



## Case report

Meningitis from invasive *Streptococcus agalactiae* in a healthy young adult

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## ARTICLE INFO

## Article history:

Received 29 June 2020

Received in revised form 2 July 2020

Accepted 3 July 2020

## Keywords:

Group B streptococcus  
*Streptococcus agalactiae*  
Acute otitis media  
Mastoiditis  
Meningitis

## ABSTRACT

*Streptococcus agalactiae* is well known to be a potential etiology of bacterial meningitis in neonates. Invasive *S. agalactiae* has been also reported in nonpregnant adults. Among adults, the incidence of invasive group B Streptococcus (GBS) has been increasing 2–4 times in the past 2 decades. Chronic medical disease was suspected to increase the susceptibility for invasive GBS, especially diabetes mellitus. There was only one case reported to have GBS meningitis from acute otitis media infection in otherwise healthy individual. Hereby, we are reporting the second invasive GBS meningitis from acute otitis media infection with mastoiditis.

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## Introduction

*Streptococcus agalactiae* was first described by Rebecca Lancefield as a micro-organism found in the vaginal tract of asymptomatic women in 1930 [1]. However, the pathogenic characteristic was not reported until 1938 where fatal postpartum infections was published [1,2]. It has been found that group B streptococcus (GBS) can cause invasive disease in both neonates and adults. The most common source of invasive GBS is bacteremia without a focus and skin/soft tissue infections [3,4]. Invasive GBS disease can manifest as meningitis and considered the most severe form of GBS [5]. It is uncommon in adults 7.3/100,000, but the incidence continues to increase. It became a significant pathogen among both neonates and adults [6,7]. Comorbidity is common with invasive GBS disease, and diabetes mellitus is the most common associated chronic medical condition [3,8].

We are reporting a young woman without past medical history who presented with meningitis, the most severe form of invasive *Streptococcus agalactiae*.

