

A Rare Case of Renal Pelvis Urothelial Carcinoma in Situ Associated with Hydronephrosis and Atrophic Kidney

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ABSTRACT

Urothelial tumours involving the renal pelvis or ureter are relatively uncommon, accounting for about 5% to 7% of all renal tumours and about 5% of all urothelial tumours. Main risk factors for urothelial tumors include smoking, high tea intake and long term use of certain analgesic like phenacetin.

Upper urinary tract urothelial tumors can be associated with renal stone disease or hydronephrosis but association is rare with only few case reports.

Upper urinary tract tumors associated with renal stone and hydronephrosis are often missed in urine cytological examination and ultrasound. Therefore it is important to carefully examine the gross specimen and microscopy, keeping in mind the possibility of upper urinary tract tumors in such cases.

Here we report a case of renal pelvis urothelial carcinoma in situ associated with hydronephrosis and atrophic kidney for its rarity.

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Introduction

Urothelial tumors involving the renal pelvis or ureter are relatively uncommon, accounting for about 5% to 7% of all renal tumors and about 5% of all urothelial tumors.^[1] It accounts for more than 90% of renal pelvic tumors. Other carcinoma type include squamous cell carcinoma and adenocarcinoma which are rare. Urothelial neoplasm have sequence of dysplasia, carcinoma in situ and invasive malignancy.^[2]

Carcinoma in situ is a neoplastic change of urothelium without breach of basement membrane. It is considered a high grade neoplasm and require specific treatment.^[3]

It occurs more commonly in elderly males. Risk factors include smoking, high tea intake and long term use of analgesic like phenacetin.^[4] Although rare, upper urothelial tumors can be associated with renal stone disease and hydronephrosis.

Here we report a case of renal pelvis urothelial carcinoma in situ associated with hydronephrosis and atrophic kidney for its rarity.

Case Report

In this case a 55 year male patient presented with left sided loin pain and 3-4 episodes of intermittent gross hematuria and there were no other significant urinary complains. There was no history of chronic analgesic intake or smoking.

On examination there was no suprapubic fullness, abdominal tenderness or prostatic enlargement. Urine examination and other biochemical parameters were within normal limits. Ultrasound was performed which showed grossly enlarged hydronephrotic left sided kidney with dilated ureter and multiple ureteric calculi (Fig 1). On right side there was large stag horn calculi with only mild hydronephrosis.

Subsequently, DTPA renal scan was done to check kidney function. On left side kidney was enlarged hydronephrotic and practically non-functioning. Right side kidney was normal in size with normal parenchymal function and non-drainage pattern.

Patient first underwent right sided PCNL to remove renal stone to prevent further damage to right side kidney followed by left sided nephroureterectomy after 3 months to remove non functioning left sided kidney.

We received formalin fixed left nephroureterectomy specimen measuring 11x9x5cm. Cortex was thinned out and kidney showed marked hydronephrotic change and atrophy of normal renal parenchyma. Entire kidney was converted into a cyst like structure (Fig. 2). There was attached ureter of 12cm in length without cuff of urinary bladder. Cyst and ureter both were filled with brownish material and pelvis mucosa was erythematous.

Microscopic examination revealed reduced normal renal parenchyma with dense lymphomononuclear infiltrate, few sclerosed glomeruli, atrophic tubules and dilated papillary system which was lined by markedly dysplastic urothelial lining (Fig 3a). Cells showing loss of polarity, high nuclear cytoplasmic ratio, nuclear hyperchromasia, pleomorphism involving whole thickness of urothelium (Fig. 3b,3c,3d). Multiple section were examined and none of them showed invasive foci. Lumen was filled with hemorrhagic and necrotic material and ureteric stump was free of tumor.

Because marked reactive changes can also mimic dysplastic cells, we performed immunohistochemistry Ki67 and p53 to check cell proliferation and neoplastic change. Ki67 and p53 are considered the progression marker of tumor and are useful to rule out reactive changes³. According to studies Ki67 and p53 are expressed in <5% of only basal layer of cells. Expression in whole thickness and in >10% of basal cells usually confirm high grade malignancy or carcinoma in situ⁵. In our case both Ki67 and p53 showed increased expression beyond basal layer cells (Fig.4a,4b).

