us with the silver staining, and the Medical Research Council for generous support.

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Spinal cord transection in utero

Several cases of cervical cord damage caused by birth trauma have been described.¹⁻³ But to our knowledge no case of antenatal cervical cord damage has been reported. We describe such a case.

Case report

A 32-year-old woman (gravida 2) was found to have a breech presentation at 36 weeks' gestation in an otherwise normal pregnancy. One unsuccessful attempt at external cephalic version was made gently at that time. The pregnancy was allowed to go to term, when the baby was delivered by elective caesarian section.

A boy weighing 3.360 kg was delivered easily and in good condition but,

Top: macroscopic view of dorsal aspect of spinal cord showing 25-mm length of softening (between broad arrows). Cervical cord is to left of picture. Bottom: transverse section of upper thoracic spinal cord showing almost complete destruction and necrosis of cord tissue with macrophage invasion (n). dr=Dorsal roots. a=Thickened arachnoid. s=Anterior spinal artery. (Luxol fast blue Nissl. $\times 15$.)

apart from some facial grimacing, made no respiratory effort. Intermittent positive pressure ventilation was started immediately and presented no difficulties. Subsequent attempts to discontinue this were, however, associated only with further facial movements; the infant made no respiratory effort despite his deteriorating condition. This persisted for the remaining 80 hours of his life, after which assisted ventilation was discontinued.

On examination at birth the baby was noticed to have normal movements of the face, palate, and sternomastoids. There was a flaccid paralysis of his arms with absent reflexes, the abdominal reflexes were absent, and there was increased tone and brisk reflexes in his lower limbs with spontaneous clonus at the hips and knees. Plantar stimulation and pin-prick on the feet produced a strong withdrawal reflex.

Sensory examination showed grimace to pin-prick on the face down to the angle of the jaw, but no response to stimulation of the arms or trunk. These neurological findings, which remained unchanged throughout the infant's life, suggested a cord lesion extending down from the level of the third and fourth cervical segments.

Necropsy showed no bony injury and no abnormality on macroscopic examination of the brain. There was gross softening and shrinkage of a 25-mm length of the lower cervical and upper thoracic cord (see figure). Microscopic examination of transverse sections of the softened areas of cord showed almost total destruction of cord tissue, with cystic spaces and invasion by macrophages. The anterior spinal roots were atrophic but the posterior roots showed no abnormality. There was thickening of the arachnoid mater (figure). The anterior spinal artery was normal. At the level of the lesion the cord showed degeneration of the ascending tracts, while below the lesion there was degeneration of the lateral corticospinal tracts with normal anterior horn cells.

Comment

The clinical and pathological findings described in this report support a diagnosis of antenatal cord trauma. At birth there was clinical evidence of a lesion at the third cervical segment, with a sensory level and presumptive affection of the phrenic nerve and intercostal muscles; the child also had well-developed spasticity of the legs. The pathological evidence of shrinkage and macrophage invasion, with degeneration of ascending and descending tracts is compatible with a lesion three to four weeks old.⁴

Although no bony injury was found, the thickening of the arachnoid mater suggests trauma as the cause, and Hadley⁵ has shown that in the normal newborn compression and shearing forces affecting the spinal cord can be produced easily without causing any bony injury.

The attempted external cephalic version, which perhaps inadvertently extended the fetal neck, occurred at a time compatible with the age of the injury and should be considered the likely cause.

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Rhabdomyosarcoma of the right ventricle associated with polyarthritis

We report a patient with a rhabdomyosarcoma of the right ventricle who presented first with a seronegative polyarthritis and later developed congestive heart failure and signs of pulmonary stenosis.

Case history

A 53-year-old woman had had mild, symmetrical, seronegative polyarthritis for four years affecting her hands and feet. In August 1975 her

arthritis was well controlled with ketoprofen. Over a period of two weeks in December 1975 her condition deteriorated. She developed palpitations, breathlessness on minimal exertion, and ankle oedema. Simultaneously her joints affected by arthritis became more inflamed and for the first time her knees and her right shoulder were affected. There was a systolic thrill palpable at the left sternal edge in the third rib interspace and a mid-systolic murmur audible over the precordium. An ECG showed sinus tachycardia (120), right axis deviation, and right ventricular hypertrophy. A chest radiograph was normal. Probable rheumatoid arthritis with rheumatoid valvulitis was diagnosed and treatment with penicillamine started, but was stopped one week later because of gastric side effects. Over the next three months she felt increasingly lethargic and unwell and lost 10 kg in weight. She remained appreciably dyspnoeic and her arthritis deteriorated further.

An acute rupture of a popliteal cyst precipitated her next referral to hospital. Her arthritis was again active. There were no rheumatoid nodules or evidence of vasculitis. Gross congestive cardiac failure was present. A chest radiograph now showed cardiomegaly and a diffuse opacity in the upper lobe of the right lung. Chest tomograms showed a thick-walled mass with central cavitation in the apical segment of the right lung. The findings of bronchoscopy were normal, and tuberculosis was excluded by examination and culture of sputum.

The results of investigations (normal ranges in parentheses, SI units throughout) were as follows: haemoglobin 10.2 g/dl; white cell count $8.2 \times 10^9/l$; platelets $300 \times 10^9/l$; plasma viscosity 1.86 centipoise (1.5-1.72). The concentrations of urea and electrolytes were normal, but the alkaline phosphatase was 215 IU/l (21-92). Electrophoretic strip showed raised gammaglobulins. Complement concentrations were normal. Anti-DNA antibodies and rheumatoid factor were not found. Echocardiography showed a well-defined mass lying in the right ventricular outflow tract. This was confirmed by angiography. An emergency operation using cardiopulmonary bypass was performed. A tumour ($4.2 \times 2.5 \times 2.5$ cm) which obstructed the pulmonary valve in systole was removed from 2 cm below the pulmonary valve. Histological examination of the tumour mass showed that it was a rhabdomyosarcoma.

