Viridans Group Streptococcus Meningitis in an Immunocompetent Child: A Case Report

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Conflict of interest: None declared

Patient: Female, 14-month-old
Final Diagnosis: Viridans group streptococcus meningitis
Symptoms: Fever • irritability • vomiting
Medication: —
Clinical Procedure: —
Specialty: Infectious Diseases
Objective: Rare disease

Background: Viridans group streptococci (VGS) are commensal organisms in humans that are considered contaminants when isolated from culture specimens. However, VGS can be pathogenic when recovered multiple times from blood cultures or when in immunocompromised hosts. VGS are the leading cause of dental abscesses and infective endocarditis in children with underlying congenital heart diseases. They are not commonly involved in meningeal infections, but meningitis due to VGS can be fatal if not treated. The onset is usually preceded by an upper respiratory tract infection or in association with neurological surgical procedures.

Case Report: Our patient was a 14-month-old girl with no significant past medical history who presented with fevers and irritability for 2 weeks. A full sepsis workup, including blood, urine, and cerebrospinal fluid (CSF) cultures, was obtained. Culture results were notable for the abundant growth of VGS in the CSF. However, brain imaging and echocardiogram were normal, with no evidence of brain abscesses or cardiac vegetations, respectively. The patient had shown marked clinical improvement after receiving 2 weeks of intravenous ceftriaxone, which was selected based on the VGS susceptibility profile.

Conclusions: Bacterial meningitis is one of the most burdensome infectious diseases worldwide, despite improvements in diagnostic methods, management, and national immunization programs. Clinicians should consider VGS in the differential diagnosis of potential infections that can cause meningitis.

Keywords: Irritable Mood • Meningitis, Bacterial • Viridans Streptococci

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/933089
Background

Viridans group streptococci (VGS) are commensal organisms that reside in humans, specifically in the oral cavity, skin, respiratory system, and gastrointestinal system [1-3]. Although they have been known as organisms of low virulence and pathogenicity, serious life-threatening infections have been described [4]. VGS can cause several invasive infections, including native valve endocarditis, dental abscess, septicemia in neutropenic patients, brain abscess, and neonatal sepsis [5-10]. Meningitis secondary to VGS is rare, with VGS possibly only responsible for 0.3% to 5% of all cases of proven cultured meningitis [11,12]. Most reported cases were in adults, and less frequently in neonates [13,14]. Predisposing factors to such infections include history of trauma, neurosurgical procedures, penetrating trauma, and malignancy [13,15-18].

Case Report

A previously healthy 14-month-old girl was presented to clinical care with vomiting, irritability, and decreased activity. These symptoms were preceded by 2 weeks of documented fevers. Prior to hospital admission, she was seen at a local clinic and received 2 doses of ceftriaxone. Her birth and medical and surgical histories were unremarkable. She was not fully immunized at the time of presentation. There was no recent history of sick contacts, travel history, or animal exposure.

At presentation, the patient was extremely irritable. Her vital signs revealed a high-grade fever (39°C), tachycardia (pulse 135 beats/min), and tachypnea (32 breaths/min). She had a normal capillary refill time and perfusion in all extremities but looked pale. She had nuchal rigidity and stiffness. Her pupils were normal in size and reactive to light, bilaterally. The oral cavity examination revealed mild pharyngeal erythema without exudate, tonsillar enlargement, or obvious dental concerns. The physical examination was otherwise unremarkable.

Laboratory testing revealed leukocytosis (27 091 WBC/mm³) along with bandemia (17.3%). A lumbar puncture yielded cloudy cerebrospinal fluid (CSF) with pleocytosis (42 WBC/μL), 43% neutrophils, normal glucose level (61.0 mg/dL; serum glucose 80 mg/dL), elevated protein (207 mg/dL), and many gram-positive cocci in chains. After cultures had been collected from blood, urine, and CSF, intravenous vancomycin (20

