Journal of Medical Microbiology (2009), **58**, 671–673 DOI 10.1099/jmm.0.006734-0

Case Report

Bartholin's abscess caused by hypermucoviscous *Klebsiella pneumoniae*

Benjamin A. Pinsky,[1] Ellen J. Baron,[1,2] J. Michael Janda[3] and Niaz Banaei[1,2]

Correspondence
Benjamin A. Pinsky
bpinsky@stanford.edu

[1]Department of Pathology, Stanford University School of Medicine, Stanford, CA 94305, USA

[2]Clinical Microbiology Laboratory, Stanford Hospital and Clinics, Palo Alto, CA 94304, USA

[3]Microbial Diseases Laboratory, California Department of Public Health, Richmond, CA 94804, USA

Received 19 September 2008
Accepted 11 January 2009

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.

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, pipericillin/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.

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, suppurative 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

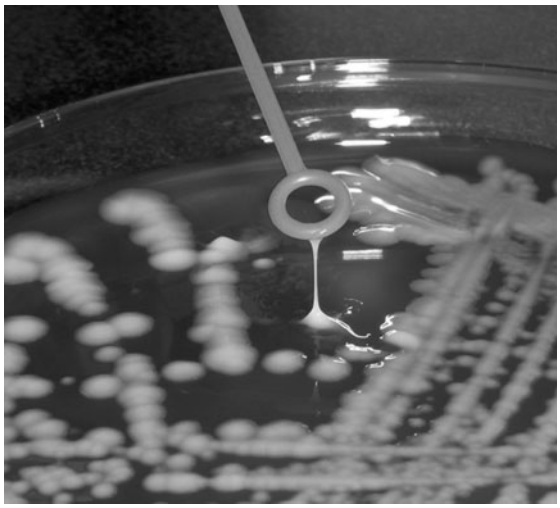

Fig. 1. The patient's *K. pneumoniae* isolate was string test positive (>5 mm string length).

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.

Acknowledgements

We thank Dr Mary Margaret O'Neill for generously providing patient information.

References

Chen, Y. J., Kuo, H. K., Wu, P. C., Kuo, M. L., Tsai, H. H., Liu, C. C. & Chen, C. H. (2004). A 10-year comparison of endogenous endophthalmitis outcomes: an East Asian experience with *Klebsiella pneumoniae* infection. *Retina* **24**, 383–390.

Chuang, Y. P., Fang, C. T., Lai, S. Y., Chang, S. C. & Wang, J. T. (2006). Genetic determinants of capsular serotype K1 of *Klebsiella pneumoniae* causing primary pyogenic liver abscess. *J Infect Dis* **193**, 645–654.

Fang, C. T., Chuang, Y. P., Shun, C. T., Chang, S. C. & Wang, J. T. (2004). A novel virulence gene in *Klebsiella pneumoniae* strains causing primary liver abscess and septic metastatic complications. *J Exp Med* **199**, 697–705.

Kawai, T. (2006). Hypermucoviscosity: an extremely sticky phenotype of *Klebsiella pneumoniae* associated with emerging destructive tissue abscess syndrome. *Clin Infect Dis* **42**, 1359–1361.

Ku, Y. H., Chuang, Y. C. & Yu, W. L. (2008). Clinical spectrum and molecular characteristics of *Klebsiella pneumoniae* causing community-acquired extrahepatic abscess. *J Microbiol Immunol Infect* **41**, 311–317.

Lee, H. C., Chuang, Y. C., Yu, W. L., Lee, N. Y., Chang, C. M., Ko, N. Y., Wang, L. R. & Ko, W. C. (2006). Clinical implications of hypermucoviscosity phenotype in *Klebsiella pneumoniae* isolates: association with invasive syndrome in patients with community-acquired bacteraemia. *J Intern Med* **259**, 606–614.

Liu, Y. C., Cheng, D. L. & Lin, C. L. (1986). *Klebsiella pneumoniae* liver abscess associated with septic endophthalmitis. *Arch Intern Med* **146**, 1913–1916.

