Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/mjafi

Case Report

Gemella sanguinis: A rare cause of native valve endocarditis in a child

Mugunthan M [a,*], *Lt Col Sharad Bhalla* [b], *Vishal Shete* [a], *Col Naveen Grover* [c]

[a] *Resident, Department of Microbiology, Armed Forces Medical College, Pune 411040, India*
[b] *Assistant Professor (Path & Micro), Military Hospital (Cardiothoracic Centre), Pune 411040, India*
[c] *Professor & Head, Department of Microbiology, Armed Forces Medical College, Pune 411040, India*

ARTICLE INFO

Article history:
Received 1 July 2015
Accepted 26 August 2015
Available online 31 December 2015

Keywords:
Gemella sanguinis
Infective endocarditis
Pediatrics

Introduction

Infective endocarditis (IE), a severe form of valvular disease, is mostly caused by Gram-positive bacteria. Lately, *Gemella* spp. have emerged as a cause of IE.[1] Five species were reported to cause human infection: *Gemella bergeri*, *Gemella haemolysans*, *Gemella morbillorum*, *Gemella palaticanis*, and *Gemella sanguinis*.[2–5] Among them, *G. morbillorum* was reported in more than 40 cases and *G. sanguinis* in four cases of IE. Early surgery with prolonged parenteral antimicrobial therapy is key for successful management.[1] Described here is a case of tricuspid valve IE due to *G. sanguinis* in a 4-year-old male patient. He was successfully treated with medical–surgical management.

Case report

A 4-year-old male patient presented with high-grade fever associated with chills and rigor, cough, weight loss, and abdominal distension. On physical examination, he was pale, febrile, and tachycardic with a pulse rate of 158 beats/min. Hepatosplenomegaly was present. His cardiovascular examination revealed a S3 gallop with no murmurs.

Complete blood count revealed anemia with hemoglobin of 5.2 gm% (RBC count 1.98×10^6/cumm, Packed cell volume 14.5%), increased RBC distribution width (16.7%), raised leukocyte count (26,900 cells/cumm with 74% neutrophils), and reduced platelet count (0.35 lakhs/cumm). Peripheral blood smear showed mild anisocytosis with moderate hypochromia.

An echocardiogram revealed huge bilobed echogenic mass of size 1.8 cm × 1.8 cm arising from atrial surface of septal tricuspid leaflet (STL) suggestive of IE vegetation with mild tricuspid regurgitation. Treatment was initially started with ceftriaxone 900 mg IV twice daily and amikacin 125 mg IV twice daily.

A diagnosis of tricuspid valve IE was made and the patient underwent successful prosthetic valve replacement surgery. A vegetation of size 5 cm × 3 cm with STL was sent for

* *Corresponding author*: Tel.: +91 8412800039.
E-mail address: dineshmugun@gmail.com (M. Mugunthan).
http://dx.doi.org/10.1016/j.mjafi.2015.08.008

microbiological analysis along with blood for culture and sensitivity.

Gram stain of vegetations showed Gram-positive cocci in pairs. The vegetation in Brain Heart Infusion (BHI) broth was incubated for 24 h and then sub-cultured in blood agar. After 48 h of incubation, blood agar showed non-hemolytic, tiny, pinpoint, smooth, translucent to grayish colonies. Gram stain showed Gram-positive cocci in pairs. The organism was identified as *G. sanguinis* by VITEK 2-Compact System (Biomerieux, France).

On performing various biochemical reactions, it was found to be catalase negative, oxidase negative, bile esculin negative, urease negative, arginine dihydrolase negative, hippurate hydrolysis negative, and showed no growth in 6.5% sodium chloride. However, pyrrolidonylarylamidase (PYRase) test was positive. It fermented maltose, mannitol, and sorbitol but not lactose. Antibiotic susceptibility testing was performed using the Kirby–Bauer disk diffusion method on 5% defibrinated sheep blood agar and was incubated for 48 h in 10% carbon dioxide. The antimicrobial susceptibility testing was interpreted according to Clinical and Laboratory Standards Institute guidelines 2014 for *Streptococcus* spp. It was found to be susceptible to vancomycin, linezolid, daptomycin, clindamycin, gentamicin, and tetracycline and was resistant to penicillin, oxacillin, chloramphenicol, ciprofloxacin, and levofloxacin.

The blood culture bottle in Versa Trek Automated blood culture system (Trek Diagnostics, USA) flagged after 72 h and was then further processed according to standard protocol. Gram stain of the positive blood culture bottle revealed Gram-positive cocci in pairs.

On subculture in blood agar, colonies similar to the growth from vegetations were seen after incubating for 48 h. The organism was again identified as *G. sanguinis* by VITEK 2-Compact System (Biomerieux, France). The biochemical reactions and susceptibility pattern were also similar to that isolated from the vegetation.

After obtaining the antimicrobial susceptibility testing report from microbiology laboratory, the patient was treated with vancomycin 500 mg IV three times daily and gentamicin 40 mg IV three times daily for 6 weeks. The patient's health status improved and was discharged after 8 weeks and was kept on regular follow-up.

