A case of acute aortic valve endocarditis due to Erysipelothrix rhusiopathiae acquired from fish

MUHAMMAD ARIF

Introduction

Infective endocarditis is characterised by the microbiological inflammation of the linings of the heart chambers, valves and great vessels. It was described for the first time by Osler in 1885. Its estimated annual incidence is 22 cases per million population in England and Wales and 49 per million per year in the US, which may be an underestimation. It is usually diagnosed by finding 'typical' organisms for endocarditis on blood cultures. But sometimes very rare or 'atypical' organisms may be seen on blood culture and may be the cause of endocarditis.[1,2] In such cases the clinical presentation may be far from classical and the course of the illness may be very dramatic and unpredictable. *Erysipelothrix rhusiopathiae* is one of these rare causes of endocarditis, which requires a high index of suspicion and awareness among microbiologists and clinicians to be able to recognise it and treat it promptly.

Br J Cardiol 2003;**10**:392–4

Case report

I present a case of acute aortic valve endocarditis due to *E. rhusiopathiae*, acquired from fish and resulting in fatal consequences. A 73-year-old man, a resident of Ascot, Berkshire, was admitted as an emergency to his local hospital, Wexham Park Hospital, Slough, with a two-day history of increasing breathlessness, orthopnoea and paroxysmal nocturnal dyspnoea. He had been mildly unwell for the last 3–4 weeks and had suffered a weight loss of two stone, with malaise and more recently night sweats. The illness had started a few days after he accidentally cut his finger while skinning and filleting fish. It was not known which type of fish he was handling and where he had been fishing. Previously, he had usually been fit and well and, apart from a prostate resection for benign prostatic hyperplasia, he had not suffered from any major illness. There was no definite history of rheumatic fever. He lived with his wife and did not smoke or drink alcohol. As he was in acute heart failure and an echocardiogram showed a mass attached to the aortic valve, he was referred to the Royal Brompton Hospital urgently for further management.

On arrival at the Royal Brompton Hospital, he was unwell, pale, and dyspnoeic, though apyrexial. There was no clubbing, Roth's spots or Janeway's lesions. His pulse rate was 94 per minute, blood pressure 130/40 mmHg and the jugular venous pressure was not raised. The apex beat was easily palpable and there was a systolic and diastolic thrill. Auscultation revealed a soft pansystolic murmur and an early diastolic murmur along the left sternal edge. There were coarse crepitations in both lung bases, posteriorly. The tip of the spleen was just palpable, but the rest of the abdominal and neurological examination was unremarkable.

Chest X-ray showed cardiomegaly and pulmonary oedema. An electrocardiogram (ECG) showed sinus rhythm and a normal PR interval. Haemoglobin was 93 g/L, mean cell volume 92 fL, white cell count 9.9 x 10^9/L, neutrophils 7.2 x 10^9/L, platelets 203 x 10^9/L, C-reactive protein 92 mg/L, sodium 137 mmol/L, potassium 3.1 mmol/L, urea 24.5 mmol/L, creatinine 236 mol/L, glucose 6.0 mmol/L, cholesterol 3.8 mmol/L, thyroxine (free) 12 pmol/L, TSH 3.15 mU/L. Urine dipstix was positive for red blood cells. An echocardiogram showed severe aortic regurgitation, with a large vegetation attached to the aortic valve leaflet. The diameter of the aortic root was 3.2 cm. The mitral valve was also abnormal and thickened, with a vegetation attached to the anterior leaflet, with a small degree of mitral regurgitation. The left ventricle was dilated and measured 4.8 cm end-systolic and 6.8 cm end-diastolic.

He was started on intravenous benzylpenicillin 2.4 g four-hourly and gentamicin 80 mg eight-hourly. For left ventricular failure, intravenous frusemide and an infusion of nitrates were given while to improve renal function prior to surgery a dopamine infusion was set up. Blood cultures (both aerobic and anaerobic) showed growth of rod-shaped Gram-positive organisms which were non-motile and catalase-negative. Hydrogen sulphide was detected after two days of incubation at 37°C. The organisms were identified as *Erysipelothrix rhusiopathiae*.

