Lateral sinus thrombosis as a complication of a superior rectus muscle abscess caused by *Gemella morbillorum* bacteremia

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**Abstract**

*Gemella morbillorum* is found in normal oral flora and is a potential bloodborne pathogen. We present a case of *G. morbillorum* bacteremia with lateral sinus thrombosis complicated by a superior rectus muscle abscess. The patient was successfully treated with adequate antibiotics without surgery or anti-coagulation therapy. The pathogen has not been previously reported to be associated with dural thrombosis. The aim of this report was to draw attention to this little-known pathogen that causes dural thrombosis.

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### 1. Introduction

*Gemella morbillorum* is a Gram-positive, catalase-negative, facultatively anaerobic coccus. It was previously named *Diplococcus morbillorum*, *Peptococcus morbillorum*, *Peptostreptococcus morbillorum*, or *Streptococcus morbillorum*. This organism was eventually transferred from the genus *Streptococcus* to the genus *Gemella* in 1988 on the basis of its biomolecular features and physiological properties [1]. Several types of infections have been identified as being caused by these pathogens, including bacteremia, infective endocarditis, pericarditis, arthritis, pleural empyema, lung abscess, peritonitis, pneumonia, Ludwig's angina, septic shock, spondylodiscitis, brain abscess, and soft tissue infection [2–12].

Thrombosis of the dural venous sinuses is generally described as occurring because of an infective etiology; however, there have been reports of it developing after nasal surgery or trauma [13]. This disorder was frequently encountered in the pre-antibiotic era but has become rarer because of the widespread use of antibiotics for the treatment of oropharyngeal infections [14]. The common organisms associated with septic thrombosis of dural sinuses are *Staphylococcus aureus*, *Streptococcus pneumoniae*, other *Streptococci*, Gram-negative bacilli, and anaerobes [15,16]. *Gemella morbillorum* had not been reported as a pathogen that causes septic thrombosis of the dural venous sinuses. We, herein, report a case of septic thrombosis of the dural venous sinuses complicated by a superior rectus muscle abscess that was caused by *G. morbillorum* bacteremia.

### 2. Case report

An 83-year-old woman with a history of hypertension for 20 years was admitted because of headache and bilateral eyelid swelling that had been present for 1 week. She had been able to walk and perform activities of daily living until 1 week ago. She then developed intermittent headaches, dizziness, and general weakness. Bilateral eyelid swelling was noted by her family, and she had attended a local clinic 1 day before admission, where analgesic agents were injected without improvement. No fever, cough, rhinorrhea, nausea, vomiting, diarrhea, dysphagia, or dysuria was noted. She did not have a history of dental procedure or trauma in the last 3 months. The symptoms persisted, and she then visited our emergency department.

Physical examination revealed a temperature of 37°C, a blood pressure of 110/90 mmHg, a respiratory rate of 18 cycles/min, and a heart rate of 105 beats/min. She was drowsy but arousable. A head and neck examination revealed bilateral eyelid swelling accompanied by chemosis. Her right eye could not move in the upper right direction. Pulmonary and cardiac examinations revealed no abnormality. A peripheral hemogram revealed a leukocyte count of \(32.3 \times 10^9/\mu L\) with 25% band form, a hemoglobin level of 12.3 g/dL, and a platelet count of \(215 \times 10^9/\mu L\). Serum aspartate aminotransferase level was 38 IU/L; urea nitrogen, 36 mg/dL; creatinine,
1.6 mg/dL; and C-reactive protein, 32.65 mg/dL. Urinalysis and chest film revealed no abnormalities. Brain computed tomography showed paranasal, ethmoid, and sphenoid sinuses.

She was admitted to our ward and treated by intravenous ceftriaxone (Roche Ltd., Switzerland) 2 g every 12 hours and metronidazole (China Chemical & Pharmaceutical Co., Ltd., Taiwan) 500 mg every 8 hours. Nonetheless, her headaches, dizziness, and diplopia persisted, and a blood culture yielded Gram-positive cocci. Antibiotics were changed to intravenous vancomycin (Gentle Pharmaceutical Corporation Kaishin Medicines Co, Ltd., Taiwan) 1 g everyday and piperacillin–tazobactam (Wyeth-Ayerst Ltd., Taiwan Branch, Taiwan) 2.25 g every 6 hours on the second day at hospital. Magnetic resonance imaging with contrast enhancement revealed fluid collection in the right paranasal sinus, clivus, right sphenoid sinus, and right ethmoid sinus, and abnormal fluid collection in the right superior rectus muscles. A magnetic resonance venogram showed a filling defect of the right internal jugular vein, right transverse sinus, and right sigmoid sinus. Three-dimensional reconstruction of the venogram indicated an obstruction of the right transverse sinus and right internal jugular vein (Fig. 1).

Two sets of blood cultures yielded Gram-positive cocci. After 48-hour incubation, an α-hemolytic organism that grew on sheep blood agar was cultured and was characterized as catalase negative and non–bile soluble. The biochemical identification system Vitek II (bioMérieux, Marcy l’Etoile, France) identified the organism as G. morbillorum. The strain was susceptible to penicillin, amoxicillin, piperacillin, and clindamycin. The patient’s antibiotics were changed to intravenous penicillin (Y F Chemical Corp., Taiwan) 3 MU every 4 hours and clindamycin (Nang Kuang Pharmaceutical Co., Ltd., Taiwan) 600 mg every 8 hours on the seventh day at hospital. Her eyelid swelling and headache subsided gradually but diplopia persisted because of impaired abduction of the right eye. Aspiration of the superior rectus muscle abscess was arranged but not performed because of her family’s hesitancy with respect to the risk of surgery. Antibiotics were continued. Her diplopia improved gradually. After a 24-day course of intravenous antibiotics, she was discharged and was maintained on oral amoxicillin/clavulanic acid (Smithkline Beecham plc, United Kingdom) 875/125 mg every 12 hours for 2 months. She had remained well when examined on her follow-up visit 3 months later.

