Teaching Point
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Acute renal failure in a patient suffering from chronic alcoholism

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Introduction

Urinary tract infections (UTI) are common in adults and are known to cause deterioration in renal function in patients with chronic renal impairment, solitary kidneys and renal transplants. Acute non-obstructive pyelonephritis is rarely considered in the differential diagnosis of acute renal failure (ARF), especially in patients with little or no evidence of previous kidney disease. Diagnosis is particularly difficult if the infection is asymptomatic. Chronic alcohol abuse increases the risk of ARF in unobstructed acute pyelonephritis [1] and is a rare cause of renal papillary necrosis [2,3].

We present an unusual case of asymptomatic acute pyelonephritis in combination with renal papillary necrosis in an otherwise well individual with a history of substantial alcohol abuse.

Case

A 62-year-old man was admitted to another hospital with an 8 week history of increasing dyspnoea and lower limb oedema. He had no urinary symptoms. Past medical history revealed varicose veins surgery a year before this admission with normal biochemistry performed at this stage. He smoked 20 cigarettes a day and drank 4–8 units of alcohol per week. However, he admitted to a much higher intake of alcohol in the past. On examination he was well, afebrile and had bilateral pitting oedema up to the knees. Investigations taken on admission were as follows: Hb 9.1 g/dl, WCC 16.0 × 10^9/l with neutrophilia and no eosinophilia; platelets 448 × 10^9/l; Na 128 mmol/l; K 4.8 mmol/l; urea 43.4 mmol/l; creatinine 730 μmol/l; HCO_3^- 20.2 mmol/l; calcium 2.54 mmol/l; phosphorus 1.82 mmol/l; bilirubin 18 μmol/l; ALT 26 IU/l; ALP 853 IU/l; albumin 28 g/l; CRP 85 mg/l; ferritin 6760 mg/l; ANCA IF positive. An ultrasound scan of the renal tract showed normal sized kidneys bilaterally with no evidence of renal artery stenosis on Doppler. Urine dipstick was positive for leucocytes and nitrites, protein trace and blood trace. The urine protein/creatinine index was high at 1448 (normal range 0–120). Urine culture grew *Escherichia coli* and enterococci, both sensitive to trimethoprim and amoxicillin. At this stage he was commenced on the latter and was transferred to our unit. A renal biopsy was performed. He was treated with oral prednisolone 60 mg and oral cyclophosphamide 1.5 mg/kg for 1 day until the result of the biopsy was available. This showed diffuse interstitial oedema together with an acute inflammatory cell infiltrate within the interstitium. There was infiltration of the tubular epithelium by neutrophils and tubules filled with pus in places (Figure 1). The immunosuppressive treatment was stopped and treatment with antibiotics was continued. Other investigations did not help in clarifying the diagnosis: immunology including ANA, complements, ELISA for MPO and PR3 and cryoglobulin were all negative, ACE normal, serum protein electrophoresis showed slight acute phase response, negative urine for Bence–Jones protein.

Five days later his creatinine was unchanged. He was well in himself with no clinical signs to correlate with the histological picture. An infective aetiology was thought to be unlikely as the sole or predominant reason for his underlying renal failure. Thus, a trial of methylprednisolone 0.5 g i.v. for 3 days was given followed by prednisolone 60 mg orally in combination with the antibiotic therapy. This failed to improve the renal function and a second renal biopsy was performed 15 days after transfer. This showed features...
E. coli growth of purulent material. On culture this showed a heavy probable papillary necrosis. A nephrostomy revealed showed mild hydronephrosis on the right side with Gram-negative bacilli were isolated from the blood glycaemic. Urinary catheterization revealed frank pus. But signs consistent with septic shock. He was hypo-

amounts of alcohol. On examination he was afebrile, his medications and that he was drinking substantial History from relatives revealed non-compliance with his medications and that he was drinking substantial amounts of alcohol. On examination he was afebrile, but signs consistent with septic shock. He was hypoglycaemic. Urinary catheterization revealed frank pus. Gram-negative bacilli were isolated from the blood culture and his urine grew E.coli. Ultrasonography showed mild hydronephrosis on the right side with probable papillary necrosis. A nephrostomy revealed purulent material. On culture this showed a heavy growth of E.coli. He was treated with intravenous cefuroxime and gentamicin and dialysis was recommenced. During his admission he became drowsy and had myoclonic jerks. Both CT scan brain and lumbar puncture were normal. He was transferred to the ICU for a day where he was treated for alcohol withdrawal with haloperidol and improved over the next few days. A ureteric stent was inserted on the right side and he was discharged on long-term cephradine. He remained dialysis-dependent.

