Chemical burns beneath tourniquets

J C Dickinson, B N Bailey

Department of Plastic Surgery, Stoke Mandeville Hospital, Aylesbury, Buckinghamshire
J C Dickinson, FRCSED, *senior registrar in plastic surgery*
B N Bailey, FRCS, *consultant plastic surgeon*

Correspondence to: Mr Dickinson.

Pneumatic tourniquets are widely used in surgery of the arms and legs; many complications have been reported, but skin damage is said to be uncommon.[1] Its causes are pressure necrosis or shearing due to inadequate padding or poor application in patients with loose or thin skin. Attention has been drawn to the possibility of burns under the cuffs of pneumatic tourniquets in young children.[2] The consensus was "that the damage was caused by spirit solutions seeping beneath tourniquets and being held tightly against the rather delicate skin of children. The problem has not been known to arise when aqueous solutions are used as skin preparation." This view was supported by Evans, who reported full thickness skin loss after a swab impregnated with spirit was left beneath a tourniquet.[3] We report four cases to show that skin burns can be a sequel of incorrect technique.

Case reports

Three patients aged less than 2 with syndactyly sustained four burns beneath the cuffs of pneumatic tourniquets. The skin preparation used was a tincture of povidone-iodine with an alcohol content of 70%. In all cases the burns were seen when the tourniquet was removed; the preparation seemed to have run down the patient's arm and been absorbed by the padding under the tourniquet cuff. In two cases the area of iodine staining corresponded exactly with the area of burn. In the other two cases the burns were smaller than this area. The burns were all partial thickness and healed within four weeks, leaving minimal scarring.

The tourniquets were checked after each incident and found to be functioning satisfactorily. They had been applied in the usual way under the supervision of the operating surgeon, as in many previous, similar cases.

Comment

These four burns show that a burn can be caused by 70% alcohol held in contact with young skin under pressure for 60-90 minutes. Skin preparations with a high alcohol content left in contact with the skin distal to the tourniquet have not caused burns in our experience. Preparations with a high alcohol content are used both for the bacteriocidal effect of the alcohol and as degreasing agents to allow better penetration of the povidone-iodine. The flammable nature of spirit preparations is well recognised, and precautions must be taken. We and our colleagues have not seen burns when other types of preparation (including aqueous iodine) have been used.

If alcohol preparations are used they must not be allowed to run down the arm and under the tourniquet. If this should happen the tourniquet should be re-applied with fresh padding. All theatre staff should be aware of the danger.

1 Palmer AK. Complications from tourniquet use. *Hand Clin* 1986;2:301-5.
2 Anonymous. Tourniquets and theatre safe guards. *Journal of the Medical Defence Union* 1985;1:2.
3 Evans DM. Tourniquets. *Journal of the Medical Defence Union* 1985;1:22.

(Accepted 7 November 1988)

Sweet's syndrome in Crohn's disease

Daniel Kemmett, David J Gawkrodger, Graeme Wilson, John A A Hunter

University Department of Dermatology, Royal Infirmary, Edinburgh
Daniel Kemmett, MRCP, *registrar*
David J Gawkrodger, MRCP, *lecturer*
John A A Hunter, FRCP, *professor*

University Department of Medicine, Western General Hospital, Edinburgh
Graeme Wilson, FRCS, *surgical registrar*

Correspondence to: Dr Kemmett.

We report on two women who developed Sweet's syndrome during exacerbations of Crohn's disease of the colon. We do not think that this association has been reported previously.

Case reports

CASE 1

A 52 year old woman presented with diarrhoea streaked with blood and epigastric pain. Her rectal mucosa was inflamed and bled easily on sigmoidoscopy. Rectal biopsy showed non-specific proctitis, and a barium enema showed changes limited to the distal colon. She subsequently developed an anal fistula, which was excised, and histological examination showed submucosal granulomas and foreign body giant cells suggestive of Crohn's disease. A further fistula was excised, and two weeks later she developed a fever and erythematous pustular lesions 2 cm in diameter over her forearms, thighs, neck, and trunk.

