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

- ¹ Main, R A, and White, M I, *Practitioner*, 1977, **219**, 456.
- ² Sneddon, I B, *Prescriber's Journal*, 1974, **14**, 1.
- ³ Winner, H I, and Hurley, R, (editors), *Symposium on Candida Infections*, p 117. Edinburgh, Livingstone, 1966.
- ⁴ Fisher, A A, *Contact Dermatitis*, 1965, **1**, 266.
- ⁵ White, M I, 1978, personal communication.

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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.

- ¹ *British Medical Journal*, 1977, **2**, 1373.
- ² Gurland, H J, et al, *Dialysis, Transplantation, Nephrology*, 1976, **13**, 45.
- ³ Vetto, R M, and Burger, D R, *Transplantation*, 1971, **11**, 374.
- ⁴ Schwartz, S I, et al, *Surgery*, 1967, **61**, 471.
- ⁵ Oschner, J L, DeCamp, P T, and Leonard, G L, *Annals of Surgery*, 1971, **173**, 933.

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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.

initial presentation she became frankly thyrotoxic with a T4 concentration of 18.2 mg/dl (normal=3.8-12.2 mg/dl) and a T3 resin uptake of 77% (normal=15-45%). Antibody to *Y enterocolitica* serotype 3 was present at a significant titre of 1/160. Over the next nine months her thyrotoxicosis was difficult to control with carbimazole and she developed a smooth goitre. Subtotal thyroidectomy was performed. At the time of thyroidectomy, antibody to *Y enterocolitica* serotype 3 was present in her serum at a dilution of 1/80. No antithyroid antibodies were detected. Immunofluorescence studies on the excised gland and normal thyroid tissue failed to detect auto-antibodies. No bound antibodies were detected.

Family study—No *Yersinia* antibodies were detected in her parents and four of her siblings, all of whom were euthyroid.

Comment

Actual infection with *Yersinia* may be related in some way to the development of thyroid disease. Partially supporting this concept is the observation that clinical infections with *Yersinia* have been followed by the development of various autoantibodies, directed against various tissues including the basement membrane of thyroid epithelial cells.^{2,3} Against this view is the finding of antibodies to *Y enterocolitica* serotype 3 in various thyroid diseases that are unlikely to have the same cause.^{1,2} These observations seem to suggest that the finding of *Y enterocolitica* serotype 3 antibodies in thyroid disease is incidental. A puzzling feature, however, is that antibody to *Y enterocolitica* serotype 3 has been detected in the sera of patients with thyroid disease from areas of both high¹ and low^{2,4} incidence.

Y enterocolitica serotype 3 has a high infectivity. The lack of antibodies in the patient's family may represent the absence of infection or the decline of antibodies to undetectable titres after infection. *Yersinia* antibodies cross-reacting with thyroid tissue were not detected. This is in contrast to the findings of Lidman *et al*³ in patients with yersiniosis and no evidence of thyroid disease.

¹ Bech, K, *et al*, *Lancet*, 1974, **2**, 951.

² Shenkman, L, and Bottone, E J, *Annals of Internal Medicine*, 1976, **85**, 735.

³ Lidman, K, *et al*, *Lancet*, 1974, **2**, 1449.

⁴ Keddie, N, Metcalfe-Gibson, C, and Tooth, J, *Lancet*, 1977, **2**, 1368.

⁵ Toivanen, P, *et al*, *Lancet*, 1973, **1**, 801.

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Motorcycle spoke injury

Spoke injury is a hitherto unreported aspect of motorcycle trauma that affects pillion riders. This report describes 21 cases seen in Karachi from October 1976 to February 1978. Typically the victim was seated astride the pillion of a lightweight motorcycle. A sudden jolt dislodged his foot from the footrest, and on the right side the heel readily slipped into the unprotected rear wheel spokes. On the left side the heel was saved by the motorcycle chain-case guarding the spokes.

Series

The victims were all males, aged 10-70 years. Five had not been on a motorcycle before. In some cases three people were riding in tandem on a motorcycle, and the back passenger was injured. A broken right footrest

was the predisposing factor in five cases. Jolts were responsible for displacing the foot in most cases. They occurred when the vehicle braked abruptly, cornered, swerved, or hit a pothole on the road.

The injuries were essentially of a stripping type at the back of the heel. Nine patients suffered heel lacerations. Five had partial heel avulsions, the heel flaps being attached caudally and medially. Complete heel avulsion with substantial loss of skin and subcutaneous tissue occurred in six patients. One patient virtually had a guillotine amputation of the back of the heel. The underlying bone and tendon were exposed and damaged to a variable extent. One case was complicated by extensive cellulitis of the leg.

Sixteen of the patients were admitted to hospital, and 14 stayed for 12 to 104 days (mean 41.9 days). Five of these patients underwent delayed cross-limb pedicle grafting and remained in hospital for 77 to 104 days (mean 86.0 days).

Rear wheel of motorcycle with suggested shield in place.

Comment

Viljanto¹ recently reported 103 bicycle and moped spoke injuries in children (1-14 years) riding pillion. Both feet were affected, the heel being injured in 22 cases. Fracture of the leg bones on the affected side occurred in 17 cases. Drewes and Schulte² were the first to draw attention to leg bone fractures occurring as a result of bicycle spoke injuries. Analysing 211 fractures of the leg bones in children aged 1 to 14 years they found that nearly 10% of the fractures were due to bicycle spoke injuries. Among the 1- to 4-year-olds (51 cases) bicycle spoke injuries accounted for one-third of the fractures. The higher speed of the motorcycle and its rigid spokes were responsible for the more extensive heel injuries reported here. There were, however, no fractures in this series, possibly because an insufficient force was transmitted upwards. Alternatively, the force might have been better withstood by the older victims.

Spoke injury has come to light following a fourfold increase in the number of registered two-wheelers in Karachi between 1966 and 1977.³ To prevent it the practice of three people riding on a motorcycle should be discouraged, as should the use of vehicles with broken footrests. Boots should offer protection but may be uncomfortable in a hot climate. Women in Karachi prefer to sit side-saddle with both their feet on the left side of the motorcycle; though it safeguards against spoke injury, this practice exposes them to other dangers.

Drewes and Schulte² found that shields covering the upper parts of the bicycle's rear wheel usually prevented spoke injuries in children. The motorcycle chain-case serves as a shield and safeguards the passenger's left heel. To protect the right heel a similar shield should be provided on that side (see figure).

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¹ Viljanto, J, *Annales Chirurgiae et Gynaecologiae*, 1975, **64**, 100.

² Drewes, J, and Schulte, H D, *Der Chirurg*, 1965, **36**, 464.

³ Office of the Excise and Taxation officer, Motor Registration, Karachi 1978. Personal Communication.

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