Ψ

- ³ Pyle RL, Mitchell JE, Eckert ED. Bulimia: a report of 34 cases. J Clin Psychiatry 1981;42(2):60-4.

 Palmer RL. The dietary chaos syndrome: a useful new term? Br J Med
- Psychol 1979;52:187-90.
- ⁵ Diagnostic and statistical manual of mental disorders, 3rd ed. Washington, DC: American Psychiatric Association, 1980.
- ⁶ Crisp AH. Anorexia nervosa at normal body weight. The abnormal normal weight control syndrome. Int J Psychiatry Med 1982;11(3): 203-33
- ⁷ Lacey JH. The bulimic syndrome at normal body weight: reflections on pathogenesis and clinical features. International Journal of Eating Disorders 1982;2(1):59-66.
- * Crisp AH, Palmer RL, Kalucy RS. How common is anorexia nervosa? A prevalence study. Br J Psychiatry 1976;128:549-54.
- Lacey JH, Chadbund C, Crisp AH, Whitehead J, Stordy J. Variations in energy intake of adolescent girls. J Hum Nutr 1978;32:419-26.
 Fairburn CG, Cooper PJ. Self-induced vomiting and bulimia nervosa: an
- undetected problem. Br Med J 1982;284:1153-5.

- 11 Halmi KA, Falk JR, Schwartz E. Binge-eating and vomiting: a survey of a college population. Psychol Med 1981;11:697-706.
- 12 Garner DM, Garfinkel PE, Bemis KM. A multidimensional psychotherapy for anorexia nervosa. International Journal of Eating Disorders 1981;2:3-46.
- ¹³ Fairburn CG. A cognitive behavioural approach to the management of bulimia. Psychol Med 1981;11:707-11.
- 14 Feighner JP, Robins E, Guza SB, Woodruff RA, Windur G, Munoz R. Diagnostic criteria for use in psychiatric research. Arch Gen Psychiatry 1972;26:57-63.
- ¹⁵ Crisp AH. Anorexia nervosa. Hospital Medicine 1967;1:713-8.
- ¹⁶ Aitkin RCB, Zealley AK. Measurement of moods. Hospital Medicine 1970;1:215-44.
- ¹⁷ Kemsley WFF. Annals of Eugenics 1953;16:316-34.
- 18 Lacey JH. The patient's attitude to food. In: Lessof MH, ed. Clinical reactions to food. Chichester: John Wiley, 1983.

(Accepted 14 March 1983)

SHORT REPORTS

Toxic shock syndrome and endocarditis

The toxic shock syndrome is known to be associated with a variety of staphylococcal infections unrelated to the use of tampons or to menstruation.1 We report a case in a patient who had staphylococcal endocarditis.

Case report

The patient was a 21 year old woman who had been having psychiatric treatment for intravenous drug abuse for the past year. She had had amenorrhoea for three months but was not pregnant. The illness started with fever of 40.4°C, severe myalgia, and non-productive cough. On admission she was drowsy, pale, and dehydrated and her blood pressure was 90/50 mm Hg (supine). On clinical examination a soft systolic murmur was heard at the left sternal edge with widespread bilateral crepitations and a harsh pleural rub at the left base. Echocardiography showed vegetations and thickening of the tricuspid valve, and electrocardiography showed a sinus tachycardia of 110 beats/min with extensive T wave inversion in anterior and lateral leads. Patchy consolidation was present at both bases on a chest x ray film, and the left diaphragm was raised.

A provisional diagnosis of endocarditis of the tricuspid valve with pulmonary embolisation was confirmed by the isolation of Staphylococcus aureus from all of four blood cultures. Laboratory investigations showed renal dysfunction with raised blood urea and creatinine concentrations (26.4 mmol/l (159 mg/100 ml) and 220 μ mol/l (2·5 mg/100 ml), respectively). There was moderate anaemia (haemoglobin concentration 8.9 g/dl), and platelets were low at 85×10^9 /l. Results of liver function tests were normal.

