Virchows Archiv A Pathol Anat (1992) 421:65–71

Virchows Archiv A
Pathological Anatomy and Histopathology

Case report

The features of glomerulitis in the acute stage of panarteritis nodosa

Developmental process of glomerulitis and correlation between glomerular and vascular lesions

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Received January 14, 1992 / Accepted February 6, 1992

Summary. In order to determine the morphological characteristics of certain vascular and glomerular lesions and the correlation between them, we attempted three-dimensional observation using serial sections of an autopsy kidney, from a patient with panarteritis nodosa in the acute phase. Fibrinoid necrotizing vasculitis spread from arcuate arteries to arterioles in a segmental, eccentric pattern, especially occurring at bifurcations. Segmental arteriolitis often originated in extraglomerular capsular arteriole and spread into the intraglomerular capsular arteriole and glomerular capillaries, directly leading to necrotizing glomerulitis. Some of the glomerulitis connected directly with extracapsular arteriolitis was segmental and eccentric in distribution. Most of the glomerulitis had a tendency to originate in the hilar arteriole, which was near bifurcations between the arteriole and glomerular capillaries. Segmental glomerulitis was found to consist of four elements: glomerular tuft necrosis with fibrin exudation, crescents, rupture or dissolution of Bowman's capsule, and pericapsulitis. It is suggested that the segmental inflammatory attacks, repeated more than twice, give rise to widespread and almost global necrotizing glomerulitis.

Key words: Pan(Poly)arteritis nodosa – Segmental necrotizing glomerulitis – Granulomatous glomerulitis

Introduction

Segmental necrotizing glomerulitis with crescents and granulomatous glomerulitis have been regarded as the characteristic glomerular lesions of panarteritis nodosa and of Wegener's granulomatosis (Heptinstall 1983; D'Agati and Appel 1989). However, little is known about the developmental process of the various glomerular lesions and the correlation between them, especially with respect to glomerular capillaries as the distal end of the arterial vascular tree.

In this study, using serial sections of the kidney from a patient with panarteritis nodosa in the acute phase, we made three-dimensional observations to reveal both the characteristics of glomerular lesions and the pathogenesis of the evolution of granulomatous glomerulitis.

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Case report

A 63-year-old man was admitted to the hospital in a state of apnoea and cardiac arrest. His health had been good until 3 months earlier, when he was first admitted to Nagano Cyu-Oh Hospital because of constipation, epigastric fullness and gradual weight loss of about 10 kg over 1 year. On admission, urinalysis revealed microscopic haematuria. Blood analysis revealed leucocytosis (WBC 10900/mm^3) and anaemia (haemoglobin 8.5 g/dl). No abnormality was discovered by examination of the upper gastrointestinal tract. No medication was administered. As the patient was doing well in general, he did not undergo a follow-up examination. One day, while he was shopping, he suddenly lost consciousness and apnoea occurred. He was brought to the hospital by ambulance and remained unresponsive despite intensive emergency treatment and soon died. Laboratory examination revealed: WBC 12300/mm^3, haemoglobin 8.4 g/dl, total protein 6.8 g/dl, albumin/globulin 0.47, albumin 32.2%, γ-globulin 34.8%, Na^+ 144 mEq/l, blood urea nitrogen 37.8 mEq/l, lactate dehydrogenase 353 mIU, Alkaline phosphatase 86 IU/l, C-reactive protein 2+. Mild anaemia, leucocytosis, hypoproteinaemia, hyper γ-globulinaemia and azotaemia were revealed. During his clinical course, corticosteroid was not administered.

Autopsy revealed that necrotizing vasculitis mainly in the acute phase was systemically distributed in medium- or small-sized arteries. These lesions were widespread and severe in the kidneys, the gastrointestinal tract, the adrenal glands, the urinary bladder, the prostate, and moderate in the coronary artery, the gallbladder, the liver and the diaphragm. Fibrous vasculitis, that is, vasculitis in healing stage, was also observed in the medium-sized arteries of the gastrointestinal tract. Sudden death seemingly resulted from acute cardiac infarction due to coronary arterial lesions.

