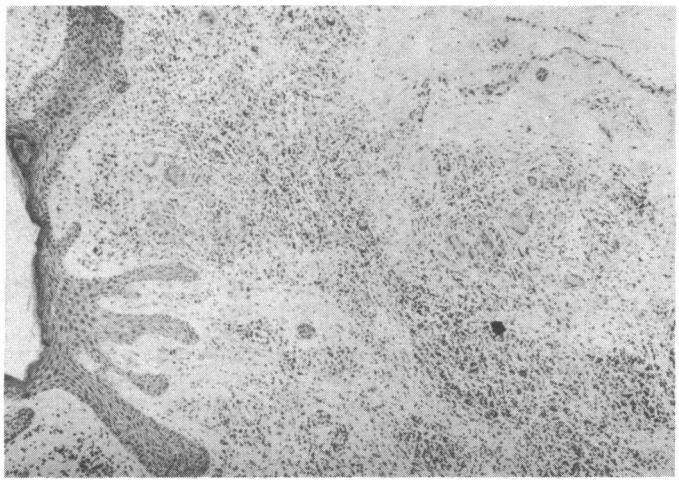
Metastatic Crohn's disease of the umbilicus

Non-contiguous disease of the skin in Crohn's disease (termed metastatic Crohn's disease[1]) is an unusual manifestation. We report on a patient with anorectal Crohn's and separate metastatic disease of the umbilicus.

Case report

A 46-year-old Caucasian woman presented with a six-month history of severe perianal discomfort and pain on defecation, associated with bleeding, alternating diarrhoea and constipation, and weight loss. Coincidentally with her symptoms she had noticed a sore at the umbilicus.

Examination showed a painful, swollen, moist, inflamed ubilicus. Perianal examination showed multiple fissures, two low-level posterior anal fistulae, and several large oedematous purple skin tags. Sigmoidoscopy disclosed friable, oedematous mucosa with ulceration. Rectal biopsy specimens showed severe chronic inflammatory changes confined mainly but not entirely to the mucosa, but no granulomata. A biopsy specimen of the umbilical lesion showed chronic inflammatory changes with several non-caseating granulomata and giant cells, but no acid-fast bacilli (figure).



Typical "sarcoid" reaction in umbilical biopsy specimen showing non-caseating granulomata, Langhan's giant cells, and epithelioid cells. ×50 (original magnification).

Other investigations showed mild hypochromic anaemia (haemoglobin concentration 10·3 g/dl), raised erythrocyte sedimentation rate (82 mm in first hour), normal white blood cell count ($6{\cdot}4 \times 10^9$), slight hypokalaemia (serum potassium concentration 3·0 mmol (mEq)/l, and hypoalbuminaemia (serum albumin concentration 29 g/l). Liver function tests, and the blood concentrations of urea, other electrolytes, and trace elements were normal. Sputum and urine cultures failed to show any acid-fast bacilli, and Mantoux test result was negative. Chest radiography showed no evidence of sarcoidosis or tuberculosis, and barium meal and follow-through showed no abnormality. A barium enema, performed with difficulty, was normal, apart from ulceration visible to the lower sigmoid region.

Topical steroid treatment could not be tolerated and in view of the severe rectal pain on defecation she was treated with total parenteral nutrition and nil by mouth for 33 days, together with prednisolone 10 mg thrice daily. She responded well to treatment with haemoglobin concentration rising to 11·7 g/dl and erythrocyte sedimentation rate falling to 32 mm in first hour. Umbilical and perianal lesions also improved and she became pain free and able to defecate. One year later she remains in remission on a maintenance oral dose of prednisolone 2·5 mg thrice daily.

Comment

Patients with skin disease from Crohn's disease fall into two groups. One group comprises several different dermatoses[2] (pyoderma gangrenosum, erythema nodosum, erythema multiforme, palmar erythema, rosacea, epidermolysis bullosa acquisita, and polyarteritis nodosa) with varying histology. The other group have a "sarcoid" reaction as described by Parks *et al*,[1] typically epithelioid cells and Langhan's-type giant cells in a non-caseating granuloma, together with a chronic inflammatory infiltrate. There have been several reports of such lesions, most of which spread outwards in a contiguous fashion from the perineum towards the scrotum and penis[3 4] or from a diseased stoma[5]; oral disease by similar lesions has been noted, but the mouth might be considered as part of the alimentary tract.

