Aortic and mitral valve endocarditis caused by *Gemella morbillorum* in a haemodialysis patient

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**ABSTRACT**

*Gemella morbillorum* is part of the commensal flora of the upper respiratory tract, intestinal tract and genitourinary tract. On rare occasions, it causes endocarditis. We report a 67-year-old Chinese man with end-stage renal disease on maintenance intermittent haemodialysis, who developed *Gemella morbillorum* endocarditis complicated by severe aortic and mitral regurgitation. Most cases of *Gemella morbillorum* endocarditis have satisfactory outcomes with antibiotics or surgical treatment.

**Keywords:** endocarditis, end-stage renal disease, *Gemella morbillorum*, haemodialysis

**INTRODUCTION**

*Gemella morbillorum* is a nutritionally-variant streptococcus that was previously classified as *Streptococcus morbillorum*.(1) It is part of the commensal flora of the upper respiratory, intestinal, and genitourinary tracts. Infections caused by this organism are unusual, but instances of endovascular infections and endocarditis have been reported. We report a patient who developed native aortic and mitral valve endocarditis caused by *Gemella morbillorum*.

**CASE REPORT**

A 67-year-old Chinese man was admitted for shortness of breath associated with chills and rigors for three days. He denied having fever, cough or chest pain. The patient was admitted three months previously for left lobar pneumonia. Blood cultures at that time grew *Streptococcus sanguis*, which was sensitive to ampicillin, and he was discharged on oral co-amoxiclav (Augmentin; Sandoz, Austria). Significant past medical history included end-stage renal disease (ESRD) secondary to hypertension that needed chronic maintenance intermittent haemodialysis, mild to moderate aortic regurgitation (ejection fraction of 65%), and treated syphilis.

On examination, there was a raised jugular venous pulse, lung crepitations and a soft diastolic murmur in the aortic area on auscultation. The patient had poor dentition but no other stigmata of infective endocarditis (IE). A left arm brachiocephalic arteriovenous fistula with a good thrill was noted. Laboratory investigations revealed leucocytosis (leucocytes 21.8 x 10⁹/L, neutrophils 73%) and raised C-reactive protein (CRP 62 mg/L). Troponin T was elevated (1.60, 1.68, 1.82 µg/L). Electrocardiography (ECG) and chest radiograph were unremarkable.

The initial impression was that of fluid overload occurring in an ESRD patient, and he was treated with increased ultrafiltration and haemodialysis. The differential diagnoses included pneumonia and endocarditis. Factors supporting diagnosis of endocarditis in the patient included recent *Streptococcus sanguis* bacteraemia, poor dentition, and an existing aortic valve abnormality. Intravenous ampicillin 2 g every 12 hours and gentamycin 80 mg every 48 hours were initiated, and further investigations were scheduled, including transoesophageal echocardiogram, colonoscopy, and dental examination.

While on haemodialysis, the patient developed central chest pain and shortness of breath. He became
hypotensive (< 100/50 mmHg) and had transient bradycardia (pulse rate 51/min). The diastolic murmur heard in the aortic area had increased in intensity. ECG revealed ectopic atrial beats, but there was no acute ST-T wave change. Seru troponin-T was elevated at 1.65 µg/L. An urgent transthoracic echocardiogram demonstrated concentric left ventricular hypertrophy and an ejection fraction of 60%. Multiple vegetations were seen attached to the aortic valve causing severe aortic regurgitation (Fig. 1). The mitral valve leaflets were thickened with multiple vegetations attached to the anterior leaflet with moderate mitral regurgitation. No pericardial effusion was noted. An angiocardiogram showed single vessel disease with a 60% stenosis in the middle of the left anterior descending artery. The patient was transferred to the cardiac intensive care unit, and aspirin and nitrates were started.

The patient started to spike temperatures up to 38°C, and *Gemella morbillorum* (which was sensitive to ampicillin) was isolated from blood cultures. Intravenous ampicillin and gentamycin were continued, and subsequent blood cultures became negative. As the patient was haemodynamically stable and not in heart failure, it was decided that intravenous antibiotics be continued for a few weeks before surgery. The patient, however, deteriorated on day 18 of admission with complaints of chest pain and shortness of breath. ECG showed supraventricular tachycardia and he was treated with intravenous amiodarone infusion and oral metoprolol, where transient improvement was observed. But the patient’s oxygenation state worsened and he developed asystole. He failed to respond to cardiopulmonary resuscitation, and efforts to revive him had to be ceased. Participating care providers conferred and the speculated cause of death was an acute myocardial infarction from worsening aortic incompetence.

