and liver enzymes were normal. Patch tests for Quinaband gave negative results. Application of Quinaband was stopped, and on 24 November 2 units of fresh blood produced a rapid rise in platelet count, which subsequently returned to normal values. The patient made an excellent recovery and remained euthyroid, though the leg ulcers posed considerable problems.

Comment

The fall in platelets and haemoglobin coincided with the application of Quinaband, and these values rose when the dressings were withdrawn (albeit aided by transfusion), suggesting a causal mechanism. The large, deep ulcers allowed considerable absorption of the quinine-like agents in the dressings and presumably led to the formation of platelet antibodies and hence thrombocytopenia. The coma after five days on the patient's regular thyroxine treatment is interesting. Firstly, she may not have actually ingested the tablets. Secondly, maximal action of L-thyroxine does not occur till after the second week. Thirdly, the anaemia may have precipitated coma in an already grossly myxoedematous patient. Finally, a combination of any of these three factors may have occurred.

The hypothermia may have been a defence mechanism to reduce the metabolic rate and counter the increased demands of the high-output state brought on by anaemia.

I thank Dr A L Wyman for invaluable help and criticism; Professor J Lee and Dr Nigel Huston for their support; and the department of medical illustration for the figure.

¹ Committee on Safety of Medicines, personal communication.

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Bronchial carcinoma with pleural spread causing unilateral thoracic hyperhidrosis

Any lesion of the sympathetic pathway may produce localised hyperhidrosis.^{1 2} Unilateral thoracic hyperhidrosis is rare and appears to have been recorded only by Pool,³ as a consequence of an osteoma of the 10th dorsal vertebra. The case reported here illustrates that unilateral thoracic hyperhidrosis may complicate bronchial carcinoma.

Case report

A 52-year-old man was admitted to hospital with a six-week history of right-sided pleuritic chest pain associated with cough and breathlessness. These symptoms had persisted despite treatment with a broad-spectrum antibiotic. He was a cigarette smoker and worked as an operating theatre technician. He was afebrile with signs of a small right-sided pleural effusion, confirmed by a chest radiograph. The pleural fluid was amber and contained 98% lymphocytes. A pleural biopsy showed no diagnostic features. The appearances at bronchoscopy (Mr R J M McCormack) were normal. A Mantoux test (1/1000 tuberculin) was positive with 15 mm induration; white cell count was $8.4 \times 10^9/l$ ($8400/mm^3$). The results of plasma protein electrophoresis and of tests for antinuclear and rheumatoid factors were all normal.

The provisional diagnosis was of tuberculous pleural effusion and he was started on rifampicin, 600 mg daily; ethambutol, 1.2 g daily; and isoniazid, 300 mg daily. Over the ensuing three months he continued to complain of right-sided chest pain and there was no further radiographic improvement. He was again bronchoscoped because the radiographic appearances were thought to represent collapse of the right lower lobe. Nevertheless, the appearances at bronchoscopy were again normal. Antituberculous chemotherapy was continued, and prednisolone was added.

Four months after presenting he complained of excessive sweating affecting the right arm, right side of the chest, and right side of the face. This occurred in paroxysms several times daily and was observed regularly during his subsequent readmission to the ward. During such an episode perspiration was visibly excessive over an area extending from the face to T10 level, and at all levels there was an abrupt cessation of sweating at the midline. During his final hospital admission his main complaint was of persistent right lower chest pain. Radiographs of the thoracic spine showed a thoracic scoliosis concave

to the right, and, although serial chest radiographs failed to show reaccumulation of fluid, there was progressive shrinkage of the right hemithorax. Needle biopsy of the pleura showed undifferentiated malignant cells invading muscle and fibrous tissue. His pain was satisfactorily relieved by a C1 cordotomy (Mr E R Hitchcock) and he died one month later.

At necropsy (Dr H M Gilmour) dense tumour tissue obliterated the right pleural cavity and totally encircled the right lung. The histological features were those of anaplastic carcinoma with a prominent fibrous stromal reaction. A nodule within the posterior basal segment of the right lower lobe showed similar histological appearances and this appeared to be the most likely primary site. Secondary deposits were also found in the peritoneum, pericardium, and tunica vaginalis of the right testis. No metastases were found in the brain and spinal cord.

Discussion

Apical carcinoma of the bronchus is a well-recognised cause of Horner's syndrome. Stanford⁴ has recently described two cases of pleural mesothelioma with interruption of sympathetic nerve supply to the ipsilateral arm. Conceivably these destructive phenomena could be preceded by a phase of local irritation causing sympathetic excitation. This appears to have been the course of events in this patient, and as intrathoracic malignant disease is common it is perhaps surprising that this particular complication has not been seen more frequently.