Based on these morphological and immunohistochemistry features a diagnosis of urothelial carcinoma in situ with hydronephrosis and atrophic kidney was given. Because finding of carcinoma in situ was incidental and no bladder cuff was removed along with ureter we advised close follow up of patient.

Discussion

Very few case report has been described having urothelial malignancy in non-functioning with nephrolithic kidney. Wani et al.^[1] described a similar case report of Rare upper urothelial malignancy in non-functioning nephrolithic kidney.

Frequency of neoplastic lesion in renal pelvis and ureter are approximately similar, but only 1/10th as common as bladder counterpart.^[6,7] Ninety-five percent of neoplasm are of epithelial origin and in that around 80% are malignant.

Common benign epithelial lesion include urothelial papilloma and inverted papilloma; while common malignant epithelial lesions are urothelial carcinoma, squamous carcinoma and adenocarcinoma. Urothelial carcinoma account for 90% of these cases.^[8]

Urothelial neoplasm have sequence of dysplasia, carcinoma insitu and invasive malignancy.^[2] Carcinoma insitu is the neoplastic change of the epithelium without breach of basement membrane. It is considered a high grade neoplasm and require specific treatment.^[3]

It is seen that mucosa adjacent to invasive pelvic and ureteral tumors is dysplastic in 95% of specimen which also

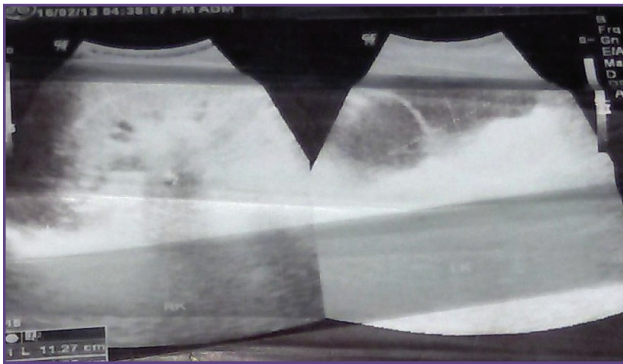


Fig. 1: Ultrasound showing left kidney hydronephrosis.

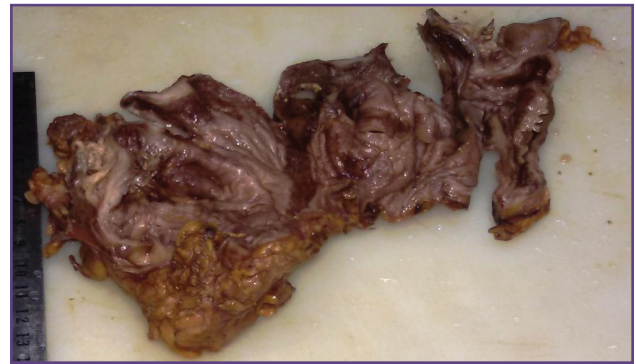


Fig. 2: Gross specimen showing markedly dilated pelvicalyseal system and thinned out cortex.

