This patient had severe salt-retaining post-streptococcal glomerulonephritis. She was perfectly well before the onset of the present illness, as a normal pregnancy and uneventful delivery testify. Further questioning revealed that the complaint of "mumps", 1 month prior to admission, was in reality pharyngitis. This is consistent with the latent period of the pharyngitis-associated post-streptococcal glomerulonephritis. Furthermore, she had all the clinical features of post-streptococcal glomerulonephritis: hematuria, hypertension, edema and circulatory congestion. A second urinalysis performed on a freshly voided morning urine confirmed the presence of an inflammatory process involving the glomeruli. It showed 2+ protein, abundant RBC (many of which were dysmorphic), 5-10 WBC/HPF and RBC casts.

Circulatory congestion dominated the clinical picture of post-streptococcal glomerulonephritis in this patient. Frank pulmonary edema and massive cardiomegaly may lead many physicians to believe that she had congestive heart failure. However, her cardiac output was 7.8 l/min, the SVR was 1,043 dyn/s/cm-5, and the systemic A-V C\(\frac{1}{3}\) difference was 4.2 vol%. An A-V O\(\frac{2}{3}\) difference of 4.2% is markedly decreased and characteristic of a congested state with a normal myocardial function (and markedly increased cardiac output). Thus, congestive heart failure was not the cause of this patient’s circulatory congestion.

We were convinced that all the symptoms in this patient were due to salt and fluid retention secondary to the glomerular injury. We treated her with high doses of intravenous furosemide plus fluid and salt restriction. In 1 week, the patient lost 20.9 kg, her blood pressure returned to normal (120/80 mm Hg), and all the other signs and symptoms of circulatory congestion disappeared. In a patient with suspected acute glomerulonephritis, complement studies are the key to establish a possible diagnosis. The causes of acute glomerulonephritis can be divided into 2 categories, one having low serum complement, the other having normal complement levels. Complement studies in this patient revealed a C3 level < 20mg\%, C4 23mg\% and CH50 100 < 21 mg%. She was hypocomplementemic; thus, the differential diagnosis is limited to postinfectious glomerulonephritis, cryo-globulinemia, systemic lupus erythematosus and mem-branoproliferative glomerulonephritis.

Antinuclear antibody, rheumatoid factor and cryo-globulins were negative. The acute ASO titer was 620 Todd units and the convalescent titer was 833 Todd units. ASO titers are very important in establishing a diagnosis of post-streptococcal glomerulonephritis; in more than 90% of patients with pharyngeal infection, ASO titers above 200 units are attained within 3-5 weeks after the infection. Renal biopsy is not indicated in post-streptococcal glomerulonephritis. We did not perform one in this patient. However, renal biopsy should be done in post-streptococcal glomerulonephritis if the patient develops nephrotic syndrome, or if the patient becomes anuric or develops oliguria of at least 1 week’s duration. In these instances, renal biopsy is very important because 5 % of patients with post-streptococcal glomerulonephritis develop rapidly progressive glomerulonephritis. The nephrotic syndrome may be the presenting sign of a totally different renal lesion.

Membranoproliferative glomerulonephritis (type II) presents with a decreased C3, and a normal C4. Twenty percent of patients with membranoproliferative glomerulonephritis have an elevated ASO titer. Consequently, the diagnosis can be difficult when healing does not occur as expected in post-streptococcal glomerulonephritis and, thus, a renal biopsy is imperative.
Most of the epidemiologic studies of post-streptococcal glomerulonephritis agree that the immediate prognosis of this disease is very good. Less than 1% of patients die in the acute phase of the disease. However, the long-term prognosis is a subject of controversy. Baldwin et al. [3] followed patients for 11 years after the initial episode of post-streptococcal glomerulonephritis. These investigators report an increased incidence of hypertension and proteinuria, and suggest that this indicates progression of the disease. By contrast, the long-term studies, epidemics of post-streptococcal glomerulonephritis in Trinidad and in Maracaibo (Venezuela), report a favorable long-term outcome with very little tendency to late progression or chronicity. While the issue is not yet settled, Brenner’s [5] recent work provides a framework which may explain the progression to renal failure in some patients with post-streptococcal glomerulonephritis. In some instances the illness may be so severe that it damages many glomeruli. As a result, the perfusion pressure increases in the remaining healthy glomeruli. With time, the intraglomerular hypertension could lead to glomerulosclerosis; the glomerulosclerosis causes the late progression of the disease with the appearance of proteinuria, hypertension and renal failure.

References

Announcement
Fifth Annual Meeting of the International Society of Blood Purification
Stockholm, Sweden, June 22-24, 1987
Topics include hemodialysis, hemofiltration, hemoperfusion, apheresis, adsorption, peritoneal dialysis, liver support, biocompatible, immunomodulation, kinetic modelling, membrane characteristics, and toxin elimination.
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