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When the Unseen Strikes: Acute Fungal Pyelonephritis with Elusive Etiology in a Healthy Paediatric Patient

[1]Dr Khushbu Agarwal (MD, PDCC-Renal &Transplant Pathology, Fellow-oncopath), Assistant Professor, Department of Pathology, Laboratory Medicine, Transfusion Services and Immunohematology, Institute of Kidney Diseases & Research Centre - Institute of Transplantation Sciences, Gujarat University of Transplantation Sciences, Ahmedabad, Gujarat, India.

[2]Dr Kamal Kanodia (MD, PDCC-Renal &Transplant Pathology, FRCpath), Professor, Department of Pathology, Laboratory Medicine, Transfusion Services and Immunohematology, Institute of Kidney Diseases & Research Centre - Institute of Transplantation Sciences, Gujarat University of Transplantation Sciences, Ahmedabad, Gujarat, India.

[3]Dr Kamlesh Suthar (MD, PDCC-Renal &Transplant Pathology), Professor, Department of Pathology, Laboratory Medicine, Transfusion Services and Immunohematology, Institute of Kidney Diseases & Research Centre - Institute of Transplantation Sciences, Gujarat University of Transplantation Sciences, Ahmedabad, Gujarat, India.

[4]Dr Minaxi Patel (MD, PDCC-renal &Transplant Pathology, FRCpath), Associate Professor, Microbiology Section, Department of pathology, Institute of Kidney Diseases and Research Centre-Institute of Transplantation Sciences, Gujarat University of Transplantation Sciences, Ahmedabad, Gujarat, India.

[5]Dr Kinnari Vala (M.D., Fellow in Pediatric Nephrology), Associate Professor, Department of Pediatric Nephrology, Institute of Kidney Diseases & Research Centre - Institute of Transplantation Sciences, Gujarat University of Transplantation Sciences, Ahmedabad, Gujarat, India.

Corresponding Author: Dr Khushbu Agarwal (MD, PDCC-Renal &Transplant Pathology, Fellow-oncopath), Assistant Professor, Department of Pathology, Laboratory Medicine, Transfusion Services and Immunohematology, Institute of Kidney Diseases & Research Centre - Institute of Transplantation Sciences, Gujarat University of Transplantation Sciences, Ahmedabad, Gujarat, India.

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Abstract

Acute fungal pyelonephritis is extremely rare in healthy children, with most paediatric cases caused by bacteria. Fungal urinary tract infections constitute only 5–7% of cases, usually in immunocompromised patients or those with urinary tract abnormalities or instrumentation. We describe an extraordinary case of a 9-year-old healthy boy with no history of urinary tract abnormalities, prolonged antibiotic use, or immunodeficiency, who presented with fever, vomiting, and abdominal pain. He was started on empirical antibiotics but failed to respond. After exhaustive evaluation, renal biopsy revealed fungal pyelonephritis. Thus, a high index of suspicion for fungal pathogens is crucial when pyelonephritis fails to respond to initial antibacterial therapy.

Keywords: Fungal Pyelonephritis, Healthy, Immunocompromised, Paediatric, Rare

Introduction

Fungal pyelonephritis is a rare but potentially life-threatening kidney infection in children. While bacterial pyelonephritis is well recognized as a cause of significant morbidity and long-term renal injury in the paediatric population, fungal infections of the kidney are far less frequently encountered and are typically associated with disseminated fungal disease or predisposing factors such as urinary tract abnormalities, prolonged antibiotic use, or immunodeficiency[1,2]. Its symptoms are often non-specific, leading to delayed diagnosis. Early and targeted therapy is crucial to prevent long-term complications such as renal scarring. Here, we report an extremely rare case of native kidney fungal pyelonephritis in a young healthy child, highlighting the diagnostic challenges, therapeutic considerations, and the importance of maintaining a high index of suspicion for atypical pathogens in paediatric urinary tract infections.

Case Presentation

A 9-year-old male, presented with a high-grade fever, vomiting, and abdominal pain for the past 3 days, for which he was admitted to a local hospital. He had no prior history of hospitalization, catheterization, diabetes mellitus (DM), or any immunosuppressive therapy. He also did not have any history of urinary tract abnormalities. He was treated with IV antibiotics and other supportive medications. However, he did not respond to the treatment and developed decreased urine output, gradually progressing to anuria.

