- ² Chew, H E R, Hanson, G C, and Slack, W K, *British Journal of Diseases of the Chest*, 1969, **63**, 113.
- ³ McAllister, T A, *et al*, in *Proceedings of 2nd International Conference on Hyperbaric Oxygenation*, ed I McA Ledingham, p 250. Edinburgh, Livingstone, 1965.
- ⁴ Watt, J, *Proceedings of the Royal Society of Medicine*, 1971, **64**, 880.
- ⁵ Neely, J, and Catchpole, B, *British Journal of Surgery*, 1971, **58**, 21.

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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 (197 mg/100 ml). 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 syndrome¹ 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 tachy-

cardia, 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.

We thank Mrs Vivian Allen for secretarial help in preparing this paper.

¹ World Health Organisation, Technical Report Series, *Salt Depletion—Heat Exhaustion. Health Factors Involved in Working under Conditions of Heat Stress*, No 412. Geneva, WHO, 1969.

² Taggart, P, Parkinson, P, and Carruthers, M, *British Medical Journal*, 1972, **3**, 71.

³ Jennings, K P, and Klidjian, A M, *British Medical Journal*, 1974, **2**, 477.

⁴ How, J, and Strachan, R W, *British Medical Journal*, 1976, **1**, 563.

⁵ Kasenally, A T, Felice, A G, and Logie, J R C, *British Medical Journal*, 1976, **2**, 21.

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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 × 10⁹/l (14 700/mm³); 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 (⁹⁹Tc-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).

The patient recovered completely and was treated with co-trimoxazole 3 tablets twice daily for a further six months. At six months x-ray examination showed sclerosis of the right sacroiliac joint and irregularities of the joint margins.

Comment

This case illustrates the facts, well-recognised by microbiologists, that most salmonella serotypes may invade the blood stream and that systemic disease need not be preceded by gastrointestinal symptoms,¹ nor need the patient have an overt susceptibility to bacteraemia. Acute suppurative arthritis or osteomyelitis is usually due to *Staphylococcus aureus*,¹ and we did not suspect a salmonella infection because of the absence of gastrointestinal symptoms in the patient and her family.

The course of salmonella infection in bone or joint may be chronic⁵ and treatment required for many weeks. Chloramphenicol is therefore unsuitable. Resistance to ampicillin may appear rapidly, as in our patient, and co-trimoxazole proved to be a suitable alternative. There have only been 32 cases reported of *S. okatie* infection in England and Wales for the years 1970-1976 inclusive. There are no reported isolates from non-human sources for the years 1973-1976 inclusive. *S. okatie* may have been originally introduced into the United Kingdom in animal foodstuff imported from Nigeria.

We are grateful to the Public Health Laboratory Service Communicable Disease Surveillance Centre for providing information about *S. okatie* infections.

¹ Saphra, I, and Wasserman, M, *American Journal of the Medical Sciences*, 1954, **228**, 525.

² Gray, J A, and Trueman, A M, *Scottish Medical Journal*, 1971, **16**, 255.

³ Roberts, A R, and Hilburg, L E, *Journal of Pediatrics*, 1958, **52**, 170.

⁴ McAllister, T A, *British Journal of Hospital Medicine*, 1974, **12**, 535.

⁵ Black, P H, et al, *New England Journal of Medicine*, 1960, **262**, 864, 921.

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Transverse myelitis after diphtheria, tetanus, and polio immunisation

Transverse myelitis from any cause is rare in infancy. We report here a case which developed in a 7-month-old girl six to seven days after her first immunisation with diphtheria and tetanus toxoid and oral poliomyelitis vaccine.

Case history

A 7-month-old girl was admitted to hospital with a four-day history of a slight cough and a hoarse cry associated with mild irritability and lassitude. On the day of admission her mother had noticed that her daughter was not moving her legs, and with hindsight thought that she had not moved them for the previous 24-36 hours. Previously the child had been normal and healthy. She had started to crawl, and could sit unsupported. She was the second child of young, healthy, non-consanguineous parents, and there were no perinatal complications. The older child was normal. Six days before the onset of the illness she had been inoculated for the first time against diphtheria and tetanus, and given her first oral polio immunisation.

