

Bartholin's Gland Abscesses Caused by *Streptococcus pneumoniae* in a Primigravida

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ABSTRACT

Bartholin gland cysts and abscesses are common problems in females during their reproductive time. Majority of Bartholin's gland abscesses described are of polymicrobial nature, but not necessarily involves opportunistic microbes that colonize the perineal region. In this report, we describe a case of Bartholin's abscess caused by *Streptococcus pneumoniae* in a 25-year-old non-diabetic primigravida.

Key words: Bartholin abscesses, primigravida, *streptococcus pneumoniae*

INTRODUCTION

Bartholin gland cysts and abscesses are common problems in females during their reproductive time. The abscess is formed due to infection of the fluid accumulated inside the blocked duct. Fluid may upsurge over many years before abscess forms.^[1] Most of Bartholin's gland abscesses have been found to be caused by microorganisms that colonize the perineal region.^[2] The infection is usually polymicrobial, with *Bacteroides spp.* and *Escherichia coli* being the predominant organisms.^[3] In this report, we describe a case of Bartholin's abscess caused by *Streptococcus pneumoniae* in a 25-year-old primigravida.

CASE REPORT

A 25-year-old non-diabetic primigravida of 31 weeks gestation presented to the outpatient obstetrics and gynecology clinic with a painful lump on the right side of the vaginal opening and pain during the

sexual intercourse of 1 week duration. She had a history of urinary tract infection 2 weeks before the presentation. On examination, there were a body temperature of 38°C and swollen and inflamed right labia majora. Pelvic examination revealed an enlarged, firm and tender 5 by 4 cm swelling of Bartholin's gland on the right side. Her laboratory work-up revealed low hemoglobin of 10.3 g%, white blood cell count of 6300/dl with differential count showing polymorphs of 73% and lymphocytes of 20%, eosinophil 1% and monocytes 6%. She has also reduced glucose-phosphate dehydrogenase (G6PD) activity. Her glucose tolerance test for pregnancy was 5.7 mmol/L (normal is <7.8 mmol/L). Here, hepatitis B and C and venereal disease research laboratory tests were negative. Her urine analysis showed pus cells 50-99 cell/HPF, with positive leukocyte esterase of 3+ and urine nitrite was negative; but her urine culture was sterile.

Complete drainage of the abscess was done through a small surgical cut under local anesthesia in the out-patient clinic. Pus drained and sent immediately to the laboratory. Gram stain of pus revealed the presence of plenty of pus cells with Gram-positive cocci in pairs and short chains with its characteristic lancet shaped appearance and no Gram-negative diplococci were seen.

Culture of the abscess material on blood and chocolate agar in 5% CO₂ atmosphere revealed alpha hemolytic

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slightly mucoid colonies with “punched-out” center as the colonies age. No growth on MacConkey agar and anaerobic culture media was observed. Identification was done using catalase, bile solubility and optochin susceptibility tests. Catalase test was negative. Bile solubility test induced lysis of the organism using the tube method while the optochin susceptibility test showed a zone of inhibition of 16 mm with a 6 mm of 0.5 µg optochin disk. Antibiotic susceptibility testing done by disk diffusion method (using Mueller Hinton agar supplemented with 5% defibrinated sheep blood and the plate was incubated at 35°C in 5% CO₂ atmosphere for 24 h) revealed resistance of the organism (no zone) to 1 µg oxacillin disk from oxoid (Unipath, Ontario, Canada). Hence, minimal inhibitory concentration (MIC) was done according to clinical and laboratory standards institute (2012)^[4] using Phoenix automated microbiology system (BD Diagnostics, Sparks, MD), which revealed penicillin MIC of 2 µg/ml that indicates susceptibility to parenteral penicillin, amoxicillin, amoxicillin-clavulanic acid, cefepime, cefotaxime, ceftriaxone and meropenem.^[4] The organism was sensitive to amoxicillin (MIC 1 µg/ml), cefuroxime (MIC ≤ 0.5 µg/ml), cefotaxime (MIC ≤ 0.5 µg/ml), cefepime (MIC ≤ 0.5 µg/ml), meropenem (MIC 0.5 µg/ml), erythromycin (MIC ≤ 0.0625 µg/ml), tetracycline (MIC ≤ 0.5 µg/ml), levofloxacin (MIC ≤ 0.5 µg/ml) and vancomycin (MIC ≤ 0.5 µg/ml). The patient was treated with oral cefuroxime 500 mg twice-a-day for 1 week and repeat culture from the wound site on follow-up was found to be sterile.