McIver, C. J. & Janda, J. M. (2008). Pathogenesis and laboratory identification of emerging hepatovirulent *Klebsiella pneumoniae. Clin Microbiol Newsl* **30**, 127–131.

Nadasy, K. A., Domiati-Saad, R. & Tribble, M. A. (2007). Invasive *Klebsiella pneumoniae* syndrome in North America. *Clin Infect Dis* **45**, e25–e28.

Nassif, X., Fournier, J. M., Arondel, J. & Sansonetti, P. J. (1989). Mucoid phenotype of *Klebsiella pneumoniae* is a plasmid-encoded virulence factor. *Infect Immun* **57**, 546–552.

Omole, F., Simmons, B. J. & Hacker, Y. (2003). Management of Bartholin's duct cyst and gland abscess. *Am Fam Physician* **68**, 135–140.

Struve, C., Bojer, M., Nielsen, E. M., Hansen, D. S. & Krogfelt, K. A. (2005). Investigation of the putative virulence gene *magA* in a worldwide collection of 495 *Klebsiella* isolates: *magA* is restricted to the gene cluster of *Klebsiella pneumoniae* capsule serotype K1. *J Med Microbiol* **54**, 1111–1113.

Tanaka, K., Mikamo, H., Ninomiya, M., Tamaya, T., Izumi, K., Ito, K., Yamaoka, K. & Watanabe, K. (2005). Microbiology of Bartholin's gland abscess in Japan. *J Clin Microbiol* **43**, 4258–4261.

Wang, J. H., Liu, Y. C., Lee, S. S., Yen, M. Y., Chen, Y. S., Wang, J. H., Wann, S. R. & Lin, H. H. (1998). Primary liver abscess due to *Klebsiella pneumoniae* in Taiwan. *Clin Infect Dis* **26**, 1434–1438.

Wiskur, B. J., Hunt, J. J. & Callegan, M. C. (2008). Hypermucoviscosity as a virulence factor in experimental *Klebsiella pneumoniae* endophthalmitis. *Invest Ophthalmol Vis Sci* **49**, 4931–4938.

Yang, C. S., Tsai, H. Y., Sung, C. S., Lin, K. H., Lee, F. L. & Hsu, W. M. (2007). Endogenous *Klebsiella* endophthalmitis associated with pyogenic liver abscess. *Ophthalmology* **114**, 876–880.

Yeh, K. M., Chang, F. Y., Fung, C. P., Lin, J. C. & Siu, L. K. (2006). *magA* is not a specific virulence gene for *Klebsiella pneumoniae* strains causing liver abscess but is part of the capsular polysaccharide gene cluster of *K. pneumoniae* serotype K1. *J Med Microbiol* **55**, 803–804.

Yeh, K. M., Kurup, A., Siu, L. K., Koh, Y. L., Fung, C. P., Lin, J. C., Chen, T. L., Chang, F. Y. & Koh, T. H. (2007). Capsular serotype K1 or K2, rather than *magA* and *rmpA*, is a major virulence determinant for *Klebsiella pneumoniae* liver abscess in Singapore and Taiwan. *J Clin Microbiol* **45**, 466–471.

Yu, W. L., Ko, W. C., Cheng, K. C., Lee, H. C., Ke, D. S., Lee, C. C., Fung, C. P. & Chuang, Y. C. (2006). Association between *rmpA* and *magA* genes and clinical syndromes caused by *Klebsiella pneumoniae* in Taiwan. *Clin Infect Dis* **42**, 1351–1358.

Yu, W. L., Ko, W. C., Cheng, K. C., Lee, C. C., Lai, C. C. & Chuang, Y. C. (2008). Comparison of prevalence of virulence factors for *Klebsiella pneumoniae* liver abscesses between isolates with capsular K1/K2 and non-K1/K2 serotypes. *Diagn Microbiol Infect Dis* **62**, 1–6.