Table 1. The number of glomeruli in the outer third and in the inner two-thirds of the cortex

Histology and subgroups	Total	Outer layer	Inner layer	RC score	*P* values
Observed glomeruli	252	88	164		
Glomeruli with any change	71	44	27		
Acute glomerulitis	56	31	25	2.69	
Central extravascular type	41	22	19	2.44	
A1	25	8	17	3.49±0.47	A1–A2: $P<0.001$ A1–M: $P<0.05$
A2	16	14	2	0.80±0.46	A2–M: $P<0.001$ A2–C1: $P<0.01$
Central intravascular type	2	1	1	4.00	
Peripheral extravascular type	5	2	3	3.73±0.50	
Peripheral intravascular type	2	1	1	4.00	
Mixed type glomerulitis	6	5	1	2.79±0.62	
Chronic glomerulitis	13	11	2	0.60	
C1	11	11	0	0.20±0.22	
C2	2	0	2	2.81	
Ischaemic global sclerosis	2	2	0	4.00	

The values of remnant capsule (RC) score are presented as mean±SD. Statistical analysis (Mann Whitney test) of the RC scores of A1, A2, M, and C1 glomerulitis group

Materials and methods

Ten kidney blocks were obtained from both kidneys which weighed 180 g each. There was moderate congestion and petechiae. After routine examination of the renal sections, a block suitable for the present examination was selected. The paraffin-embedded kidney block, square in shape in the coronal plane, about 12×14 mm in size, was cut at 2–3 μm intervals and about 120 serial sections were made. The specimens were divided into 24 sets of 5, in which the specimens 1–5 were stained respectively with haematoxylin-and eosin, periodic acid-Schiff, periodic acid-methenamine silver, Masson's trichrome and van Gieson's elastic stain. Using these serial sections, we attempted to reveal the three-dimensional structure of the vascular and glomerular lesions.

The renal cortex was divided into two areas: the outer third and the inner two-thirds. Eighty-eight glomeruli in the outer layer and 164 glomeruli in the inner layer were observed in these serial sections (Table 1). We especially evaluated glomeruli situated at the distal part of the vascular tree in view of the correlation between glomerular and vascular lesions.

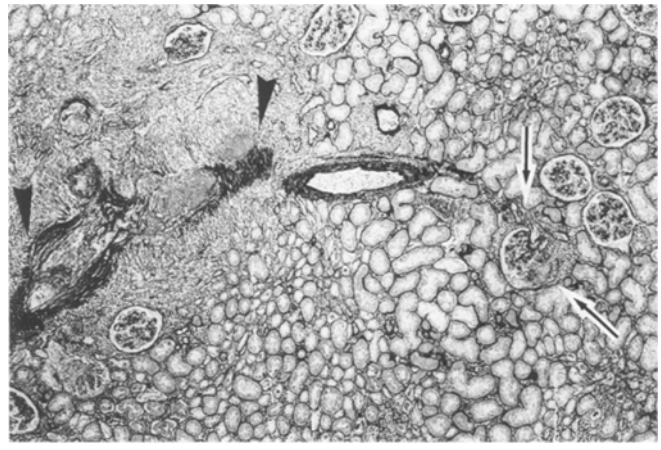


Fig. 1. Arteries of arcuate and interlobular arteries (*arrowheads*) and glomerulitis of central extravascular type (*arrows*) are observed in the same vascular tree, with intervening unaffected arteries. Periodic acid-methenamine silver, ×20

Results

Interlobar, arcuate and interlobular arteries and arterioles were affected. The acute phase of vasculitis, composed of fibrinoid necrosis with infiltration of inflammatory cells, was mainly seen, and in some sites, fibrous vasculitis was also evident. The vasculitic lesions were segmental in distribution, involving a short length of the vessel. Segmental and eccentric vasculitic lesions were apt to be observed at bifurcations of arcuate and interlobular arteries with a high frequency.

Not only arterioles but also the glomeruli situated in the distal portion of these lesions were involved in various sites with intervening skip zones of unaffected vessel wall (Fig. 1). It was observed that some vasculitis was located at the end of the arteriole and spread into necrotizing glomerulitis (Fig. 2a, b). When vascular lesions were eccentric, only one or two glomerular capillaries were continuously involved on the same side as the hilar arteriolar lesion in contrast to the other capillaries which were unaffected or only slightly affected. When hilar arteriolitis was nearly circumferential, almost all capillaries were involved.

Two hundred and fifty-two glomeruli were observed, including 71 affected glomeruli (Table 1). In these, fibrin exudation was observed in 56 but was not seen in the other 15.

Glomerulitis with fibrin exudation was termed acute glomerulitis. In acute glomerulitis, at least five histological varieties could be distinguished (Table 1, Fig. 3).

