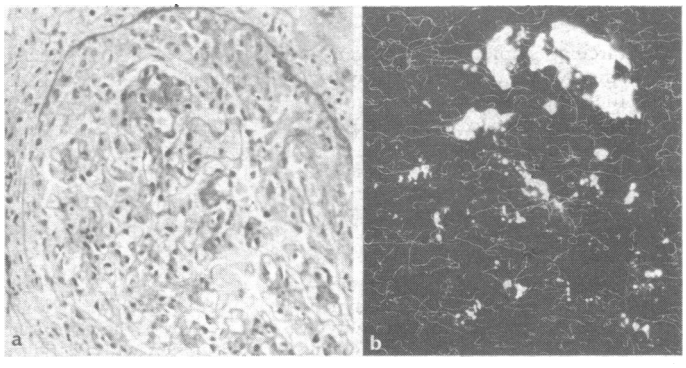
Extracapillary proliferative glomerulonephritis in Russell's viper bite

Renal changes in snake bite include tubular necrosis, cortical necrosis, interstitial nephritis, arteritis, and thrombophlebitis.[1-3] Glomerular changes are usually mild,[1] and severe glomerulonephritis is uncommon.[4] We report the case of a patient bitten by a Russell's viper who presented with acute renal failure. A renal biopsy specimen showed extracapillary proliferative glomerulonephritis in addition to tubular necrosis.

Case report

The patient, a 33-year-old farmer, was bitten on the left foot by a Russell's viper. When brought to a local hospital within two hours he had gross haematuria and oliguria. Except for some pain at the site of the bite there were no abnormal physical signs. Only conservative treatment was instituted, and antivenom was given. One day after the bite his blood urea concentration was 11·1 mmol/l (66·6 mg/100 ml) and serum creatinine concentration 0·2 mmol/l (2·26 mg/100 ml). He was therefore transferred to our unit for the management of renal failure. Laboratory investigation two days after the bite showed haemoglobin 11 g/dl, blood urea 31·8 mmol/l (191 mg/100 ml), serum creatinine 0·3 mmol/l (3·4 mg/100 ml), normal serum electrolytes, factor V 58%, factor VIII 62%, plasma fibrinogen 120 mg/dl, platelets 80×10^9/l (80 000/mm^3), coagulation time over 30 min, serum C3 70 mg/dl, plasma haemoglobin 2·2 mg/dl, and serum fibrin degradation products 11·6 mg/dl. A peripheral blood smear showed burred cells consistent with disseminated intravascular coagulation. The urine contained numerous red blood cells and 2+ protein. The patient remained very oliguric, and after six days his blood urea concentration rose to 57·1 mmol/l (343 mg/100 ml) and serum creatinine to 0·6 mmol/l (6·8 mg/100 ml). Because of uraemic symptoms he underwent peritoneal dialysis on the seventh and 16th hospital days. Diuresis occurred in the third week with gradual increase in the urine flow. Coagulation factors and platelets returned to normal 12 days after admission. His serum creatinine concentration was 0·13 mmol/l (1·46 mg/100 ml) 30 days after admission. A renal biopsy specimen obtained two weeks after admission showed focal tubular degeneration, arteritis, and extracapillary proliferative glomerulonephritis in half of the glomeruli (fig (a)). The other glomeruli showed only mesangial proliferation. Neither immunoglobulins nor C3 could be detected in the renal lesion but immunofluorescence showed fibrin deposition in the glomeruli (fig (b)).



Extracapillary proliferative glomerulonephritis in Russell's viper bite. (a) Glomerulus with proliferation of epithelial cells (haematoxylin-eosin, original magnification ×252). (b) Glomerular fibrin deposition (fluorescent antifibrin, original magnification ×252).

