A 61-year-old female was admitted for fever and acute renal failure 6 weeks after receiving a kidney transplant. Her past medical history was notable for CKD stage 5 due to chronic idiopathic tubulointerstitial nephritis. This was diagnosed in 1998 when she presented with an elevated creatinine, sterile pyuria and proteinuria, and underwent native kidney biopsy confirming the diagnosis. She was initially treated with corticosteroids and remained on 5 mg daily of prednisone up until the date of her pre-emptive living related kidney transplant. She had a low-risk immunologic profile with a negative T and B cell crossmatch prior to transplant. Per our center protocol, she received basiliximab for induction, and was maintained on tacrolimus, mycophenolate and prednisone for immunosuppression, with trough FK506 levels ranging between 8 and 12 ng/mL. She was discharged on a standard antimicrobial prophylaxis regimen of trimethoprim-sulfamethoxazole and valacyclovir and had a nadir serum creatinine of 1.2 mg/dL following her transplant.

Her post-operative course was uneventful until her presentation to the hospital, when she complained of fevers, nausea and vomiting. Her physical examination and review of systems did not suggest an obvious source for the fever. Admission labs demonstrated evidence of acute kidney injury with an elevated serum creatinine level of 4.1 mg/dL. There were no abnormalities on her chest radiograph, but her urinalysis was suggestive of infection, so empiric ciprofloxacin was initiated. On hospital day 2, her blood cultures turned positive for *Candida krusei* and she was started on voriconazole. Despite therapy, she remained persistently fungemic. An extensive evaluation for a source of her candidemia was largely unrevealing, including a transesophageal echocardiogram. However, computed tomography of the abdomen and pelvis demonstrated mild hydronephrosis and perinephric stranding of the allograft. As there was persistent renal dysfunction with fungemia, a biopsy of her allograft was performed on hospital day 5.

Sections of the allograft biopsy showed marked neutrophilic inflammation with the formation of microabscesses as seen in Figure 1a. Periodic acid Schiff stains highlighted intratubular budding yeast forms.

Fig. 1. (a) H&E stain (magnification of ×100) of the kidney allograft biopsy, demonstrating marked neutrophilic inflammation with the formation of microabscesses. (b) PAS stain (magnification of ×600) highlighting intratubular budding yeast forms.
intratubular budding yeast forms as seen in Figure 1b. A Gomori methenamine silver stain confirmed the diagnosis of candida pyelonephritis.

The patient’s therapy was changed from voriconazole to non-liposomal-based amphotericin B. Immunosuppression was significantly reduced, including cessation of mycophenolate. Repeat biopsy 4 weeks after discharge showed no evidence of fungal infection, but did show grade IB cellular rejection. This was treated with pulse corticosteroids, and her antifungal therapy was transitioned to micafungin and flucytosine. She completed another 7 weeks of therapy with these agents. She is currently 14 months out from her transplant, but unfortunately has a stable creatinine of ~4 mg/dL and has been listed for a repeat transplant.

Solid organ transplant recipients are known to have high rates of invasive fungal infections, with mortality due to candidemia reported to be between 21 and 35% [1, 2].

Patients with candidemia due to C. krusei have associated mortality rates >50%. To our knowledge, this is the first documented case of C. krusei pyelonephritis leading to fungemia in a renal transplant recipient.

Conflict of interest statement. None declared.

References

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