3. Discussion

The cerebral veins and venous sinuses have no valves, and therefore, blood within them can flow in either direction depending on the pressure gradient. Uncontrolled infections of the facial area frequently led to septic thrombosis of the intracranial venous sinuses in the pre-antibiotic era. Three types of dural venous sinus thrombosis have been established by anatomy, and they are cavernous sinus thrombosis, lateral sinus thrombosis (LST), and superior sagittal sinus thrombosis [15]. The thromboses in lateral and transverse sinuses in our case belonged to the LST group.

LST is a rare but feared intracranial complication of otitis media and associated mastoiditis. Modern reported mortality rates are lower
than earlier ones but still range from 2% to 10% [17–20]. The pathogenesis seems to be almost exclusively because of the spread of infection to the lateral and sigmoid sinuses from the mastoid air cells, either through the emissary vein or by direct invasion. LST most frequently occurs as a complication of ear infection [15–21]. Our patient did not have any of the typical signs of otitis media, such as otorrhea and otalgia, and therefore, this cannot be considered to be the primary site of infection. The superior rectus muscle abscess complicated by *G. morbillorum* bacteremia was considered as the primary site of infection for LST in this case. This abscess, along with thrombosis, could spread the bacteria through the superior ophthalmic vein, which could be followed by cavernous sinus, superior petrosal sinus, transverse sinus, sigmoid sinus, and internal jugular vein.

In only 28% of the cases in the era of antibiotics have the bacterial pathogens causing LST been adequately demonstrated [16]. The principal organisms associated with LST reflect the bacteriology of chronic otitis media, namely, *Proteus* species, *S. aureus*, *Escherichia coli*, and anaerobes [16]. In a pediatric study of LST, streptococci were the predominant pathogens [19]. *Gemella morbillorum* has not been previously reported to be associated with LST. However, this pathogen may be a more common cause of LST than is presently recognized because this species of bacteria is sometimes incorrectly identified or remains unidentified.

*Gemella morbillorum* is an aerobic or aerotolerant Gram-positive coccus that is usually found as paired cells. It is often isolated on rich, nonselective media, for example, blood agar plates, where it may exhibit no hemolysis or *α*-hemolysis. It does not grow on 6.5% NaCl and is positive for 1- pyrrolidonyl-β-naphthylamide hydrolysis and leucine aminopeptidase tests [1]. However, during Gram staining, the cells are easily decolorized and may, therefore, appear to be Gram variable and even Gram negative. It is likely that the Gram-staining abnormality and morphological polymorphism are responsible for the misidentification of this pathogen. This, in turn, may have led to the fact that so few cases of *G. morbillorum* infection are reported. *Gemella morbillorum* usually behaves as a commensal organism of the mucous. Invasive infection caused by this pathogen often occurred in patients with sinusitis or in those who had poor dental condition [22–24]. Members of the genus *Gemella* share with the viridans group of *Streptococci* and other mucosal pathogens the ability to produce specific immunoglobulin A1 protease [25], which enables the bacteria to evade the adherence inhibitory activity of secretory immunoglobulin A in vitro. By this mechanism, this pathogen is able to cause bloodstream infection from a local mucosal infection. The pathogenesis of thrombosis caused by *G. morbillorum* has been rarely investigated. In a case of shunt nephritis [26], this organism was proven to act as a trigger for the production of auto-antibodies against proteinase 3, which are widely recognized as being able to induce the small-vessel vasculitis associated with focal necrotizing damage. Focal vascular damage can induce the release of coagulative substances and cause thrombosis [27].

*Gemella morbillorum* is generally susceptible to penicillin and other antibiotics, but resistance to penicillin has been reported [22]. Empirical antibiotic treatment should be selected carefully. In addition to culture-directed antibiotic therapy, surgery has been frequently required for the treatment of LST. Lateral sinus recanalization usually follows the removal of the inflammatory focus. A superior rectus muscle abscess was considered as the primary site of infection for LST in our case. Although our case had a good outcome without surgery, nonetheless, early aspiration or debridement of the abscess is a superior treatment of LST.

The efficacy of anticoagulation is controversial in the treatment of LST. A review of cases from the otorhinolaryngological literature supports anticoagulation in selected patients who have a clot burden that extends beyond the sigmoid sinus, who have neurologic changes, who have undergone embolic events, or who have persistent fevers despite appropriate surgical intervention [28]. Although the clot burden extended to the internal jugular vein and transverse sinus in our case, there were no embolic events, persistent fever, or neurologic changes associated with the LST. Antibiotics alone without anticoagulation appeared to be sufficient to treat the LST under these conditions. In conclusion, this is the first case of LST caused by *G. morbillorum* to be reported. Furthermore, in addition to otitis media, it seems that a superior ocal muscle abscess can be the primary site of infection for LST. Although *G. morbillorum* is primarily characterized in the literature as a pathogen related to septicemia, this case report emphasizes the need to consider the possibility of dural thrombosis in patients when *G. morbillorum* is isolated.

References


