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Group B Streptococcus and upper respiratory tract infection – report of *S. agalactiae* associated with bacteraemic tonsillitis.

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Abstract

Group B streptococcal infection is a well known cause of sepsis in the neonatal and obstetric setting but is rarely associated with upper respiratory tract infections. This brief report describes a case of group B streptococcal bacteraemia secondary to acute tonsillitis, together with a short case series of pharyngitis associated with the same organism. Discussion follows regarding the organism's role in throat infection.

Keywords

Group B Streptococcus; bacteraemic tonsillitis; pharyngeal infection

Introduction

Streptococcus agalactiae (Group B Streptococcus [GBS]) can be found as part of normal mucosal flora in the gastro-intestinal and genital tracts. It can also become pathogenic especially in the obstetric and neonatal settings. More recently, this organism's ability to cause true infection in other patient groups and with other clinical diseases has been promoted. The role of GBS in upper respiratory tract infection, where other haemolytic streptococci are much more commonly involved, has been largely overlooked. This case report describes a bacteriologically confirmed case of bacteraemic tonsillitis caused by GBS and a short series of other recently diagnosed cases of pharyngitis where this organism was considered the likely pathogen. The prevalence of these cases occurring mainly in young adults raises a possible association with sexual acquisition, given the organism's known human habitat.

Case Report

A 50 year old woman presented to the Emergency Department with a 2 day history of acutely painful, swollen tonsils. She had no significant past medical history and did not take any medication. She denied any genitourinary or gastrointestinal symptoms.

On examination she was febrile (38.5°C) with a tachycardia of 110 but her blood pressure and oxygen saturations were normal at 110/70 and 97% respectively. Her throat revealed an exudative tonsillar inflammation with pus present bilaterally. Lung examination was clear on auscultation and a thorough systems examination failed to show any clinical soft tissue, joint or abdominal findings of note. The white blood cell (WBC) count was significantly raised at 18.7 x 10^9/L but C-reactive protein was only slightly elevated at 13 mg/L (normal range <5 mg/L). Both renal and liver function tests including urea, creatinine, bilirubin, alanine transferase and alkaline phosphatase levels were all within normal limits.

Blood cultures and a tonsillar swab were taken at the time of admission. A diagnosis of probable acute bacterial tonsillitis was made and she was given a stat dose of IV Benzyl penicillin 1.2g along with IV fluids and analgesia. Her tachycardia and fever reduced within a few hours such that she could be discharged from the Emergency Department to complete a 10 day course of oral Phenoxymethyl penicillin (penicillin V 500mg 6 hourly).

After 5 hours incubation in the microbiology laboratory, Gram positive cocci in chains were identified from both bottles of the blood culture. Inoculation and then incubation of the appropriate blood agar plates for 24 hours revealed grey colonies surrounded by beta haemolysis and typical of GBS. Identification of the organism was subsequently confirmed via MALDI-TOF as *Streptococcus agalactiae*. Antibiotic sensitivity testing, using standard laboratory methods, showed that the isolate was sensitive to penicillin, erythromycin, ceftriaxone and resistant to tetracycline. More prolonged incubation of one blood agar plate in anaerobic conditions failed to grow any other haemolytic streptococcus or recognised pharyngeal pathogen e.g. *Arcanobacterium spp., Fusobacterium spp.*.

Due to the uncertain association of GBS with true throat/tonsillar infection and the failure to grow the same organism from the throat itself, the Emergency Department was asked to recall the patient for clinical review. She returned after 3 days and felt much improved. Repeat clinical assessment showed her to be afebrile with only a residual mild sore throat. After careful review of her presentation, symptoms and general progress, there was no focus of infection to explain the GBS bacteraemia other than acute tonsillitis. On completing the course of penicillin therapy, a repeat blood test revealed a normal WBC count of 5.9×10^9/L and the patient was considered to have made a full recovery.

