Abstract Partial fungal obstruction of the renal collecting system is an unusual finding among infants that poses specific management problems. We report a patient with sepsis and fungal infection of the kidneys post surgery who presented with bilateral fungus balls and was successfully managed by conservative measures. Sonography is the imaging technique of choice in the diagnosis and follow-up of such patients. The need for prompt diagnosis in high-risk patients and the role of sonography are discussed.

Key words Candida tropicalis · Candidiasis · Fungal bezoar · Renal fungus balls

Introduction Mycotic infections of the kidney, and particularly fungus balls of the urinary tract, are an extremely rare finding among pediatric patients. They mainly occur in premature infants, in the presence of immunosuppression due to chemotherapy or corticosteroids, as well as in some postoperative patients [1]. Factors implicated in the pathogenesis include prolonged application of broad-spectrum antibiotics [2], immunosuppressive agents, intravenous catheters [3], and genitourinary abnormalities affecting urinary flow [4]. Infectious organisms usually belong to the Candida albicans or Aspergillus species.

Although Candida albicans accounts for the majority of Candida infections [5], the number of infections caused by other species is rising [2]. Diagnosis is based on clinical symptoms and detection of fungi in a catheterized or suprapubic urine specimen or blood culture. Although the sonographic features of renal candidiasis are nonspecific, high-resolution sonography can make a valuable contribution to the early diagnosis of fungal bezoars [6–11].

We report an infant presenting post surgery with refractory sepsis in connection with candiduria and associated bilateral fungal bezoars.

Case report

The patient was male and 6 months old (body weight 6,000 g) at the time of presentation. Three months before he had undergone surgical reconstruction of vesical exstrophy. The exposed vesical mucosa was dressed preoperatively with sterile swabs; there was no infection and no antibiotic coverage. Surgery was uneventful, involving bladder closure with bilateral antireflux surgery, reconstruction of the penis, and symphysisal adaptation. Ureteral stents and a suprapubic catheter were also placed. The postoperative course was satisfactory, including effective treatment of two urinary tract infections caused by Enterococcus faecalis (10^5/ml) and Klebsiella pneumoniae (10^2/ml) with oral amoxicillin/clavulanic acid (25 mg/kg). The sonograms routinely obtained in the postoperative course (7.5-MHz transducer) showed no abnormalities other than grade 1 hydronephrosis, which is a common phenomenon related to surgery. Because of this uneventful development, antibiotic prophylaxis was considered dispensable.

The ureteral stents were removed 3 months after surgery. Immediately after the patient developed fever (40°C), feeding problems, and emesis. His serum showed elevated parameters of inflammation [leukocytes 27.4 x 10^9/l, C-reactive protein (CRP) 4.2 mg/dl], while serum blood urea nitrogen (BUN) and creatinine were in the normal range. Suprapubic urine culture revealed Enterococcus faecium and E. faecalis, which were successfully treated with vancomycin (40 mg/kg body weight over 3 days). Follow-up urine cultures were sterile. The child then received long-term prophylaxis with oral cefixime and was discharged from hospital; he was in good general health, was afebrile, and showed normal parameters of inflammation.
Two weeks after being discharged he again developed a fever despite cefixime prophylaxis, and was readmitted 1 week later. He presented with clinical signs of acute respiratory infection, showing elevated parameters of inflammation and a temperature of up to 40°C. Microscopic examination of urinary sediment was negative. After obtaining blood and urine cultures, cefixime was discontinued and clarithromycine given. The urine culture obtained on admission was contaminated. The ensuing temporary stabilization of clinical parameters ended in a relapse 4 days later, with temperature peaks of up to 39.8°C. Subsequent treatment consisted of three doses of imipenem and placement of a central venous catheter for parenteral alimentation because of feeding problems and weight loss. This is when *Candida tropicalis* [10⁶ colony-forming units (cfu/ml)] was first isolated from urine. With resistance testing still pending, imipenem was discontinued and 5-fluorocytosin (5-FC) was initiated.

![Flowchart](chart.png)

**Fig. 1** Clinical course, culture results, and imaging studies. Renal ultrasound studies 1–4 were performed with a 7.5-MHz transducer and 5–7 with a 10-MHz high-resolution transducer (OP1 bladder closure, bilateral antireflux surgery, penis plastic, symphysial adaptation, ureteral stents, and suprapubic catheter placement; OP2 removal of ureteral stents; OP3 central venous line placement (CVL); OP4 new CVL; SPCex removal of suprapubic catheter; n within normal range; B bronchitis; CHA *Candida* hemagglutination titer; BUN blood urea nitrogen; CRP C-reactive protein; Cr creatinine; MRSA methicillin-resistant *Staphylococcus aureus*; E. faecalis *Enterococcus faecalis*; S. intermedius *Streptococcus intermedius*)

We also replaced the central venous catheter, but culture of the catheter was negative.

The sonogram (7.5-MHz transducer) now revealed a shadowless hyperechoic mass in the lower calyces of the left kidney that had a delimited map-like structure. This finding was interpreted as pyelonephritis (Fig. 1, ultrasound 4).

Despite antifungal therapy, the clinical manifestations did not improve: leukocyte numbers and the CRP level remained elevated, and the temperature remained near 40°C. A new urine culture obtained during 5-FC therapy showed that *C. tropicalis* was still growing (10⁸ cfu/ml). The CHA titer had increased to 1:2,560, and serum creatinine (1.03 mg/dl) and BUN (27 mg/dl) were elevated.

Further sonographic examinations with a high-resolution (10-MHz) transducer showed hyperechoic masses with a heteromorphic roundish shape taking up most of the space in the middle and lower-pole calyces of both renal collecting systems (Fig. 2a, b). This characteristic sonographic pattern in association with candiduria and a significant rise of the CHA titer provided the basis for diagnosing renal candidosis with fungal sludge accumulations in the left and, to a lesser degree, right kidneys with superimposed bacterial sepsis.

Magnetic resonance urography and contrast-enhanced computerized tomography (CT) of the kidneys were performed to verify these sonographic findings. Both CT and magnetic resonance imaging (MRI) showed bilateral moderate hydronephrosis (Fig. 3), but failed to delineate the hyperechoic masses in the renal collecting system. DMSA (dimercaptosuccinic acid) scintigraphy indicated that the parenchyma of the left kidney was damaged but supplied no clues for a causal explanation. The presence of a foreign body (residual stent fragment) as the cause or catalyst of the protracted infection was excluded by sonography. Abdominal ultrasonography and CT of the thorax and cranium were performed to exclude metastatic fungal colonies elsewhere in the body.

Immediately after diagnosis, resistance to 5-FC was established and therapy switched to amphotericin B (1 mg/kg per day). After urine cultures had remained positive for *C. tropicalis* for the first 5 days of amphotericin B treatment, fluconazole (6 mg/kg per