

*Nephroquiz for the Beginner*  
(Section Editor: M. G. Zeier)

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## Acute renal failure in a single kidney with previous obstruction

### Case

A 68-year-old man was admitted to our unit in January 1996 because of acute renal failure. The patient had been a heavy smoker for more than 40 years. In 1986, he underwent bilateral ureterolysis because of hydronephrosis caused by retroperitoneal fibrosis. One year later an abdominal computerized tomography (CT) showed a non-functioning atrophic right kidney.

In 1995, the patient underwent cystectomy with bilateral percutaneous nephrostomy because of invasive transitional cell carcinoma. During the following 6 months he experienced two episodes of reversible anuria due to left nephrostomic obstructions. In November, his plasma creatinine level was 1.4 mg/dl. At the end of December 1995 he developed a sore throat and fever. A few days later he noted a progressive reduction in urine output. After unsuccessful antimicrobial therapy (ceftriaxone and erythromycin) he was admitted to our unit.

The patient complained of asthenia and dyspnea on admission. His body weight had increased by 10 kg due to overhydration, and his blood pressure was 160/90 mmHg with a pulse of 92 beats/min. His

temperature was 38°C. Physical examination revealed oedema of his legs, and pleuric and pericardial rubs. A chest X-ray and an echocardiography confirmed the presence of pericardial and bilateral pleuric effusions. Relevant abnormalities found in the results of the laboratory tests were haemoglobin 6.6 g/dl, serum creatinine 12 mg/dl and blood urea 186 mg/dl. Urine analysis showed proteinuria 3.8 g/l (urine volume 500 ml/24 h), and ~100 red blood cells and 20 white blood cells per high power field, together with granular and erythrocyte casts in the urinary sediment. Urine culture was positive for *Pseudomonas aeruginosa*. A pharyngeal culture was positive for *Staphylococcus aureus* and *Candida albicans*. Haemodialysis and antibiotic treatment were started.

### Questions

What is your diagnosis? Which further investigations would be helpful?

## Answer to the quiz on the preceding page

As a first hypothesis, this patient might have had a new episode of obstruction of the left urethral catheter, similar to those he suffered previously. Other possible causes of obstructive uropathy might also be considered, such as progression of the retroperitoneal fibrosis or metastasis of carcinoma in the retroperitoneal space [1]. However, an abdominal CT failed to demonstrate hydronephrosis or progression of the retroperitoneal fibrosis or bladder carcinoma metastases (Figure 1). It is well known that in a single kidney, hydronephrosis could be absent in the case of acute obstruction [2]. For this reason, despite negative imaging, urethral catheters were replaced but the patient remained oliguric. A vascular accident was excluded as the flow in the main kidney vessels was shown to be normal by Doppler ultrasonography.

Parenchymal disease of the left kidney had to be taken into account. The presence of proteinuria and active urinary sediment, and in particular the presence of erythrocyte casts, strongly suggested a glomerular disease [3], although erythrocyte casts may also be seen in acute interstitial nephritis [4]. The history of fever and sore throat 2 weeks before the diagnosis of renal insufficiency could suggest a post-infective glomerulonephritis, although serum complement levels were normal. Alternatively, because of the rapid deterioration of renal function, an extracapillary glomerulonephritis could be suspected. Finally, an acute interstitial nephritis could not be excluded even if cutaneous manifestations and eosinophilia were absent. As a matter of fact, the patient had been treated with cephalosporines that may induce interstitial nephritis [5]. In order to obtain more information, anti-neutrophil cytoplasmic antibody (ANCA) and anti-glomerular basement membrane (GBM) antibodies were tested. The ELISA assay result for ANCA was negative, while that for anti-GBM antibodies was positive. In view of the rare possibility of false positive results of

