Huge fungal perinephric abscess masquerading as malignancy

Sharath P Madhyastha,[1] Raviraja V Acharya,[1] Barnini Banerjee,[2] Koteshwara Prakashini[3]

[1]Department of Internal Medicine, Kasturba Medical College Manipal, Manipal, Karnataka, India
[2]Department of Microbiology, Kasturba Medical College Manipal, Manipal, Karnataka, India
[3]Department of Radio diagnosis, Kasturba Medical College Manipal, Manipal, Karnataka, India

Correspondence to
Dr Sharath P Madhyastha, dr.sharathymc@gmail.com

Accepted 16 December 2018

DESCRIPTION

A 60-year-old woman with diabetes, hypertension for the past 15 years, with two episodes of complicated urinary tract infection in last 6 months treated with broad-spectrum antibiotics (imipenem) in a local hospital, presented with complaints of fever, vomiting, loin pain and breathlessness. On examination, she had tachycardia, tachypnoea, hypotension (blood pressure (BP) of 90/60 mm Hg), O_2 saturation of 84% on room air, decreased breath sounds on left hemithorax and a large non-tender, palpable mass in the left lumbar region of abdomen, measuring around 10×10 cm.

On evaluation, she had anaemia (haemoglobin (Hb) 8.4 g/dL with microcytic hypochromic blood picture), polymorphonuclear leucocytosis of 22 750 cells/mm^3 and thrombocytosis. Serum creatinine was 5.1 mg/dL. Urine microscopy showed the full field of WBCs and yeast cells. Random blood sugar was 283 mg/dL at admission with glycated haemoglobin of 13.8%. The patient was started on empirical broad-spectrum antibiotics (Meropenem with modified dose according to creatinine clearance) after sending blood (in a BacT/ALERT FA Plus bottle) and urine for culture and antibiotic susceptibility testing. She was supported with inotropes, mechanically ventilated and received one session of haemodialysis. Chest x-ray (figure 1) was suggestive of left massive pleural effusion. Pleural fluid aspiration showed neutrophilic exudative pleural fluid (total count of 18 500 cells/mm^3, 99% neutrophils, protein 3.4 g/L, lactate dehydrogenase (LDH) 1814 IU/L, serum total protein 6.5 g/L, serum LDH 581 IU/L). Non-contrast CT of kidneys, ureters and bladder (KUB) (figure 2 and figure 3) showed a large heterogeneous lobulated collection 8.6×6.5×10.3 cm in the perinephric region which was drained with image-guided pig tail insertion and the aspirate was also sent for culture. Both the BacT bottles signalled positive for *Candida kefyr*. In urine, it was 10 000 colony forming unit/mL and in the pleural fluid and perinephric collection, there was a heavy growth of *C. kefyr*. *C. kefyr* was identified by Matrix Assisted Laser Desorption Ionization Time-of-Flight (MALDI-TOF) VITEKMS and antimicrobial susceptibility testing for the isolates was done by VITEK2 system (bioMérieux, Inc, Durham, North Carolina, USA). The isolated *C. kefyr* was sensitive to echinocandins, amphotericin and fluconazole. But the patient had the septic shock which was refractory and succumbed to the illness within 48 hours of hospital stay.

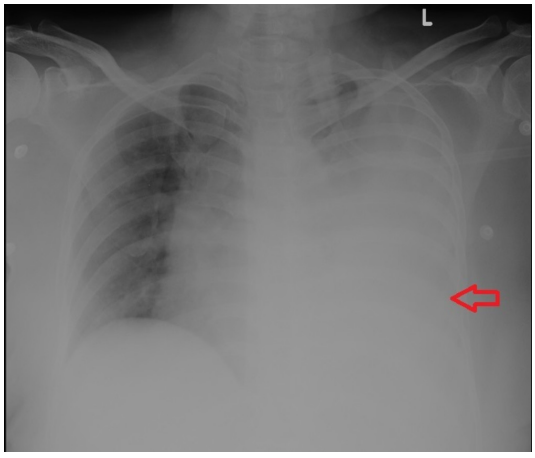

Figure 1 Chest X-ray showing moderate to massive left pleural effusion with mediastinal shift to right (arrow).