Despite aggressive treatment to achieve volume expansion and correct shock, systolic blood pressures below 90 mm Hg were recorded several times in the 24 hours after admission. Thereafter the blood pressure stabilised. She was treated with intravenous cloxacillin (12 g daily) and gentamicin (240 mg daily) but remained feverish for nine days. Blood cultures collected during this period remained sterile. Rifampicin 900 mg daily was then substituted for gentamicin and she became afebrile two days later. Eleven days after the onset of illness an erythematous rash appeared over her fingers, and two days later the skin desquamated over the distal two phalanges of the fingers of both hands. She discharged herself on the 28th day but was believed to have remained well.

Bacteriology—The S aureus isolated was phage group II (type 3A/3C/55) 71) and produced enterotoxin F. It was resistant to penicillin but sensitive to methicillin, vancomycin, gentamicin, and rifampicin. Minimal inhibitory and bactericidal concentrations for the isolate (table) showed in vitro toler-

Sensitivity of S aureus associated with the toxic shock syndrome

Antibiotic	Minimal inhibitory concentration (mg/l)	Minimal bactericidal concentration (mg/l)
Cloxacillin	0·25	> 32
Gentamicin	0·35	0·35
Vancomycin	0·25	0·25
Rifampicin	0·007	0·007

ance to cloxacillin. Bacterial synergy could not be shown in vitro for either of the combinations of antibiotic ultimately used. Carriage of S aureus at other sites was not investigated until after treatment had started and proved negative.

Comment

We believe this to be the first recorded case of the toxic shock syndrome in association with staphylococcal endocarditis. Although the case was unusual as blood cultures yielded S aureus the patient had the classic features of the disease with fever, hypotension, desquamation, and evidence of multisystem involvement (myalgia, renal impairment, thrombocytopenia, and cardiac abnormalities) as defined by Bergdoll et al.2 The staphylococcus isolated was unusual since it belonged to phage group II, a group classically associated with production of epidermolytic toxins A and B and the scalded skin syndrome. It failed to produce these toxins but did elaborate enterotoxin F, which is characteristic of strains associated with the toxic shock syndrome.²

Tolerance to both nafcillin and vancomycin has been documented in staphylococci associated with the toxic shock syndrome but is rare.4 The clinical importance of in vitro tolerance remains controversial, but this phenomenon is more important when the syndrome is associated with endocarditis than it is in most cases, in which a bactericidal antimicrobial regimen is not mandatory.

The toxic shock syndrome has emerged as a multifactorial staphylococcal disease and should be considered in all patients with appropriate signs and symptoms regardless of their sex or menstrual state.

We thank Dr Maureen De Saxe, Central Public Health Laboratory, Colindale, and Dr Merlin Bergdoll, University of Wisconsin, Madison, for testing this staphylococcal isolate for epidermolytic toxins and enterotoxins.

- Reingold AL, Hargrett NT, Dan BB, Shands KN, Strickland BY, Broome CV. Non-menstrual toxic shock syndrome. Ann Intern Med 1982;96:871-
- ² Bergdoll MS, Crass BA, Reiser RF, Robbins RG, Davis JP. A new staphylococcal enterotoxin, enterotoxin F, associated with toxic shock syndrome. Lancet 1981;i:1017-21.
- ³ Bergdoll MS, Crass BA, Reiser RF, Robbins RG, Davis JP. An enterotoxin-like protein in Staphylococcus aureus strains from patients with toxic shock syndrome. Ann Intern Med 1982;96:969-71.
- ⁴ Chesney PJ, Crass BA, Pulyak MB, et al. Toxic shock syndrome—management and long term sequelae. Ann Intern Med 1982;96:847-51.

(Accepted 15 March 1983)

Departments of Bacteriology and Medicine, Royal Infirmary, Ĝlasgow G4 0SF

M WHITBY, MB, FRACP, senior registrar in microbiology S FRASER, MB, MRCP, registrar in medicine G GEMMELL, PHD, MIBIOL, senior lecturer in bacteriology P A WRIGHT, MB, MRCPATH, consultant microbiologist

Correspondence to: Dr M Whitby.