

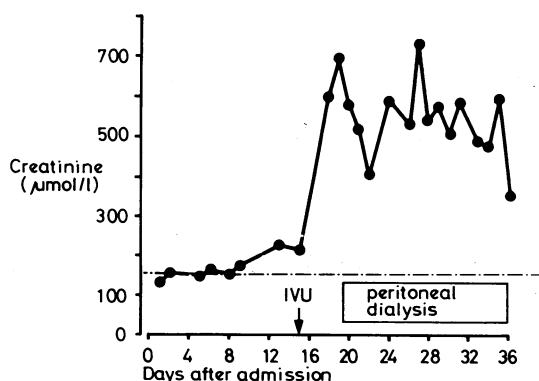
Acute renal failure precipitated by radiographic contrast medium in a patient with rhabdomyolysis

Myoglobinuria is an uncommon but important cause of acute tubular necrosis.¹ It was originally described in association with trauma but also occurs in cases of non-traumatic rhabdomyolysis, when diagnosis may be more difficult.²⁻³ Infusion intravenous urography is a standard investigation for acute renal impairment of obscure cause¹; we report a case of acute renal failure precipitated by administration of radiographic contrast medium in a patient with undiagnosed rhabdomyolysis and renal impairment.

Case report

A 57-year-old man presented with malaise and fever. His history was not helpful, but he mentioned that his urine was the colour of "oxtail soup." Biochemical data were: sodium 134 mmol(mEq)/l, potassium 4.3 mmol(mEq)/l, urea 9.3 mmol/l (55.9 mg/100 ml), creatinine 147 μ mol/l (1.7 mg/100 ml), calcium 1.93 mmol/l (7.7 mg/100 ml), phosphate 1.71 mmol/l (5.3 mg/100 ml), aspartate transaminase 690 U/l (normal 13-35), lactate dehydrogenase 1670 U/l (normal 240-525), γ -glutamyltransferase 45 U/l (normal <50), bilirubin 12 μ mol/l (0.7 mg/100 ml), uric acid 518 μ mol/l (8.7 mg/100 ml). Creatinine clearance was 40 ml/min. Haemoglobin concentration was 13 g/dl and white cell count 22×10^9 /l (80% polymorphs). A midstream specimen of urine showed no casts or red cells, although the results of ward testing had been strongly positive for blood. Urine was not tested for myoglobin.

Plasma creatinine concentration rose slowly from 147 to 221 μ mol/l (1.7 to 2.5 mg/100 ml) by day 14 (figure), when renal biopsy was performed after drip infusion pyelography using 250 ml of 30% Urografin. As the nephro-



Plasma creatinine concentrations before and after intravenous urography (IVU).

Conversion: SI to traditional units—1 μ mol/l = 11.3 μ g/100 ml.

graphic phase was poor a further 150 ml of 30% Urografin was infused, after which the kidney was visualised. The patient was not dehydrated before the procedure. Urine output in the 12 hours preceding the biopsy was 75 ml/h, but in the next 12 hours only 250 ml was passed. He was anuric thereafter. There was no response to an infusion of 500 ml physiological saline, 20 g mannitol, and 500 mg frusemide, and he was therefore transferred for haemodialysis. Azathioprine and prednisolone were started after polyarteritis nodosa was provisionally diagnosed. His muscles were noted to be tender, and aspartate transaminase activity remained raised.

Haemodialysis was started, but two days later he developed *Escherichia coli* septicaemia, bronchopneumonia, and respiratory failure. In the interim the renal biopsy specimen was reported as showing mild and patchy acute tubular necrosis with slight interstitial oedema. Glomeruli and blood vessels were normal. Acute tubular necrosis secondary to myoglobinuria was then suspected, and creatine phosphokinase and aldolase activities were measured in stored blood samples. Creatine phosphokinase activity was found to have been 7500 U/l (normal 0-75) and aldolase 70 U/l (normal 0.5-3.1) one week after the initial admission. Myoglobin was not detected in the serum. A muscle biopsy specimen showed an acute necrotising myopathy. Despite ventilation and appropriate antibiotic treatment respiratory failure progressed, and he died three weeks after the onset of renal failure.