There was a central extravascular type which appeared to be derived from hilar arteriolitis, resulting in segmental necrotizing glomerulitis. This was composed of glomerular tuft necrosis covered with cellular crescents, with evidence of ruptures or the dissolution of Bowman's capsule and a pericapsular interstitial inflam-

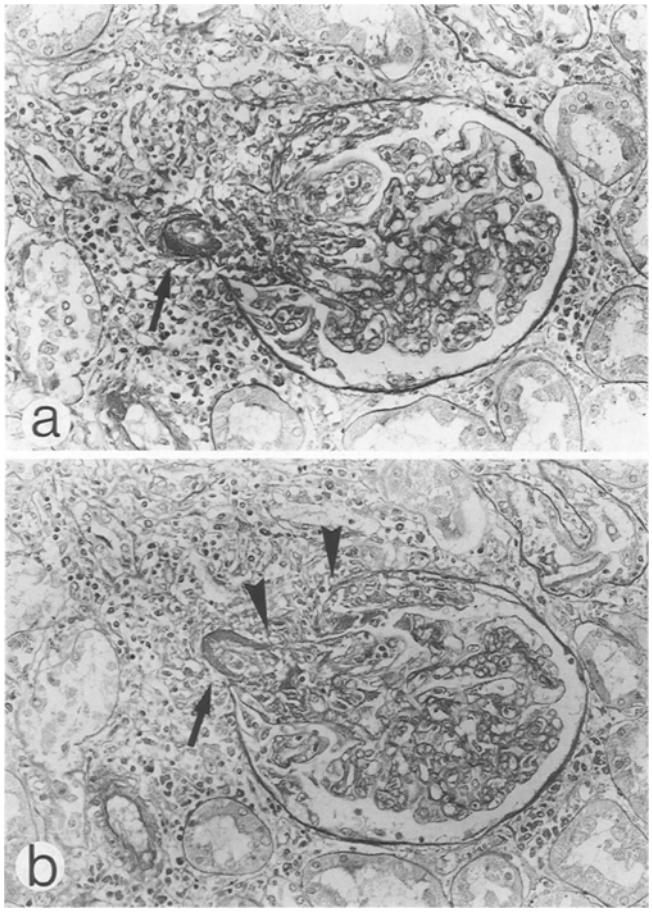


Fig. 2a, b. Segmental necrotizing glomerulitis originating in extraglomerular capsular arteriolitis. Serial sections of the glomerulus are as follows: **a** extraglomerular capsular arteriolitis (*arrow*); **b** hilar arteriolitis (*arrow*) with segmental dissolution of Bowman's capsule (*arrowheads*). Periodic acid Schiff, ×170

mation associated with inflammatory cell infiltration (Figs. 3a, b). Each component of the segmental lesions was located on the same side as eccentric hilar arteriolitis and fused with each other. Central extravascular type acute glomerulitis was divided into two subtypes: A1 (Fig. 3a) and A2 (Fig. 3b). In A1 glomerulitis, the pathological changes of Bowman's capsule were localized adjacent to the site of fibrin exudation. However, the extent of the capsular change was greater than that of fibrin exudation in A2 glomerulitis. There were 25 glomeruli in the A1 group. The necrotizing and destructive lesions of the glomerular tuft were observed in and around the region of fibrin exudation, the amount of which was relatively large (estimated semiquantitatively, from + to 2+) except for 2 glomeruli. In these glomeruli, nearly unaffected capillaries except for a mild degree of collapse were observed in addition to the necrotizing lesion. Fibrinoid necrosis of the glomerular tuft was covered with cellular crescents accompanied by capsular dissolution and pericapsular interstitial nephritis infiltrated by inflammatory cells. There were 16 glomeruli in the A2 group. The structure of the glomerular tuft was almost completely destroyed, resulting in hardly any capillaries being left. Glomeruli of this group, except for 1 glomerulus, were associated with only a small amount of fibrin exudation (estimated semiquantitatively, ±), but with fibrocellular or fibrous crescents, comparatively broad dissolution of Bowman's capusle and interstitial inflammatory cell infiltration with mild fibrosis. Even segmental sclerosis was observed in these glomeruli.

The central intravascular type of lesion typically showed intra-arteriolar fibrin thrombus and fibrinoid necrosis of the glomerular tuft, without large crescents or manifest changes of Bowman's capsule (Fig. 3c).

