Severe Forms of Glomerulonephritis: Subacute Malignant and Rapidly Progressive


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Clinicomorphological comparisons were made in two groups of patients who died of renal failure within a period of 3 to 24 months. Group 1 (15 cases) showed extracapillary proliferation and membranous changes; group 2 (31 cases) had nephritic corrugated kidney. These comparisons served to separate two severe forms of glomerulonephritis: subacute malignant and rapidly progressive. The rapidly progressive form differs from subacute malignant nephritis by its less sudden beginning, longer duration, longer survival (4 ± 0.8 and 19 ± 0.8 months, respectively) and also by the morphological changes indicated.

Recently, subacute malignant nephritis has been frequently observed and some investigators single out still one more variant of the disease: rapidly progressive glomerulonephritis. Some authors identify the notions “subacute malignant nephritis” and “rapidly progressive glomerulonephritis”. This fact can be related, to some extent, to the variety and depth of changes (proliferation, membranous and fibroplastic processes) detected during the investigation of renal biopsy material both in subacute malignant nephritis and rapidly progressive glomerulonephritis. In the 1960s, extra-intracapillary proliferation was considered to be a morphological equivalent of subacute malignant nephritis. However, in the publications of the seventies, in subacute malignant nephritis alongside with proliferation and membranous impairment a fibroplastic process is described which is an end stage of morphological evolution of nephritis.

Wilson [12] separates rapidly progressive glomerulonephritis as its malignant variant which is characterized by less rapid onset, longer duration of disease (from 1 to 5 years) and morphological changes of the membranoproliferative type.

Bacani et al. [3] described 8 cases of rapidly progressive nephritis of poststreptococcal origin: it presented as severe glomerulonephritis and within 5 to 27 weeks the patients died of uremia and cardiac insufficiency. Histological examination of biopsy material showed extracapillary cellular proliferation and rapid development of interstitial tissue. The authors are inclined to separate rapidly progressive glomerulonephritis as an independent disease with unknown etiology and pathogenesis. Bohle et al. [4] single out rapidly progressive glomerulonephritis in their description of renal biopsy tissue.
Enumerating the forms of glomerulonephritis, Sarre [8] singled out rapidly progressive glomerulonephritis and related to it the cases of renal insufficiency developing in about two years.

Arieff and Pinggera [2] reported on 6 cases of rapidly progressive glomerulonephritis with acute onset, oliguria or anuria, rapid decrease in creatinine clearance and increase in azotemia. The study of renal biopsy tissue in all cases demonstrated cellular proliferation of glomerular loops, and thickening of capsules due to epithelial crescents.

Wojnarowski et al. [13] state that the progressive course of glomerulonephritis more often happens in cases of streptococcal origin stipulated by extracapillary proliferation and its clinical manifestations are nephrotic syndrome, hypertension, erythrocyturia and development of terminal renal failure within 1 year.

Stejskal [9] studied 44 renal biopsy specimens from 33 patients with severe post-streptococcal glomerulonephritis, and concluded that the rapidly progressive course of the disease contributed to discontinuities of the glomerular capillary wall.

Couser [6] believes that rapidly progressive course of glomerulonephritis is due to antibody circulation in blood to the basement membrane of glomerular capillaries.

Georgescu [7] analysing the data of renal biopsy, singles out proliferative-progressive glomerulonephritis resulting from persistent acute glomerulonephritis for which it is difficult to make prognosis.

Sue et al. [10] collected 14 observations of diseases of various etiology (post-streptococcal nephritis, systemic lupus erythematosus, Goodpasture’s syndrome, etc.) when proteinuria, hematuria and rapid decrease in renal function are present.

Consequently, the description of rapidly progressive glomerulonephritis is mainly based on the findings of morphological studies and retrospective clinicomorphological comparisons of a small number of observations. The notion “rapidly progressive glomerulonephritis” comprises variable morphological alterations. Unification is based on their common clinical manifestations, renal failure develops rapidly on the background of nephritic syndrome and death ensues within some months to two years.

In clinicopathological comparisons of cases of glomerulonephritis, when the patients died within some months to two years from onset of the disease, we set up 2 groups. Group I comprised cases of nephritic corrugated kidney (31 patients), and group 2 cases with morphologically detected intra-extracapillary proliferation (15 patients). The indicated difference in morphological manifestations was the basis for differential clinicolaboratory analysis. Out of 31 patients, who died from renal insufficiency within 6 to 24 months, 30 showed secondary corrugated kidneys at autopsy. At biopsy made five months after onset of the disease, before renal insufficiency developed, one patient showed proliferative-fibroplastic type of glomerulonephritis (the relative percentage of involved glomeruli was 100%).

We consider the cases developing renal insufficiency within two years