

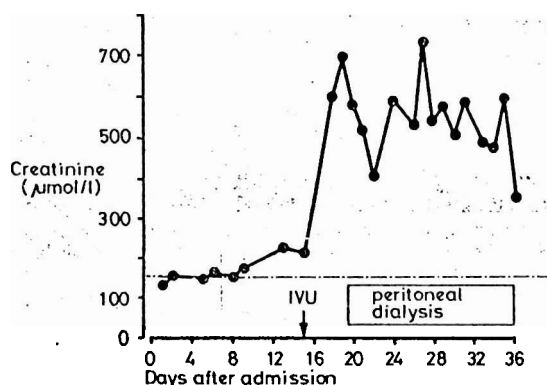
Acute renal failure precipitated by radiographic contrast medium in a patient with rhabdomyolysis

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.²⁻⁴ 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.

The following details were not stated: the type of sphygmomanometer (61 papers), the diastolic end-point (62), the position of the subject (49), and the number of measurements per assessment (70). Virtually no information was given for the remaining data. We could find no obvious difference between reporting in 1969 and 1979, but the number of studies (36) for 1969 was relatively small.

Comment

The important finding of this study is that in two-thirds of the papers relating to blood pressure published in four prestigious general medical journals one or more of the following were not mentioned: type of equipment used, the diastolic end-point chosen, or the number of readings per measurement. Other pertinent information was also not given. The implications are clear. Firstly, in the interests of scientific accuracy and comparability editors and referees must apply to blood pressure measurement reporting the same critical standards given to other measurement methods. Secondly, if the prevailing carefree attitude to blood pressure measurement is to be corrected to enable more accurate diagnosis and treatment of hypertension the incentive and example for a general reappraisal of measurement teaching must come from those who profess a special interest and skill in the subject.

¹ O'Brien ET, O'Malley K. The ABC of blood pressure measurement: technique. *Br Med J* 1979;ii:982-4.

² Anonymous. Mild hypertension. *Br Med J* 1980;280:1062.

³ Anonymous. The treatment of mild hypertension. *Lancet* 1980;i:1283-4.

⁴ Hypertension Detection and Follow-up Program Cooperative Group. Five year findings of the hypertension detection and follow-up program. 1. Reduction in mortality of persons with high blood pressure, including mild hypertension. *JAMA* 1979;242:2562-71.

(Accepted 17 October 1980)

Blood Pressure Clinic, the Charitable Infirmary, Jervis Street, Dublin 1, and Department of Clinical Pharmacology, Royal College of Surgeons in Ireland, St Stephen's Green, Dublin 2

ANTONIA LEHANE, medical student

E T O'BRIEN, MRCP, FRCPI, consultant physician, cardiology

K O'MALLEY, MD, FRCPI, professor of clinical pharmacology

Coeliac plexus block for control of pain in chronic pancreatitis

Disruption of the nerve supply to the pancreas has not been widely used to control pancreatic pain, despite several studies¹ showing that operative splanchnicectomy, with or without coeliac ganglionectomy, results in satisfactory control of pain in most patients. Denervating the pancreas by injecting alcohol into the region of the coeliac ganglion has been useful for pain associated with pancreatic cancer,^{2,3} but long-term studies in chronic pancreatitis have not been reported. We reviewed 16 consecutive patients with chronic pancreatitis treated by coeliac plexus block; the results indicate that satisfactory control of pain may be achieved by this technique.