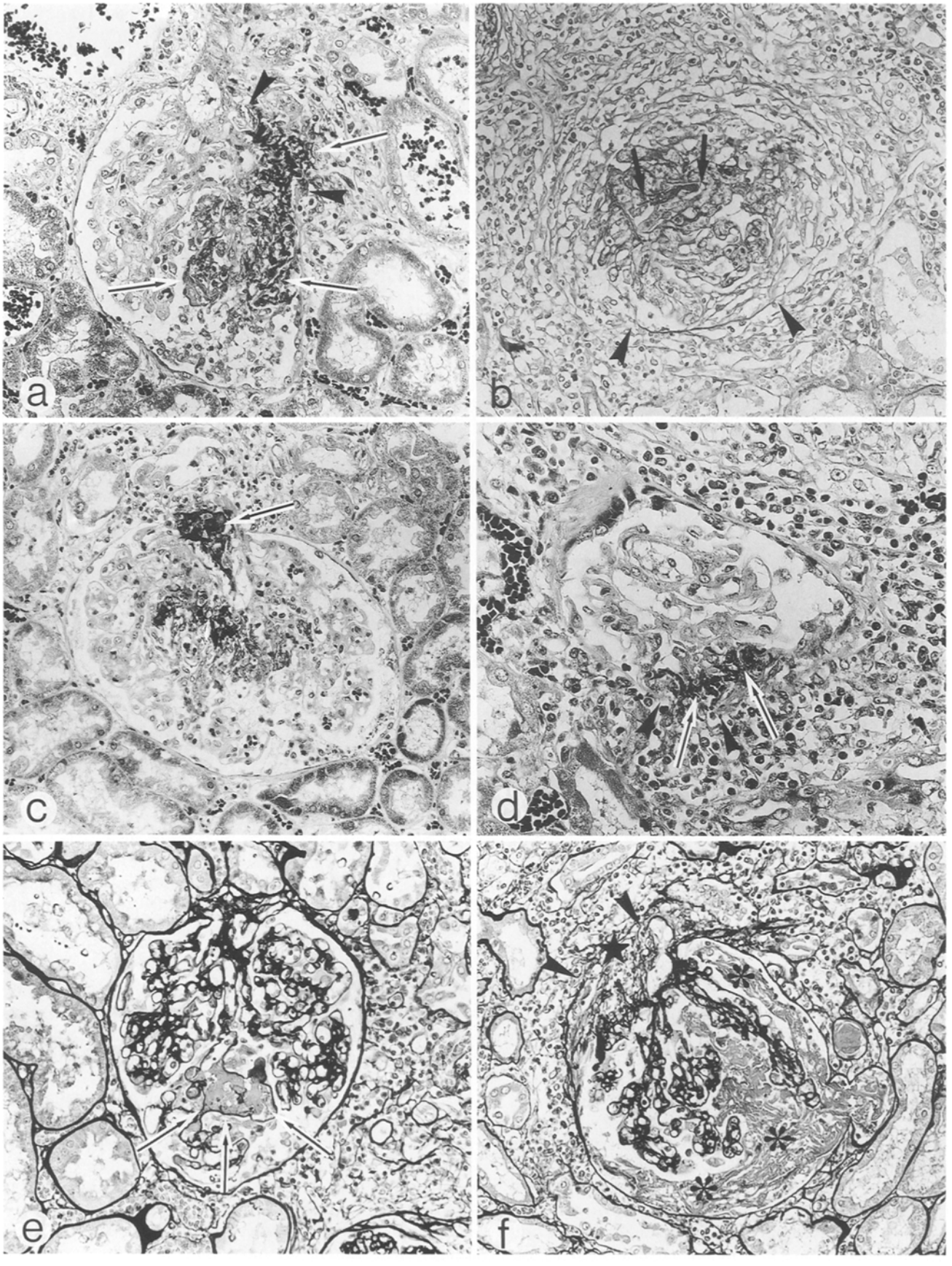
The peripheral extravascular type, originated in glomerular capillaries without any involvement of the hilar arteriole (Fig. 3d). This type was composed of peripheral glomerular tuft necrosis with fibrin exudation and small crescents. Rupture or dissolution of Bowman's capsule were observed adjacent to fibrin exudation. These capsular lesions were located in the peripheral area of the capsule and did not continue to the hilar arteriole.

The peripheral intravascular glomerulitis was a small lesion composed of fibrin thrombus within the capillary lumen, sometimes associated with small crescents (Fig. 3e).

In mixed type glomerulitis (M glomerulitis), there was a mixed acute and chronic glomerulitis (Fig. 3f). Fibrous and sclerosing segmental glomerular lesions were associated with the dissolution of Bowman's capsule involving in and around the hilar arteriole.