**DISCUSSION**

ESRD patients on haemodialysis usually develop endocarditis associated with vascular access infections.  

Temporary haemodialysis catheters are associated with the highest rates of infectious complications, followed by tunnelled dialysis catheters, arteriovenous grafts, with arteriovenous fistulae having the lowest rates. The organisms that are typically isolated are skin commensals such as *Staphylococcus aureus*. This patient, however, developed endocarditis caused by an unusual organism, *Gemella morbillorum*, which prompted further investigations for the infective source. Moreover, he died when most patients with *Gemella* endocarditis have good outcomes.

*Gemella morbillorum* is a nutritionally-variant streptococcus that was previously classified as *Streptococcus morbillorum*. It was transferred to its present genus in 1988 based on DNA homology, physiological properties, and 16S RNA cataloguing.  

It is an anaerobic to aerotolerant Gram-positive coccus, and is part of the commensal flora of the upper respiratory tract, intestinal tract and genitourinary tract. Infections caused by this organism are unusual, other than rare instances of endovascular infections and endocarditis. Predisposing factors include poor dental hygiene, dental procedures, colon diseases, colonoscopy, steroid therapy, diabetes mellitus, hepatorenal dysfunction and prexisting cardiac conditions, such as valvular lesions, hypertrophic cardiomyopathy and cardiac myxoma. Any of the valves in both sides of the heart can be affected. The tricuspid valve is involved in drug abusers, and prothotic valve infections have also been reported.

*Gemella morbillorum* share some physiological characteristics with other nutritionally-variant streptococci, and thus endocarditis by this organism should be treated with more aggressive combination therapies, using a regimen similar to that for enterococcal endocarditis. For *Gemella morbillorum* susceptible to penicillin and aminoglycosides (as in our patient), intravenous ampicillin or penicillin G, and gentamycin for 4–6 weeks is recommended, according to American Heart Association guidelines. Gentamycin trough levels have to be monitored to prevent underdosing, and inadvertent overdosing with its attendant risk of ototoxicity, especially in a patient with renal failure. Combination therapy with vancomycin and gentamycin is recommended only for patients unable to tolerate penicillin or ampicillin, but this method is associated with an increased risk of ototoxicity and nephrotoxicity. Streptomycin is used in place of gentamycin for isolates that are susceptible to penicillin but resistant to gentamycin. Linezolid is used when the organism is resistant to penicillin, aminoglycosides and vancomycin.

Cardiac valve replacement may be necessary.

Most cases of *Gemella morbillorum* endocarditis will have satisfactory outcomes from antibiotics or surgical treatment. However, complications such as congestive heart failure (CHF), embolisation and periannular extension of infection can occur. Of all the complications, CHF has the greatest impact on prognosis. In native valve IE, acute CHF occurs more frequently in aortic valve infections (29%) than with mitral (20%) or tricuspid (8%) disease. CHF in IE portends a grave prognosis with medical therapy alone,
and is also the most powerful predictor of poor outcome with surgical therapy. The decision to operate is driven primarily by the severity of CHF. Poor surgical outcome is predicted by New York Heart Association Class III or IV, CHF, renal insufficiency and advanced age. In any patient, a decision to delay surgery to extend preoperative antibiotic treatment carries the risk of permanent ventricular dysfunction and should be discouraged. Valve surgery for patients with complicated left-sided native valve endocarditis was associated with reduced six-month mortality, especially in moderate to severe CHF. Despite a higher operative mortality rate in patients with CHF than in those without CHF, patients with CHF who undergo valve surgery have a reduced mortality rate compared with those on medical therapy alone. The incidence of reinfection of newly-implanted valves in patients with active IE is about 2%-3%, which is far less than the mortality rate for IE and CHF without surgical therapy, which can be as high as 51%. Surgical intervention is also warranted when there is echocardiographical evidence of large vegetations (> 10 mm), severe valvular insufficiency, large perivalvular abscess, valvular perforation/dehiscence/rupture/fistula and decompensated heart failure.

This patient was unusual in that endocarditis in ESRD patients is usually associated with vascular access infections and the organism would usually be skin commensals such as Staphylococcus aureus. An unusual organism should prompt a search for the source, and with Gemella morbillorum, a dental and gastrointestinal examination should be performed. He was at risk from oral commensal bacteraemia as he had developed Streptococcus sanguis infection in a prior admission, and probably should have received a dental examination and treatment at that time, as well as antibiotic prophylaxis in view of his pre-existing valvular disease. His death may have been averted by earlier surgery, although he did not have compelling indications for immediate surgery.

REFERENCES