This rare case led to a search for other potential cases of pharyngitis/tonsillitis associated with GBS, via microbiological samples sent to the laboratory over the previous 2 years. The clinical and microbiological records of all individuals with GBS grown from blood cultures were reviewed. During this period there were no other GBS bacteraemic episodes detected where the upper respiratory tract had been considered the likely source. Stored laboratory data was also used to detect throat or tonsillar swabs from which pure heavy growths of GBS had been isolated and 6 cases were found. None of these individuals had had accompanying blood cultures taken as all specimens had been collected in the primary care (GP) setting. It is therefore possible that one or more of this group of patients may also have had a bacteraemic phase to their infection. Clinical details from GP records of the six cases were gathered from around the time when the throat swabs were obtained and in at least 5 patients, there was a specific clinical description of tonsillitis with the presence of purulence or pus recorded for at least 3 individuals (see Table). Excluding one patient aged 51, all other cases were seen in young adults aged 18-20 years.

Discussion

Streptococcus agalactiae has long been recognised as a cause of sepsis in pregnant women and neonates but since the introduction of antenatal screening in some countries and the wider use of intrapartum antibiotic prophylaxis, the incidence of maternal/neonatal GBS infection has reduced dramatically. Infection in non-pregnant adults now accounts for more than 75% of invasive GBS disease where significant morbidity and mortality may still occur [1]. Since the 1980's, a wide variety of clinical diseases associated with GBS have been reported. These include skin/soft tissue infections, urinary and lower respiratory tract infections in the elderly, discitis and vertebral osteomyelitis and more rarely

conditions such as meningitis, endocarditis, peritonitis and endophthalmitis [2].

The role of GBS as a true pathogenic cause of pharyngitis or tonsillitis has been more controversial, partly due to the finding that it may colonise mucosal surfaces and can be isolated from infected sites along with other more recognised virulent organisms [3]. Group A streptococcus (GAS) is accepted as the most frequent major bacterial pathogen causing pharyngitis and tonsillitis. Evidence to support non-Group A haemolytic streptococci (i.e. Groups B, C & G isolates) as aetiological agents of pharyngitis comes mainly from reports of outbreaks, although cases of heavy pure growths of these organisms from the throat swabs of affected individuals has been well described [4-6]. Their pathogenicity is supported by the findings that non-Group A streptococci share virulence factors with GAS including M proteins, streptokinase, and superoxide dismutase amongst others [7,8]. These all help to overcome defensive barriers and aid survival in the host.

An association between GBS and pharyngitis/tonsillitis was reported nearly 50 years ago when a series of 4 young adults (aged 19-21) with severe exudative pharyngitis, tonsillitis and cervical adenopathy had heavy pure GBS isolated from throat cultures [9]. It may be pertinent to note that in 5 of the 6 cases described in this current series, the age of affected individuals was 18 to 20, suggesting a specific link with young adults. One explanation for this possible association could be increased sexual activity amongst this age group leading to transmission of GBS through orogenital contact. One of the patients had had GBS isolated from a genital culture swab taken 5 months before the episode of pharyngitis. Another published series looked at the throat cultures of over 1,000 patients with pharyngitis and GBS was found as a heavy pure growth in 4.4% of cases although there was no age breakdown given. These individuals were reported as being more likely to have had enlarged tonsils, exudate and tender cervical adenopathy [10].

A GBS bacteraemia associated with pharyngitis has been previously reported, occurring in a middle-aged woman who subsequently developed endocarditis [11]. Examination of our patient on two occasions failed to identify any other potential source for the bacteraemia apart from her severe tonsillitis. The rarity of this case is supported by the huge published series (nearly 20,000 episodes) of invasive GBS infection occurring over a 17 year period in non-pregnant adults which failed to record a single episode of pharyngitis/tonsillitis as an underlying cause although a few cases of otitis media were included [12].