At necropsy the kidneys were slightly enlarged by interstitial oedema and pigment casts were present in some distal convoluted tubules and collecting tubules. There was evidence of fairly extensive acute tubular necrosis with many dilated tubules lined by flattened regenerating epithelium. In the lungs there was a diffuse interstitial pneumonia, and cells containing cytomegalovirus inclusion bodies were present in the alveolar spaces.

Comment

The combination of dark brown urine, positive for blood on a reagent strip but without red cells, high creatinine phosphokinase activity, and necrotising myopathy suggest myoglobinuria as the cause of initial tubular damage and renal impairment. The abrupt onset of anuria after infusion intravenous urography, coinciding with a rise in plasma creatinine concentration (figure), indicates that the contrast medium precipitated the acute renal failure, as myoglobinuria had probably been present for four weeks. The myoglobin and the contrast medium probably acted synergistically to produce severe acute tubular necrosis, because each alone may cause it.¹ Indeed, some tubular damage was seen in the biopsy specimen. This association has not been documented, and it may be prudent to exclude myoglobinuria before performing infusion urography in patients with acute renal failure of obscure cause. The kidney might perhaps be protected from the effects of myoglobinaemia by infusion of mannitol and sodium bicarbonate.⁴

We thank Dr M Esiri, who examined the muscle biopsy specimen.

¹ Kerr DNS. Acute renal failure. In: Black D, Jones NF, eds. *Renal disease*. 4th ed. Oxford: Blackwell Scientific Publications, 1979:437-93.

² Koffler A, Friedler RM, Massry SG. Acute renal failure due to non-traumatic rhabdomyolysis. *Ann Int Med* 1976;85:23-8.

³ Grossman RA, Hamilton RW, Morse BM, Penn AS, Goldberg M. Non-traumatic rhabdomyolysis and acute renal failure. *N Engl J Med* 1974; 291:807-11.

⁴ Eneas JF, Schoenfeld PY, Humphreys MH. The effect of infusion of mannitol—sodium bicarbonate on the clinical course of myoglobinuria. *Arch Intern Med* 1979;139:801-5.

(Accepted 4 November 1980)

Renal Unit, Churchill Hospital, Oxford OX3 7LJ

C G WINEARLS, MRCP, DPHIL, registrar in medicine

J G G LEDINGHAM, DM, FRCP, May reader in medicine

Department of Histopathology, John Radcliffe Hospital, Oxford OX3 9DU

A J DIXON, BM, BCH, registrar in pathology

Reporting of blood pressure data in medical journals

The technique of blood pressure measurement is all too often taken for granted.¹ As the benefits of treating milder forms of hypertension become apparent²⁻⁴ there is an increasing tendency to diagnose and treat it at lower levels than before. To avoid misdiagnosis and over treatment it is imperative that the methodology of blood pressure measurement be standardised or at least carefully described in reports of studies where blood pressure levels are of central interest. Therefore it might reasonably be assumed that the method on which research conclusions in scientific papers are based would be carefully examined by editors and referees of medical journals. We aimed at discovering whether the criteria usually applied to scientific methods were in fact being applied to the reporting of blood pressure measurements.

Methods and results

We selected four prestigious general medical journals for study—the *British Medical Journal*, the *Lancet*, the *New England Journal of Medicine*, and the *Journal of the American Medical Association*. The articles were found through the subject index using the words or phrases "blood pressure," "hypertension," "hypotension," and "antihypertensive agent" or "drug." Correspondence, editorials, leading articles, commentaries, and book reviews were excluded as being unlikely to contain relevant data. Thus most were original articles, short papers, or progress reports. We compared data for 1969 and 1979 to assess possible differences that might have emerged with the recent increased awareness of the importance of measurement techniques. For 1969 there were 36 such papers and for 1979 there were 60. Out of this total of 96 articles 85 bore the words blood pressure (15) or hypertension (70).

Each paper was read so as to identify the type of sphygmomanometer used, whether the diastolic pressure was taken as phase 4 or 5, the position of the patient during measurement, the number of readings taken, the bladder size, the cuff size, details of the personnel (nurse, doctor, patient) who took the blood pressure, clinical setting (hospital, clinic, etc), the presence of obesity or arrhythmias, the limb used, and the time of day.