Comment

Renal failure due to acute tubular necrosis or cortical necrosis is common in a patient bitten by Russell's viper. Multiple factors including direct tubulotoxicity, intravascular haemolysis, hypovolaemia, and disseminated intravascular coagulation are responsible for its development.[1] Glomerular changes are usually benign, and an immunological mechanism has been implicated in their pathogenesis.[3] In our patient glomerulonephritis was severe, there was no evidence of any immunological process, and hence the glomerular changes were presumably due to the direct effect of venom, the mesangial proliferation being a non-specific pathological reaction. Since Russell's viper's venom is vasculotoxic[2] it could cause rupture of the glomerular basement membrane with fibrin deposition and secondary epithelial proliferation. This is an uncommon finding which might result from a large amount of envenomation. Nevertheless, it expands the range of renal lesions in snake bite and represents a non-immunological cause of glomerulonephritis.

[1] Sitprija V, Boonpucknavig V. Snake venoms and nephrotoxicity. In: Lee CY, ed. *Snake venoms*. Berlin: Springer, 1979:997-1018.
[2] Sitprija V, Benyajati C, Boonpucknavig V. Further observations of renal insufficiency in snakebite. *Nephron* 1974;**13**:396-403.
[3] Sitprija V, Boonpucknavig V. The kidney in tropical snakebite. *Clin Nephrol* 1977;**8**:377-83.
[4] Seedat YK, Reddy J, Edington DA. Acute renal failure due to proliferative nephritis from snake bite poisoning. *Nephron* 1974;**13**:455-63.

(*Accepted 4 March 1980*)

Department of Medicine, Chulalongkorn Hospital Medical School, Bangkok, Thailand
VISITH SITPRIJA, MD, PHD, professor of medicine
Department of Pathology, Ramathibodi Hospital, Bangkok, Thailand
VIJITR BOONPUCKNAVIG, MD, associate professor of pathology

High-pressure water jet injury

Complex high-pressure water jet guns have been developed for underwater cutting and cleaning. They operate at pressures up to 100 MPa (1000 bar; 14 500 lbs/in^2; 1019 kg/cm^2) with a jet velocity of 900 miles (559 km) per hour. The jet therefore causes injury like a high velocity missile. The entrance wound tends to be small and give little indication of the damage to the deeper tissues. For a variable extent along the track of the jet the surrounding tissues will be damaged if not killed by the shock waves developed as the kinetic energy of the jet is dispersed. Deeper tissues will be lacerated depending on the depth of penetration and the anatomy of the area.

Case report

The patient, a 32-year-old experienced diver, was lowered to 150 feet (46 metres) under the sea to clean by high-pressure jet one of the "sea legs" of an oil rig. The jet gun had been left at the site by a previous diver. The water was murky and he could not see the gun. He picked up the hose and began to withdraw it when the gun fired for a moment causing him to drop the hose. He felt he had received a blow on the abdomen but did not realise quite what had happened. He groped for the gun and used it for a minute or two. The pain in his abdomen became more severe and he asked to be taken up. He was decompressed routinely with a pause of three minutes at 50 feet (15 metres). The time from the start of the descent to the return to the surface was 14 minutes. He was wearing an inner tight-fitting Neoprene suite about 3 mm thick and an outer loose-fitting, 7-mm thick Neoprene suit with a tough "anti-scuff" surface. The jet had drilled a neat hole through both suits and entered the abdominal wall. He was transferred by helicopter to hospital.

On admission three hours later the patient's general condition was quite good. His blood pressure was 140/75 mm Hg, pulse 74 and regular. There was a small puncture wound about 0·25 cm in diameter on the anterior abdominal wall 6 cm below the umbilicus and 3 cm to the right of the midline. There was tenderness and guarding about the wound but the left side of the abdomen was soft. Bowel sounds could be heard but were diminished. A chest radiograph was normal and *x*-ray examination of his abdomen showed no evidence of free intraperitoneal gas and no abnormal soft tissue shadows. Because of the mechanism of the injury laparotomy was done through a right paramedian incision excising the entrance wound. The wound enlarged as it went deeper. There was a hole 2 cm in diameter in the anterior rectus sheath. The underlying rectus muscle had been torn, leaving a gap of about 4 cm, and there was a hole 3 cm in diameter in the peritoneum. There was quite extensive oedema of the subcutaneous tissue. The gut was not injured but there was an area 1·5 cm in diameter of bruising of the mesentery and a 1-cm tear in the omentum. The posterior peritoneum was intact. Some blood and, presumably, sea-water in the peritoneal cavity was sucked out. The patient had an uneventful convalescence and went home after eight days.