The author is grateful to Dr G J R McHardy for permission to report this case.

¹ Ellis, H, *British Journal of Hospital Medicine*, 1972, 7, 641.

² Telford, E D, *British Medical Journal*, 1942, 2, 96.

³ Pool, J L, *Journal of Neurosurgery*, 1956, 13, 111.

⁴ Stanford, F, *British Journal of Diseases of the Chest*, 1976, 70, 134.

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Hypothermia and Wernicke's encephalopathy

Hypothermia is well known as a complication of alcoholism, but there are few reports of its association with Wernicke's encephalopathy. A case of Wernicke's encephalopathy in which hypothermia was the presenting feature is reported.

Case report

A 54-year-old woman was admitted on the evening of 24 December 1975 with a rectal temperature of 29.5°C. The outdoor temperature that afternoon was about 4°C and she had been lying, fully clothed, for not more than seven hours on an internal common stair. She was incapable of giving a history, but according to her relatives had been a heavy drinker for five years. She had been ataxic for some months, and one week before her admission she had been found cold and drowsy, but had recovered at home.

She was drowsy, obese, cold, and hyperventilating. She did not smell of alcohol, moved all four limbs spontaneously, and responded to painful stimuli. Cranial nerve function could not be tested fully. She was normotensive (blood pressure 140/80 mm Hg) with a regular pulse of 80/minute. Emergency investigations showed hypokalaemia (serum potassium 2.3 mmol (mEq/l), blood sugar of 16.4 mmol/l (295 mg/100 ml) and a compensated metabolic acidosis. She was given ampicillin, oral fluids, and potassium supplements and rewarmed passively. Thirty-six hours later her blood sugar had returned to normal and her oral temperature was 39.5°C. She was conscious, but apathetic and hallucinated. Bilateral sixth nerve palsies, bilateral vertical nystagmus, peripheral neuropathy with bilateral foot-drop, and ataxia of all limbs were then obvious. Wernicke's encephalopathy was diagnosed and she was given intramuscular thiamine 100 mg and intravenous parenterol HP daily. She continued to have hallucinations for several days; as this resolved, severe memory impairment with confabulation became evident. The peripheral neuropathy improved very slowly; the ataxia persisted. In view of the Korsakoff's psychosis she was transferred to psychiatric care.

Discussion

This patient's low temperature seemed incompatible with her moderate exposure to cold, and I believe that her hypothermia was directly attributable to Wernicke's encephalopathy. The cerebral lesions in this disease are predominantly in the hypothalamus, which is concerned in thermoregulation. Clinical evidence suggests that hypothalamic lesions can induce spontaneous hypothermia,¹ though there are no studies of thermoregulation in Wernicke's encephalopathy itself. Koeppen,² Victor *et al*,³ Philips and Smith,⁴ and Ackerman⁵ have described hypothermia in association with Wernicke's encephalopathy, but the rarity of this complication is emphasised by Victor's study, in which only two out of 245 patients with Wernicke's encephalopathy had hypothermia. In many of the earlier cases described hypothermia was mild (35-36.6°C) and a coincidental finding. Profound hypothermia is an unusual presentation of Wernicke's encephalopathy, and by masking the classical signs and symptoms may lead to delay in diagnosis, as in this patient. At worst, the patient may die and the diagnosis be made post mortem.³ Our patient had begun to improve before receiving thiamine but in other cases early treatment could be life-saving. Hence thiamine is indicated in any alcoholic developing or presenting with severe hypothermia.

I thank Dr D H A Boyd for permission to report on a patient under his care.

¹ Fox, R H, *et al*, *Lancet*, 1970, **2**, 185.

² Koeppen, A H, Daniels, J C, and Barron, K D, *Archives of Neurology*, 1969, **21**, 493.

³ Victor, M, Adams, R D, and Collins, G H, *The Wernicke-Korsakoff Syndrome*. Philadelphia, Davis, 1971.

⁴ Philips, G, and Smith, J F, *Lancet*, 1973, **2**, 122.

⁵ Ackerman, W J, *Western Journal of Medicine*, 1974, **121**, 428.

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Benign intracranial hypertension after withdrawal of topical steroids in an infant

Although there have been numerous reports of the potential hazards and of the actual biochemical abnormalities induced by topical steroids, there have been few clinical reports of adverse systemic effects.¹ There appears to have been one report of benign intracranial hypertension occurring during topical steroid treatment.² Although this condition is regarded as benign, some patients develop severe visual loss. The case reported here describes the development of benign intracranial hypertension after stopping potent topical steroid treatment which had been limited to the napkin area.