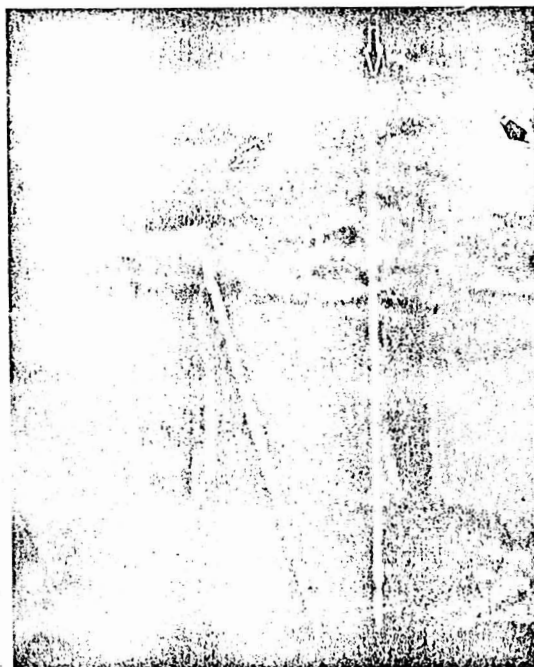
Patients, methods, and results

Coeliac plexus block was performed in 16 patients (14 men, two women; mean age 49 years) with advanced chronic pancreatitis associated with disabling abdominal pain. Pain had begun two to 25 years (mean six years) before the procedure, and, at the time of nerve block, 12 of the 16 patients required regular injections of narcotic analgesics. Four patients were managed with high doses of simple analgesics and narcotic analgesic supplements. Pancreatitis was attributed to alcohol abuse in 12 patients, of whom seven had calcification on plain abdominal x-ray examination. Three patients had previously had gall stone disease. Advanced chronic pancreatitis had been confirmed at laparotomy in six patients and by retrograde pancreatography in 12. Fourteen patients were alive for review in December 1979. These patients completed a questionnaire and subsequently attended for interview to clarify their answers. A mean of 3.5 years (range nine months to seven years) had elapsed since the coeliac plexus block. The efficacy of coeliac plexus block in the two patients who had died was evaluated from the medical record.

The technique of coeliac plexus block has been described previously.⁴ A recent modification involves injecting radiological contrast material (Hypaque 60%) before using alcohol to ensure that the points of both

needles are positioned correctly (figure) with the point of the needle on the right side being more cephalad than that on the left.⁴

Thirteen of the 16 patients (81%) had experienced a substantial reduction in the severity of pain within two weeks of nerve block. Pain relief had lasted for less than six months, however, in three of these patients. Ten of 16 patients (63%) showed long-term improvement: three are completely free of pain and seven had only mild pain, which is readily controlled by non-narcotic analgesics. Five of the six patients with continuing pain underwent a second coeliac plexus block and two subsequently improved. Two patients with ineffective blocks underwent pancreatic resection and subsequently died. Postural hypotension was common for one to two weeks after coeliac plexus block but thereafter resolved spontaneously. The only long-term complication was persistent nerve root pain in one patient, possibly related to the tracking of alcohol along the needle track to the L1-L2 nerve root.



Lateral x-ray film showing needles positioned across body of L1. The needle on the right (long arrow) is more cephalad than that on the left, and the position of alcohol in the retroperitoneal space (short arrows) has been outlined by a second injection of radiological contrast.

Discussion

The degree of pain control achieved by coeliac plexus block may be similar to that achieved by pancreatic surgery.⁵ Furthermore, the procedure is fairly simple and safe and does not appear to increase the degree of difficulty of subsequent pancreatic resection. Severe pain recurred within six months of nerve block in three patients, but recurrence of severe symptoms at a later stage was not observed. These findings suggest that coeliac plexus block (carefully performed under radiological control) has a place in the management of pain associated with chronic pancreatitis and should be considered before pancreatic surgery.

¹ Vosschulte K, Wagner E. Splanchnicectomy in chronic pancreatitis. Application and effect. *Min Med* 1970;53:1053-9.

² Thompson GE, Moore DC, Bridenbaugh LD, Artin RY. Abdominal pain and alcohol coeliac plexus nerve block. *Anesth Analg (Cleve)* 1977; 56:1-5.

³ Kune GA, Cole R, Bell S. Observations on the relief of pancreatic pain. *Med J Aust* 1975;2:789-91.

⁴ Ward EM, Rorie DK, Nauss LA, Bahn RC. The coeliac ganglia in man: normal anatomic variations. *Anesth Analg (Cleve)* 1979;58:461-5.

⁵ White TT, Slavotinek AH. Results of surgical treatment of chronic pancreatitis. *Ann Surg* 1979;189:217-24.

(Accepted 22 October 1980)

Royal Melbourne Hospital, Victoria, 3050 Australia

SIMON N BELL, MD, FRACS, surgical registrar

RUSSELL COLE, FRACS, FFARCS, anaesthetist

IAN C ROBERTS-THOMSON, MD, FRACP, assistant gastroenterologist