Discussion

G. sanguinis was first described in 1998.[6] It is part of normal flora of human oropharynx, urogenital, and gastrointestinal tract.[6] Previously reported cases of endocarditis caused by *Gemella* species were often associated with dental disease or an invasive procedure, but there was no identifiable source of infection in our patient.[5,7]

G. sanguinis are Gram-positive, non-spore-forming cocci that occur singly, in pairs, or in short chains. Colonies on blood agar plates after 48 h of incubation are small, circular, entire, low convex, translucent, smooth, and non-pigmented. Some strains can show hemolysis. They are facultative anaerobes and catalase and oxidase reaction were negative. According to the API systems, esculin, hippurate, and gelatin are not hydrolyzed. Fermentative pattern and the enzymes produced are given in Tables 1 and 2, respectively. Voges–Proskauer test is variable and nitrate is not reduced.[8] Most cases of *Gemella* species endocarditis have been successfully treated with a combination of penicillin or vancomycin and an aminoglycoside for 2–4 weeks.[4,7,9–12]

Table 1 – Characteristic fermentative pattern of *Gemella sanguinis*.[8]

Sugar	Acid production
Glucose	+
Lactose	–
Sucrose	+
Mannitol	+
Maltose	+
Sorbitol	+
Trehalose	–
Ribose	–
Raffinose	–
D-Arabitol	–
L-Arabinose	–
Glycogen	–
D-Xylose	–

+, Fermented; –, not fermented.

In the present case, the diagnosis of *G. sanguinis* causing IE was made based on the isolation of the organism from both vegetations and blood sample. Identification was done by Vitek 2 and confirmed by biochemical reactions. The patient was treated with vancomycin and gentamicin for 6 weeks. He was discharged and was on regular follow-up.

To conclude, this is a rare case of tricuspid valve IE caused by *G. sanguinis*, which was treated successfully with early surgery and prolonged IV antibiotics.

Table 2 – Characteristic enzymes produced by *Gemella sanguinis*.[8]

Enzymes	Results
Alkaline phosphatase	++
Acid phosphatase	++
Ester lipase C8	++
Pyrrolidonyl arylamidase	+
Pyrazinamidase	+
Esterase C4	+
β-Mannosidase	–
Trypsin	–
Valine arylamidase	–
Urease	–
Arginine dihydrolase	–
Chymotrypsin	–
Cystine arylamidase	–
α-Fucosidase	–
β-Galacturonidase	–
Leucine arylamidase	–

++, produced by all strains; +, produced by some strains; –, not produced by any strains.

Conflicts of interest

The authors have none to declare.

REFERENCES

1. Yang CH, Tsai KT. *Gemella sanguinis* endocarditis: first case report in Taiwan and review of the literature. *J Formos Med Assoc.* 2014;113:562–565.
2. 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.
3. Collins MD, Hutson RA, Falsen E, Sjöden B, Facklam RR. Description of *Gemella sanguinis* sp. nov., isolated from human clinical specimens. *J Clin Microbiol.* 1998;36:3090–3093.
4. Collins MD, Jovita MR, Foster G, Sjöden B, Falsen E. Characterization of a *Gemella*-like organism from the oral cavity of a dog: description of *Gemella palaticanis* sp. nov.. *Int J Syst Bacteriol.* 1999;49:1523–1526.
5. La Scola B, Raoult D. Molecular identification of *Gemella* species from three patients with endocarditis. *J Clin Microbiol.* 1998;36:866–871.
6. Rousseau-Gagnon M, Riopel J, Desjardins A, Garceau D, Agharazii M, Desmeules S. *Gemella sanguinis* endocarditis with c-ANCA/anti-PR-3-associated immune complex necrotizing glomerulonephritis with a ''full-house'' pattern on immunofluorescence microscopy. *Clin Kidney J.* 2013;6 (3):300–304.
7. Vasishtha S, Isenberg HD, Sood SK. *Gemella morbillorum* as a cause of septic shock. *Clin Infect Dis.* 1996;22:1084–1086.
8. Collins MD, Hutson RA, Falsen E, Sjöden B, Facklam RR. Description of *Gemella sanguinis* sp. isolated from human clinical specimens. *J Clin Microbiol.* 1998;36(10):3090–3093.
9. Fresard A, Michel VP, Rueda X, Aubert G, Dorche G, Lucht F. *Gemella haemolysans* endocarditis. *Clin Infect Dis.* 1993;16: 586–587 [Letter].
10. Breathnach AS, Gould FK, Bain HH, Aucken HM. *Gemella haemolysans* endocarditis associated with a raised anti-streptolysin-O titre. *J Infect.* 1997;34:87–88.
11. Farmaki E, Roilides E, Darilis E, Tsivitanidou M, Panteliadis C, Sofianou D. *Gemella morbillorum* endocarditis in a child. *Pediatr Infect Dis J.* 2000;19:751–753.
12. Kaufhold A, Franzen D, Lutticken R. Endocarditis caused by *Gemella haemolysans*. *Infection.* 1989;17:385–387.