the severe night sweats stopped after two courses of treatment, and his temperature returned to normal over one month. No other active treatment was given and his symptoms remained well controlled for two months before the disease eventually progressed and he died.

Discussion

We attribute the relief of symptoms to levamisole. No other active form of treatment for Hodgkin's disease was used in the second patient during the asymptomatic period, and in the first patient three months of the same combination chemotherapy had completely failed to alleviate her symptoms. Control of severe night sweats occurred after two courses of levamisole in both cases.

The pathogenesis of the systemic manifestations of Hodgkin's disease is obscure and the mechanism of action of levamisole in abolishing the night sweats and fever in our patients is not clear. There is indirect evidence of circulating immune complexes in the plasma of patients with Hodgkin's disease[2 3] and their association with systemic symptomatology has been suggested.[3] Nephrotic syndrome in association with immune complexes and active Hodgkin's disease has been described.[1]

We speculate that levamisole potentiated the activity of phagocytes and thymus-dependent lymphocytes in these patients, with subsequent ingestion of immune complexes[3] or temporary elimination of disease-associated antigen.[5] We propose that levamisole may have a place in the management of Hodgkin's disease as an adjunct to radiotherapy and chemotherapy, especially in symptomatic cases.

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Use of hyperbaric oxygen in paralytic ileus

Paralytic ileus may complicate major abdominal surgery or present as a sequel to peritoneal infection. Rarely it may be purely neurogenic, usually after abdominal injury. Conventional treatment occasionally fails and abdominal distension and general toxicity increase. Inhalation of 100% oxygen, which has been used to treat abdominal distension for many years,[1] is thought to act by reducing the partial pressure of nitrogen in the blood and therefore increasing the speed of absorption of nitrogen from the bowel. There is every reason to believe that hyperbaric oxygen would have a similar action. While it is generally known that hyperbaric oxygen has a profound effect on anaerobic bacteria,[2] it is not so well known that it also kills or slows the growth rate of aerobic pathogenic bacteria.[3 4] It is thus reasonable to expect hyperbaric oxygen to be of value in paralytic ileus, whatever the cause, as part of treatment.

Patients, methods, and results

During the past five years 12 cases of paralytic ileus with varying and increasing degrees of distension and toxicity have been treated with hyperbaric oxygen at this hospital (see table). Ten cases occurred after acute infection—five from acute appendicitis, two from perforations of the large intestine (stab wound and caecal carcinoma), two with no obvious cause found at laparotomy, and one from empyema of the gall bladder—one after vagotomy and pyloroplasty, and one after laparotomy for abdominal trauma, at which a retroperitoneal haematoma was the only finding. All the patients had shown either no improvement or a worsening of their condition despite continuous gastric suction, intravenous fluids, and correction of biochemistry. When indicated laparotomy had been performed, the cause treated, sepsis drained, and antibiotics given.

Hyperbaric oxygen was begun on the second day of illness in four cases, the third day in five cases, and the fourth, seventh, and eight days in the remaining cases. It was given over one hour twice daily in a Vickers single-person chamber at two-and-a-half atmospheres, gastric suction and intravenous fluids being continued.

The patients received four to 10 hours of hyperbaric oxygen, and all were improved, 11 recovering completely (table). The remaining patient (case 7), with a perforated carcinoma of the caecum, died on the third day of treatment, although his bowel function was recovering. There were no complications referable to the hyperbaric oxygen.

Comment

It was surprising how quickly recovery occurred and how well the patients felt after even the first treatment. Measurement of abdominal girth gave no real indication of success, as an abdomen as tight as a drum at the beginning might be of the same size but quite soft after one hour's treatment. Only a post-traumatic neurogenic distension in a young girl (case 12) diminished (by 8·5 cm in 48 hours).

I do not suggest that any steps in the conventional treatment of paralytic ileus should be omitted—which should possibly include giving sympathetic nervous blocking agents[5]—but that when improvement in the patient's condition is not obvious after a reasonable interval hyperbaric oxygen should be tried.

I thank Mr D W Bracey, consultant surgeon, for enthusiasm and help, and Sister J Burbage and her nursing staff in the intensive care unit, who administered the hyperbaric oxygen.

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Clinical details and outcome of treatment

Case No	Age (years)	Sex	Cause of paralytic ileus	Symptoms: Distension	Symptoms: Toxicity	Day of illness hyperbaric oxygen begun	Hours of treatment	Outcome
1	11	F	Appendicitis	+ + + +	+ + +	3	8	Temperature normal in 24 hours; distension resolved in three days
2	58	M	Appendicitis	+ + +	+ + +	2	7	Temperature 38 C and pulse 120 min falling to normal in 48 hours; bowels open in 48 hours
3	58	M	Appendicitis	+ + + +	+ +	8	3	Pulse 100 min falling to 80 min and distension subsiding in 48 hours
4	14	M	Appendicitis (abscess)	+ +	+ + + +	3	8	Temperature 38·4 C and pulse 120 min falling to normal in 48 hours
5	12	M	Appendicitis	+ +	+ + + +	7	4	Temperature 38·5 C and pulse 130 min falling to normal; distension reduced in 24 hours
6	26	M	Perforated colon (stab wound)	+ +	+ + + +	2	8	Pulse 120 min falling to 80 min in 24 hours
7	84	M	Perforated caecum (carcinoma)	+ +	+ +	2	6	Bowel sounds started; patient died on third day
8	26	M	Peritonitis (no cause found)	+ +	+ + + +	4	4	Pulse 140 min falling to 80 min in 24 hours
9	1 12	M	Peritonitis (no cause found)	+ + +	+	2	6	Bowels open and patient hungry in 24 hours
10	64	F	Empyema of gall bladder	+ +	+ + + +	3	10	Temperature 39·5 C and pulse 140 min falling to 36 C and 90 min respectively in 48 hours
11	58	M	Vagotomy and pyloroplasty	+ + +	+ +	3	4	Pulse 110 min falling to 78 min, no distension, and bowels open in 48 hours
12	8	F	Retroperitoneal haematoma	+ + + +	+ +	3	4	Temperature 38 C and pulse 130 min falling to 36·5 C and 100 min respectively in 48 hours; girth 68·5 cm decreasing to 60·0 cm in 48 hours

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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.

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

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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).