## A case of *Gemella morbillorum* endocarditis

*Sir,*

We report a case of *Gemella morbillorum* endocarditis. A 75-year-old man was admitted to hospital for investigation of weight loss and lassitude of two months duration. He had a history of rhematic fever as a child. Examination revealed a temperature of 38.5°C, mild ankle oedema and a pansystolic murmur radiating to the axilla. There were no other signs of endocarditis. He had a haemoglobin of 9.3 g/dl, white cell count of $9.7 \times 10^9$/l and an erythrocyte sedimentation rate of 48 mm/h. An echocardiogram revealed mitral valve vegetations with regurgitation. A diagnosis of endocarditis was made, three sets of blood cultures were taken and he was started on benzylpenicillin 1.2 g iv 4 hourly and gentamicin 80 mg iv 12 hourly. His temperature returned to normal after 24 hours. At 48 hours all three sets of blood cultures grew *G morbillorum* (confirmed by the Streptococcal Reference Laboratory). The organism proved sensitive to penicillin on disc testing but failed to survive subculture for further sensitivity studies. After 24 days of benzylpenicillin a bright red maculopapular rash appeared over the patient's trunk and legs and a leucopenia of $1.8 \times 10^9$/l was noted. This was consistent with an allergic reaction and treatment was stopped. His temperature rose to 39°C within 48 hours. Further cultures were taken and he was given teicoplanin. After the first dose of this he became dizzy, wheezy and breathless. His blood pressure fell to 90/60 mmHg. He was given iv hydrocortisone, chlorpheniramine and salbutamol. In view of his reaction to teicoplanin we decided not to risk treatment with vancomycin and opted instead for rifampicin 600 mg orally 12 hourly and erythromycin 500 mg orally 6 hourly. The organism had appeared sensitive to both these drugs on disc testing. The patient received a total of four weeks treatment and made a good recovery. He remains well six months later.

*G morbillorum* is closely related to the 'viridans' type streptococci and is considered a member of the normal upper respiratory tract flora. Infections caused by this organism seem to resemble those caused by viridans streptococci. Bacteraemia and endocarditis seem to predominate[1] although meningitis[2] and septic arthritis[3] have been reported. Osman[4] reports five cases of *G morbillorum* endocarditis. We report a sixth. Therapy with penicillin and gentamicin is the treatment of choice as most isolates show synergy although tolerance has been demonstrated.[5] Coto[6] presents a case in which penicillin plus rifampicin was successful after penicillin plus amikacin had failed. Glycopeptide therapy is recommended in those allergic to beta-lactams. Since our patient could not tolerate these, a combination of rifampicin plus erythromycin was selected and success achieved. Peard[7] reports one case where this combination has been used successfully against *Staphylococcus aureus* endocarditis in which good synergy was demonstrated. We believe this combination is unique in the treatment of 'viridans' type streptococcal endocarditis.

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**Learning points**

- *Gemella morbillorum* is closely related to the streptococci and is a rare cause of endocarditis
- penicillin plus an aminoglycoside is the treatment of choice
- rifampicin plus erythromycin is an alternative combination in penicillin-allergic patients

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## *Gemella haemolysans* prosthetic valve endocarditis

*Sir,*

We report a case of prosthetic valve endocarditis caused by *Gemella haemolysans* in a 34-year-old man who has had four cardiac operations including valve replacement for complex heart disease. We believe this to be the first reported case in the UK.

A 34-year-old man presented with a short history of myalgia, fever, and general malaise. He recognised these symptoms as possibly being endocarditis, having had four previous episodes. The only positive findings on examination were three sites of asymptomatic dental sepsis. Transoesophageal echocardiography showed a lesion on his prosthetic aortic valve suggestive of a vegetation. *G haemolysans* was isolated from several blood cultures. *G haemolysans* is sensitive to penicillin and synergy with gentamicin has been demonstrated.[1] This option was not open to us as, three years previously, the patient had profound neutropenia, including bone marrow eosinophilia, during another episode of endocarditis treated by benzylpenicillin and gentamicin. The neutropenia resolved on withdrawal of benzylpenicillin. Based on blood culture sensitivities treatment was begun with intravenous cefuroxime and tobramycin; the latter was stopped after 14 days and oral ciprofloxacin substituted. After a further five days the cefuroxime was stopped because the patient developed a profound neutropenia, and oral erythromycin started. Marrow examination revealed a decreased number of myeloid cells, with very little development beyond the myelocyte stage, and an increased number of eosinophils. Erythroid cells and megakaryocytes were normal. The neutrophil count recovered spontaneously over the next four days.

Six cases of endocarditis due to *G haemolysans* have been reported since 1982,[2] but only one case of prosthetic valve endocarditis.[3] The recommended treatment is a combination of penicillin and aminoglycoside.[1,2] In this case management of the patient was complicated by drug-induced neutropenia forcing a change in therapy. Despite this the patient made a good recovery and remains well with no deterioration in his prosthetic valve function.

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## Aplastic anaemia or aplastic pancytopaenia?

*Sir,*

The term aplastic anaemia is an unsuitable and confusing term extensively used in the medical literature. It is still present in the recent editions of textbooks of internal medicine and haematology,[1,2] and in most medical journals.[3–5]

The term 'aplastic anaemia' refers to conditions in which a markedly hypocellular bone marrow results in pancytopaenia (anaemia, leukopaenia, and thrombocytopaenia).[1] However, from a terminological point of view, aplastic anaemia means anaemia due to aplastic bone marrow where this anaemia in the medical literature is termed pure red cell aplasia. So I suggest that aplastic anaemia should be used only when the aplasia involves a selective failure in the production of erythroid elements in the bone marrow causing anaemia and reticulocytopaenia. When pancytopaenia due to bone marrow aplasia is present, instead of the classic term aplastic anaemia, the term aplastic pancytopaenia should be used; it is both more descriptive, and more suitable.

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