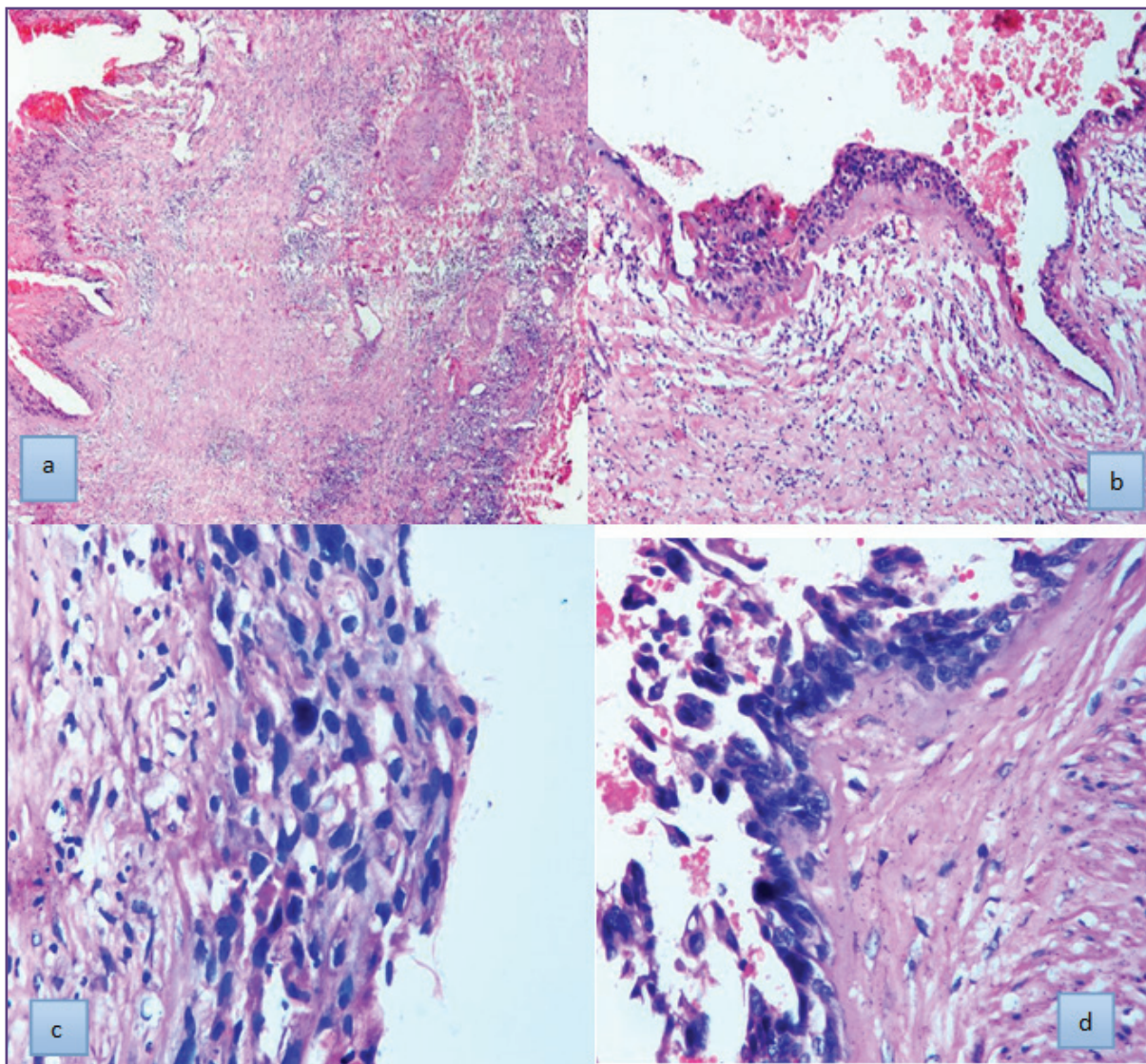


Fig. 3: Low power view showing atrophic kidney with sclerosed glomeruli, thick walled blood vessels and dysplastic urothelium. (40X)(a). Figure showing full thickness severely dysplastic urothelial cells 100X (b), 400X (c,d)