In chronic glomerulitis, partial or widespread sclerosis of the glomerular tuft without fibrin exudation and fibrous crescents with widespread dissolution of Bowman's capsule were observed (Fig. 4). There were 13 glomeruli in this chronic stage (Table 1). Chronic glomerulitis was classified into two subtypes: C1 (Fig. 4a) and C2 (Figs. 4b, c). No fibrin exudation was found, though inflammatory cell infiltration in and around the glomerulus was obvious in C1 glomerulitis. Neither fibrin exudation nor cell infiltration was observed, but sclerosis and fibrosis were observed in C2 glomerulitis (Fig. 4b, c). Eleven glomeruli in the chronic stage were classified in the C1 group (Fig. 4a). Segmental sclerosis of the glomerular tuft, fibrous crescent and fibrous interstitium infiltrated by inflammatory cells were observed associated with widespread capsular changes seemingly occurring in the vicinity of the hilar arteriole in this group. Only 2 glomeruli were classified in the C2 group (Figs. 4b, c). Both showed segmental sclerosis, fibrous crescent and dissolution of capsule, while the remaining part of each glomerulus, including Bowman's capsule, remained almost intact.

The extent of the remnant of Bowman's capsule was measured in the following manner: a capsule of each section was divided into four equal parts, and the number of quadrants was the extent of the section: a remnant of only one-quarter of the original capsule was designated 1, about half a capsule was 2, an almost totally circumferentially intact capsule was 4. Then, the average of each number of more than ten sections in each glomerulus was regarded as representative of the remnant surface area of the glomerulitis. This average was designated as the remnant of Bowman's capsule, that


a
b
c
d
e
f

is, the remnant capsule (RC) score. The average RC score in each group of glomerulitis is shown in Table 1.

The average RC score described above was analysed statistically by the Mann-Whitney test (Table 1). The difference between A1 and A2 was statistically significant well beyond the 0.1% level, A2 and M was statistically significant well beyond the 0.1% level, A1 and M was statistically significant beyond the 5% level, and A2 and C1 was statistically significant well beyond 1% level.

The prevalence of the involved glomeruli was 44 of 88 (50%) in the outer third, and 27 of 164 (16.5%) in the inner two-thirds of the renal cortex (Table 1). Out of the involved glomeruli in each part, A2+C1 glomeruli, which were affected widely, were 25 of 44 (56.8%) in the outer third, and 2 of 27 (7.4%) in the inner two-thirds.

Based on the histopathological characteristics described above, including RC score, we concluded that the A1 glomerulitis was the most prevalent in acute glomerulitis, suggesting that the initial segmental inflammation predominantly originates in and around the hilar arteriole (Fig. 5). In some glomeruli, only a single inflammatory attack occurred. With time, these glomeruli progressed to the healed stage of segmental necrotizing lesions, manifested by C2 glomerulitis. After some undetermined interval, however, a second segmental inflammation occurred in a large number of glomeruli, frequently on the other side of the hilar arteriolar wall or in the most proximal area within the capillaries free from the initial inflammation, manifested by a mixed type glomerulitis. With the passage of time, there were decreases in the amount of fibrin exudation with spreading of dissolution of Bowman's capsule (A2 glomerulitis), resulting in the almost global destruction of the glomerular structures (C1 glomerulitis).

Fig. 3a–f. Acute glomerulitis. **a** A1 type central extravascular acute glomerulitis. Segmental fibrinoid necrosis of glomerular capillaries originating in the hilar arteriole. A part of Bowman's capsule adjacent to the hilar arteriole is ruptured (*arrowheads*) by fibrin exudation (*arrows*) originated in the hilar arteriole. Masson trichrome, ×150. **b** A2 type glomerulitis. Segmental glomerular sclerosis and widespread dissolution of Bowman's capsule (*arrowheads*) associated with interstitial inflammatory cell infiltration. Fibrin exudation (*arrows*) is localized and scanty. Periodic acid Schiff, ×100. **c** Central intravascular type. Segmental fibrinoid necrosis of glomerular capillaries derived from fibrin thrombus (*arrow*) in hilar arteriole. Masson's trichrome, ×150. **d** Peripheral extravascular type. Fibrinoid necrosis of the peripheral part of glomerular capillaries (*arrows*) and cellular crescent with fibrin exudation. A part of Bowman's capsule adjacent to fibrin exudation is destroyed (*arrowheads*). Masson's trichrome, ×150. **e** Peripheral intravascular type. Intracapillary fibrin thrombus (*arrows*) is observed. Periodic acid-methenamine silver, ×120. **f** Mixed type glomerulitis. Segmental glomerular sclerosis, fibrous small crescent (*star*) and dissolution of Bowman's capsule (*arrowheads*) involving one side of the hilar arteriole are observed in the upper-left part of the glomerulus. Central extravascular type segmental acute glomerulitis with a large amount of fibrin exudation (*snow stars*) is observed in the right part of the glomerulus. Periodic acid-methenamine silver, ×150

