Cardiology in the Young

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Gemella bergeri infective endocarditis: a case report and brief review of literature

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Brief Report

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Abstract

Gemella is a genus of Gram-positive bacteria found in the digestive tract of humans. They rarely cause systemic illness but have been recently implicated in several serious infections. We report infective endocarditis caused by *Gemella bergeri* in a 23-year-old with a bicuspid aortic valve status post-intervention in infancy.

The genus *Gemella* is a group of Gram-positive cocci found in the oral cavity and digestive tract of mammals, including humans. The genus has six known species: *haemolysans*, *morbillorum*, *bergeri*, *sanguinis*, *palaticanis*, and *cuniculi*. They rarely cause systemic illness but it may be severe when it does occur. *Gemella hemolysans*, *Gemella morbillorum*, and *Gemella sanguis* are usually the most frequent culprits. We present an extremely rare case of endocarditis caused by *Gemella bergeri*.

Case report

The patient is a 23-year-old male with a bicuspid aortic valve, post-surgical aortic valvotomy at 5 months of age with subsequent mild aortic stenosis and moderate insufficiency. He was lost to follow-up for the past 5 years. He reported being asymptomatic until 2 weeks before presenting to an emergency room with a 2-week history of night sweats, and a 2-day history of bilateral knee and ankle pain and pedal oedema. In the past 2 days, he reported having episodes of palpitations lasting <2 minutes each. His last dental cleaning was 1.5–2 years before and he always took prophylactic Amoxicillin.

At presentation, he was afebrile, alert, and comfortable with normal vital signs. His cardiovascular examination revealed a grade 3/6 holosystolic murmur at the apex, not previously noted, along with a 2/6 diastolic murmur at the right mid-sternal border radiating to the apex. He had mild bilateral non-pitting pedal edema. The rest of his systemic exam was within normal limits, showing no signs suggestive of endocarditis.

Admission bloodwork revealed a normal white blood cell count and differential, normal electrolytes, and mild anaemia (haematocrit 29%). Troponin I was 0.23 ng/ml, brain natriuretic peptide was 112 pg/ml, C-reactive protein was elevated at 11.3 mg/dl (normal <1.0 mg/dl), pro-calcitonin was normal at 0.13 ng/ml (normal <0.5 ng/ml), and erythrocyte sedimentation rate was elevated at 39 mm (normal <15 mm/hour). He was started on ceftriaxone and vancomycin and a blood culture grew Gram-positive cocci within 24 hours.

A transthoracic echocardiogram showed a thickened anterior mitral valve leaflet with an echogenic nodule measuring 1.67 × 1.09 cm (Fig 1), highly suggestive of an infectious vegetation. There was mild mitral stenosis with a mean gradient of 4 mmHg and trivial insufficiency. He had a bicuspid, thickened aortic valve with a mildly dilated aortic root and moderate aortic stenosis (peak gradient 58 mmHg and mean gradient 27 mmHg) with moderate insufficiency. He previously had only mild aortic stenosis with trivial insufficiency. The remaining findings were new.

An MRI of the brain showed a few non-specific punctate lesions in the white matter. Renal ultrasound, lower-extremity venous Doppler, and abdominal ultrasound were unremarkable except for mild splenomegaly. Ophthalmologic exam was normal.

The original blood culture was identified as *Gemella bergeri* on day 4. Multiple subsequent blood cultures were negative and inflammatory markers declined. Imaging at 6 days showed that the vegetation was larger (2.03 × 0.98 cm) with worsening aortic insufficiency. At 12 days, it was 2.4 × 2.6 mm with central clearing suggestive of abscess formation (Fig 2). There was a new narrow jet of mitral insufficiency. The mitral stenosis and aortic insufficiency had both worsened. Biventricular systolic function remained normal but the left ventricle slowly dilated with a maximum diastolic dimension of 7.3 cm and a z score of 3, with mild hypertrophy.

After 14 days of treatment with ceftriaxone and vancomycin, the sensitivities were obtained on the *Gemella bergeri* from his original blood culture and antibiotics were changed to ampicillin and gentamicin; sensitivity results took several days because of slow growth of the organism. The patient was discharged on day 15 to complete 6 weeks of IV antibiotics.