She had a temperature of 38.2 °C but looked well and alert. She had a slightly hoarse cry, and her respiration was diaphragmatic. She had complete flaccid paralysis of her legs and lower trunk. The knee and ankle jerks were exaggerated bilaterally with ankle clonus, and the plantar responses were extensor. Abdominal reflexes were absent. Her bladder was palpable up

to the umbilicus. Sensation to pinprick was absent up to the level of T4/5; at this level the child cried and tried to push the pin away. The power, tone, and reflexes in her arms were normal and the cranial nerves were normal. All other systems were normal.

Initial investigations showed a white cell count of $16.2 \times 10^9/l$ ($16\,200\text{ mm}^3$), with 58% lymphocytes and 34% mature neutrophils. Her cerebrospinal fluid (CSF) was clear and under pressure of 160 mm of water. It contained four polymorphs, two lymphocytes, two red cells, and a protein concentration of 0.3 g/l (30 mg/100 ml). A myelogram gave a normal result. Virology of the CSF and throat showed nothing abnormal. Poliovirus type 2 was isolated from the stools.

She was started on dexamethasone 1 mg four times daily for four days, followed by prednisolone 5 mg three times daily continued for a six week period. The bladder was catheterised for two days, after which bladder function was automatic and recatheterisation was not required. Her temperature fell to normal within 24 hours, and her general condition remained good, though there was no obvious improvement in her paraplegia. She was discharged from hospital 10 days later.

After discharge there was slight improvement in the child's condition. The power returned to her lower trunk muscles and she had some power in the hip flexors that enabled her to maintain the sitting position and to get into the crawling position. She crawled around the floor by pulling herself along on her arms. The reflexes in her legs remained brisk with extensor plantar responses, and abdominal reflexes were absent. She developed some tricks for swinging her legs around, and would lift them with her hands. She was wearing splints at night to keep her limbs in an optimum position. Her sensory level was at T7/8, and her bladder emptied automatically. Bowel function was normal and the urine was not infected. Her social and intellectual development remained entirely normal.

Comment

Although myelitis may have occurred by chance in this child, the onset of her symptoms occurred at the time at which other reactions to immunisation are most frequently found. Many complications of immunisation have been described in infants and young children, but these are usually attributed to the pertussis component of triple vaccine,¹ or smallpox vaccination. Even in these conditions, or in association with serum administration or antirabic treatment, myelitis is a rare type of neurological complication. Only one case of myelitis has been reported in an infant receiving triple vaccine, and in that child the illness was complicated by cytomegalovirus infection.² One further case of transverse myelitis in a 22-year-old man after diphtheria immunisation given as alum-precipitated toxoid has been reported.³

Careful studies after the introduction of live oral polio vaccination⁴ showed no cases of myelitis after over 30 million doses of live oral polio vaccine, and only rare cases of other types of neurological disease. Holt *et al.*,⁵ however, have recently reported two cases of transverse myelitis after immunisation with live rubella vaccine. Our case is therefore the first one of transverse myelitis reported in association with the use of either oral poliomyelitis vaccine or with the diphtheria and tetanus inoculation in an otherwise well child. Without other evidence, however, we cannot be sure which of these antigens is the one most likely to have been responsible for the myelitis.

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¹ Dick, G W A, *Proceedings of the Royal Society of Medicine*, 1974, **67**, 371.

² Kulenkampf, M, Schwartzman, J S, and Wilson, J, *Archives of Disease of Childhood*, 1974, **49**, 46.

³ Miller, H G, and Stanton, J B, *Quarterly Journal of Medicine*, 1954, **23**, 1.

⁴ Miller, D L, Reid, D, and Diamond, J R, *Public Health* (London), 1970, **84**, 265.

⁵ Holt, S, et al, *British Medical Journal*, 1976, **2**, 1037.

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