Conclusion

In summary, and after reviewing the literature, there appears to have been little interest for many years in the role of GBS as an upper respiratory tract pathogen despite the organism's undoubted ability to cause disease elsewhere. *Streptococcus agalactiae*, when isolated from the throat, is often dismissed as representing colonising flora rather than having a potential causative role even though prevalence studies have indicated low carriage rates of GBS in the throat. The bacteraemic patient presented here, together with the further 6 cases of pharyngitis/tonsillitis seen in primary care, warrant attention and heavy pure growths of this organism when isolated from throat or tonsillar swabs in patients with appropriate signs and symptoms should not be dismissed as insignificant, particularly in young adults presenting with a purulent upper respiratory tract infection.

Table

Table: Clinical and microbiological details of six patients found to have GBS from throat swabs, taken at time of acute sore throat (April 2013 - March 2015).

Patient Age / Gender	Clinical Details	Culture Result from Throat swab	Sensitivities	Other relevant history and results
18/M	Diabetic, sore throat with bilateral inflamed tonsils.	Heavy pure GBS	*Sensitive to penicillin, erythromycin & ceftriaxone. Resistant to tetracycline.	EBV IgM –ve, IgG +ve. Consistent with past EBV infection.
20/F	Unwell, pus seen on both tonsils. History of recurrent tonsillitis.	Heavy pure GBS		Group A Strep from throat 2 months earlier.
18/F	Unwell, sore throat, grossly swollen purulent tonsils.	Heavy pure GBS		Nil
18/F	Unwell, sore throat, injected, red pharynx.	Heavy pure GBS		Genital culture from 5 months earlier showed GBS carriage.
51/M	Bilateral purulent, swollen tonsils.	Heavy pure GBS		Nil
19/M	Acute bilateral follicular tonsillitis.	Heavy pure GBS	Sensitive to penicillin & ceftriaxone. Resistant to erythromycin & tetracycline.	Nil

*Most common overall sensitivity pattern for *Streptococcus agalactiae* with approx 80% resistance to tetracycline but universal sensitivity to penicillins and cephalosporins.

References

1. Blancas D, Santin M, Olmo M, Alcaide F, Carratala J, Gudiol F. Group B streptococcal disease in nonpregnant adults: incidence, clinical characteristics and outcome. Eur J Clin Micro Infect Dis 2004; 23: 168-73.

2. Edwards MS, Baker CJ. Group B streptococcal infections in elderly adults. Clin Infect Dis 2005; 41: 839-47.

3. Farley MM. Group B streptococcal disease in nonpregnant adults. Clin Infect Dis 2001; 33: 556-61.

4. Cimolai N, Elford RW, Bryan L, Anand C, Berger P. Do the β-haemolytic non-group A streptococci cause pharyngitis? Rev Infect Dis 1988; 10: 587-601.

5. Turner J, Fox A, Fox K, Addy C, Garrison C, Herron B, et al. Role of group C beta-hemolytic streptococci in pharyngitis: epidemiologic study of clinical features associated with isolation of group C streptococci. J Clin Micro 1993; 31: 808-11.

6. Tiemstra J, Miranda RL. Role of non-group A streptococci in acute pharyngitis. J Am Board Fam Med 2009; 22: 663-69.

7. Efstratiou A, Pyogenic streptococci of Lancefield groups C and G as pathogens in man. J Appl Micro 1997; 83 (S1): 72S-79S.

8. Burnham CD, Tyrrell GJ. Virulence factors of group B streptococci. Rev Med Micro 2003; 14: 109-18.

9. Braunstein H, Tucker EB, Gibson BC. Identification and significance of Streptococcus agalactiae (Lancefield Group B). Am J Clin Path 1969; 51: 207-13.

10. Chretien J, McGinniss C, Thompson J, Delaha E, Garagusi V. Group B beta-hemolytic streptococci causing pharyngitis. J Clin Micro 1979; 10: 263-66.

11. Mannik M, Baringer J, Stokes J. Infections due to group B beta-hemolytic streptococcus — report of three cases and review of the literature. New Eng J Med 1962; 266: 910-13.

12. Skoff TH, Farley MM, Petit S, Craig AS, Schaffner W, Gershman K, et al. Increasing burden of invasive group B streptococcal disease in nonpregnant adults, 1990-2007. Clin Infect Dis 2009; 49: 85-92.

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