Bilateral Renal Papillary Necrosis Due to Candida Infection in a Diabetic Patient Presenting as Anuria

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Abstract
A 38 years insulin-dependent diabetic male, with nephropathy on antituberculous treatment presented with painless frank hematuria followed by anuria for a day which was associated with fever. Ultrasonogram of the abdomen showed bilateral hydroureteronephrosis. Necrotic papillae were retrieved after ureteroscopy which on histopathological examination and culture showed Candida albicans. This was successfully treated with fluconazole and ureteroscopic removal of necrotic papillae.

INTRODUCTION
Among various etiopathological causes of acute renal failure obstructive uropathy is a potentially reversible form. Early recognition and treatment are essential as the outcome is related to the duration and completeness of obstruction. We present a patient with acute renal failure and obstructive uropathy due to bilateral renal papillary necrosis caused by Candida albicans infection localised to the renal pelvis and ureters.

CASE HISTORY
A 38 years diabetic male presented with painless frank hematuria for two days and subsequent anuria for a day associated with fever. The patient had mild renal impairment due to diabetic nephropathy and was on insulin and maintenance anti-tuberculous drugs for pulmonary tuberculosis. The drugs included isoniazid, rifampicin and ofloxacin. The patient also had diabetic neurogenic bladder with incomplete voiding.

On examination patient was afebrile. Vital signs were normal. There was slight tenderness in the right loin. Laboratory investigations showed blood urea nitrogen 55 mg/dl and creatinine 4.1 mg/dl. His haemoglobin was 8.3 g/dl and fasting blood sugar 204 mg/dl. He had negative serology for HIV, HBsAg and HCV. Fresh urine examination showed 10-12 RBCs, plenty of WBCs per HPF and budding yeasts. Ultrasonogram of the abdomen showed bilateral hydroureteronephrosis with internal echoes in the pelvicalyceal system suggesting the possibility of papillary necrosis.

On the following day, the patient underwent cystoscopy and bilateral ureteroscopy. On cystoscopy, the left ureteric orifice was blocked with necrotic tissue, projecting out of the ureteric orifice and was removed using grasping forceps. Left ureteroscopy revealed a tortuous and dilated ureter. Ureteroscope was taken upto the left renal pelvis and multiple small flaky tissues were removed. The left pelvi calyceal system had a fungal ball with associated pus. The toileting of the dilated pelvicalyceal system was done with normal saline and a DJ stent was left in situ. Right ureteroscope was taken up to renal pelvis. Two pieces of necrotic tissue were removed. The ureter was again dilated and tortuous. DJ stenting was done. The pus was sent for culture and sensitivity and the necrotic tissue was subjected to histopathological examination.

Light microscopic examination showed necrotic papillae with numerous yeast like budding organisms resembling Candida (Fig. 1 and Fig. 2). The pus from the pelvicalyceal system also grew Candida albicans in culture. Blood culture yielded no growth. The patient was started on parenteral fluconazole 400 mg intravenous daily for one week followed by oral fluconazole. After the ureteroscopic removal of the debris the patient started to pass urine and he had polyuria for next few days. Renal functions on discharge showed a serum creatinine 1.2 mg/dl and blood urea nitrogen 19 mg/dl. Patient continued on oral fluconazole 200 mg daily for three months.

DISCUSSION
Fungi produce characteristic patterns of tissue injury which are modified by the special structure of the tissue in which they occur. Systemic candidiasis is an opportunistic mycosis...
that usually involves the GI tract, kidneys, heart and CNS but can involve almost any organ. Primary infection of the urinary tract by the Candida spp results in cystitis and ascending pyelonephritis that may be complicated by papillary necrosis. Sloughed necrotic papillae containing fungal elements from fungus balls can cause ureteropelvic obstruction with secondary hydronephrosis.2

Diseases which pose a risk factor for candidial infection of the urinary tract include diabetes mellitus, obstructive uropathy, congenital genitourinary abnormalities, neurogenic bladder, ileal conduit, malignancies and immunosuppressed states. Urinary catheterization, stenting, lithotripsy and other invasive procedures are added risk factors. European studies indicate that fungi are responsible for 17% of nosocomial urinary infection in intensive care units.3 Surveillance data in the United States indicate that candidiasis constitutes 10% of intensive care unit infections.

In a series of study of fungal infections of kidney by Raghavan R et al candidiasis was the commonest mycosis encountered.4 Involvement was usually bilateral. Necrosis of papillary tips was present in two out of 16 cases. It has been shown in experimental candidiasis that the blastospores of Candida attach to the glomerular and peritubular capillary endothelium through surface fibrills. Soon after localization in the kidney, these blastospores extend germ tubes which penetrate directly through the tubular cells. Within 24 hours there is necrosis and abscess formation. This necrosis can manifest as papillary necrosis.5

Fluconazole in patients with renal candidiasis at a dosage of 200-400 mg once daily for a minimum of four weeks or for at least two weeks following the resolution of symptoms along with corrective endoscopic surgical intervention in the presence of obstructive uropathy can lead to cure. But persistent candidial pyelonephritis mandates intravenous amphotericin B with the total dosage of 1 gram.3

In conclusion we are presenting a diabetic patient who presented with neurogenic bladder, bilateral obstructive uropathy and anuria which was the result of papillary necrosis due to Candida albicans. With the advent of minimally invasive procedure like ureteroscopy it is not only possible to undertake the procedure for diagnostic reasons but also do difficult therapeutic interventions with good results and minimum post-operative morbidity.

REFERENCES