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.

We thank Dr A St J Dixon, Dr D W Barritt, and Mr G Keen for permission to report on their patient; Dr F G M Ross for echocardiography studies; Mr K Jeyasingham for bronchoscopic examination; and Mrs L F Davis for secretarial help.

¹ Goodwin, J F, *Lancet*, 1963, **1**, 464.

² Dixon, A St J, *Progress in Clinical Rheumatology*, 1965, **1**, 362.

³ Currey, H L F, Matthews, J A, and Robinson, J, *British Medical Journal*, 1967, **1**, 547.

(Accepted 21 April 1978)

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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

This report seems to be the first to describe adverse reactions to drugs accidentally transmitted from one person to another by physical contact.

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(Accepted 26 April 1978)

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Development of cytotoxic antibodies after a venous allograft

The maintenance of adequate vascular access remains one of the major problems associated with long-term haemodialysis.¹ For those patients for whom neither the creation of a Cimeno-Brescia fistula nor the insertion of a Scribner shunt is possible the insertion of a venous allograft has become increasingly common: in Europe, during 1975, 285 such operations were recorded.²

We report the development of cytotoxic antibodies after the insertion of a saphenous vein allograft in a patient awaiting renal transplantation. A positive cross-match result against lymphocytes from a potential cadaveric donor having three HLA antigens in common with the patient caused a transplant operation to be abandoned.

Case report

A 54-year-old woman, with a history of two pregnancies, had been transfused with 12 units of whole blood and concentrated red cells over four years. She had been on maintenance haemodialysis for eight years but had not been transfused for six years. A saphenous vein allograft was successfully inserted into her forearm. The vein, which had been removed from a patient undergoing varicose vein surgery, had been washed with isotonic saline and stored at 4°C for 24 hours. ABO grouping and HLA typing of the donor were not performed before the allograft, but subsequent typing showed him to be group A HLA-A3, B7, B14. The recipient was known to be group O HLA-A1, A2, B17, BW21. The vein graft remained patent until the patient received a compatible kidney transplant 11 months later.

Before the vein transplantation the result of routine monthly testing for cytotoxic antibodies was negative, but one month afterwards her serum contained cytotoxic antibodies against 75% (18/24) of the regular lymphocyte panel, selected to represent a wide range of HLA-A and B antigens; no blood had been transfused at operation.

Three months later a cadaveric kidney—group O HLA-A1, A2, BW16, B17—became available but proved incompatible on cross-matching, and transplantation was not performed. One month later a second cadaveric kidney—Group O HLA-A1, A2, B8, B15, CW4—also proved incompatible, but after a further six months a compatible cadaveric kidney—group O HLA-A1, A9, BW21, BW35, CW4—was found and was successfully transplanted.

Further investigation of the patient's serum showed that all cytotoxic activity against the lymphocyte panel was removed by absorption with lymphocytes (5% by volume) from the donor of the venous allograft. Cytotoxicity remained, however, after the serum was absorbed with lymphocytes from three unrelated subjects whose HLA types were as follows: (1) HLA-A2, AW30, B15, BW21; (2) HLA-A1, A3, B17, B27; (3) HLA-A2, A11, B15, BW35.

Comment

The development of cytotoxic antibodies after the insertion of a venous allograft has not been reported. In dogs transplantation antigens have been detected in vascular endothelium,³ but they appear to be only weakly antigenic.⁴ In man little information is available, and possibly, of course, in this case "passenger" leucocytes trapped in the interstices of the graft stimulated the antibody response.

It has been suggested that ABO compatibility between donor and

recipient may prolong the survival of venous allografts,⁵ but there is no information on the effect of HLA matching. In this case no such tissue typing was performed before operation and cytotoxic antibodies were produced with important consequences to a patient awaiting transplantation.

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(Accepted 21 April 1978)

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Antibodies to *Yersinia enterocolitica* serotype 3 in thyroid disease

Though the presence of serum agglutinins to *Yersinia enterocolitica* serotype 3 has been reported in various thyroid disorders,^{1,2} their role is not clear. This association has not been described in these islands, an area where infection with *Y enterocolitica* serotype 3 is unusual. We report the presence of antibodies to *Y enterocolitica* serotype 3 in a girl with thyrotoxicosis, and the absence of these antibodies in her family and in 19 other patients with thyroid disease.

Patients and methods

Twenty (19 women, one man) consecutive new patients with thyroid disease were investigated (aged 14-75). Thyroid function was assessed by measuring T4, TSH, and T3 resin uptake, and by the T3 suppression test. Thyroid scan was performed in selected cases. Fourteen patients had Graves's disease; two a single toxic nodule; two a multinodular toxic goitre; one Hashimoto's thyroiditis; and one idiopathic hypothyroidism. Serological tests were performed for antibodies: (1) to thyroid cytoplasm and colloid by an immunofluorescent method; (2) to thyroglobulin by the tanned cell agglutination test; (3) to *Y enterocolitica* serotype 3 by an agglutination test. These serological tests were also carried out on sera from 20 matched controls (19 women, one man; ages 16-76). Results are shown on the table.

One patient had *Yersinia* antibodies in her serum.

Case report—A 13-year-old schoolgirl (patient 2 in table) had a four-month history of unilateral exophthalmos. Clinically and biochemically she was euthyroid and she had no goitre. Tests with thyroglobulin-tanned cell agglutinins were positive at a dilution of 1/40. The results of tests for other thyroid autoantibodies were negative. The exophthalmos progressed over the next three months, but improved subsequently. Fifteen months after her

Titres of thyroid antibodies where present

Patient No	Clinical diagnosis	Thyroid antibodies		
		Thyroglobulin	Colloid	Cytoplasmic
2	Graves's disease	1/40	0	0
3	Multinodular toxic goitre	1/40	0	0
5	Graves's disease	0	0	< 1/20
8	Graves's disease	0	0	< 1/20
10	Single toxic nodule	0	0	< 1/20
16	Graves's disease	0	0	< 1/20
17	Hashimoto's disease	ND*	ND	ND
18	Graves's disease	1/320	0	0
Controls (9)		7: 1/20 2: 1/80	0	0

*ND = Not done.