



Subdural Empyema due to *Streptococcus constellatus*

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Most subdural empyemas (SE) are caused by '*Streptococcus milleri group*', which is now termed '*Streptococcus anginosus group*' (SAG). However, in the literature there is paucity of information on which species of SAG cause SE. We present the case of a 26-year-old female with subdural empyema (SE) caused by *Streptococcus constellatus*. *Strep. constellatus* is a species of SAG. The patient was successfully treated with burr hole evacuation of the subdural empyema and antibiotic therapy. When subdural empyemas are caused by members of SAG, it would be useful to identify the causative species. Different species of SAG might be associated with different outcomes.

***Streptococcus constellatus* • subdural empyema • *Streptococcus anginosus group* • antibiotics**

OBJECTIVE AND IMPORTANCE

Subdural empyema (SE) is a focal collection of pus between the dura mater and the arachnoid mater. About 95% of the subdural empyemas occur intracranially with most in the frontal region; and 5% involve the spinal subdural space.

It has a tendency to spread rapidly through the subdural space until limited by specific boundaries (falx cerebri, tentorium cerebelli, base of the brain, foramen magnum). SE is usually unilateral. SE account for 15-20 % of all localized intracranial suppurations [1,11].

Before the advent of antimicrobial therapy, the disease was essentially fatal, the mortality rate approached 100% before the introduction of penicillin in 1944, but with current methods of diagnosis and treatment, mortality rates are approximately 10% to 20% [1,11].

With progression, SE has a tendency to behave like an expanding mass lesion resulting in increased intracranial pressure (ICP). Cerebral edema and hydrocephalus also may be present secondary to

disruption of blood flow or cerebrospinal fluid (CSF) flow caused by the increased ICP. Cerebral infarction may be present from thrombosis of the cortical veins or cavernous sinuses or from septic venous thrombosis of contiguous veins in the area of the SE [1].

In infants and young children, SE most often occurs as a complication of meningitis. In such cases, SE should be differentiated from reactive subdural effusion (sterile collection of fluid due to increased efflux of intravascular fluids from increased capillary wall fenestrations into the subdural space). In older children and adults, it occurs as a complication of paranasal sinusitis, otitis media, or mastoiditis. Most cases of SE in adults are associated with some form of sinus infection or sinus abnormality [1].

The infection usually enters into the subdural space from the frontal or ethmoid sinuses or, less frequently, from the middle ear, mastoid cells, or sphenoid sinus. This often occurs within two weeks after a sinusitis episode, with the infection spreading intracranially through thrombophlebitis in the venous sinuses. Infection also may extend directly through the cranium and dura from an erosion of the

posterior wall of the mastoid bone or frontal sinus.

Direct extension also could be from an intracerebral abscess. Rarely, infection spreads hematogenously from distant foci, most commonly from a pulmonary source or as a complication of trauma, surgery, or septicemia [1,2,11].

Common causative organisms are anaerobic and aerobic *Streptococci*, *Staphylococci*, *Haemophilus influenzae*, and other gram-negative bacilli [1].

The most of SE are caused by 'Streptococcus milleri group', which is now termed 'Streptococcus anginosus group' (SAG). They are anaerobic or micro-aerophilic streptococci. Although these bacteria are part of the normal flora in the oral cavity, urogenital region and intestinal tract, they frequently cause purulent infections in various body sites [5, 6].

Streptococcus constellatus, *Streptococcus anginosus*, *Streptococcus intermedius* are three distinct species that constitute the SAG. However, in the literature there is paucity of information on which species of SAG cause SE [2,10,11].

In this report we report a case of SE caused by the *Strep. constellatus* a species of SAG, in a previously healthy female.

CLINICAL PRESENTATION

A 26-year-old woman was transferred to us with fever, headache, and confusion from another hospital. For few days prior to the transfer she had been experiencing from nasal discharge, vomiting and left eye swelling. Per oral Amoxicillin/clavulanate had been commenced two days earlier at the referring hospital.

On admission, she was agitated, stuporous and had nausea and vomiting. On general examination the patient appeared ill. Her temperature was 38.7°C, blood pressure 110/80 mm Hg, pulse 68 per minute and respiratory rate 18 per minute. Glasgow Coma Scale of the patient was 8/15. She had left periorbital edema and hyperaemia and posterior limiting subconjunctival haemorrhage. The pulmonary and cardiac examinations were normal. Severe neck stiffness and other meningeal signs were present. There was no papilloedema.

