Letters to the Editor

Gemella haemolysans: a rare and unusual cause of infective endocarditis

Sir,

Bacterial endocarditis is a serious complication of cardiac disease with a mortality of between 10% and 20%.¹ Mitral valve prolapse has been reported as the underlying cardiac abnormality in 10% of cases.² We report a case of endocarditis caused by *Gemella haemolysans* in a 74 year old man with mitral valve prolapse. We believe this to be the first case reported in the United Kingdom.

A 74 year old man diagnosed 10 years previously as having mitral regurgitation secondary to mitral valve prolapse was admitted with a 2-month history of non-specific myalgia and headaches. For 2 weeks he had had night sweats and rigors. He had not undergone any invasive procedures. On examination his temperature was 38.5°C. There was a faint macular rash on both legs and signs of mitral regurgitation. Echocardiography showed prolapse of the posterior leaflet of the mitral valve with a small mass attached suggestive of a vegetation. A Gram-variable coccus was isolated from all 6 blood cultures and endocarditis of the mitral valve was diagnosed.

Identification of the isolate was attempted using the API 20 Strep system and this suggested *Gemella haemolysans* or *Streptococcus morbillorum*. It was confirmed as *G. haemolysans* by the National Collection of Type Cultures, Colindale. The organism was sensitive to penicillin (minimal inhibitory and bactericidal concentrations of 0.015 mg/l and 0.03 mg/l respectively) and he was given intravenous benzyl penicillin and intravenous gentamicin daily. He became afebrile within 48 hours and remained so. After 2 weeks the penicillin was replaced by oral amoxycillin for 4 further weeks and the gentamicin given intramuscularly for 2 further weeks. Following cessation of antibiotics he continued to remain afebrile, with negative blood cultures.

Mitral valve prolapse is well recognized as predisposing to infective endocarditis. In this case the organism responsible for the infection, *G. haemolysans*, is most unusual. The organism is considered to be a commensal of the upper respiratory tract. The exact taxonomy of this species is disputed but *G. haemolysans* is related to the Streptococci and is biochemically similar to *Streptococcus morbillorum*,³ an organism which has also been reported as a cause of endocarditis.⁴ Infections caused by *G. haemolysans* were unknown until 1978–9 when three cases of endocarditis were reported in France.⁵ There have been further reports of *Gemella* infections from France, including two cases of septicaemia and a further case of endocarditis.⁶ A case of post-neurosurgical meningitis caused by *G. haemolysans* was reported from Oxford in 1985.⁷ The strains isolated in all of the reported cases were highly sensitive to penicillin and synergy between penicillin and gentamicin has been demonstrated.⁸ In this case the infection rapidly responded to antibiotic therapy and the patient continues to remain well with no deterioration of his mitral valve disease.

Acknowledgement

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Chlorhexidine-induced gastritis

Sir,

Acute gastritis is caused by a variety of ingestants and systemic conditions which have in common either a direct action on the surface epithelial cells or an indirect action on the mucous membrane through a reduction in blood flow and inhibition of the normal cellular turnover.¹ We report a case of gastritis induced by self ingestion of chlorhexidine gluconate 4% (Hydrex). To our knowledge, this has not previously been reported.

A 72 year old man was admitted to St Charles' Hospital

for investigation of palpitations and postural hypotension. He also suffered from Parkinson's disease but was well controlled on treatment.

The positive findings on examination included the classical features of Parkinson's disease and a postural drop in blood pressure. The latter quickly resolved on withdrawal of his diuretic therapy. Two days after admission he started vomiting, initially four times on one day and then intermittently over the following 10 days. This usually occurred in the mornings. He was not receiving any new medication.

Fibreoptic gastroscopy showed multiple erosions in the lower part of his stomach and first part of the duodenum. His oesophagus appeared normal. Histology confirmed an active atrophic gastritis with many helicobacter-like organisms present. A full blood count, biochemical screen and liver function tests were all normal. It transpired that every morning he washed himself on the ward with Hydrex. However, he also used this preparation as a mouth wash and then swallowed it. He said that he felt nauseated each time he did this.

He was advised to discontinue this practice and was given ranitidine. He made a full recovery with no further vomiting and repeat endoscopy after 6 weeks revealed resolution of his mucosal erosions with some mild residual antral gastritis.

Chlorhexidine is a bisbiguanide disinfectant which is effective against a wide range of vegetative Gram-positive and Gram-negative bacteria. It is most active at a neutral or slightly acidic pH. Hydrex is a 4% solution of chlorhexidine gluconate in detergent that is used in pre-operative skin preparation and hand washing.² Skin sensitivity, transient taste disturbance and oral desquamation have occasionally been reported with a variety of preparations of chlorhexidine.

There has been one report of an acute hepatitis and liver necrosis following ingestion of chlorhexidine gluconate in a suicide attempt.³ Because it is irritant, it is recommended that it should not be used on the brain, meninges, eyes, middle ear or other sensitive tissues.

There have been no previous reports of chlorhexidine-induced gastritis. This is presumably because the taste and consistency of concentrated preparations of chlorhexidine render them sufficiently unpalatable to discourage ingestion in the majority of cases. We have informed the manufacturers of Hydrex of our unusual observation in the hope that a recurrence of this problem can be avoided by providing a more visible written warning on the container.

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The cardiovascular effects of beta adrenergic agonist drugs administered by nebulization

Sir,

I read with interest the report of Flatt *et al.*¹ reporting the cardiovascular effects of inhaled bronchodilators. They clearly demonstrate the greater cardiovascular effects of fenoterol 5 mg compared with equal doses of salbutamol and terbutaline. They conclude that this is due to 'greater β_1 adrenoreceptor stimulation following fenoterol'. However, I think a more satisfactory explanation of their findings is that fenoterol has a greater systemic bioavailability than salbutamol or terbutaline (i.e. more fenoterol is absorbed).

The authors have assumed that the tachycardia and positive inotropic effects of the drugs are primarily due to cardiac β_1 adrenoreceptor stimulation. In studies of human subjects and tissues, the chronotropic and inotropic effects of salbutamol have been shown to be purely due to cardiac β_2 adrenoreceptor stimulation.^{2,3} Consequently, all the cardiovascular effects in the study of Flatt *et al.* could be attributable to cardiac β_2 adrenoreceptor stimulation and the greater responses seen after fenoterol could be due to greater absorption. Conclusive evidence of greater absorption of fenoterol comes from a previous study published by this group of workers,⁴ where fenoterol by metered dose inhaler produced more hypokalaemia than equal doses of salbutamol. Hypokalaemia induced by β -agonists is purely due to β_2 adrenoreceptor stimulation.⁵

The references cited by Flatt *et al.* to show β_1 -mediated effects of fenoterol also assume that chronotropic and inotropic effects are not mediated by β_2 adrenoreceptors whereas evidence exists to disprove that assumption in the species studied, i.e. in man^{2,3} and guinea pigs.⁶

Although the findings of differential cardiovascular effects of equal doses of these commonly used bronchodilators are important I think it should be recognized that probably all the cardiovascular effects can be attributed to β_2 adrenoreceptor stimulation. Consequently in order to avert these important side effects of bronchodilator treatment it would not be necessary to develop an agent with 'greater β_2 selectivity' but to develop a β_2 agonist which is less well absorbed.

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