolosclerosis on electron microscope study. Amer. J. Pathol. 44: 349-535.
8- Burkholder, P. M. and Bradford, W. D. (1969). Proliferative glomerulonephritis in children. Amer. J. Pathol. 56: 423-445.
9- Camerini-Daualos, R. A., Offermann, W., Treser, G., Reich, T. E. and Lange, K. (1968). Glomerulosclerosis in a strain of genetically diabetic mic. J. Amer. Diabetes 17: 301.
10- Crasnier, J. and Slama, R. (1960). Les lupo-erythematonephrites; diagnostic differentiel, nosologique et traitement. Presse Med. 68: 371-374.
11- Deysage, A., Jr. and Dobson, H. L. (1961). Renal glomerular and vascular lesions in prediabetes and diabetes mellitus; a study based on renal biopsies. Ann. Int. Med. 54: 672-684.
12- Edelmann, C. M., Jr., Greifer, I. and Barnett, H. L. (1964). The nature of kindney disease in children, who fil to recover from apparent acute glomerulonephritis. J. Pediat. 64: 881-887.
13- Farmer, C. D. and Ferguson, R. H. (1966). Diagnostic and treatment of lupus nephritis. Disease of the kidney 50: 1119-1125.
14- Fillastre, J. P. and Morel-Maroger, L. (1970). Atteint renal du purpura rhumatoid chez I'adulte. La presse Med. 78: 2375-2378.
15- Forshaw, J. W. B. and Moorhouse, E. H. (1964). Amyloidosis Secondary to chronic ulcerative colitis. Brit. Med. J. 2:94-95.
16- Freedman, P., Meister, H. P. and Siu-co, B. (1966). Subclinical renal response to streptococcal infection. New Eng. J. Med. 275: 795-808.
17- Goettsch, E. (1971). Kidney disease in the young. I. W. B. Saunders Co., Philadelphia, London, Toronto.
18- Gottschalk, B. C. W. (1971). Function of the chronically diseased kidney. Girculation Research 28: 10-13.
19- Grishmann, E., Sohen, S., Salomon, M. I. and Churg, J. (1967). Renal lesion in acute rheumatic fever. Amer. J. Pathol. 51: 1045-1053.

20- Hepinstall, R. H. (1969). The Enigma of chronic pyelonephritis. J. Infec. Dis. 120: 104-108.
21- Heptinstall, R. H. (1968). Pathology of end-stage kidney disease Amer. J. Med. 44: 656-663.
22- Hennigar, G. R., Cohne, R. J. and Katz, H. P. (1961). Nodular glomerulosclerosis; clinico-pathological correlation of 40 advanced cases. Amer. J. Med. Sicence 241: 131/89-137/95.
23- Horris, H. J. and Wiener, J. (1961). The renal lesions in leukemia. Amer. J. Med. Science 241: 140/512-145/517.
24- Ireland, J. T., Patnaik, B. and Ducan, L. J. (1967). Glomerular Ult astructure in secondary diabetic and normal subjects.
25. Kessner, D. M. and Florcy, C. D. V. (1967). Mortality trends for acute and chronic nephritis and infections of the kidney. Lancet 11: 978-980.
26- Lagrue, G. and Milliez, P. (1964). La nephrologie 1964. La Revue de practicien 14: 2271-2309.
27- Lelong, M., Vialatte, J., Canlorbe, P., Stage, P. S., Vinh, L. T. and Samarco, P. G. (1960). La biopsie renale chirugicale dans les syndromes nephrotiques de L'enfant. Archive Francaise de pediatrie 17: 1-13.
28. Lemieux, G., Cuveller, A.A. and Lefebvre, R. (1967). The clinical spectrum of renal in sufficiency during acute glomerulonephritis. Le Journal d'Association Medicale canadienne **96:** 1129-1134.
29. Macadam, R. F. (1970). Glomerular fine structure in an adult case of so-called mixed membranous and proliferative glomerulonephritis. J. Pathol. **100:** 149.