Then, he was shifted to a paediatric hospital, where he was put on IV antibiotics, and 5 cycles of haemodialysis were done. At that time, his serum creatinine (S.Cr), high sensitivity C-reactive protein (hsCRP), total leukocyte count (TLC) and serum LDH levels were high (Table 1). Serum C3 was slightly low. Routine urine microscopy revealed plenty of RBCs and 35–40 pus cells/hpf. Urine culture revealed a significant colony count of Enterococcus faecium (2 million CFU/ml). Patient was put on IV antibiotic but was still not responding. S. Cr, hsCRP and TLC were still rising. Blood culture was negative. His USG abdomen showed B/L acutely swollen kidneys with increased echotexture and impaired corticomedullary differentiation, suggesting B/L acute renal parenchymal disease.

Table 1: Laboratory parameters

Parameter	Values	Reference range
S. Cr (mg/dl)	2.26	0.3-0.7
hsCRP(mg/L)	119.2	<1
TLC/cmm	29,590	4000-11,000
S.C3 (mg/dl)	86	90-207
LDH (U/L)	1505	128-287

Then he was further referred to a higher center, where a renal biopsy was performed which was reported as features suggestive of acute pyelonephritis. His beta-D-glucan level was done and found to be little high at 8.951 pg/ml (< 7 pg/ml negative). Suspecting some invasive fungal infection, patient was started on IV fluconazole with IV antibiotics. He was still anuric and on haemodialysis.

For additional management, he was subsequently moved to our center, almost 1 month after the initial presentation. At the time of admission, his procalcitonin level, TLC, S. Cr and hsCRP levels were still high, all of which indicated a stage of sepsis.

Our histology department reviewed the kidney biopsy (Fig 1) which revealed that around 35–40% of the cortex had necrosis with renal parenchyma heavily infiltrated by neutrophils, lymphocytes, and fungal hyphae (both septate and non-septate) with microabscess formation. Tubular damage was substantial. Neither vessels nor

glomeruli were noteworthy. Since both septate and non-septate fungal hyphae were seen, mucormycosis and aspergillus were suspected. The absence of fungal buds and pseudo-hyphae reduced the likelihood of candida. These histology findings were not reported by the previous centre.

An MSCT scan revealed multiple non-enhancing hypodense lesions involving parenchyma of all the poles.

Liposomal amphotericin B was started. Patient started responding with urine output of 100-200 ml/day. But he was still on haemodialysis. A repeat biopsy was performed almost 10 days after starting amphotericin which revealed single fungal hyphae but necrosis had progressed involving about 45-50% of the cortex (Fig 2)

However, the patient was discharged against medical and lost to follow up.

Discussion:

Fungal pyelonephritis is a crippling illness that can have disastrous effects. Predisposing factors are diabetes mellitus, indwelling catheters, recent antibiotic usage, urinary tract disease, congenital or structural abnormalities of the urinary tract, prolonged hospitalization, immunocompromised status, malignancy, and renal transplantation [1, 3-5]. Without prompt diagnosis and treatment, it can quickly worsen into multi-organ dysfunction syndrome (MODS) [6].

The infection is thought to spread primarily through two pathways: (1) hematogenous spread and (2) retrograde spread via the urethra. Hematogenous infections typically manifest bilaterally, whereas ascending infections typically manifest unilaterally and typically affect the medulla and renal pelvis [7]. Candida genus, which tends to invade and colonize the urinary tract, is most commonly implicated [8].

In a recent study on fungal pyelonephritis by Husain et al from north India, found the majority of the patients were diabetics (60%). Thirty-six percent of patients were known cases of systemic hypertension, 4% of patients were previously diagnosed cases of chronic kidney disease, 24% had indwelling catheters, and 4% had nephrolithiasis. The most common species isolated was C. albicans [9].

Only few cases of fungal urinary tract infections in children have been reported where either the urinary tract abnormality/obstruction or prolonged hospitalisation were the predisposing factor [10,11]. In our case, the child did not have any structural urinary tract abnormalities, no previous hospitalization or on any immunosuppressive therapy. His blood cultures and urine cultures were negative. The source of invasive fungal infection could not be determined. The disease progressed within a month, as his first USG did not show any infarcts in the kidney. Later he developed multiple infarcts.

This highlights the need for a high index of suspicion for fungal pathogens, especially when a patient fails to respond to standard antibacterial treatment. The case is notable because it occurred in a young, healthy individual with no risk factors, challenging the typical understanding of the disease.

Conclusion

Acute fungal pyelonephritis can occur even in immunocompetent children without urinary tract abnormalities. Delayed recognition may lead to ineffective antibacterial therapy and increased morbidity. Early suspicion, supported by urine microscopy, fungal culture, and biopsy, is crucial for timely antifungal treatment and better outcomes.

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