Blood analysis showed a white-cell count of

18,400/mm³ (polymorphonuclear leukocytes 85%, lymphocytes 8%), hemoglobin of 10.3 g/dl, platelet count of 106,000/mm³. Erythrocyte sedimentation rate was 120 mm in the first hour; C-reactive protein was 35.3 mg/dl (normal value, 0.5 mg/dl). Serum biochemistry showed normal results.

A cranial computed tomography (CT) revealed a subdural hypodense collection (about 7mm thick and spanning about 40 mm) over the left frontoparietal region, effacement of left lateral ventricle and shift of the midline structures to the right.

The patient was promptly taken to operating theater. While under general anaesthesia the patient had a burr hole over the left coronal suture and about 50 ml of purulent material was evacuated from the subdural space.

Microscopic examination of the pus showed mostly polymorphonuclear leucocytes but, no organisms. Post-operatively empirical treatment with intravenously meropenem, which has a broad spectrum, was commenced.

Cultures of the purulent material on day 3 grew streptococci; alpha-hemolytic *streptococci* were isolated on blood agar and identified as *Streptococcus constellatus* using the automated Phoenix System (BD, USA). Antimicrobial susceptibility testing was done using the disc diffusion test in-addition to Phoenix system according to the guidelines of the National Committee for Clinical Laboratory Standards [13]. The isolate was susceptible to penicillin G, cephalosporins, carbapenems, clindamycin and vancomycin. Minimum inhibitory concentrations were determined using the E Test (AB Biodisk, Sweden) and the results were as follows: penicillin G, 0.045 g/ml; cefotaxime, 0.012 g/ml; and meropenem, 0.032 g/ml. Subsequent to this the antibiotic treatment was changed to penicillin G on the fifth day of admission (4 million units intravenously 6 times a day).

The patient's condition rapidly improved, and on the fifteenth day of treatment, CT of the head was repeated. This scan did not show any residual subdural collection. The patient was discharged after 4 weeks of penicillin G treatment. At the sixth-month follow-up the patient was well and had no neurological deficits.

DISCUSSION

Subdural empyema refers to collection of pus in the space between the dura and arachnoid. In 60-90 percent [1] of the cases there is (often asymptomatic) concurrent sinusitis or otitis. Aerobic streptococci, staphylococci, gram-negative organisms, and anaerobic organisms, including anaerobic and microaerophilic streptococci (in particular the SAG) have been reported as the causative agents of SE [1].

Strep.constellatus is Gram-positive, catalase-negative cocci, and often requires carbon dioxide for growth [8]. *Strep.constellatus* strains may be beta-hemolytic, alpha-hemolytic or nonhemolytic and mainly possess Lancefield group F antigens, although some strains may be of group A, C or G or may be non-groupable [8]. The isolate of *Strep. constellatus* from our patient was alpha-hemolytic and belonged to Lancefield group F.

Streptococcus anginosus group of organisms can produce dental abscesses and localized infections in the brain, the sinuses, the lungs, the heart, the abdomen, and other sites, and have been isolated in approximately 50-80% of brain abscesses [7,10]. On Medline, reported cases of SE due to *Strep. constellatus* is very rare [5].

Strep. constellatus could produce alpha-glucosidase and hyalurodinase. Production of hyaluronidase may be an important determinant in the pathogenicity of *Strep. constellatus* [11,12]. The capsular material produced by encapsulated strains of SAG also might be a pathogenic factor [13].

Okayama et al. (6) recently showed that supragingival dental plaque is a source of the infectious pathogens that cause abscess formation. In their study, the majority of isolates of dental plaque belonged to SAG, particularly *Strep. constellatus* [6]. In our patient we were not able to ascertain the source of the infective *Strep. constellatus*.

Most isolates remain fully susceptible to penicillin G and other antibiotics, mainly beta-lactam agents and macrolides [4]. Penicillin G is the antibiotic of choice for treatment of *Strep. constellatus* infections. Vancomycin and erythromycin have also been used effectively in patients with allergy to penicillin [14].

Recently, Limia et al. (4) found that the minimum inhibitory concentration of penicillin was in the

intermediate range for 5.6% of the strains of SAG [4]. Our strain was susceptible to penicillin and other antibiotics (cephalosporins, carbapenems, clindamycin and vancomycin).

Often antibiotic treatment and surgical drainage of subdural empyemas are necessary [10]. Our patient also was successfully treated by burr-hole drainage of SE and four weeks of treatment with penicillin G.

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