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Hazards of the sauna

We report a case of severe dehydration, complicated by acute gastric dilatation and renal failure, after prolonged sauna bathing. Nevertheless, the patient made a complete recovery.

Case report

A previously healthy 26-year-old West Indian man mistakenly considered himself overweight, but could not tolerate a conventional weight-reducing diet. He tried to lose weight rapidly by fasting for two days (taking fluids normally), and followed this by five hours of sauna bathing, when he developed lassitude, a dry, sore mouth, and generalised abdominal pain. He was muscular, non-obese, but drowsy and severely dehydrated; pulse 76/min; blood pressure unrecordable; temperature 36°C; abdomen normal. The results of investigations included: haemoglobin 21 g/dl, packed cell volume 0·58, Na 140 mmol(mEq)/l, K 4·4 mmol(mEq)/l, urea concentration 9·6 mmol/l (58 mg/100 ml).

Treatment was immediately started with intravenous physiological saline (and potassium supplements as necessary), and continued for six days. After the first litre, given within 30 minutes, the blood pressure rose to 80/60, but the abdominal pain worsened, the abdomen became distended, and bowel sounds were absent; plain radiographs showed acute gastric dilatation. Gastric aspiration yielded 5·5 l clear fluid in the first 24 hours, but none thereafter; however, his intake was only 5·1 l, resulting in a further negative fluid balance. He was anuric on days two and three, and passed only 650 and 250 ml urine on days four and five, respectively. By this time he was fully rehydrated, normotensive (110/90), and bowel sounds had returned, but the blood urea concentration had risen to 32·7 mmol/l (197 mg/100 ml)/l (serum Na 130 mmol(mEq)/l, K 4·0 mmol(mEq)/l. Careful control of fluid balance, protein prohibition, and 8·4 MJ/day (2000 kcal/day) carbohydrate diet led to a diuresis reaching 3·0 l on day 11. The blood urea was then 43·5 mmol/l (262 mg/100 ml), but by day 18 had fallen to 7·7 mmol/l (46 mg/100 ml), when all dietary restrictions were lifted. He was discharged fit on day 21.

Comment

A healthy young man developed a salt depletion and heat exhaustion syndrome[1] as a result of exposure to a temperature of 43°C (that of an average sauna bath) for the incredibly long period of five hours. He also developed the unusual complication of gastric dilatation, and the resulting combination of hypovolaemia and hypotension led to acute renal failure. He made a full recovery, but such a fortunate outcome does not always occur.[2]

The salt depletion and heat exhaustion syndrome result from lack, or inadequate replacement, of salt lost during profuse sweating. Typical features are fatigue, muscle cramps, subnormal temperature, and dehydration, with haemoconcentration, frequently leading to acute renal failure. The most important aspect of treatment is the restoration of blood volume and of the balance of electrolytes to water by giving physiological saline.[1]

Acute gastric dilatation is an uncommon, life-threatening condition that may arise in different circumstances, including the postoperative recovery period, diabetic coma, anorexia nervosa,[3] a late complication of drug overdosage,[4] and trauma.[5] We do not know precisely why it occurred in our patient, but disturbances in the balance of fluid and electrolytes were probably important factors.

Little is known about the physiological changes produced by sauna bathing. Taggart *et al*,[2] prompted by deaths in the sauna, studied healthy controls and patients with coronary artery disease; after only five minutes in the sauna both groups developed tachycardia, electrocardiogram abnormalities, and raised plasma adrenaline concentrations. We do not know whether such changes were relevant in our patient, as he was exposed to the high temperature of the sauna for much longer. Like Taggart *et al*,[2] we trust that this report will alert the public and relevant authorities to the dangers of prolonged sauna bathing and lead to the adequate supervision of such establishments.

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Acute sacroiliitis due to Salmonella okatie

Although the commonest manifestation of salmonella infection is acute gastroenteritis, spread to the blood stream may occur and the illness present with focal lesions in almost any organ with or without septicaemia.[1] Several factors predispose to blood-stream invasion—namely, infections with salmonella serotypes such as *Salmonella choleraesuis*,[1] loss of gastric acidity,[2] immunosuppression, and sickle-cell disease.[3] We report a case of acute sacroiliitis in a patient without evidence of predisposition, due to *S okatie*—a serotype not reported to have affected bones or joints.

Case report

A 14-year-old West Indian girl was admitted to hospital as an emergency case. She gave a three-day history of severe low back pain radiating to the right leg. There had been no trauma to the back nor any previous serious illness. She had not had any disturbance of bowel action, vaginal discharge, or eye symptoms. She was feverish (38·7°C), in severe pain, and unable to bend or stand. There was considerable tenderness over the lumbosacral region, particularly over the right sacroiliac joint. Straight-leg raising was limited to 70° on the left and 20° on the right. No other abnormality was detected on systematic examination. The sacroiliac joints, pelvis, lumbosacral spine, and hip joints were radiologically normal. There was neutrophil leucocytosis (white cell count 14·7 $\cdot 10^9$/l (14 700/mm^3); 79% neutrophils). The erythrocyte sedimentation rate (Westergren) was 110 mm in the first hour. *S okatie* was isolated from blood and stool cultures. A bone scan (^{99}Tc-pyrophosphate) showed increased uptake of the radioisotope over the right sacroiliac joint with no indication of adjacent bone involvement. Haemoglobin electrophoresis showed nothing abnormal, thus excluding haemoglobinopathy. Serum immunoglobulin concentrations and lymphoblastic transformation to phytohaemagglutinin were normal when measured during her convalescence, showing that she did not have obvious immunodeficiency. *S okatie* was also isolated from the stools of two other members of the patient's family and from a neighbour, all of whom were symptomless.

Initially she was treated with flucloxacillin and ampicillin to cover the possibility of either staphylococcal infection or infection with Gram-negative organisms. Once the results of blood culture were known, ampicillin was continued at a dose of 1 g six-hourly. Eight days later she was without fever, her pain relieved, and mobility improved. When first isolated the organism was fully sensitive to ampicillin, the minimum inhibitory concentration (MIC) being 1 mg/l, but sensitivities of later faecal isolates showed ampicillin resistance (MIC 100 mg/l) due to the acquisition of a plasmid transferable to a suitable *Escherichia coli* K12. Nevertheless, the organism was sensitive to co-trimoxazole (MIC: sulphamethoxazole 25 mg/l; trimethoprim 0·6 mg/l).