Discussion

The relationships between vascular and glomerular lesions recognized in this case are summarized as follows. Arcuate and interlobar arteritis and glomerulitis was observed in the same vascular tree. Some arteriolitis, originated in the extraglomerular capsular region, spread through hilar arteriole into glomerular capillaries, leading to segmental necrotizing glomerulitis. In such lesions, both eccentric arteriolitis and segmental glomerulitis were contiguous. The morphological features of the central extravascular type of glomerulitis, which was the most common in this study, was closely similar to those of glomerulitis originating in the extracapsular arteriolitis. The process of morphogenesis of the global glomerulitis probably results from segmental glomerulitis occurring more than twice in each glomerulus, resulting in a fully developed glomerulitis. In the developmental process of glomerulitis, each glomerular lesion showed a segmental and eccentric distribution, having a tendency to occur at or near bifurcations, that is, around the vascular poles.

The vascular lesions were segmental, eccentric in distribution and located predominantly at bifurcations, as already reported by many authors (Heptinstall 1983; D'Agati and Appel 1989). Some arteriolitis spreading continuously into the glomerulus resulted in necrotizing glomerulitis (Fig. 2a, b). When an intracapsular (hilar) arteriole was spared, there was no inflammation in the glomerulus. In contrast, when intracapsular arterioles were affected by vasculitis, glomerulitis was observed to be associated with arteriolitis. When intracapsular arterioles were affected circumferentially, all capillaries were involved, resulting in widespread or nearly global glomerulitis. When intracapsular arterioles were affected predominantly in an eccentric fashion, glomeruli were also involved in a segmental manner and the glomerular lesion spread continuously on the same side of arteriolitis. With these findings, it seems that the glomerulitis derived from arteriolitis can be regarded as the distal end part of arteriolitis.

The most prominent glomerular alteration was focal segmental necrotizing glomerulitis with crescents, especially of the central extravascular type (Fig. 3a). Glomerulitis of this type displayed glomerular tuft fibrinoid necrosis, cellular crescents, rupture or dissolution of Bowman's capsule and pericapsular interstitial inflammatory cell infiltration. Each component was located on the same side, fusing one with another and deriving from eccentric hilar arteriolitis. These findings are quite similar to granulomatous inflammation in Wegener's granulomatosis, which originates in hilar arteriolitis as suggested by McManus and Hornsby (1951) and Yoshikawa and Watanabe (1984). Weiss and Crissman (1984) suggested that prior necrosis was the cause of sclerosis based on the observation of fragmented glomerular basement membrane within the healing glomerulus in Wegener's granulomatosis. Serra et al. (1984) also reported that in patients with vasculitis who had received serial biopsies a tendency toward sclerosis of the previously necrotizing glomerular lesions was seen. Thus,