Three months after her operation the patient developed multiple pulmonary metastases and died.

Comment

The development of cardiac symptoms and signs simultaneously with the exacerbation of previously mild arthritis led initially to consideration of rheumatoid granulomatous disease of the pulmonary valve. The later development of an apical lung mass subsequently presumed to be infarction distal to tumour embolism also resembled a cavitating rheumatoid granuloma.

The interest in this case lies in the undescribed association of a malignant intracardiac neoplasm with an immunologically mediated systemic illness. Goodwin,¹ in a comprehensive review of the presentation of cardiac myxomas, categorised three major features: embolism, valve obstruction, and constitutional effects. Similarly this malignant tumour was associated with all three. Arthralgia and arthritis have been described in association with a left atrial myxoma.² The association between cardiac tumours, constitutional changes, and immunological abnormalities has not been explained conclusively. Currey *et al*³ suggested that the combination of tumour necrosis and the trauma of compression with systole may feed into the circulation a continuous supply of material which is similar to but not antigenically identical with the normal body tissues and hence gives rise to the immunoglobulin abnormalities and "immunological" constitutional manifestations.

Arthritis preceded the development of cardiac symptoms by four years, and may have been related to the ventricular neoplasm. The rapid deterioration of affected joints and the inflammation of unaffected joints simultaneously with the development of cardiac symptoms supports this concept.

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Adverse reaction to a topical steroid transferred by physical contact

The adverse effects of topical steroid preparations have recently been emphasised.¹ These agents are occasionally used injudiciously and in excessive amounts, and many also contain sensitising agents, which may promote infections and infestations while altering the characteristic morphology of the eruption.²

Occasionally a patient may use and react adversely to a drug which has been prescribed for another person. This is especially likely with dermatological preparations because the general public do not usually consider them to induce untoward effects when applied externally. Topical steroids may be used in this way in the mistaken belief that "what is good for one rash is good for all." Topical applications may also be transferred accidentally from the skin or mucous membrane of one person to the skin or mucous membrane of another during physical contact, especially in members of a family or during sexual activities. Potent topical steroids are particularly likely to induce adverse reactions if transferred in this way. They are cosmetically acceptable to the patient for whom prescribed and after application may easily be forgotten and not readily noticed by friends and relatives. Moreover, they are pharmacologically active even when present in small amounts.

We describe here two cases that illustrate the hazards of an adverse reaction after transfer of a topical steroid.

Case reports

Case 1—A 30-year-old man was referred with a rash confined to the genitalia. He stated that a small red patch had appeared on the tip of the penis seven months previously. He knew of no precipitating factors and the patch was itchy especially after sexual intercourse. His doctor prescribed betamethasone valerate cream to be applied twice daily and just before intercourse. The rash initially improved but the patient continued to use the steroid as a lubricant to aid sexual activity. Two weeks later the patch became more irritable, especially after sexual intercourse, and spread to involve the shaft of the penis and scrotum. The patient's wife had experienced mild pruritus vulvae associated with a slight non-foul-smelling vaginal discharge for about one year. The symptoms became worse seven months before she was examined, at the time her husband started to use the steroid cream. The pruritus and discharge were particularly severe after sexual intercourse, but she had not sought medical advice.

On examination the man was found to have a weeping eczematous rash on the penis and scrotum, from which a swab failed to grow any organisms. A high vaginal swab from his wife grew *Candida albicans* sensitive to nystatin, and candida were identified on a vaginal smear. The patient's original penile rash was probably an allergic reaction to his wife's candidiasis. The topical steroid he used would certainly have been transferred to his wife during sexual contact, worsening her candidiasis, which in turn aggravated his allergy. The patient was treated with Nystaform-HC cream twice daily and just before sexual activity, and his wife was treated with nystatin pessaries BPC. One month later both husband and wife were symptom free.

Case 2—A 50-year-old man attended the skin department with a lesion on the penis, which had been present for five months. He stated that two months after its appearance he had attended another hospital, where lichen planus was diagnosed and he was asked to apply betamethasone valerate cream twice daily. The condition did not respond to this treatment during the following three months. When seen at the skin department his wife stated that she had suffered from pruritus vulvae and a slight vaginal discharge for three months. On examination the patient was found to have classical lesions of lichen planus on the penis, and his wife had candidal vulvovaginitis. The steroid had probably been transferred from husband to wife during sexual intercourse, thereby promoting her candidal infection. Treatment of his wife with antifungal agents cleared her condition in four weeks. The patient was treated with Nystaform-HC cream and his lesions were slightly improved but still present after three months' treatment.

Comment

Topical steroid applications may encourage candida infection, which may not be recognised easily because the symptoms can be suppressed by the steroid.² In both of these patients a topical steroid was thought to have been transmitted from the man to his wife by physical contact during sexual activity. In case 1 the transferred steroid worsened existing candidiasis in the patient's wife, followed by exaggeration of the allergic reaction in the patient.³ In case 2 the steroid cream prescribed for the lichen planus was probably responsible for causing or aggravating vaginal candidiasis in the patient's wife.

There are several reports of skin reactions in men who are sensitive to their wives' cosmetics,⁴ and a transferred sensitivity reaction to his wife's Nivea cream has been confirmed in one man by patch testing.⁵