



CORRESPONDENCE

Malakoplakia in a patient with complicated urinary tract infection caused by extended-spectrum β -lactamase-producing *Escherichia coli*



Dear Editor,

With great interest, we read the article by Pacheco et al¹ in the *Journal of Microbiology, Immunology and Infection*, which reported that multidrug-resistant, extended-spectrum β -lactamase (ESBL)-producing *Escherichia coli* is an important pathogen in nosocomial urinary tract infection, causing great concern in therapeutic failure. Here, we report a patient who was diagnosed with complicated urinary tract infection caused by ESBL-producing *E. coli* and complicating obstructive uropathy by malakoplakia. We performed multiple-site biopsies of the bladder and ureter to verify the diagnosis and observed Michaelis–Gutmann bodies microscopically, which is the characteristic of malakoplakia. It is a challenge for the clinician for managing such a case infected with emerging resistant strains complicating with malakoplakia.

This 83-year-old male patient had a history of rectal adenocarcinoma, stage T3N0M0, status postoperation, and chemotherapy. He was suffering from fever, left flank pain, and dysuria on admission. The results of the laboratory data were as follows: hemoglobin, 10.6 mg/dL; white blood cells count, 14,800 cells/mL; and platelet count, 178,000 cells/mL. The urine routine examinations showed pyuria and bacteriuria. He empirically received antibiotic therapy with 1-g cefazolin intravenous (iv) drip every 8 hours. The image study of abdominal sonogram revealed left-sided hydronephrosis. The percutaneous nephrostomy plus antegrade pyelography showed hydronephrosis and dilatation of the lower third ureter (Fig. 1A). Endoscopic findings of the bladder and ureter showed some soft yellowish tumor-like plaques over the bladder and ureter mucosa. The pathological findings of biopsy and resection specimens revealed

the typical finding of malakoplakia (Fig. 1B). His fever persisted even after the 3-day cefazolin therapy. However, the urine culture revealed ESBL-producing *E. coli* with a multidrug resistant strain, but susceptible to carbapenems. Therefore, his cefazolin therapy was discontinued and 1-g iv drip of ertapenem was administered every day for a complete 7-day therapeutic course. A follow-up visit with cystoscopic examination revealed that his condition was resolved with urologic surgical intervention and the complete course of antibiotics therapy.

Malakoplakia is a uncommon tumor-like xanthogranulomatous disease that occurs commonly in the urinary tract, followed by the gastrointestinal tract.^{2–4} The clinical manifestation of urinary tract malakoplakia is diverse, from asymptomatic to chronic cystitis, pyelonephritis, renal mass-like lesion, and complicated with obstructive uropathy.^{2–4} Endoscopically, malakoplakia commonly presents with soft yellow to gray polyp-like lesions.² Histopathologically, it is characterized by distinctive Michaelis–Gutmann bodies and with positive von Kossa staining.³ The etiology and pathogenesis of malakoplakia is still not fully understood, but multiple possible mechanisms have suggested that it may be related to chronic infection. The common organisms included *E. coli*, *Proteus*, *Mycobacterium tuberculosis*, and *Staphylococcus aureus*.^{2–4} The macrophages or histiocytes incompletely digest microorganisms accumulated in lysosomes, leading to the mineralization of calcium and iron salts deposited in the cytoplasm and emulating biofilm formation, which is difficult to treat.⁵ The Michaelis–Gutmann bodies of malakoplakia can be highlighted by the Periodic acid–Schiff (PAS) for iron and von Kossa stain for calcium, and this is the diagnostic characteristic (Fig. 1B).