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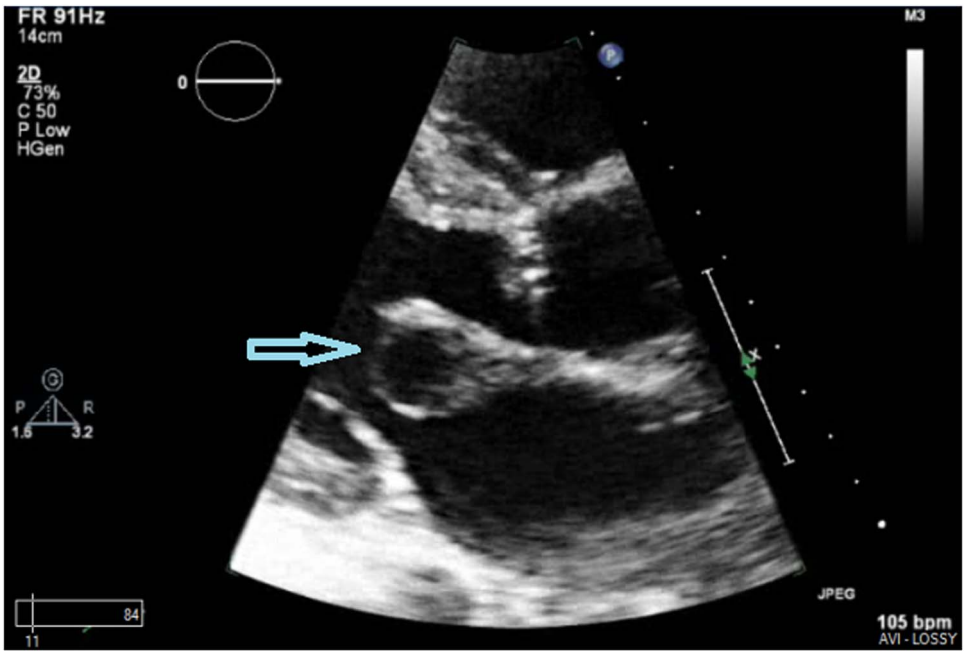

Figure 1. Parasternal long-axis view of the heart on echocardiogram showing the echogenic nodule with a hypoechoic centre on the anterior mitral leaflet.

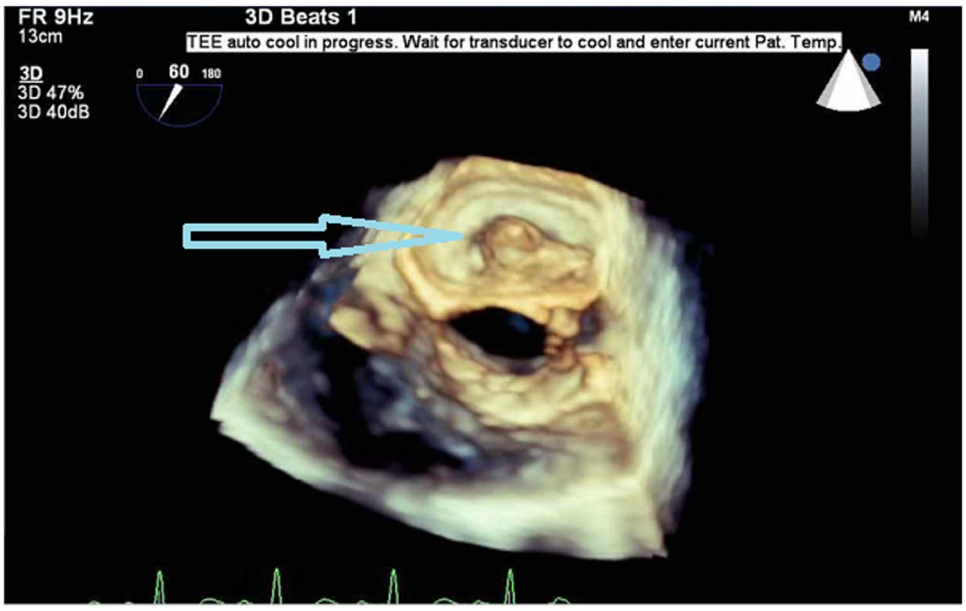

Figure 2. Three-dimensional transoesophageal echocardiogram view of the anterior mitral vegetation with central clearing suggestive of abscess formation.