After 48 hours of initial resuscitation, he was operated upon: his aortic valve was replaced by a St. Jude 27 mm prosthetic valve and the vegetation from the anterior leaflet of the mitral valve was cauterised. Post-operatively, he suffered a grand mal fit and transient left hemiparesis. A brain computerised tomogram scan was normal, and an echocardiogram showed no thrombus in the left ventricle. His urea improved to 9.6 mmol/L and his serum creatinine returned to normal. A few days after this initial improve-

Medical Assessment Unit, Wexham Park Hospital, Wexham Street, Slough, Berkshire, SL2 4HL.
Muhammad Arif, Staff Physician
Correspondence to: Dr M Arif
(email: marif62@hotmail.com)

ment, unfortunately, he collapsed into severe pulmonary oedema and shock. He later died in the intensive treatment unit on day 16 of his admission.

Discussion

The clinical pattern of infective endocarditis has changed considerably over the years, mainly due to the introduction of antibiotics and the decreased incidence of the major risk factor (rheumatic heart disease) but new risk factors have emerged, such as intravenous drug abuse, prosthetic valves and an increase in the elderly population. Echocardiography, especially transoesophageal echocardiography, has become a very useful diagnostic tool in endocarditis.[3,4]

The commonest 'typical' bacteria of endocarditis i.e. streptococci and staphylococci alone are responsible for 60% and 25% of cases, respectively. There have been few reports of endocarditis due to atypical and rare organisms.[2] In the UK, underlying heart disease will not have been previously detected in up to 40% of patients with endocarditis.[5] *Erysipelothrix rhusiopathiae* is a very rare cause of endocarditis: fewer than 60 cases have been reported in the literature. Most recently, cases have been reported from Germany and Holland.[6,7] Although *E. rhusiopathiae* is found worldwide, I believe there have been no case reports of *E. rhusiopathiae* endocarditis acquired from the waters surrounding the British Isles.

The first member of *Erysipelothrix* was described by Koch in 1880 in the blood of mice, and the first human case was described in 1909 by Rosenbach. A major reservoir is believed to be domestic swine. This organism is found in the slime surrounding the body of various fish, in the sewage effluent from abattoirs and in the tonsils and faeces of pigs and other animals.[8] It may live long enough in soil to cause infection months after initial contamination. A case of *E. rhusiopathiae* bacteraemia after dog bite has been reported.[9] Our patient most probably became infected while skinning and filleting fish, during which he accidentally cut his finger.

Erysipelothrix rhusiopathiae is a rod-shaped, Gram-positive, non-motile, non-sporing, aerobic (or facultative anaerobic) organism, which grows better with 5–10% CO_2. Although it grows on standard culture media, its identification can be a challenge to the microbiologist.[10] It may appear Gram-negative because it decolourises readily. It may be arranged in any form. It may be haemolytic on blood agar and most strains produce hydrogen sulphide, a diagnostically important reaction. There have been recent advances in molecular techniques for diagnosing *E. rhusiopathiae*. Two PCR assays have been described for the diagnosis of swine erysipelas, one of which has been applied successfully to human samples.[11]

The most common form of infection by *E. rhusiopathiae* in humans is a skin lesion called erysipeloid: septicaemia occurs in less than 1% of cases[12] but over 90% of these cases are characterised by endocarditis.[13] All reported cases of endocarditis, except one case involving a Starr-Edwards prosthetic aortic valve, have involved native valves.[14] Overall, in 60% of cases, endocarditis developed on normal heart valves. The organism exhibits significant aortic valve tropism (involved in 70% of patients).[13] Only 36% of patients with *E. rhusiopathiae* endocarditis have a history of a preceding skin lesion. *Erysipelothrix* is highly susceptible to penicillin, cephalosporins and clindamycin and variably susceptible to erythromycin, chloramphenicol and tetracycline.[13] Most strains are resistant to aminoglycosides, vancomycin, trimethoprim-sulphamethoxazole, sulphona-mides, polymyxins, streptomycin and novobiocin.

Key messages

- *E. rhusiopathiae* should be suspected as a cause of endocarditis, if the patient has been involved in the handling of animals especially fish
- Most strains are resistant to amino glycosides and vancomycine, commonly used for empiric treatment of endocarditis
- Over 60% of cases involve previously normal valves
- Poorly treated cases may deteriorate very rapidly

Infective endocarditis can be complicated by renal disease, due mainly to immune complex deposition, and also by emboli. Our patient had evidence of renal impairment on admission. A progressive increase in serum creatinine with haematuria and proteinuria can be due to acute interstitial nephritis caused by antibiotics like penicillin and aminoglycosides. Cerebral emboli have been reported to occur in up to 17% of cases and rupture of a mycotic aneurysm may occur up to two years after apparent cure of the infection.[15]

The presence of cardiac failure and severe aortic regurgitation were indications for surgery in our patient. The expected mortality with medical treatment alone in endocarditis with gross cardiac failure is up to 89%. This reduces to approximately 23% with a combined medical and surgical approach. It has been found that the risk of developing infection of a prosthetic valve is only 4%, even when surgery is performed before completion of the full course of antibiotics.[5] Overall mortality is quoted at about 20% for native valve endocarditis and it rises to 38% in cases of *E. rhusiopathiae*.[16] Of the survivors 20% may develop some incapacity due to complications, namely congestive heart failure, myocardial abscess, meningitis and glomerulonephritis.