Comment

Nearly all the reported cases of high-pressure jet injury have been from jets of oil, grease, or paint and usually to the hand. Neill and George[1] described multiple perforations of the ileum and caecum caused by high-pressure water jet and Gardner[2] reported a case in

which a high-pressure water jet struck the abdominal wall at a tangent causing a wound 10-cm long together with surgical emphysema. Recovery was uneventful. My patient's heavy protective clothing decreased the velocity of the jet and dispersed the reduced kinetic energy in the abdominal wall, so that it caused no internal injuries. Since the jet was fired only momentarily the mass of water was minimal.

[1] Neill RWK, George B. Penetrating intra-abdominal injury caused by high-pressure water jet. *Br Med J* 1969;ii:357-8.
[2] Ward Gardner A. High-pressure water injury. *Trans Soc Occup Med* 1966;**16**:30.

(*Accepted 4 March 1980*)

Dr Gray's Hospital, Elgin, Morayshire
J L M DE BEAUX, MB, FRCS, consultant surgeon

Inadequate information about drug overdosage in manufacturers' data sheets

Pharmaceutical companies are required[1] to supply doctors with data sheets giving information about their drugs, including the likely effects and treatment of overdosage. We reviewed the information about overdosage in the current *Data Sheet Compendium*.[2]

Analysis of data sheets

Out of the 1035 data sheets about oral drugs, only 736 (71%) contain information about overdosage. Thirty-four of these give some information about the possible effects but no advice about treatment, and 207 (28%) advise about treatment without stating the clinical features of toxicity. Three hundred and forty-seven data sheets concern drugs for injection but 96 (28%) contain no information about overdosage. Four hundred and twenty-nine data sheets concern products for topical use. Most (72%) of these give no information about toxicity when taken by mouth, although some products contain potentially dangerous substances, such as salicylates, phenol, or malathion. Nevertheless, a few data sheets give useful information about the likely effects of swallowing ophthalmic ointment (for example, Vira-A, Parke Davis) or suppositories (Anusol, Warner), or even intrauterine injection of disinfectant (Cetavlon, ICI). Twenty manufacturers (16%) provide no information about overdosage of any of their products.

Unfortunately, the information about overdosage in many data sheets is incomplete, vague, misleading, or wrong, and in some the treatment recommended is frankly dangerous. There are 40 data sheets about products containing paracetamol but five give no information about overdosage and only 21 (52%) mention that liver damage may occur. Effective specific treatment for paracetamol poisoning has been available since 1974[3] but is recommended in only 15 data sheets, whereas 10 advise supportive or symptomatic treatment and three actually state that there is no specific antidote. One data sheet advises alkaline diuresis, which is ineffective and potentially dangerous. Many data sheets advise the use of "antidotes" but do not mention intensive supportive therapy. Noradrenaline or metaraminol are recommended for hypotension, but not sensible measures such as raising the foot of the bed, restoration of plasma volume, and correction of hypoxia. Obsolete drugs recommended for treatment include tannic acid, analeptics (picrotoxin, bemegride, leptazol, amphetamine), and paregoric. Emetics, "universal antidote," subcutaneous pilocarpine, and respiratory stimulants are advised for overdosage of the anti-emetic combination of dicyclomine and doxylamine, but supportive treatment is not discussed. Overdosage of cardiac glycosides may cause severe hyperkalaemia[4] but the data sheet about lanatoside C advises treatment with potassium chloride. Those for digoxin and medigoxin, however, suggest glucose and insulin for treating hypokalaemia. Fluid restriction is advised for overdosage of the diuretic bendrofluazide and correction of electrolytes with ammonium chloride, potassium chloride, and hypertonic saline. It is implied that artificial respiration may be needed for overdosage of hydrochlorothiazide. Overdosage of pentaerythritol tetranitrate may cause severe headache, which one manufacturer suggests should be treated with caffeine, neostigmine, or amphetamine.