After his 6-week course of antibiotic therapy, he underwent surgical repair – removal of vegetation, mitral valvuloplasty and annuloplasty, and mechanical aortic valve replacement. Intraoperatively, there was a large hole in the anterior leaflet of the mitral valve covered by a large vegetation. There was no obvious abscess or purulent fluid. He has been followed-up clinically and has been doing well 2 years after this procedure.

Discussion

Gemella bergeri are Gram-positive cocci that occur in pairs or short chains. Contrary to popular belief that *Gemella* is a harmless commensal, there have been reports of meningitis, cerebrospinal fluid shunt infection, cerebral abscess, and septic shock, as well as other bone and soft tissue infections. Endocarditis has been reported in individuals with and without pre-existing CHD. Other *Gemella* species, such as *morbillorum*, *hemolysans*, and *sanguis*, have been reported to cause prosthetic and native valve endocarditis.

Gemella bergeri was first detected by Collins et al[1] from the blood cultures of six patients, three of whom had subacute bacterial endocarditis. Subsequently, it was reported in a 32-year-old male with a bicuspid aortic valve[2] and then in a 15-year-old boy with tetralogy of Fallot and pulmonary atresia.[3] In May, 2014, a 24-year-old had a fulminant course of endocarditis with *Gemella bergeri* complicated by an embolic stroke, as well as intracerebral and subarachnoid haemorrhage secondary to rupture of a mycotic aneurysm.[4] All previous reports (seven

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cases) of *Gemella bergeri* infective endocarditis involved the left side of the heart except for the last, which involved a tricuspid valve cleft.[5]

In this case, it is likely that the endocarditis involved the bicuspid aortic valve first. The aortic insufficiency jet striking the anterior mitral leaflet may have resulted in contiguous spread as the mitral valve had previously appeared normal on transthoracic echocardiography.

Most cases of *Gemella* endocarditis have been successfully treated with a combination of penicillin or vancomycin and an aminoglycoside for 4–6 weeks. Although there have been rare reports of resistance, consideration for an initial empirical combination therapy – a β-lactam agent and an aminoglycoside – or vancomycin treatment is still recommended.

Summary and conclusions

Infections caused by *Gemella* species are rare but have been reported more frequently in recent years owing to advances in laboratory techniques. Some of these bacteria are difficult to grow, grow very slowly, or grow only on special media and will continue to be a challenge for clinicians to diagnose and laboratories to identify and treat in a timely manner.

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Conflicts of Interest. None.

Ethical Standards. The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national guidelines on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

References

1. Collins MD, Hutson RA, Falsen E, Sjöden B, Facklam RR. *Gemella* bergeriae sp nov, isolated from human clinical specimens. J Clin Microbiol 1998; 36: 1290–1293.
2. Elsayed S, Zhang K. *Gemella bergeriae* endocarditis diagnosed by sequencing of rRNA genes in heart valve tissue. J Clin Microbiol 2004; 42: 4897–4900.
3. Logan LK, Zheng X, Shulman ST. *Gemella bergeriae* endocarditis in a boy. Pediatr Infect Dis J 2008; 27: 184–186.
4. Hussain K, Abubaker J, Ahmed R. Unreported neurological complications of *Gemella bergeriae* infective endocarditis. BMJ Case Rep 2014; 2014: pii: bcr2014204405.
5. Orathai Pachirat O, Watt G, Pussadhamma B. First case of tricuspid valve endocarditis caused by *Gemella bergeri*. Case Rep Med 2015; 2015: 704785.

https://doi.org/10.1017/S1047951118000070 Published online by Cambridge University Press