True non-contiguous metastatic skin disease is extremely rare, usually occurring where there is skin apposition within flexures such as the sub-mammary,[1] retroauricular,[2] or anterior abdominal-wall skin fold[4] regions. Metastatic Crohn's disease of the umbilicus has not been reported, but this area is similarly one of skin apposition.

It is not clear whether metastatic Crohn's disease may precede alimentary tract disease, as may perianal lesions, but in our patient it developed simultaneously with the rectal disease, and both responded in parallel to treatment. It is important, nevertheless, to consider Crohn's disease in patients presenting with sarcoid-like skin lesions, even in the absence of overt alimentary symptoms or signs. As the histology is non-specific, tuberculosis and sarcoidosis must also be excluded. The cause of metastatic Crohn's disease remains an enigma.

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St Mary's Hospital, London W9
R K S PHILLIPS, FRCS, surgical registrar
GEOFFREY GLAZER, FRCS, consultant surgeon

Burns caused by striking underground electricity cables

Effective preventative measures are the most important factor in dealing with the problem of burns. Industrial accidents are still a regular and preventable cause. When such accidents entail damage to underground electricity cables the result may be immediate death or severe injuries, including burns. A few of those injured in this way are referred to burns units and a review of patients admitted to our unit shows the type of avoidable injury which these accidents can cause.

Case reports

From June 1979 to December 1980, eight previously fit men aged 16-55 years were admitted for treatment of burns caused by striking underground cables. Six had damaged the cables with pneumatic drills and two with forks. Three, with 9%, 12%, and 18% burns affecting the face and hands were inpatients for 12 to 28 days and their burns healed with regular dressings. One with 23% superficial burns required resuscitation with intravenous fluids but healed with regular dressings as an inpatient for eight days. Three men, with 25%, 25%, and 40% burns were inpatients for 37 to 61 days; they needed resuscitation with intravenous fluids and two operations each for débridement of the wounds and skin grafting, in addition to regular dressings. One patient in this group had an inhalation injury, requiring temporary endotracheal intubation and ventilation, and another had a pulmonary embolus after operation. One 30-year-old man had a 44% burn and despite prompt adequate resuscitation developed a severe haemolytic anaemia with acute renal failure, and died three days after his accident. All patients had flash burns, with or without additional flame burns from burning clothes. The man who died also had signs of electrical, as opposed to heat, burns on his hands.[1]

Comment

Each year there are about 20 000 incidents due to underground electricity cables in Britain.[2] Although only a few of these accidents

result in injury,[2] the consequences can be severe; death, long hospital admissions, painful operations, and permanent physical and mental scarring. The National Joint Utilities Group (Post Office Telecommunications, Electricity Supply Industry, British Gas, and the Water Industry) publish advisory booklets and also cards suitable for use at work sites; these recommend how to avoid underground electricity cables and give information on cable locating devices. Legislation to prevent injury from electricity cables is contained in the Construction (General Provisions) Regulations 1961, the Health and Safety at Work Act 1974, and the Electric Lighting (Clauses) Act 1899. It has however been stated that "apathy is the greatest single obstacle to progressive improvement" in safety and health at work (Robens Report, 1972[3]) and that more education rather than legislation is required.[3]

The experiences of our patients show the need for even more knowledge and understanding of the dangers of striking underground electricity cables.

I thank Mr D H Harrison for permitting me to report on patients under his care, and Mr A P Gifford of the Health and Safety Executive for the statistics and details of relevant legislation.

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Mount Vernon Hospital, Northwood, Middlesex
M J TIMMONS, MA, FRCS, registrar in plastic surgery

Spinal cord degeneration in a case of "recovered" spinal decompression sickness

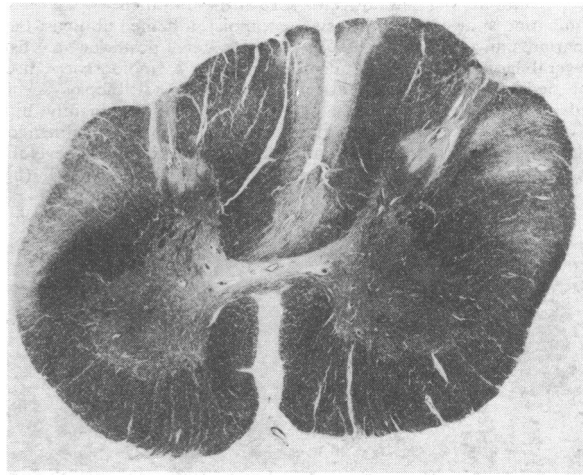
Degeneration in the spinal cord of goats after experimental decompression sickness is well documented[1 2] but opportunities to examine the long-term effects in man are rare.[3] We describe severe damage in the spinal cord of a man who had made an almost complete functional recovery from spinal decompression sickness.