Comment

Drug overdosage is common and accurate information about the likely consequences and necessary treatment is essential. The *Data Sheet Compendium* should be a useful source of up-to-date information, but unfortunately some data sheets are inadequate or dangerously wrong. They should contain accurate and relevant advice about the acute toxicity of all products, including topical preparations which are occasionally taken by mouth. It is helpful to know if a product is non-toxic so that unnecessary anxiety and treatment may be avoided. We know the difficulties of providing reliable information about overdosage: we look after poisoned patients ourselves and give advice through the poisons information services. Some drug manufacturers have been concerned and extremely helpful with our inquiries about overdosage of their products. Others, however, have been unwilling or unable to provide useful information, especially about animal toxicity studies of new drugs, and seem to believe that no problem exists. Pharmaceutical companies should supply doctors and the poisons information services with adequate information about their drugs.

[1] Statutory Instrument 1972 No 2076. *The medicines (data sheet) regulations 1972*. London: HMSO, 1972.
[2] Association of the British Pharmaceutical Industry. *Data sheet compendium 1979-80*. London: Pharmind Publications, 1979.
[3] Prescott LF. Prevention of hepatic necrosis following paracetamol overdosage. *Health Bull (Edinb)* 1978;**36**:204-12.
[4] Ekins BR, Watanabe AS. Acute digoxin poisonings: review of therapy. *Am J Hosp Pharm* 1978;**35**:268-77.

(*Accepted 21 February 1980*)

Accident and Emergency Department, General Infirmary, Leeds LS1 3EX
R N ILLINGWORTH, BM, MRCP, senior registrar

Regional Poisoning Treatment Centre and University Department of Therapeutics and Clinical Pharmacology, Royal Infirmary, Edinburgh EH3 9YW
L F PRESCOTT, MD, FRCPED, consultant physician and reader in clinical pharmacology

Late disruption of initially satisfactory stapled anastomoses

The circular staple gun has been successfully used for colorectal anastomosis[1] and devascularisation of oesophageal varices.[2] We have used the American EEA staple gun for 97 intestinal procedures, and we report here four cases of late dehiscence which occurred despite a complete staple ring being seen at radiology in the first week after operation.

Case reports

Case 1—A 73-year-old woman underwent an anterior resection for diverticular disease under gentamicin and metronidazole prophylaxis with a well-prepared bowel. Intestinal continuity was restored with the American EEA staple gun: two complete rings of intestine were present in the cartridge. On the sixth day after operation a radiograph showed that the staple line was complete. She was discharged on the ninth day after operation but had had a small rectal bleed 24 hours earlier. She was readmitted nine days later with peritonitis. A radiograph showed a break in the staple line, and a barium enema showed a large leak at this site. Dehiscence of the anastomosis with purulent peritonitis and a large pelvic abscess was found at a second laparotomy.

Case 2—A 68-year-old diabetic with a rectal carcinoma had an anterior resection under gentamicin and metronidazole prophylaxis with a satisfactorily prepared colon. Colorectal anastomosis was performed with the American EEA staple gun. Two complete rings of colon were present in the cartridge. On the eighth day after operation a radiograph showed a complete staple ring. Subsequently the patient developed a rectal haemorrhage and on the 12th day a radiograph showed a break in the staple ring and a water-soluble barium enema showed a small leak at this site.

Case 3—A 66-year-old woman with a rectal carcinoma had an anterior resection using the EEA staple gun under cefoxitin prophylaxis in a well-prepared colon. Two complete intestinal rings were present in the cartridge. A radiograph on the seventh day after operation confirmed a complete staple ring. A contrast study on the 10th day showed a complete staple ring and no anastomotic leak. Postoperative pyrexia persisted until the 17th day when pus discharged per rectum and a radiograph then showed a posterior disruption of the staple ring; pus was seen to discharge from the anastomosis at sigmoidoscopy.