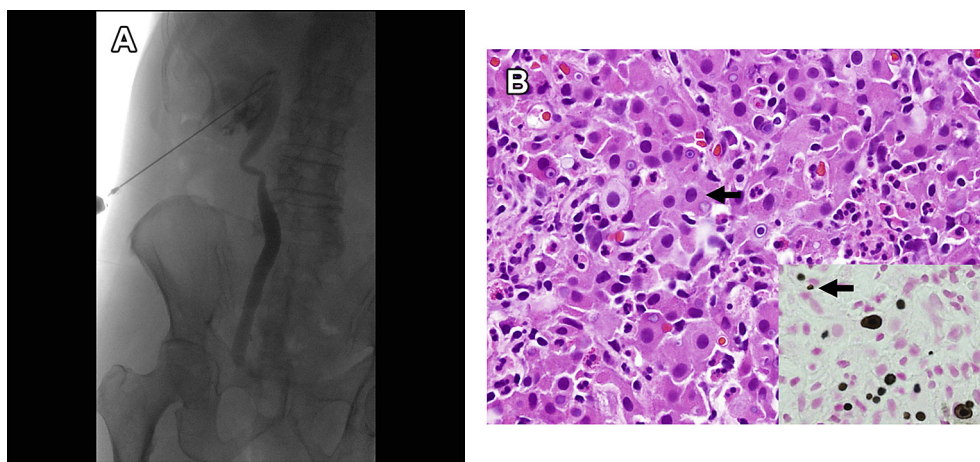


Figure 1. (A) Percutaneous nephrostomy and antegrade pyelography reveals the obstruction of the lower third ureter with hydronephrosis and hydroureter. (B) Pathology of the urinary tract mucosa shows malakoplakia (Michaelis-Gutmann bodies; arrow) in some of the histiocytes as rounded, concentrically-layered inclusions. The inset picture shows positive staining for calcium by von Kossa stain (original magnification 400 \times).

The treatment of malakoplakia involves antibiotics therapy and/or surgical resection.^{2–4} If it is a susceptible pathogen, the antibiotics trimethoprim/sulphamethoxazole, rifampicin, and ciprofloxacin are the drugs of choice for biofilm-related organisms⁵ because these drugs have good penetration into macrophages and contribute to intracellular bacteria death. For a multidrug-resistant microorganism such as ESBL-producing *E. coli*, carbapenems antibiotics plus surgical resection may be an alternative therapy.

Ethics approval

Ethics approval was not required for this study.

Conflicts of interest

The authors have no conflicts of interest to declare.

References

- Pacheco R, Correia S, Monteiro R, Goncalves A, Radhouani H, Ramos S, et al. Multiresistant extended-spectrum β -lactamase producing *Escherichia coli* in human urine samples in Portugal. *J Microbiol Immunol Infect* 2013;46:399–404.
- Nabeshima A, Yamada S, Xin G, Tasaki T, Kitada S, Noguchi H, et al. [A case of malakoplakia of the urinary bladder]. *J UOEH* 2012;34:265–70 [In Japanese].
- Hyun KH, Shin HD, Kim DH. Malakoplakia in a healthy young female patient. *Korean J Intern Med* 2013;28:475–80.
- Abolhasani M, Jafari AM, Asgari M, Salimi H. Renal malakoplakia presenting as a renal mass in a 55-year-old man: a case report. *J Med Case Rep* 2012;6:379.
- Wu WS, Chen CC, Chuang YC, Su BA, Chiu YH, Hsu HJ, et al. Efficacy of combination oral antimicrobial agents against biofilm-embedded methicillin-resistant *Staphylococcus aureus*. *J Microbiol Immunol Infect* 2013;46:89–95.

Wen-Sen Lee

Division of Infectious Diseases, Department of Internal Medicine, Wan Fang Medical Center, Taipei Medical University, Taipei, Taiwan

Wei-Yu Chen

Department of Pathology, Wan Fang Medical Center, Taipei Medical University, Taipei, Taiwan

Tsong-Yih Ou

Fu-Lun Chen

Division of Infectious Diseases, Department of Internal Medicine, Wan Fang Medical Center, Taipei Medical University, Taipei, Taiwan

Yung-Wei Lin*

Division of Urology, Department of Surgery, Wan Fang Medical Center, Taipei Medical University, Taipei, Taiwan

*Corresponding author. Division of Urology, Department of Surgery, Wan Fang Medical Center, Taipei Medical University, Taipei 116, Taiwan.
E-mail address: 89425@wanfang.gov.tw

20 November 2013