Figure 1. Viridans streptococci on (A) blood agar and (B) chocolate agar plates; alpha hemolysis (partial or green discoloration), minute to small, gray, convex shaped, smooth, or matte colonies. (C) Disc diffusion plate of antibiotics susceptibility on blood agar; resistant to optochin disk, sensitive to vancomycin, ceftriaxone, and oxacillin.
mg/kg every 6 h) and ceftriaxone (100 mg/kg/day) were empirically started for suspected bacterial meningitis. A computed tomography scan and brain magnetic resonance imaging (MRI) were normal, with no evidence of brain abscesses. The CSF culture grew abundant colonies of bacteria identified as VGS using VITEK 2 GP ID CARD for identification, ST03 card for susceptibility test, and VITEK 2 Compact instrumentation (Biomerieux, France). The identification of VGS was further supported based on colony morphology, Gram stain, optochin resistance, bile solubility, no growth in 6.5% NaCl, and negative biochemical reaction for catalase (Figure 1). Antibiotic susceptibilities were determined according to the Clinical and Laboratory Standard Institute guidelines [19]. Her isolate was intermediate to ampicillin (minimum inhibitory concentration [MIC]=4 μg/mL) but sensitive to ceftriaxone (MIC ≤0.5 μg/mL); therefore, ceftriaxone was continued for the whole duration of therapy. Blood culture results were negative, and an echocardiogram showed no evidence of vegetations or valve pathology. Immune workup results were interpreted according to her age. The immunoglobulin levels (IgE=37.37 mg/dL, IgA=29 mg/dL, IgG=6024 mg/dL, and IgM=72 mg/dL) and lymphocyte subset comprehensive panel (absolute lymphocyte count=4731 cells/μL, T lymphocytes [CD3+]=3311 cells/μL, T-helper cells [CD3+/CD4+]=1892 cells/μL, cytotoxic T cells [CD3+/CD8+]=946 cells/μL, B lymphocytes [CD19+]=1040 cells/μL, natural killer cells [CD16+/CD56+]=283 cells/μL, CD4+/CD8+ ratio=2) were normal for her age. The patient was discharged home after she received 2 weeks of intravenous ceftriaxone. She had a normal hearing test upon discharge.

**Discussion**

The principal clinical syndromes associated with VGS are bacteremia, subacute bacterial endocarditis, upper respiratory infection, and sepsis in patients with hematological malignancies [1,2,5,7]. VGS recovered from the CSF are often consistent with specimen contamination, rather than being true pathogens [3]. Nachamkin and Dalton reported that only 8 of 43 adult patients whose CSF cultures were positive for α-hemolytic streptococci other than *Streptococcus pneumoniae* were determined to be clinically significant and treated as meningitis [20]. Given the CSF parameter abnormalities, the number of organisms isolated, and the response to therapy, we believe that VGS was most likely the cause of meningitis in our patient’s case. The CSF glucose level can be normal in these infections [3].

The source of these infections for most cases is endogenous flora. Poor oral hygiene and dental procedures are known risk factors for VGS bacteremia and subsequent endocarditis [3]. In a single review of 55 adult cases of VGS meningitis, upper respiratory infection was found in 31% of patients, extracranial infection and endocarditis in 13%, and head trauma or neurosurgical procedures, including lumbar puncture, in 8%. In approximately 35% of these patients, a source of entry was not identified [11]. Our patient did not have a positive blood culture for VGS; however, we hypothesize transient bacteremia may have occurred secondary to upper respiratory infection prior to presentation to our hospital.

VGS have the tendency to develop brain abscesses and infective endocarditis [7,8]. Our patient did not have evidence of fluid collection on the brain MRI, and echocardiography showed no vegetation or valve disease. The basic immune workup, including immunoglobulin level and lymphocyte subset, did not reveal any abnormality, although VGS meningitis in the absence of cardiac involvement has been described in immunocompromised patients [21,22]. VGS has not been shown to be associated with immunodeficiencies beyond profound neutropenia, cytarabine therapy, and mucositis [23].

VGS are generally susceptible to β-lactams, macrolides, tetracyclines, and aminoglycosides [24]. However, VGS have become increasingly resistant, which has created significant problems for clinicians in recent years. The percentage of resistance against penicillin and erythromycin in the medical literature ranges from 30% to 40% [21,22,25]. This resistance is believed to be due to the alteration of penicillin-binding protein [25]. The subtype isolated in our case was intermediate to penicillin, leading to the use of ceftriaxone for the entire 2 weeks of therapy.

**Conclusions**

VGS should be considered to be a true pathogen in patients with signs and symptoms of meningitis. Clinicians should be aware of the pathogenicity of this organism and the different invasive diseases that it can cause.

**Acknowledgements**

We thank Dr. Samah Abdelhamid (Security Forces Hospital) for her clinical evaluation, which ultimately led to this patient’s diagnosis, and for continuing outpatient management.

**Conflicts of Interest**

None declared.

**Declaration of Figures Authenticity**

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