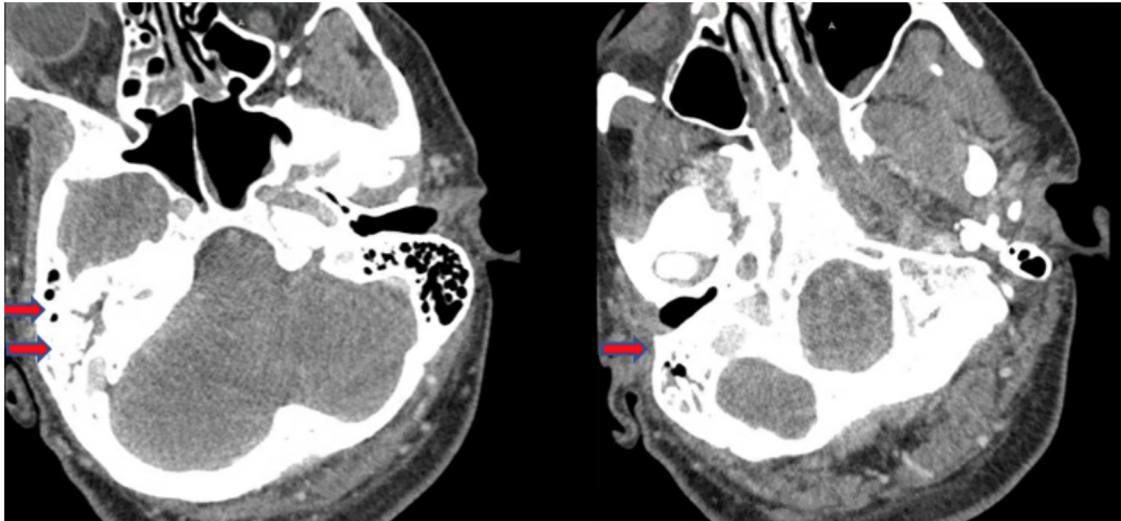
## Case presentation

A 38-year-old female with a medical history of anxiety and gestational diabetes presented to the emergency department (ED) for altered mental status noted by her family. She recently was started 2 days ago on amoxicillin 500 milligrams for an ear infection by her primary care doctor and was complaining of headaches and nausea for the week prior to admission. As per her family, she was on a 6-h camping road trip with her spouse and came home and took a nap. She was later found in the driveway lethargic and speaking in random, unorganized words. Emergency medical services (EMS) was called and found the patient to have a temperature of 102 degrees Fahrenheit, other vitals were within normal limits, physical exam was only remarkable for lethargy and mumbled speech however neurological exam was otherwise unremarkable.

She was brought to the ED where her vitals were a temperature of 102.1 degrees Fahrenheit, blood pressure of 133/65 mm Hg, heart rate of 82 beats per minute, and respiratory rate of 48 breaths per minute. Her oxygen saturation was 97 % on room air. On physical exam, she was obtunded in apparent respiratory distress, her cardiovascular and pulmonary exam was unremarkable, she was not following any commands but moving all extremities without any motor deficits and had no nuchal rigidity. She was ultimately intubated for airway protection in the ED and was started on broad spectrum antibiotics with vancomycin and piperacillin-tazobactam. Tick-borne disease panel was obtained

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**Fig. 1. CT scan of head.** Near complete opacification of the right mastoid air cells and right middle ear. There is normal appearance of the ossicles, semicircular canals, cochlea and vestibule. No ossicular chain erosions. The tegmen tympani is intact. No evidence of erosion in the roof of the sinus tympany or pneumocephalus.

which resulted negative and computed tomography (CT) scan of the head, chest, and abdomen was unremarkable, with no pathology. CT scan of the temporal bones revealed near complete opacification of the right mastoid air cells and right middle ear (Fig. 1). Laboratory results revealed a white blood cell count of  $18.0 \times 10^3/\mu\text{L}$  (normal range:  $4.5\text{--}11.0 \times 10^3/\mu\text{L}$ ) and a lactic acid of  $4.2 \text{ mmol/L}$  (normal range:  $0.5\text{--}2.0 \text{ mmol/L}$ ).

Due to high suspicion for meningitis, the patient was switched to vancomycin and ceftriaxone, was started on steroids and acyclovir then transferred to the intensive care unit (ICU) for further management. She underwent a lumbar puncture with cerebrospinal fluid (CSF) analysis which was turbid in appearance and straw colored. CSF results showed a glucose level of  $59 \text{ mg/dL}$  (normal range:  $50\text{--}75 \text{ mg/dL}$ ), protein of  $366 \text{ mg/dL}$  (normal range:  $14\text{--}45 \text{ mg/dL}$ ), WBC count of  $17,982/\mu\text{L}$ , segmented neutrophil count of  $83 \%$  (normal range:  $0\text{--}6 \%$ ), and monocyte count of  $2 \%$  (normal range:  $26\text{--}55 \%$ ). CSF and blood cultures grew *Streptococcus agalactiae* and infectious disease recommended antibiotic coverage with intravenous penicillin G for a total of 14 days based on culture sensitivities. Otolaryngology was also consulted, and the right ear middle ear effusion was drained with a tympanostomy. She was weaned off the ventilator and transferred to the medical floors on day 5 where she received a peripherally inserted central catheter (PICC) line. She was ultimately discharged home after a 7-day hospital course to continue her intravenous antibiotic regimen and on outpatient follow-up one week later remained asymptomatic and medically stable.

## Discussion

*Streptococcus agalactiae* (Group B streptococcus, GBS) is a normal floral bacterium that colonizes the gastrointestinal and genitourinary tracts of healthy adults [9]. It is a well-known culprit for neonatal invasive diseases [3]. Moreover, it has a potential to cause morbidity and mortality among non-pregnant and elderly adults [3]. Invasive GBS disease is defined as isolation of GBS from a normally sterile site (e.g., blood or cerebrospinal fluid) in an individual who is  $>18$  years and not pregnant or  $<30$  days postpartum. Invasive GBS bacterial meningitis include  $0.3\text{--}4.3 \%$  of all bacterial meningitis [10]. It has been reported that GBS does not

readily penetrate the blood-brain barrier, except in young infants [11].