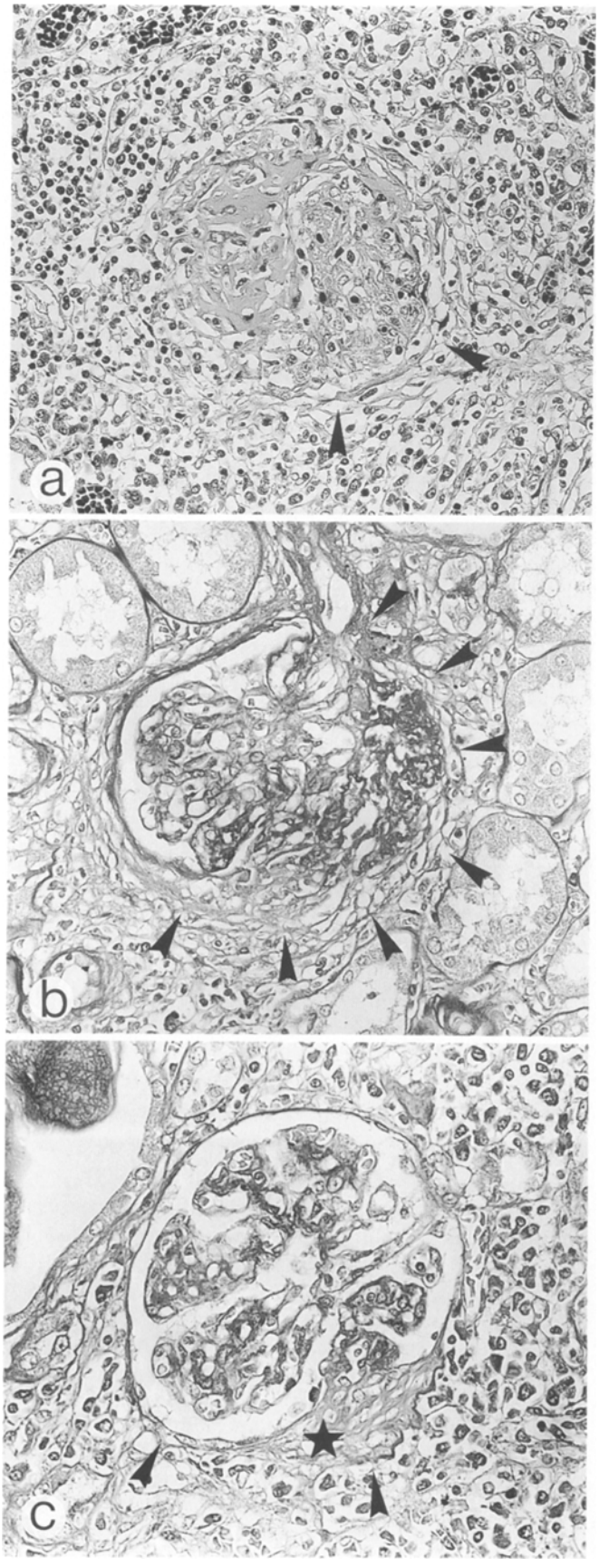


Fig. 4a–c. Chronic glomerulitis. **a** C1 type. Segmental glomerular sclerosis with widespread dissolution of Bowman's capsule (*arrowheads*) associated with inflammatory cell infiltration. Periodic acid Schiff, ×120. **b** C2 type. Segmental sclerosis, fibrous crescent and dissolution of capsule (*arrowheads*) are continuous with the hilar arteriole, which is thought to be the origin of necrotizing inflammation. Periodic acid Schiff, ×120. **c** C2 type. Segmental sclerosis with small fibrous crescent (*star*) is observed in the periphery of glomerular capillaries. Bowman's capsule adjacent to this sclerosing lesion is destroyed (*arrowheads*). Periodic acid Schiff, ×150

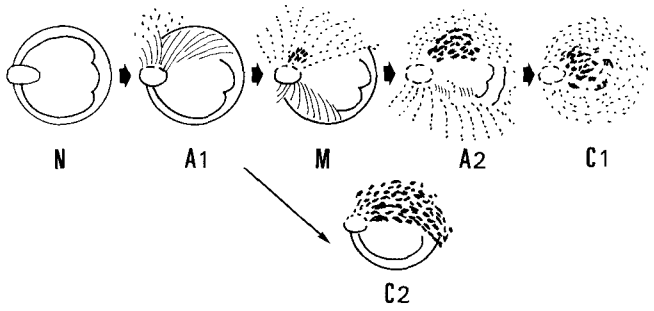


Fig. 5. Schematic representation of the developmental process of inflammatory glomerular changes. Recurrence of segmental inflammation more than twice in each glomerulus leads to the whole glomerular involvement. *N*, Normal glomerulus, Bowman's capsule, and hilar arteriole; *A1*, A1 type acute glomerulitis; *M*, mixed type glomerulitis; *A2*, A2 type acute glomerulitis; *C1* C1 type chronic glomerulitis; *C2*, C2 type chronic glomerulitis. Fibrin exudation ( ), sclerotic lesion ( ), inflammatory cell infiltration ( )

the sequential lesion in each glomerulitis was apparently determined by the amount of fibrin exudation, the character of crescents and of the glomerular and interstitial changes. In 6 mixed type glomeruli in this case, all segmental dissolution of Bowman's capsule occurring around the hilar arteriole was associated with fibrous crescents. This finding indicates that segmental necrotizing lesions originated first in hilar arteriolitis, that is, that the initial inflammatory attacks were all of the central extravascular type.