C. kefyr, previously called as *Candida pseudotropicalis*, was first reported by Morgan *et al*, in an elderly woman with malignancy.[1] *C. kefyr* is a rare cause of disease. Among 2019 patients with invasive fungal infections reported from North America between 2004 and 2008, 11 isolates were *C. kefyr*.[2] This is the first reported case of disseminated *C. kefyr* from the eastern part of the world.

Risk factors for candida urinary traction infection are diabetes, urinary tract abnormalities, malignancy, urinary tract drainage devices and prior antibiotic therapy.[3] Our patient had uncontrolled diabetes with glycated haemoglobin of 13.3%, prior use of broad-spectrum antibiotics twice. Among the previously reported cases, most common risk factors were underlying malignancy or immunosuppression.

Our isolate was sensitive to all the three drug groups. But our patient presented late and received only one dose of antifungals, so it is difficult to make any conclusions of antifungal efficacy in our case. In 10.5-year worldwide surveillance study, resistance to fluconazole for *C. kefyr* ranged from 3.3% to 1.7% in the different time periods, 1997–2000 and 2005–2007, respectively.[4]

Our patient presented with severe sepsis with septic shock. 'Sepsis is defined as the presence of infection together with systemic manifestations of infection. Severe sepsis is defined as sepsis plus sepsis-induced organ dysfunction or tissue hypoperfusion. Sepsis-induced hypotension is defined as a systolic BP (SBP) <90 mm Hg or mean arterial pressure (MAP) <70 mm Hg or a SBP decrease >40 mm Hg. Septic shock is defined

To cite: Madhyastha SP, Acharya RV, Banerjee B, *et al*. *BMJ Case Rep* 2018;**11**:e226753. doi:10.1136/bcr-2018-226753

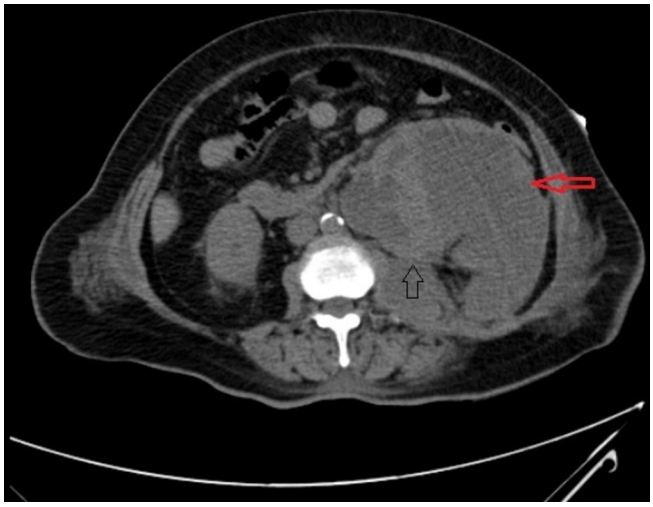

Figure 2 Axial image of plain CT KUB showing large heterogeneous lobulated collection (red arrow) in the left perinephric and paranephric space causing indentation and medial displacement of kidney and collection is extending up to the left psoas muscle (black arrow). KUB, kidneys, ureters and bladder.