DISCUSSION

Infection of Bartholin's glands is one of the most common infections in gynecologic field.^[5] The type and the frequency of the causative microbes of Bartholin's gland abscess varied over the years. In the second half of the last century; gonococci played a significant role and involved in more than one-third of cases.^[6] Infection with anaerobic bacteria was also reported to be quite common.^[7] However, *Chlamydia trachomatis* had been implicated in quite number of cases with Bartholin's gland abscess.^[8] The bacteriogram in the last two decades showed implication of other bacteria in causing Bartholin's gland abscess. *E. coli* was the most frequently isolated bacteria from this abscess. Poly microbial infections with both aerobes and anaerobes were detected with high frequency. Anaerobes would be derived from vaginal flora and might strengthen the pathogenicity of aerobes.^[9] Interestingly there is an increasing isolation rate for respiratory tract-associated infectious organisms, such as *S. pneumoniae* and *Haemophilus influenzae* from Bartholin's gland abscesses.^[9]

In this case report, we isolated *S. pneumoniae* from Bartholin's gland abscesses in a young primigravida non-diabetic lady. In particular, *S. pneumoniae* known to be a common component of the indigenous flora of the oropharynx and is associated with both upper and lower respiratory tract infections and is one of the most common causes of bacterial meningitis in adults and young adults. There are more than 91 known serotypes of *S. pneumoniae*.^[10] There were quite number of the previous reporting *S. pneumoniae* as a cause of Bartholin's gland abscesses. Quentin *et al.* 1999 described three cases of Bartholin's gland abscesses caused by *S. pneumoniae* isolates of serotype 3 or 4.^[3] Mikamo *et al.* 2005 described one case of *S. pneumoniae* and *Fingoldia magna* causing Bartholin's gland abscesses. They showed that the isolated *S. pneumoniae* was penicillin-resistant *S. pneumoniae*.^[2] Parvathi *et al.* 2009 described a case of acute Bartholin's abscess caused by *S. pneumoniae* in a primigravida.^[11]

It is possible that infection of Bartholin's gland with *S. pneumoniae* can be a primary focal abscess that could disseminate causing more serious infections. Danilova and Vdernikova 2006 described two cases of pelviperitonitis caused by *S. pneumoniae*.^[10] The increasing rate of genital infection with *S. pneumoniae* and other respiratory organisms may be due to orogenital contact as a result of increasing tendency to orosexual activity. This was proved by the presence of the same resistance patterns for *S. pneumoniae* isolated from both Bartholin's gland abscesses and respiratory tract infections.^[8]

Our patient was diagnosed to have severe reduction of G6PD activity. Patients with severe G6PD reduction may have associated severe leukocyte G6PD deficiency. They may present with impairment of nicotinamide adenine dinucleotide phosphate-oxidase activity and a history of recurrent infections, mimicking the phenotype of chronic granulomatous disease.^[12]

There is a wide range of treatment spectrum for Bartholin glands abscesses. It varies from silver nitrate gland ablation; laser abscess fenestration, ablation, or excision, marsupialization, needle aspiration with or without alcohol sclerotherapy, fistulization using a Word catheter, Foley catheter or Jacobi ring, gland excision and or incision and drainage followed by primary suture closure.^[13]

From this case, three possible hypotheses were elaborated and are in a need for further studies. The first hypothesis is about the possible role of pregnancy in increasing the risk of Bartholin's gland abscess. The second hypothesis is the possible relationship between oral sex and the

increased incidence of genital infections with microbes that usually cause upper respiratory tract infections. The third hypothesis is the effect of G6PD deficiency in increasing the incidence and tendency of abscess formation.

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