As for acute glomerulitis, the average RC score was 3.5 in the A1 group, suggesting that about five-sixths of the area was preserved in A1 glomeruli (Table 1). The average RC score was 0.8 in the A2 group, suggesting that only a quarter of the area was preserved in A2 glomeruli. In the C1 group, the average RC score was 0.2, suggesting that almost the whole capsule had disappeared. In mixed type glomeruli, the average RC score was 2.8, suggesting that about a quarter of the area was destroyed by the initial segmental necrotizing glomerular inflammation. In the early stages of segmental glomerulitis, rupture of the capsule is usually adjacent to the site of origin of the fibrin exudation. In an advanced stage, associated with fibrosis of crescents and interstitium, the dissolution of the capsule became more widespread. Thus, the RC score is throught to vary not only according to the size but also to the stage of evolution of segmental necrotizing glomerulitis. A small RC score may indicate two possibilities. One is that the lesion of segmental glomerulitis is small; the other is that

the lesion is in an early stage of segmental glomerulitis. Similarly, a large RC score may also indicate two possibilities. One is that the lesion is large; the other is that the lesion is old or in the healed stage.

The progress from segmental lesion to nearly global granulomatous lesion by continuous evolution, originating in hilar arteriolitis extending along the pericapsular space, was suggested by Yoshikawa and Watanabe (1984), based on their investigations of granulomatous glomerulitis in Wegener's granulomatosis. However, in this study, it was concluded that there was a disparity of age between A1 glomerulitis and A2 glomerulitis, which was well documented by the statistical difference between the RC scores of each type of glomerulitis (Table 1). In the mixed type (M glomerulitis), it was suggested that after the initial segmental glomerulitis had occurred, the second occurred at some undetermined interval. M glomerulitis proceeded into A2 glomerulitis and consequently resulted in C1 glomerulitis with the disappearance of fibrin exudation. Further, segmental necrotizing glomerulitis, accompanied by the dissolution up to half of the surface of Bowman's capsule (as in one glomerulitis in the C2 group, Fig. 4b), occurred more than twice at some undetermined intervals, leading to involvement of the whole glomerulus. When the initial inflammation originated on one side of the hilar arteriolar wall, the subsequent episode was apt to occur on the other side (Fig. 3f). When the initial inflammation involved the arteriolar wall almost circumferentially, the second was apt to occur in the most proximal area, within the preserved capillaries. That is, the second episode seemed to have a tendency to occur adjacent to the site of origin of the first. Segmental necrotizing glomerulitis was eccentric in distribution, had a tendency toward healing, and a high affinity to the hilar arteriole, which was near bifurcations of arteriole and glomerular capillaries. This area is thought to be vulnerable to segmental inflammation, which recurs, evolving into the global destruction of the glomerular structures.

Some authors regard segmental necrotizing glomerulitis as a vasculitis of glomerular capillaries, which are the smallest vessels. Davson et al. (1948) reported for the first time that glomerular lesions were the only evidence of vasculitis within the kidney from a careful autopsy study of two patients with panarteritis nodosa. Yoshikawa and Watanabe (1984) reported, in 24 cases of Wegener's granulomatosis, that vasculitis in the kidney was noted in all 13 patients with granulomatous glomerulonephritis, whereas it was infrequent in those without granulomatous crescents, occurring in only 3 of 11 kidneys. Serra et al. (1984) reported that after prolonged follow-up, a substantial portion of patients presenting with necrotizing and crescentic glomerulonephritis developed full expression of systemic vasculitis. Shibata (1981), after a thorough investigation of autopsy findings and the clinical course of systemic lupus erythematosus, pan (poly) arteritis nodosa, and primary glomerulonephritis in many cases reported that glomerulonephritis could be a manifestation of systemic vasculitis. Thus, glomerulonephritis itself could be a manifestation of vasculitis of the smallest vessels.

Finally, in relation to the glomerular changes in particular sites in the cortex, it was observed that the ratio of involved glomeruli in the outer layer (50.0%) was higher than that in the inner layer (16.5%) in our study (Table 1). In addition, the ratio of A2+C1 glomeruli in the outer layer (56.8%) was higher than that in the inner layer (7.4%): the extent of the lesions in each invovled glomerulus in the outer layer was comparatively larger than that in the inner layer. In Wegener's granulomatosis, Watanabe et al. (1981) reported that granulomatous glomerular lesions were more prevalent in the outer layer of the cortex. Considering these findings, it can be speculated that in the outer cortex there is an area having an affinity for glomerulitis. In this area, with the passage of time, many glomeruli become involved in a segmental pattern with some glomeruli repeatedly affected. Thus in patients in the advanced stage, glomerular involvement is large in number and more widespread or global in each glomerulus, especially in the outer cortex.

Acknowledgements. The authors are grateful to Miss T. Nishizawa, Miss N. Aoshima and Mrs. M. Watanabe for their technical assistance.

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