toxigenic strains of Staph aureus that produced enterotoxin F were isolated from high vaginal swabs from each patient (see table). Treatment with intravenous fluids and appropriate anti-staphylococcal antibiotics was followed by complete clinical resolution.

**Cases of toxic-shock syndrome: clinical features and properties of Staphylococcus aureus strains isolated**

<table>
<thead>
<tr>
<th>Case</th>
<th>Date of presentation</th>
<th>Age (years)</th>
<th>Temperature &gt;38°C</th>
<th>Scarletinaiform rash</th>
<th>Myalgia</th>
<th>Conjunctival hyperaemia</th>
<th>Vaginitis on admission</th>
<th>Symptoms associated</th>
<th>Tampon present</th>
<th>Phage type</th>
<th>Enterotoxin detected</th>
<th>Staph aureus strain isolated</th>
<th>Site of isolation</th>
<th>Presence of Enterotoxins in swabs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case 1</td>
<td>Dec 1980</td>
<td>18</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>High vaginal swab</td>
<td>A+F</td>
<td>B+D+F</td>
<td>A+F</td>
<td></td>
</tr>
<tr>
<td>Case 2</td>
<td>Oct 1981</td>
<td>18</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>High vaginal swab</td>
<td>A+F</td>
<td>B+D+F</td>
<td>A+F</td>
<td></td>
</tr>
<tr>
<td>Case 3</td>
<td>Nov 1981</td>
<td>18</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>High vaginal swab</td>
<td>A+F</td>
<td>B+D+F</td>
<td>A+F</td>
<td></td>
</tr>
<tr>
<td>Case 4</td>
<td>March 1982</td>
<td>35</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Breast abscess</td>
<td>A+F</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* A phage group II strain which produced epidermolysis toxin A was also isolated from vaginal and nose swabs.
* A non-typable strain that produced enterotoxin F alone was isolated from the nose of this patient.

**Case 4—A 35-year-old Filipino woman presented with a two-day history of pain in the right breast, fever, vomiting, and profuse watery diarrhea three weeks after forcible delivery of a healthy infant. While in the maternity hospital, the baby was reported to have developed a sticky eye, but no bacteriological report was available. On examination the patient had a fever of 39°C, her blood pressure was 80/50 mm Hg, and her right breast was diffusely tender. She had severe muscle tenderness but normal results of vaginal examination were normal for three weeks post partum. Investigations showed a neutrophil leucocytosis with a platelet count of 75 × 10⁹/l, abnormal electrolyte values (sodium 128 mmol/l, urea concentration 29.5 mmol/l, (177 mg/100 ml), and creatinine concentration 583 mmol/l (5.9 mg/100 ml). Serum creatinine phosphokinase activity was raised at 332 IU/l, calcium concentration at 1.6 mmol/l (6.5 mg/100 ml), and alkaline phosphatase activity 234 IU/l. Blood cultures, high vaginal swabs, faeces, and throat swabs collected on admission yielded only Staph aureus or other pathogens. Septicaemic illness or toxic-shock syndrome was diagnosed and she was given intravenous antibiotics and fluids. On day 1 she passed only 300 ml urine and developed a macular rash on face and trunk. On day 3 her breast became more swollen and an abscess was drained: the pus yielded a pure heavy growth of *Staph aureus* (phage type 29/2). She improved rapidly and on day 9 showed the characteristic skin desquamation on palms, soles, and face. On discharge her renal function was normal and abscess wound dry. The strain was subsequently shown to produce enterotoxins A and F.

**Comment**

That three girls presented with tampon-associated toxic-shock syndrome in this area within 11 months indicates that the syndrome may not be as rare as generally believed. There is, however, a much higher proportion of young women than average in the St Stephen's Hospital catchment area. There has been a suggestion that the cellular activity of certain bacteria on coagulase-negative staphylococci may occur in vivo and that this may contribute to the pathogenesis of toxic-shock syndrome. Interestingly coagulase-negative staphylococci was not contained in the tampons used by our patients. Enterotoxin F is considered to be important in the aetiology of toxic-shock syndrome. Enterotoxin of the *Staph aureus* isolates from our patients showed that they belonged to different enterotoxin-F-producing strains.

There have been reports from the United States of toxic-shock syndrome postpartum. These cases were often associated with the use of tampons. Our case 4 was not associated with tampons and is the first reported case during the peripuerium in Britain.

We thank Dr B G Gazzard, Dr E N Coomes, and Professor A F Lant for allowing these cases to be reported, and Miss A A Wieneke, of the Food Hygiene Laboratory, Colindale, for testing our strains for enterotoxin production.


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**Failure of rifampicin and co-trimoxazole in Q fever endocarditis**

Information on the response of Q fever endocarditis to the newer antibiotics is slow to accumulate because of the rarity of the disease. Recently, the primacy of tetracycline in its treatment has been challenged, at least in vitro, and by one clinical success with rifampicin, and by co-trimoxazole as a potential first choice. We report the complete failure of these and other antibiotics even to suppress Coxiella burnetii infection in a patient in whom tetracycline could not be used.