Case report

In June 1976 a 34-year-old scuba diver surfaced probably rather rapidly from about 110 feet (33·5 m), where he had remained for 20-25 minutes. Shortly afterwards he suffered pain and tightness in a girdle distribution around the mid-chest and had difficulty speaking and breathing. By the time of admission (11 am) symptoms had disappeared but he was nevertheless put into a pressure chamber, which was adjusted to 28 lb/in^2 (2 kg/cm^2) guage pressure. Ten minutes later he still had no complaints and began decompression over one and a half hours. That night his right thigh and foot became numb with some patchy loss of sensation to pinprick, and there was weakness in the left leg. He was repressurised to 30 lb/in^2 (2·1 kg/cm^2). After two hours 20 minutes he improved; co-ordination, sensation, and reflexes were recorded as normal. He was then decompressed on an oxygen table (US Navy, 6; Royal Navy, 6B). At the time there was only indefinite weakness in the left leg and a slightly abnormal gait. The paraparesis improved gradually over about three weeks.

In January 1980 the patient was re-examined neurologically. He felt no fatigue after exercise but during cold weather his legs tended to shake when standing. No evidence of mental impairment was found and cranial nerve functions were normal apart from brisk snout and palmomental reflexes. Muscle power, tone, and co-ordination were normal in all limbs but deep tendon reflexes in the legs were abnormally brisk, with sustained clonus at the ankles and knees and bilateral extensor plantar responses. Reflexes in the arms were also brisk but within normal limits. Superficial abdominal reflexes were present. The left cremasteric reflex was less active than the right. Sensory testing showed only slight impairment of joint position sense at the toes. It was concluded that there was evidence of residual corticospinal tract damage and perhaps some higher involvement, because of the unexpectedly active primitive facial reflexes. Twelve days later he met with a violent death not connected with diving.

Neuropathological changes were confined to the spinal cord. Between C1 and T4 there was bilateral degeneration of the fasciculus gracilis, the subpial myelin often being spared (figure). The lateral funiculi were also affected between C1 and L4, the most severe damage being between C6 and T10, where the lateral corticospinal tracts and spinocerebellar tracts were principally affected. Between C1 and C6 this degeneration was confined to the dorsal spinocerebellar tracts, decreasing in severity at the rostral levels. Degeneration in the ventral columns was present between T3 and T5; this was more prominent on the left side and affected the deep fibres. Overall focal damage was most severe between C7 and T4, where there were changes compatible with a past ischaemic episode.



Spinal cord at C5 showing degeneration of fasciculus gracilis and lateral funiculi. Methasol fast blue × 10 (original magnification).

Comment

The distribution of lesions was similar to that reported in other cases of spinal decompression sickness. Haymaker[3] described symmetrical softening and later sclerosis of the dorsal and lateral columns, particularly in the thoracic cord. In our patient tract degeneration may have stemmed from an area of infarction between C7 and T4. In view of the extent of the damage it was remarkable that he had made such a good recovery.

This case poses major questions concerning the definition of clinical recovery from spinal decompression sickness and the advice that should be given about subsequent diving activities. Neurophysiological tests would perhaps be more likely to disclose functional derangement than the more routine neurological assessment.

We thank Professor A Usher for helpful co-operation and Dr C S Treip for advice.

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Department of Clinical Veterinary Medicine, University of Cambridge, Cambridge CB3 0ES
A C PALMER, SCD, FRCVS, assistant director of research

Department of Morbid Anatomy, London Hospital Medical College, London E1 1BB
I M CALDER, MD, honorary lecturer

University Department of Industrial Health, Newcastle upon Tyne
R I McCALLUM, MD, DSC, reader in industrial health

Department of Medicine, University of Western Australia
F L MASTAGLIA, MD, FRACP, associate professor of neurology