Conclusions

Erysipelothrix rhusiopathiae is a rare but serious and life-threatening cause of infective endocarditis. It is treatable if recognised early and if appropriate antibiotics are used. A high index of suspicion should be maintained if the patient belongs to a high-risk group. Survival and prognosis after surgery are largely determined by preoperative left ventricular function. Therefore it is very important to recognise patients who may deteriorate rapidly, and early surgical opinion should be sought.[17] *Erysipelothrix* is an occupational hazard, therefore protective gloves and shoes are highly recommended when involved in handling of animals, especially fish.

References

1. Raoult D. Editorial: Afebrile blood culture-negatives endocarditis. *Ann Intern Med* 1999;**131**:144-6.

2. Gubler JGH, Kuster M, Dutly F *et al*. Whipple endocarditis without overt gastrointestinal disease: report of four cases. *Ann Intern Med* 1999; **131**:112-16.
3. Daniel WG, Mugge A, Martin RP *et al*. Improvement in the diagnosis of abscess associated with endocarditis by trans-oesophageal echocardiography. *N Engl J Med* 1991;**324**:795-800.
4. Durack DT, Lukes AS, Bright DK. New criteria for the diagnosis of infective endocarditis: utilisation of specific echocardiographic findings. *Am J Med* 1994;**96**:200-09.
5. Gribbin B, Crook DWM. Infective Endocarditis. In: *Oxford Textbook of Medicine*. Oxford: Oxford University Press, 1996;2436-50.
6. Artz AL, Szabo S, Zabel LT, Hoffmeister HM. Aortic valve endocarditis with paravalvular abscesses caused by *Erysipelothrix rhusiopathiae*. *Eur J Clin Microbiol Infect Dis* 2001;**20**:587-8.
7. Heidrich JP, Stahl M, Dittman R, Maass M, Solbach W. Mitral valve endocarditis caused by *Erysipelothrix rhusiopathiae*. *Dtsch Med Wochen Schr* 2001;**126**:431-3.
8. Smith GR, Melaughlin J, Taylor AG. Erysipelothrix and listeria. In: Parkes MT, Duerden BI (eds.). *Topley and Wilson's principles of bacteriology, virology and immunity. 8th ed. Vol 2: Systemic bacteriology*. London: Edward Arnold, 1990;60-2.
9. Abedini S, Lester A. *Erysipelothrix rhusiopathiae* bacteraemia after dog bite. *Ugeskr Laeger* 1997;**159**:4400-01.
10. Dunbar SA, Clarridge JE. Potential errors in recognition of *Erysipelothrix rhusiopathiae*. *J Clin Microbiol* 2000;**38**:1302-04.
11. Brooke CJ, Riley TV. *Erysipelothrix rhusiopathiae*: bacteriology, epidemiology and clinical manifestations of an occupational pathogen. *J Med Microbiol* 1999;**48**:789-99.
12. Nandish S, Khardori N. Valvular and myocardial abscesses due to *Erysipelothrix rhusiopathiae*. *Clin Infect Dis* 1999;**29**:1351-2.
13. Gorby GL, Peacock JEJ Jr. *Erysipelothrix rhusiopathiae* endocarditis: microbiologic, epidemiologic and clinical features of an occupational disease. *Rev Infect Dis* 1988;**10**:317-25.
14. Reboli AC, Farrar WE. *Erysipelothrix rhusiopathiae*: an occupational pathogen. *Clin Microbiol Rev* 1989;**2**:354-9.
15. Steckelberg JM, Murphy JG, Ballard D *et al*. Emboli in infective endocarditis: the prognostic value of echocardiography. *Ann Intern Med* 1992;**114**:635-40.
16. Hill DG, Ghassemian JN. *Erysipelothrix rhusiopathiae* endocarditis: clinical features of an occupational disease. *South Med J* 1997;**90**:1147-8.
17. Tornos MP, Permanyer-Miralda G, Olona M *et al*. Long-term complications of native valve infective endocarditis in non addicts. A 15 year follow up study. *Ann Intern Med* 1992;**117**:567-72.