Pseudomonas aeruginosa was repeatedly isolated. The *P aeruginosa* was treated with a 10-day course of gentamicin. Radical curettage of the subtalar joint was performed and during the next year the discharge stopped and radiography showed diminution of cavity size with ankylosis. She subsequently walked well.

Intradermal skin tests for *M fortuitum* were performed using ranin 1, 2, and 4 and chelonin, which are antigens specific for *M fortuitum* serotypes 1, 2, and 4 and *M chelonei* respectively. A 0·1 ml volume of each was injected, and after 72 hours she had areas of induration 6 mm in diameter at the site of the serotype 4 injection but less than 3 mm at the *M chelonei* site, and no response at the other two sites. Induration of 5 mm or more is considered to be positive.

Comment

M fortuitum is the commonest of the Runyon group IV rapidly growing mycobacteria. It may cause pulmonary, lymph-gland, soft-tissue, corneal, heart-valve, and prostatic infections.[1] In bone vertebral abscesses, medial sternotomy infections, and osteomyelitis of a phalanx after a scraping and of a calcaneum after a puncture wound have been reported. Ohry *et al*[3] described a case in which *M fortuitum* osteomyelitis occurred after major trauma with open fractures. Wegmann *et al*[2] reported the case of a woman who had suffered from peritoneal and cervical tuberculosis, poliomyelitis, hepatitis, and ankylosing spondylitis: she had received several cortisone injections to her right shoulder over some years. She presented with *M fortuitum* arthritis in her right shoulder and ankle.

Antibiotic resistance is always a problem with *M fortuitum*. The standard drugs for *M tuberculosis* are rarely useful. Ethionamide, capreomycin, cycloserine, and the aminoglycosides[4] may occasionally be useful. In our case there was borderline sensitivity to cycloserine and also to kanamycin, so possibly the gentamicin, given for pseudomonas infection, may have helped to eliminate the *M fortuitum*. In a review of published reports Halpern and Nagel[5] concluded that incision and debridement are the mainstay of effective treatment. We assume that the infection in our case was due to contamination of an arthritic joint at the time of the first operation.

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1 Wolinsky E. Nontuberculous mycobacteria and associated diseases. *Am Rev Respir Dis* 1979;**119**:107-159.
2 Wegmann T, Jutz P, Pagon S. Atypische Mykobakteriose, hervorgerufen durch Mycobacterium fortuitum. *Schweiz Med Wochenschr* 1978;**108**: 684-6.
3 Ohry A, Brooks M, Steinbach T, Rozin R. Osteomyelitis caused by Mycobacterium fortuitum. *Infection* 1977;**5**:46-8.
4 Nicholson DP, Savier WR. Mycobacterium fortuitum as a pathogen. A case report. *Am Rev Respir Dis* 1971;**104**:747-50.
5 Halpern AA, Nagel DA. Mycobacterium fortuitum infections: a review with two illustrative cases. *Clin Orthop* 1978;**136**:247-52.

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Prostacyclin infusion in haemolytic-uraemic syndrome of children

Pathological intravascular platelet aggregation plays a major role in the pathogenesis of the microangiopathy of the haemolytic-uraemic syndrome and thrombotic thrombocytopenic purpura. Deficiency of endogenous prostacyclin (PGI_2), a potent inhibitor of platelet aggregation, has been shown in both conditions,[1 2] and this could favour the formation of platelet thrombi in the microcirculation. PGI_2 replacement seems a logical therapeutic manoeuvre, but results in adults have been variable.[1-3] We report our experience with PGI_2 infusion in three children with the haemolytic-uraemic syndrome.

Case reports

Case 1—An 18-month-old girl was admitted after a diarrhoeal illness. On admission the blood pressure was 90/50 mm Hg, she was oliguric (20 ml/day), serum creatinine concentration was 400 μmol/l (4·52 mg/100 ml), platelet count was 25×10^9/l, serum concentration of fibrin degradation products (FDP) was 80 mg/l, and burr cells were present on the peripheral blood film. Peritoneal dialysis was carried out for seven days. On the sixth day in hospital PGI_2 infusion was started at an initial dose of 2·5 ng/kg/min and increased to 30 ng/kg/min within nine days. The total duration of the infusion was 12 days. Diuresis started on the 15th day in hospital, with progressive improvement in renal function. The platelet count rose to 150×10^9/l by the 14th day and the serum FDP concentration fell to normal by the 19th day. Five weeks after admission both blood pressure and renal function were normal.