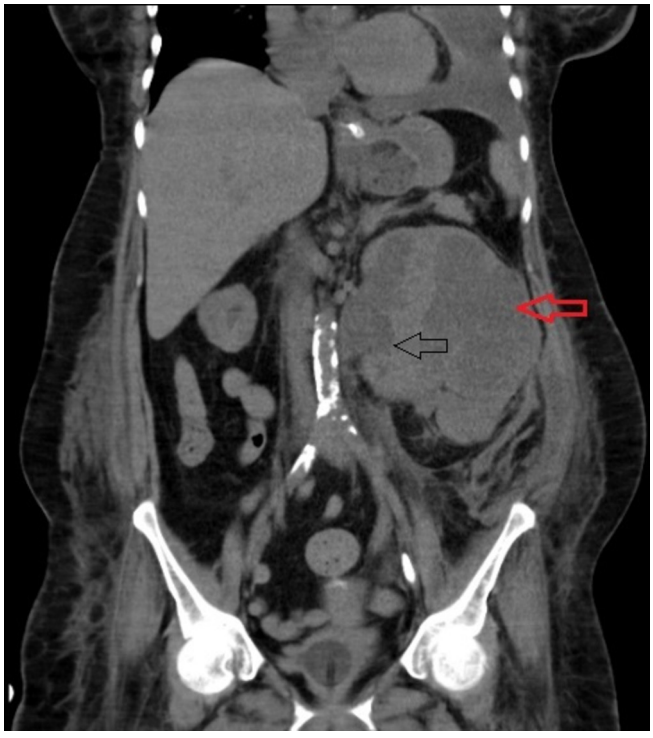

Figure 3 Coronal image of plain CT KUB showing left perinephric abscess (red arrow) with moderate hydronephrosis (black arrow) with abrupt termination at PUJ which possibly represents PUJ obstruction secondary to stricture. Also, note there is moderate to gross left pleural effusion. KUB, kidneys, ureters and bladder; PUJ, pelviureteric junction.

as sepsis-induced hypotension persisting despite adequate fluid resuscitation'.[5]

The above treatment efforts follow the surviving sepsis campaign. The campaign aimed at reducing mortality by 25% from sepsis in the next few years. Key recommendations mentioned by category, include: 'early resuscitation of the septic patient during the first 6 hours after recognition; blood cultures before antibiotic therapy; imaging studies to confirm a potential source of infection; administration of broad-spectrum antimicrobials therapy; initial fluid resuscitation; protocols for inotropes to maintain adequate BP; protocols for mechanical ventilation, weaning and sedation; a protocolised approach to blood glucose management; provision of continuous haemofiltration or intermittent haemodialysis; prophylaxis for deep vein thrombosis; use of stress ulcer prophylaxis; oral or enteral (if necessary) feedings, as tolerated; and addressing goals of care, including treatment plans and end-of-life planning (as appropriate), as early as feasible, but within 72 hours of intensive care unit admission'.[5]

In conclusion, with increasing isolation of non-*Candida albicans* and emergence of azole resistance, with an increasing use of antifungal prophylaxis, knowledge about emerging pathogens and antifungal sensitivities is of importance.

Learning points

- *Candida kefyr* is a rare cause of disease.
- Most common risk factors were underlying malignancy or immunosuppression.
- A high index of clinical suspicion, efforts towards early diagnosis and initiation of antifungal therapy are required to prevent complications.

Contributors SPM and BB: wrote the draft of the manuscript. PK: provided the CT pictures and commented on it. RA: involved in patient management and revised the manuscript critically for important intellectual content. All authors contributed to the literature review and approved the final manuscript for submission.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent Next of kin consent obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

1 Morgan MA, Wilkowske CJ, Roberts GD. Candida pseudotropicalis fungemia and invasive disease in an immunocompromised patient. *J Clin Microbiol* 1984;20:1006–7.
2 Horn DL, Neofytos D, Anaissie EJ, *et al*. Epidemiology and outcomes of candidemia in 2019 patients: data from the prospective antifungal therapy alliance registry. *Clin Infect Dis* 2009;48:1695–703.
3 Kauffman CA, Vazquez JA, Sobel JD, *et al*. Prospective multicenter surveillance study of funguria in hospitalized patients. The National Institute for Allergy and Infectious Diseases (NIAID) Mycoses Study Group. *Clin Infect Dis* 2000;30:14.
4 Pfaller MA, Diekema DJ, Gibbs DL, *et al*. Results from the ARTEMIS DISK Global Antifungal Surveillance Study, 1997 to 2007: a 10.5-year analysis of susceptibilities of Candida species to fluconazole and voriconazole as determined by CLSI standardized disk diffusion. *J Clin Microbiol* 2010;48:1366–77.
5 Dellinger RP, Levy MM, Rhodes A, *et al*. Surviving sepsis campaign: international guidelines for management of severe sepsis and septic shock: 2012. *Crit Care Med* 2013;41:580–637.