Bartholin’s abscess caused by hypermucoviscous Klebsiella pneumoniae

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Klebsiella pneumoniae serogroups displaying the hypermucoviscosity phenotype are associated with a distinct clinical syndrome characterized by liver abscesses, bacteraemia and metastatic lesions. We describe here what we believe to be the first reported case of hypermucoviscous K. pneumoniae causing a superficial Bartholin’s abscess in the absence of systemic involvement.

Discussion

Hypermucoviscous K. pneumoniae is a virulent Klebsiella subtype that was first recognized in Taiwan and is now an emerging cause of community-acquired invasive infections worldwide (Fang et al., 2004; McIver & Janda, 2008; Nadasy et al., 2007; Wang et al., 1998). In particular, this organism is associated with pyogenic liver abscesses in both immunocompetent and diabetic patients often without apparent underlying hepatobiliary disease (Lee et al., 2006). This invasive syndrome can result in numerous metastatic complications, including endophthalmitis, suppurrative meningitis and pleural empyema (Chen et al., 2004; Lee et al., 2006; Liu et al., 1986; Wiskur et al., 2008; Yang et al., 2007). We present here a case of Bartholin’s gland abscess with hypermucoviscous K. pneumoniae. While community-acquired extra-hepatic abscess with hypermucoviscous K. pneumoniae has been described (Ku et al., 2008), to our knowledge, this is the first reported case of an isolated, superficial infection with this organism.

Bartholin’s glands are located bilaterally at the posterior introitus and provide lubrication for the vaginal vestibule. It is estimated that 2 % of all women will develop a Bartholin’s duct cyst or gland abscess in their lifetime, making the diagnosis and treatment of this infection a relatively common occurrence in gynaecological practice (Omole et al., 2003). In a recent study of the microbiota of Bartholin’s gland abscess in Asia, Klebsiella species accounted for only a small fraction of cases (7/224, ~3 %) and the presence of hypermucoviscosity was not noted (Tanaka et al., 2005). As the klebsiellae are members of the Enterobacteriaceae, it is probable that this patient’s

Case report

In April 2008, an otherwise healthy 36-year-old Asian American woman presented to her gynaecologist with a 3-day history of vulvar swelling. She reported a similar episode in 2002 that resolved with antibiotic treatment. Her medical and surgical history was otherwise unremarkable. The patient was born in Vietnam and emigrated to the United States as a child but denied any recent travel to Asia.

On physical exam, she had a firm, left-sided Bartholin’s abscess and no other abnormal findings. At this initial visit, she was prescribed a 7-day course of ciprofloxacin, 500 mg per day, and was scheduled to return to the clinic for incision and drainage 2 days later. At that time, a swab of the abscess material was sent for routine bacteriological work up. The patient’s abscess healed well following the drainage procedure and completion of the course of ciprofloxacin. On follow-up, she had no signs or symptoms of local or disseminated recurrence of infection.

Culture of the abscess material on blood agar revealed numerous large, dull-grey, hypermucoid colonies positive for the string test (Fig. 1). Growth on MacConkey agar indicated lactose fermentation. Identification and antibiotic susceptibility testing by microdilution performed on the MicroScan Walkaway (Siemens, Dade-Behring) identified Klebsiella pneumoniae resistant to ampicillin and susceptible to cefazolin, piperioclillin/tazobactam, gentamicin, ciprofloxacin, trimethoprim–sulfamethoxazole and levofloxacin. Serological testing revealed that the isolate had the K2 capsule type and PCR evaluation indicated that the strain carried the plasmid-encoded regulator of mucoid phenotype A (rmpA) gene. Consistent with the serogrouping results, the mucoviscosity-associated gene A (magA), a marker of the K1 serotype, was not present. The causative agent of this Bartholin’s gland abscess was therefore K. pneumoniae expressing the K2 capsular serotype and the hypermucoviscosity phenotype.
infection arose from faecal contamination. Given her history of a previous incidence of the same syndrome, we hypothesize that the patient and/or her sexual partner carried this organism in their gastrointestinal tract.

The majority of invasive infections with hypermucoviscous *K. pneumoniae* have been reported in Asia and in Asian patients living abroad (Kawai, 2006). Consistent with this, our patient was Vietnamese. While these observations suggest that Asian ancestry may be an important risk factor for both invasive and superficial disease, the basis for this apparent ethnic specificity remains unknown. Host genetic susceptibility, limited geographical distribution of hypermucoviscous subtypes, or contamination of unique dietary elements may all play a role in the epidemiology of this infection.

The hypermucoviscosity phenotype is thought to contribute to invasive virulence by impairing phagocytosis and enhancing resistance to serum killing (Fang *et al.*, 2004). The underlying molecular mechanism involves multiple factors, including the antigenicity of the capsule itself, in particular the K1 and K2 serotypes (Chuang *et al.*, 2006; Fang *et al.*, 2004; Struve *et al.*, 2005; Yeh *et al.*, 2006, 2007), as well as the expression of the rmpA gene, whose protein product positively regulates extra-capsular polysaccharide synthesis (Nassif *et al.*, 1989; Yu *et al.*, 2006, 2008). Although the *K. pneumoniae* strain isolated in our case expressed the K2 capsule and carried the plasmid-borne rmpA gene, the patient showed no signs or symptoms of systemic illness. It may be that this isolate lacks additional virulence factors important for invasive disease (Yu *et al.*, 2008). Alternatively, it is possible that the Bartholin’s gland represented the primary focal abscess and only rapid identification and treatment prevented this infection from further spread.

It is our hope that this case report heightens awareness of hypermucoviscous *K. pneumoniae* as a cause of superficial infection. While this patient did not progress to invasive disease, identification of this organism from an isolated site should suggest the potential for spread and prompt a thorough clinical evaluation for systemic involvement.

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**References**