**Case report**

A male nurse aged 38 years was referred to this unit because of the recurrence of fever with embolic phenomena four weeks after a culture-negative endocarditis had apparently been successfully treated with penicillin and gentamicin. The underlying lesion was a discrete sub-valvar aortic stenosis. Blood culture results again were negative but echocardiography showed massive highly mobile echoes in the aortic root; the complement fixation test result for Q fever was positive at a titre of 1/320 (phase 1 and 2).

Although the initial response to tetracycline was favourable the drug had to be withdrawn after a week because of a severe pancreatitis and hepatic and renal failure. Treatment was continued with co-trimoxazole alone (960 mg three times daily). The immediate clinical response seemed very satisfactory, but after 10 months of continuous treatment there was relapse with fever, Osler's nodes, and a rise in Q fever titres to 1/640 (phase 1) and 1/5120 (phase 2). Lincomycin 2 g daily was added to his regimen with a dramatic initial response followed by relapse after one month of treatment. Rifampicin 600 mg daily was substituted for the lincomycin, the co-trimoxazole being continued throughout. The immediate clinical response was good, but since this seemed likely to be due to the regimen, the patient was switched to the alternative new drug. Blood cultures were again negative but echocardiography showed massive highly mobile echoes in the aortic root; the complement fixation test result for Q fever was positive at a titre of 1/320 (phase 1 and 2).

Although the increase in Q fever titres to 1/640 (phase 1) and 1/5120 (phase 2). Lincomycin 2 g daily was added to his regimen with a dramatic relapse two months after operation he felt well but the Q fever titre remained high and a diastolic murmur, not heard before the operation, persisted throughout. The immediate clinical response was good, but since this seemed likely to be due to the regimen, the patient was switched to rifampicin, and the co-trimoxazole was continued and after two weeks the murmur cleared and the Q fever titre fell to 1/160. Although the patient was readmitted with florid endocarditis and gross aortic reflux. There was no response to either lincomycin or rifampicin and the patient died two weeks later, after 16 months of continuous treatment with co-trimoxazole and five months with rifampicin and co-trimoxazole in combination. At necropsy there was extensive dehiscence of the prosthesis with massive clots and many fresh vegetations composed microscopically of clumps of *C. burnetii*.
Nitrofurantoin-induced parotitis

Nitrofurantoin is known to cause toxic and type III cell-mediated allergic reactions. The risk of these increases with age and is more likely to occur in women than in men. Holmberg at al.1 divided the side effects of nitrofurantoin into six categories: (a) acute pulmonary reactions; (b) chronic pulmonary reactions; (c) allergic reactions, including various types of cutaneous manifestations (exanthema, erythema, urticaria), fever, and anaphylactic reactions; (d) liver damage and gastrointestinal disturbances; (e) blood dyscrasias; and (f) neuromuscular. We report a parotitis-like clinical condition induced by nitrofurantoin.

Case report

A 78-year-old woman was admitted with acute onset of bilateral painful swelling of the parotid gland, dry mouth, and fever of 39°C. She gave a history of allergic reactions to tetracycline, doxycycline, trimethoprim, and lincomycin. On admission she had a parotid swelling. Pulmonary auscultation indicated minor basal rales. Thoracic x-ray films showed an enlarged heart, pulmonary venous congestion, and interstitial extravasation. The only abnormal laboratory findings were raised erythrocyte sedimentation rate (35 mm/hour), moderate leucocytosis (12.4 x 10⁹/l), and raised serum amylose activity (553 U/l; normal range < 300 U/l). The fever, parotid swelling, leucocytosis, and raised serum amylose activity disappeared within two days; pulmonary auscultation showed no abnormalities. Hydrochlorothiazide was substituted for frusemide 80 mg daily. The patient had no history of mumps, and this was the diagnosis made on discharge four days after admission.

We are most grateful to Dr W P G Turck, for his advice in this case, to Dr I Weinbren and Dr F Costello, for their help in the management of the hepatic and renal failure, and to Dr D Harry for his detailed pathological studies.


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Comment

That Q fever endocarditis can be intractable is well recognised and recurrence after valve replacement well known. Nevertheless, good results are reported with tetracycline1 even in the difficult field of prosthetic endocarditis, though treatment may have to be continued for five or more years. Tetracycline could not be used in our patient, and the striking feature was the florid nature of the relapses occurring after an initial response to each of the antibiotics used. At operation and necropsy vegetation was luxuriant and C burnetii present in abundance despite prolonged and continuous treatment with co-trimoxazole, rifampicin, and lincomycin.

Where success has been reported with these drugs—2,3 they have been used in combination with or after treatment with tetracycline. We consider that there is still no acceptable substitute for tetracycline as the mainstay of treatment in Q fever endocarditis. Should circumstances arise again where tetracycline could not be used in a patient with Q fever endocarditis increased doses of rifampicin in the presence of normal liver function or even the use of chloramphenicol might be considered.


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