Case 2—A 2-year-old girl was admitted after a diarrhoeal illness. On admission the blood pressure was 120/70 mm Hg, she was anuric, serum creatinine concentration was 520 μmol/l (5·87 mg/100 ml), platelet count was 120×10^9/l, serum FDP concentration was 160 mg/l, and burr cells were present on the peripheral blood film. Peritoneal dialysis was carried out for 10 days. On the fourth day in hospital PGI_2 infusion was started at an initial dose of 2·5 ng/kg/min, increasing to 40 ng/kg/min within seven days. The total duration of the infusion was 10·5 days. Diuresis began on the 14th day with progressive improvement in renal function. The platelet count rose to 300×10^9/l and the serum FDP concentration fell to normal by the 16th day. Ten weeks after admission the blood pressure was normal and the ^{51}Cr-EDTA clearance was 69 ml/min/1·73 m^2.

Case **3**—A 5-year-old girl was admitted after a diarrhoeal illness. On admission the blood pressure was 125/85 mm Hg, she was oliguric (10 ml/day), serum creatinine concentration was 540 μmol/l (6·1 mg/dl), platelet count was 25×10^9/l, serum FDP concentration was 20 mg/l, and burr cells were present on peripheral blood film. Peritoneal dialysis was carried out for seven days. On the second day in hospital PGI_2 infusion was started at an initial dose of 2·5 ng/kg/min, increasing to 55 ng/kg/min within 12 hours. The total duration of the infusion was eight days. Diuresis began on the seventh day in hospital, with progressive improvement in renal function. The platelet count rose to 250×10^9/l on the ninth day and the serum FDP concentration fell to normal on the 12th day. Four weeks after admission the blood pressure was normal and the ^{51}Cr-EDTA clearance was 58 ml/min/1·73 m^2.

Comment

Activity of the prostacyclin was verified by testing the ability of aliquots of the solution to inhibit aggregation of normal platelets induced by adenosine diphosphate. The infusions were delivered via a central venous catheter, no antiplatelet agents were used, and no antihypertensive agents were required during treatment. Side effects were minimal and controllable. One patient developed intermittent drowsiness and vomiting; hypotension and bradycardia occurred only at a dose of more than 50 ng/kg/min and rapidly resolved after reduction in the rate of the infusion. The dose range (30-50 ng/kg/min) was appreciably greater than the maximum dose tolerated by adult patients with the haemolytic-uraemic syndrome (6 ng/kg/min)[1] and thrombotic thrombocytopenic purpura (16 ng/kg/min).[2]

Although these data are preliminary, they show the safety of this treatment in association with frequent blood-pressure monitoring. It is not possible to draw any conclusions on its success; the short oliguric phase in the last case, however, may have been related to the early introduction of the infusion at high dose. We feel sufficiently encouraged at this stage to continue with further evaluation of this treatment. Further experience is, however, essential, preferably on a controlled basis, before any recommendations on its use can be made.

Prostacyclin was synthesised by the Upjohn Company, formulated by the Wellcome Foundation, and kindly supplied by Dr J O'Grady.

1 Webster J, Rees AJ, Lewis PJ, Hensby CN. Prostacyclin deficiency in haemolytic uraemic syndrome. *Br Med J* 1980;**281**:271.
2 Hensby CN, Lewis PJ, Hilgard P, *et al*. Prostacyclin deficiency in thrombotic thrombocytopenic purpura. *Lancet* 1979;ii:748.
3 Budd GT, Bukowski RM, Lucas FV, *et al*. Prostacyclin therapy of thrombotic thrombocytopenic purpura. *Lancet* 1980;ii:915.

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