Case report

A boy aged 7 months was admitted with a history of 24 hours' drowsiness, irritability, and vomiting. During this period the mother had noted that the anterior fontanelle was bulging. Previously he had been quite well and developing normally. Since the age of 3 months a persistent napkin rash had been treated regularly with 0.1% betamethasone -17- valerate ointment. The mother had not been aware that it should be used sparingly, and in her own words had "tended to plaster it on" at each napkin change. The 0.1% betamethasone -17- valerate ointment had been prescribed in 100-g quantities. A total of 300 g had been used up over three months until one week before admission, when it was stopped completely. In the week preceding admission he had had no medication.

On admission he was ill looking and very fretful. There were no Cushingoid features. The weight was 6.6 kg (tenth centile) and the head circumference was 43.5 cm (just below 50th centile). The temperature was normal. The anterior fontanelle was tense and bulging. The fundi were normal and there were no other abnormal neurological signs. General examination showed normal features except for oral thrush and a red dry scaly rash confined to the napkin area. The results of full laboratory examinations on

blood and spinal fluid were normal, while the skull x-ray film showed splaying of sagittal and occipital sutures. The EEG was normal.

The anterior fontanelle tension became less day by day, and the boy was discharged home after five days—when he was quite well, the anterior fontanelle tension was almost back to normal, and the results of a full neurological examination were normal. The only treatment he received was 0.1% betamethasone -17- valerate cream to the napkin rash for the first two days of admission, this was then changed to Nystatin cream as *Candida albicans* had been grown from the napkin area. At follow-up a week after discharge the anterior fontanelle tension was normal. A repeat skull x-ray film eight weeks after admission showed that the sutures were back to normal. Follow-up at the age of 19 months showed normal development and the head circumference remained on the 50th centile.

Discussion

In children intensive treatment with 0.1% betamethasone -17- valerate ointment can significantly suppress the hypothalamic-pituitary-adrenal axis function.¹ I suggest that in this boy enhanced absorption of 0.1% betamethasone -17- valerate ointment probably occurred because of the liberal application of topical steroid and the napkin acting as an occlusive dressing. The sudden onset of symptoms and signs of raised intracranial pressure six days after stopping topical steroid treatment is similar to the clinical presentation described in cases of benign intracranial hypertension precipitated by withdrawal of systemic steroids.³

The napkin area is a vulnerable area for systemic absorption of topical steroids. Hence prescriptions should be for the smallest amount compatible with effective treatment, the weakest preparation which will control the disease being the ointment of choice.

I thank Dr John Wilson and Dr Richard Wilson for their advice and help.

¹ Munro, D D, *British Journal of Dermatology*, 1976, **94**, Supplement, 12, 67.

² Benson, R F, and Pharoah, P O D, *Guy's Hospital Reports*, 1960, **109**, 212.

³ Neville, B G R, and Wilson, J, *British Medical Journal*, 1970, **3**, 554.

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Oxytocin and neonatal jaundice

It has been stated that jaundice in newborn infants has reached epidemic proportions in recent years.¹ Ghosh and Hudson² associated this increase with oxytocin. Chalmers *et al*³ demonstrated this effect whether oxytocin was given to initiate labour or to accelerate labour which had already started; they postulated that if there is an association between neonatal jaundice and oxytocin it may be due to a toxic effect of synthetic oxytocin.

The present study was designed to establish whether oxytocin infused during labour that has already started affects bilirubin metabolism in the newborn. A policy of active management of labour results in about half all primigravidae receiving oxytocin during labour in this hospital.⁴ In contrast, a policy of selective induction of labour makes the use of oxytocin to initiate labour comparatively rare.⁵

Patients, methods, and results

A prospective study was conducted on 200 consecutive first-born infants, whose birth weight was over 2500 g and gestation over 37 weeks. The serum bilirubin concentration was measured in capillary blood, obtained on the fourth day in conjunction with the Guthrie test, by a direct reading bilirubinometer (American Optical Corporation). Method of delivery and type of infant feeding were recorded as well as oxytocin infusion. Three patients received oxytocin to initiate labour; they were excluded from the study, which was confined to those who had started labour spontaneously. None of the remaining 197 patients received more than 10 units of oxytocin.⁴ A total of 109 patients received oxytocin and 88 received no oxytocin. The results (table) show that the infusion of 10 units of oxytocin during labour has no significant effect on mean bilirubin levels on the fourth day. The method of delivery and type of infant feeding did not influence the result (table). The