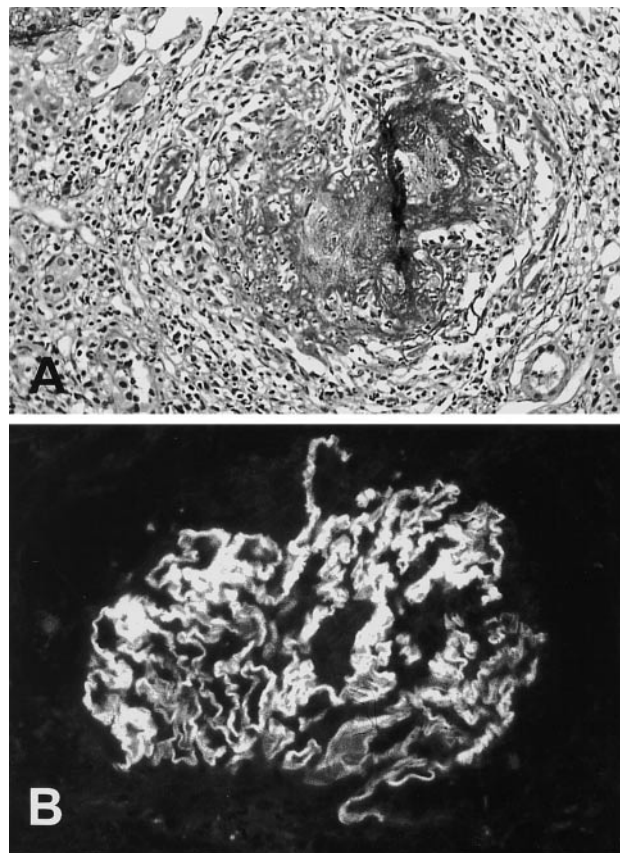
anti-GBM antibodies [6] and in order to evaluate whether the lesions could potentially be reversible before giving an aggressive treatment in such a frail patient, we decided to perform a renal biopsy.

Of the 16 glomeruli studied, eight were obsolescent. The remaining eight showed circumferential epithelial crescents containing some multinucleated giant cells, with disruption of the Bowman's capsule and conspicuous periglomerular inflammation with a granulomatous appearance. The glomerular tufts were collapsed and presented areas of fibrinoid necrosis and infiltration of polymorphonuclear leukocytes (Figure 2A). Diffuse and severe interstitial inflammation was also present. Immunofluorescence microscopy showed linear deposits of IgG (3+) and C3 (2+) along the GBM (Figure 2B), the Bowman's capsule, and some tubular basement membranes.

A diagnosis of rapidly progressive anti-GBM disease was made. The patient was treated initially with plasmapheresis and intravenous methylprednisolone pulses (0.5 g each) followed by oral prednisone (25 mg/day). Renal function did not recover. Considering the clinical conditions of the patient, the history of bladder neoplasia, the severe histological changes at



**Fig. 1.** CT scan (January 1996) showing a small right kidney (single arrow) and a normal left kidney (two arrows), and the absence of retroperitoneal masses or fibrosis at the level of the main left renal vessels.



**Fig. 2.** (A) Light microscopy showing total necrosis of the glomerular tuft, which has been infiltrated by polymorphonuclear leukocytes, and surrounded by a circumferential cellular crescent with disruption of the Bowman's capsule and periglomerular infiltration. (B) Immunofluorescence microscopy: linear deposits of IgG along the glomerular capillary walls.

renal biopsy and the oliguric state, which is associated with a poor prognosis [6], we decided not to insist on an aggressive treatment. The patient continued chronic haemodialysis.

In anti-GBM disease, the globular domain  $\alpha 3$  non-collagenous (NCI) of type IV collagen has been identified as the target antigen [7]. Our patient had a bladder cancer. Lymphomas and other cancers may be associated with anti-GBM disease [8–11] as a possible presentation of GBM antigens by the tumour invasion. However, although different types of glomerulonephritis can be associated with vesical tumours [12], no association with anti-GBM disease was reported. Moreover, as the existence of the 'Goodpasture's antigen' in the bladder basal membrane has not been documented [8], the association between the two diseases is unlikely. Our patient had a retroperitoneal fibrosis, which may be associated with other glomerulopathies [13], but not with anti-GBM disease. Weber *et al.* [14] reported one case of anti-GBM disease that developed 4 weeks after acute hydronephrosis and pyelonephritis. Our patient too had previous episodes of infection and hydronephrosis. As suggested by Weber *et al.*, it is possible that in urinary tract obstruction,  $\alpha 3$ (NCI) of collagen IV excreted in urine may enter the renal interstitium. The acidic pH associated with inflammatory infiltrate in the renal interstitium can dissociate  $\alpha 3$ (NCI) hexamer with exposition of the Goodpasture's epitopes. This could stimulate the autoantibody production in genetically predisposed individuals.

Whatever the pathogenesis, it is important to stress that an acute deterioration of renal function associated with proteinuria and a 'nephritic sediment' should alert one to the possibility of an underlying glomerular disease, even in patients with a history of obstructive nephropathy.

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