The rate of invasive GBS disease is increasing among non-pregnant adults by 2–4-fold over the past 2 decades [3,10,12]. This is evident by the large study that has been conducted during 1990–2007 which showed almost doubling the incidence of invasive GBS from  $3.6\text{--}7.3$  cases per 100,000 persons ( $P < 0.001$ ) [3]. Furthermore, it is still not clear why there is an increase incidence of the invasive GBS infection among nonpregnant adults [9]. Stratification of many studies and data did not show relevance of the age nor the different virulence subtype of the bacteria [12,13].

The immunocompromised and the elderly were the most susceptible to invasive GBS infection with a mortality reported to be more than  $50 \%$  [9]. Per Crespo-Ortiz et al., at least one underlying chronic disease was associated with most of the invasive GBS cases ( $91 \%$  of his study). The most common chronic disease was diabetes mellitus ( $44.4 \%$ ) [3,9]. Other chronic underlying diseases include metastatic cancer, and transplant history. However, very few cases of invasive GBS has been reported in healthy individuals without any underlying chronic disease or medical history, as reported in our case [9,11].

The clinical syndromes associated with *Streptococcus agalactiae* infections ranges from skin and soft tissue infections (cellulitis) ( $8.2 \%\text{--}25.6 \%$ ), pneumonia ( $12.6 \%$ ), peritonitis ( $22.8 \%$ ), and arthritis ( $5.5 \%$ ) [3,9]. However, mixed bloodstream infections were also reported ( $20.6\%\text{--}39.3 \%$ ) and cases of endocarditis due to this organism have been reported [3,9,14]. In an analysis of 21 studies, where invasive GBS was reported in 1167 episodes,  $<1 \%$  ( $1\text{--}12$  cases) have been reported. [10–31].

Moreover, it was agreed that the growing incidence with age was related to the chronic medical conditions [15]. Which makes acute otitis media, as in the presented case, a rare source of invasive GBS, and worthy to consider. As the trend and behavior of invasive GBS in nonpregnant adults is still underestimated and overlooked, delayed recognition of the illness may lead to detrimental patient outcomes [32,33]. Treatment with penicillin G remains the first line therapy for *Streptococcus agalactiae* infections as resistance to non-beta-lactamase antibiotics, including vancomycin, has been increasing [14]. Generally, antibiotics are given for a minimum of 10 days with meningitis requiring a 14-day course [14].

## Conclusion

*Streptococcus agalactiae* can cause invasive disease in both neonates and adults. Although the incidence is still very low in adults, it still has a significant pathological consequence. Diabetes and other co-morbidities are common with invasive GBS disease, but for unknown reasons it can occur in otherwise healthy individuals such as our case presented with acute otitis media. Regardless of the cause and source of infection, GBS meningitis should be immediately identified and aggressively treated.

## Funding statement

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

## CRedit authorship contribution statement

**Asseel Al-Bayati:** Visualization, Writing - original draft, Writing - review & editing. **Steven Douedi:** Visualization, Writing - original draft, Writing - review & editing. **Ghadier Alsaoudi:** Visualization, Writing - original draft, Writing - review & editing. **Maurice Mosseri:** Visualization, Writing - original draft, Writing - review & editing. **Vandan Upadhyaya:** Writing - review & editing. **Nancy Gornish:** Writing - review & editing. **Mohamed Elsawaf:** Writing - review & editing.

## Declaration of Competing Interest

The authors declare that there is no conflict of interests regarding the publication of this paper.

## Acknowledgments

Not applicable.

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