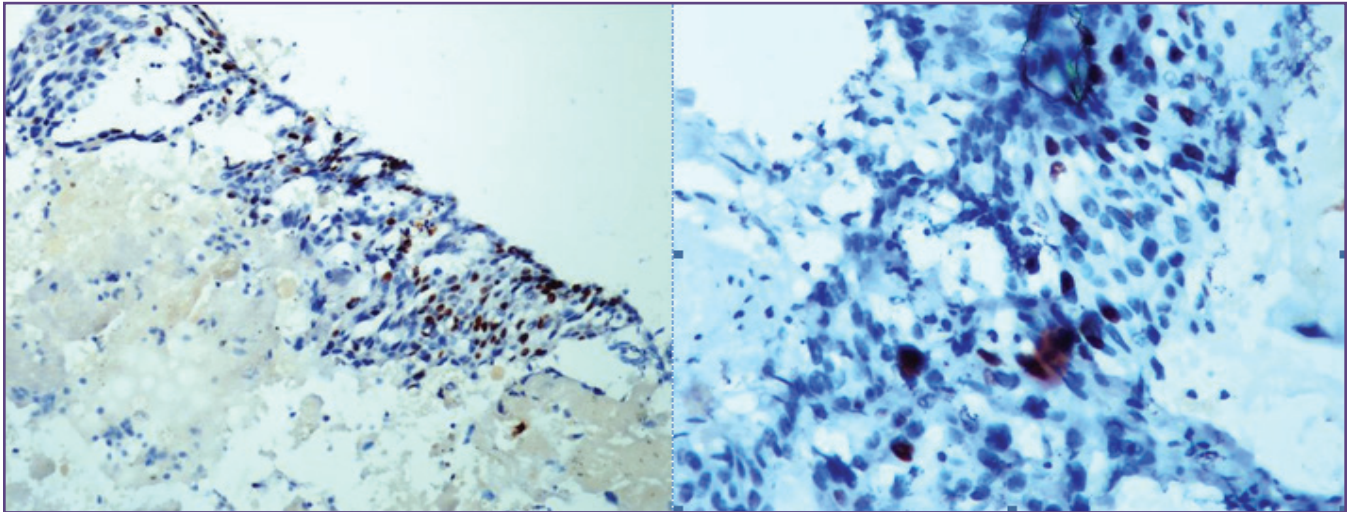


Fig. 4: Immunohistochemistry showing increased expression of Ki67 (200X)(a)and p53 (400X) (b) in superficial layer.

support this progressive change. The severity of dysplasia correlate with the grade of adjacent tumor and identifies patient at risk of metachronous tumours of other sites.^[9-12]

Carcinoma in situ occur more commonly in elderly males.^[13,14] They can present with hematuria or loin pain.^[15] Our patient also had complain of left sided loin pain and 3-4 episodes of intermittent gross hematuria. Risk factors include smoking, high tea intake and long term use of analgesic like phenacetin^[4] There were no such history in this patient. In our case probable cause of neoplastic change was long standing renal pelvic stone with hydronephrosis due to chronic irritation. This association is rare and only few cases are reported. Upper urinary tract tumors associated with renal stone and hydronephrosis are often missed in urine cytological examination because degenerative changes in cells produced by chronic irritation of epithelium. We have performed urine cytology which did not show any abnormalities. As patient underwent nephroureterectomy on left side to remove non functional kidney, there was no suspicion of neoplastic pathology and bladder cuff was not removed. Only after microscopic examination we found sever dysplastic changes of renal pelvis epithelium amounting to carcinoma insitu. Therefore it is important to carefully examine the urine sample, gross specimen and microscopy, keeping in mind the possibility of upper urinary tract tumors in such cases, so that better treatment and follow up can be provided to these patients. Prognosis is usually excellent if patient with carcinoma insitu is treated with radical nephroureterectomy.

The gold standard of treatment for patients with upper urinary tract urothelial neoplasm and normal contralateral kidney is complete nephroureterectomy with removal of cuff of urinary bladder. It is important to remove the

cuff of urinary bladder due to high rate of ureteral stump recurrence, which has been reported to be between 30-75%.^[16] In our case bladder cuff was not removed because carcinoma in situ was detected incidentally after surgery. Therefore close follow up of patient was advised.

Conclusion

Although rare renal pelvic stone with hydronephrosis and atrophic kidney can be associated with neoplastic transformation of epithelium, therefore it is important to keep in mind the possibility of such association with proper preoperative investigation especially urine cytology and CT/MRI scan, so that appropriate treatment can be provided to patient.

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Competing Interests

None Declared

Reference

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