30. Martinez, J. and kohiler, P. F. (1971). Variant Goodpasture's syndrome. Ann. Int. Med. **75:** 66-67.
31. Milliez, P., Lagrue, G. Samarcq, P. and kaplan, K. C. (1961). Le rein des collagenoses. La Revue de praticien **11:** 941-944.
32. Morel-Maroger, L., Fillastre, J. P., Corin, F., Gall, P. and Richet, G. (1970). Evolution histologique inhabituelle de trois glomerulonephrites de L'aulte. La pres. Med. **78:** 55.
33. Owen, K. (1953). Hypertension and renal artery disease. Brit. J. Urol. **31:** 414-418.
34. Pearl, M.A., Jr., Carvajal, E. and Mccracken, B. H. (1963). The Renal aspects of the collagen disease. Amer. J. Med. Sci. **246:** 118/84-137/104.
35. Pirani, C. L., pollak, V. E., Follis, G. and Lonnigan, R. (1963). The Renal glomerular lesion of pre-ecampsia; eectron microscopic studies. Amer. J. Obst. Gynec. **87:** 1047-1070.
36. Pollak, V. E. and nettless, J. B. (1960). Preliminary observations on the differential diagnosis of toxemias of pregnancy; by means of renal biosy. Amer. J. Obst. Gynec. **70:** 866-870.
37. Pollak, V. E. and Nettless, J. B. (1960). The kidney in toxemia of pregnancy, pathological studies based on renal biopsies. Medicine 39: 469-526.
38. Pollak, V. E. and Mark, R. M. (1961). The Toxemias of pregnancy and the renal lesion of pre-eclampsia. Amer. J. Med. **30:** 181-184.
39. Rabson, S. M. (1969). Renal scars and peylonephritis. Amer. Heart J. **78:** 427-428.
40. Richard, J. Sherman, R. S. and Diamond, H. D. (1962). Renal lesions associated with malignant lymphoma. Amer. J. Med. **32:** 184-207.
41. Robbins, S. L. (1967). Pathology. Third ed., 1015, W. B. Saunders, philadelphia, London.
42. Rothfield, N.F., Mckluskey, R. T. and Walwin, D. S. (1963). Renal disease in systemic lupus erythematosis. New Eng. J. Med. **269:** 537-544.
43. Roy, S., Wall, H. P. and Etteldorf, J. N. (1969). Second attacks of acute glomerulonephritis. J. Pediat. **75:** 758-767.
44. Selye, H. (1970). Mercury poisoning; prevention by spironolactome. Science **169:** 3947.
45. Shillingford, J. P. (1964). A Case of rheumatic heart disease with an inschaemic kidney. Brit. Med. J. **2:** 40-43.
46. Sokmen, C. and Ozdermir, A. L. (1967). The Spectrum of renal disease

found by kidney biosy in Turkey. Ann. Int. Med. **67:** 603-605.
47. Spiro, R. G. (1967). Studies on the renal glomerular basement membrane. J. Biochem. Chem. **242:** 1923-1932.
48. Steele, R. E., Leadbetter, G. W. and Crawford, J. D. (1963). Prognosis of childhood urinary tract infection. New Eng. J. Med. **269:** 883-888.
49. Tu, W. H. and Shearn, M. A. (1967). Systemic lupus erythematosis and latent renal tubular dysfunction. Ann. Int. Med. **67:** 100-109.
50. Turgeon, C. and Sommers, S. C. (1961). Juxta-glomerular cell counts and human hypertension. Amer. J. Pathol. **38:** 227-237.
51. Ural, L., Munkacsi, I. and Szinay, G. (1961). New data on the pathology of true scleroderma kidney. Brit. Med. J. **1:** 713-715.
52. Velican, D. and velican, C. (1970). Structural heterogenaty of kidney basement membranes. Nature 226: 1259-1260.
53. Vernier, R. L. (1963). The Structure and function of renal glomerular capillaries in health and disease. Pediatrics **82:** 480-496.
54. West, C. D., Mcadams. A. J. and Northway, J. D. (1968). Focal flomerunephritis in children. J. Pediat. 73: 184-194.
55. White, R.H.R. (1970). Histo-pathological studies in post-strptococcal glomerulonephritis. Arch. Dis. Child. **45:** 712.