Her white cell count was $11{\cdot}6\times10^9$/l (88% neutrophils, 10% lymphocytes, and 2% monocytes), haemoglobin concentration 86 g/l, and erythrocyte sedimentation rate (Westergen) 86 mm in the first hour. Biopsy of a representative lesion from her neck showed the typical histological features of Sweet's syndrome with an infiltrate predominantly of polymorphonuclear cells with nuclear fragmentation in the upper and mid dermis. Prednisolone 30 mg daily was started. Her temperature returned to normal within 24 hours, and the skin eruption cleared within 10 days. One month later a proctocolectomy was performed for her colonic Crohn's disease. The dose of prednisolone was reduced and then stopped within three months. She remained well at one year with no recurrence of Sweet's syndrome.

CASE 2

A 25 year old woman presented with a one year history of intermittent bloody diarrhoea and an anal fissure. The fissure was excised, and histological examination showed submucosal granulomas. Nine months later she had a further acute episode of bloody diarrhoea associated with bilateral iridocyclitis and an erythematous papular rash on her legs. Her temperature was normal. Prednisolone was started at 40 mg daily, and azathioprine 150 mg daily was added 10 days later. After six weeks the dose of prednisolone was reduced to 20 mg daily, but she then developed a fever and erythematous lesions 1 cm in diameter, some showing pustulation. Her white cell count was raised at $13\,400\times10^9$/l (90% neutrophils), and her erythrocyte sedimentation rate was 57 mm in the first hour. Biopsy of a typical lesion showed a predominantly neutrophilic, perivascular infiltrate in the upper dermis with fragmentation of white cells. Prednisolone was increased to 60 mg daily, and the skin lesions resolved within seven days. She had a panproctocolectomy three months later, and histological examination confirmed colonic Crohn's disease. She remained well at four years and did not have any further skin disease.

Comment

Sweet described a dermatosis characterised by tender erythematous plaques and a dense dermal, perivascular infiltrate consisting predominantly of polymorphonuclear leucocytes often with nuclear dust.[1] Patients with Sweet's syndrome have leucocytosis of peripheral blood and usually a concurrent fever. Other systemic features include a raised erythrocyte sedimentation rate, arthritis, myalgia, and ocular signs.[2]

Infection may be an aetiological factor, and the onset is often preceded by an upper respiratory tract infection. In 10% of patients acute myeloid or myelomonocytic leukaemia is present,[3] and Sweet's syndrome has also been recorded in patients with subacute lupus erythematosus, Sjögren's syndrome, and subacute thyroid disease. Its occurrence with ulcerative colitis has been reported in three patients, including one of Sweet's original patients.[1,4] Crohn's disease and ulcerative colitis may be similar clinically, especially if Crohn's disease is confined to the distal colon. In our patients the diagnosis was firmly established by histological examination of bowel that had been resected.

Although concurrence of Sweet's syndrome and Crohn's disease may be coincidental, the presence of the syndrome in two patients with active Crohn's disease suggests a causal link.

1 Sweet RD. An acute febrile neutrophilic dermatosis. *Br J Dermatol* 1964;**76**: 349-56.

2 Gunawardena DA, Gunawardena KA, Ratnayaka RMRS, Vasanthanathan NS. The clinical spectrum of Sweet's syndrome (acute febrile neutrophilic dermatosis)—a report of eighteen cases. *Br J Dermatol* 1975;**92**:363-73.

3 Cooper PH, Innes DJ, Greer KE. Acute febrile neutrophilic dermatosis (Sweet's syndrome) and myeloproliferative disorders. *Cancer* 1983;**51**: 1518-26.

4 Benton EC, Rutherford D, Hunter JAA. Sweet's syndrome and pyoderma gangrenosum associated with ulcerative colitis. *Acta Derm Venereol (Stockh)* 1985;**65**:77-80.