He was readmitted 4 weeks later with sepsis and mild hydronephrosis of the left pelviccalyceal system. Cystoscopy and retrograde ureterogram revealed a soft-filling defect in the mid-ureter on the left side. A stent was inserted and a purulent discharge was noted. Stains and cultures for acid-fast bacilli were negative. The diagnosis was bilateral ureteric obstruction secondary to sloughed papillae which were complicated by infection. The decision was taken to keep the stents in long-term but bilateral nephrectomy was considered in view of persisting sloughing and recurrent sepsis. Unfortunately, he developed a pneumonia and was too unwell for any surgical intervention. He decided to discontinue any further dialysis and passed away 4 days later. A post-mortem was performed which revealed inspissated pus-like material within the pelvicalyceal system.

Discussion

Acute pyelonephritis is not a common cause of ARF especially in patients with previously normal kidneys. There are four main mechanisms by which UTI can lead to ARF [1]: (i) UTI and obstructive uropathy, (ii) septicaemia, (iii) antibacterial drug therapy and (iv) bacterial tubulointerstitial nephritis (pyelonephritis). The first three mechanisms are well recognized, but it is not clear how frequently ARF complicates non-obstructive pyelonephritis especially in patients with previously normal kidneys. In 1979, Baker et al. [4] described five cases over a period of 7 years giving an incidence of 2–3% of patients with ARF. The first cases were described in 1978 by Richet and Mayaud [5]. They described 30 cases of ARF with biopsy-proven acute tubulointerstitial nephritis. Twenty-one of these cases were attributed to an infective origin based on an acute polymorphonuclear cell infiltrate and all had clinical evidence of sepsis. The outcome of these cases was as follows: seven died, four recovered, 10 had recurrences associated with deteriorating renal function.

The asymptomatic nature of the underlying UTI added to the diagnostic dilemma and led to a considerable delay in reaching a definite diagnosis. In 1992, Jones [6] reviewed the cases of ARF caused by acute pyelonephritis that have been reported in the last quarter century. Patients with acute papillary necrosis were excluded. Twelve cases were included in this series. The predisposing factors included solitary kidney, urethral catheterization, NSAID administration and chronic alcoholism, the latter being present in our patient. The underlying UTI was clinically silent in 50% of these patients. Asymptomatic acute pyelonephritis causing ARF has also been described in the elderly [7].

Chronic alcohol abuse has been described as one of the risk factors for ARF to occur in unobstructed acute pyelonephritis [1]. In 1982, Bailey and Mailing [8] described three female patients with ARF and non-obstructive pyelonephritis. All three were chronic alcoholics. Alcohol abuse is also associated with papillary necrosis [2]. It has been previously debated whether pyelonephritis leads to papillary necrosis or vice versa. Edmondson et al. [2] reported papillary necrosis in 20% of alcoholics with pyelonephritis. Eknovan et al. [9] noted that over half of the 27 cases of papillary necrosis had more than one possible aetiological factor and concluded that papillary

![Fig. 1. Low-power view showing interstitial oedema, infiltration of tubular epithelium by neutrophils and tubules filled with pus.](http://ndt.oxfordjournals.org/).
necrosis is often multifactorial and uncommon when only one pathogenic factor is present [9]. In our case, the combination of alcohol and infection most probably predisposed to the development of papillary necrosis.

**Teaching points**

(i) Acute bacterial tubulointerstitial nephritis (pyelonephritis) should be included in the differential diagnosis of ARF even if there are no symptoms of UTI.

(ii) Chronic alcohol abuse may be associated with UTI and/or papillary necrosis, thus predisposing to an increased risk of ARF.

**References**