(Accepted 31 August 1988)

Correction of severe hyponatraemia by continuous arteriovenous haemofiltration before liver transplantation

Andrew J Larner, Christopher R Vickers, D Adu, J A C Buckels, Elwyn Elias, James Neuberger

Queen Elizabeth Hospital, Edgbaston, Birmingham B15 2TH

Andrew J Larner, BM, *surgical house officer, liver unit*
Christopher R Vickers, MB, *research fellow, liver unit*
D Adu, FRCP, *consultant physician, renal unit*
J A C Buckels, FRCS, *consultant surgeon, liver unit*
Elwyn Elias, FRCP, *consultant physician, liver unit*
James Neuberger, MRCP, *consultant physician, liver unit*

Correspondence to: Dr A J Larner, Intensive Therapy Unit, St Thomas's Hospital, London SE1 7EH

Profound hyponatraemia is often encountered in end stage liver disease. Rapid correction increases the risk of precipitating central pontine myelinolysis[1]: one such case associated with rapid correction during liver transplantation has been described.[2] We describe the use of continuous arteriovenous haemofiltration to correct severe hyponatraemia slowly before liver transplantation in two patients with end stage liver disease.

Patients, methods, and results

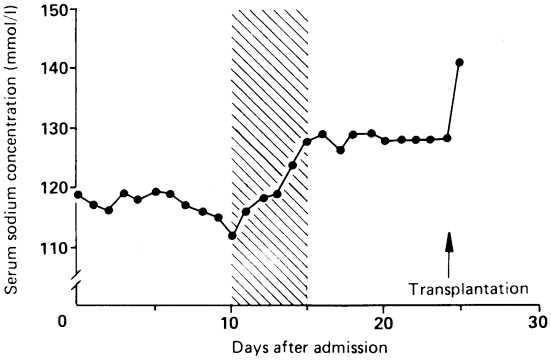
Case 1—A 43 year old man with acute Budd-Chiari syndrome was referred for liver transplantation. Serum sodium concentration on admission was 119 mmol/l (normal range 134-146); despite a restricted fluid intake (500 ml/day) this fell over 10 days to 112 mmol/l, by which time urine output had declined to <10 ml/hour. A Quinton-Scribner arteriovenous shunt was inserted into the right ankle under local anaesthesia. Continuous arteriovenous haemofiltration was started with a Gambro AV55 haemofiltration kit (Gambro Lundia, Sweden). Heparin was infused into the arterial arm of the haemofilter at 500 units/hour. Fluid was removed by the haemofilter at a variable rate (50-100 ml/hour) and isotonic saline was infused into a peripheral vein at the same rate. Haemofiltration was continued for five days, and serum sodium concentration rose to 129 mmol/l (see figure). Orthotopic liver transplantation was performed 24 days after admission. Serum sodium concentration before operation was 128 mmol/l, rising to 141 mmol/l during the operation and remaining in the normal range afterwards. No rebound hypernatraemia or neurological sequelae were observed. The patient remained well 13 months after operation.

Case 2—A 34 year old woman with acute on chronic Budd-Chiari syndrome was referred for liver transplantation. Serum sodium concentration had fallen despite fluid restriction and stopping of diuretics and was 100 mmol/l on admission, with urinary sodium concentration <10 mmol/l. Urinary output was <5 ml/hour. A Quinton-Scribner arteriovenous shunt was inserted into the left ankle under local anaesthesia. Intravenous infusion of colloids and inotropes ensured that blood pressure was adequate to start continuous arteriovenous haemofiltration with a Gambro AV55 haemofiltration kit. Heparin was infused into the arterial arm of the haemofilter at 1000 units/hour. Fluid was removed at a variable rate, and isotonic saline was infused into a peripheral vein at the same rate. By the fifth day, when orthotopic liver transplantation was performed, serum sodium concentration had steadily risen to 129 mmol/l. There were no postoperative neurological complications, and serum sodium concentration remained within the normal range. The patient remained well 10 months after operation.

Comment

Hyponatraemia complicating hepatic insufficiency results chiefly from the kidney's intense conservation of water and sodium, resulting in an inability to produce dilute urine. This may be exacerbated by the use of intravenous fluids, particularly dextrose solutions, to maintain euglycaemia. Spontaneous hyponatraemia is considered to indicate a poor prognosis.[3]

Treatment of dilutional hyponatraemia by fluid and salt restriction may suffice in patients whose urine output is adequate. In oliguric patients a fixed intake of fluid may prove inadequate and hyponatraemia progressively worsen. When water was removed by



Serum sodium concentration before liver transplantation (day 24). Continuous arteriovenous haemofiltration (shaded area) was started on day 10 and continued to day 15