

Coexistence of Primary Renal Squamous Cell Carcinoma and Tuberculosis in a Kidney with Nephrolithiasis: A Case Report and Review of the Literature

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ABSTRACT

Primary squamous cell carcinoma (SCC) of the renal pelvis is a rare but recognised disease, representing 0.5–0.8% of all malignant renal tumours. Concomitant tuberculosis (TB) and SCC of the kidney have never been reported. We present a case of a 55-year male patient presenting with flank pain and dysuria. The patient was diagnosed with a non-functioning right kidney following preliminary baseline investigations, which included a CT scan of the kidney, ureter, and bladder (KUB), as well as a dimercaptosuccinic acid (DMSA) scan. He subsequently underwent a right open nephrectomy. Histopathological examination revealed the presence of both primary SCC and TB within the same kidney. Anti-tuberculosis treatment (ATT) was initiated four weeks post-surgery. However, after eight weeks, the patient returned to the clinic with a fungating lesion at the nephrectomy scar site, and a biopsy confirmed moderately differentiated SCC. Despite eight weeks of intensive ATT, the patient's poor performance status prevented the administration of adjuvant chemotherapy. Unfortunately, the patient passed away two months after the commencement of ATT. SCC of the kidney carries a poor prognosis, and the presence of concurrent TB makes it even more challenging.

Key Words: Nephrolithiasis, Squamous cell carcinoma, Renal tuberculosis.

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INTRODUCTION

A primary squamous cell carcinoma (SCC) of the renal pelvis is a strikingly uncommon yet significant disease, accounting for 0.5–0.8% of all malignant renal neoplasms.^{1,2} Due to its atypical presentation and elusive imaging features, it is often diagnosed incidentally on histopathology. It is commonly associated with chronic irritation secondary to nephrolithiasis. Concomitant tuberculosis (TB) and SCC of the kidney are rare findings. However, the co-existence of TB with SCC of the bladder has been reported before.³ To the best of our knowledge, concurrent primary SCC and TB in the same kidney have never been reported in the literature before. We report a case of a 55-year male patient who underwent right open nephrectomy with an impression of xanthogranulomatous pyelonephritis (XGP) with nephrolithiasis and was found to have primary SCC and TB of the kidney.

CASE REPORT

A 55-year male with no known comorbidities presented to the clinic in July 2023 with right flank pain and burning micturition that had persisted for four weeks. He reported no fever, haematuria, or lower urinary tract symptoms. His medical history included an open pyelolithotomy for a left renal stone in 2011. There was no family history of renal stones or malignancy, and he did not smoke or consume alcohol.

Clinical examination revealed mild tenderness in the palpable right kidney and a scar in the left flank area consistent with his previous surgery. The rest of the abdominal examination was unremarkable. Urine analysis was reported as having 2 + leucocytes and 1 + protein. No bacterial growth was obtained on the urine culture. His complete blood count revealed haemoglobin (Hb) of 9.1 g/dL and white cell count (WCC) of $5.6 \times 10^9/L$. Renal functions were normal, and his C-reactive protein (CRP) was high at 94 mg/L (normal: <10 mg/L).

Ultrasound of the kidney, ureter, and bladder (KUB) revealed an echogenic right kidney measuring 14.2 cm, with gross hydronephrosis and hydroureter containing echoes (Figure 1). A 2.1 cm calculus was observed in the lower calyx along with a localised peri-nephric collection of approximately 257 ml. The left kidney measured 12.6 cm with mild residual fullness and a few calculi in the lower pole, the largest one measuring 0.9 cm. A CT KUB scan confirmed gross hydronephrosis in the right

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kidney due to a stone at the pelvic-ureteric junction and a lower pole sub-capsular collection with echoes. The left kidney showed a tiny stone at the midpole, residual fullness, and delayed contrast excretion. A radionuclide dimercapto-succinic acid (DMSA) scan indicated a non-functioning right kidney and a normally functioning left kidney with a clinical diagnosis of XGP. Given the non-functioning, obstructed right kidney with urolithiasis, the patient underwent a right nephrectomy.



Figure 1: Coronal plane of CT showing gross hydronephrosis of right kidney secondary to pelviureteric junction stone (white arrow) and subcapsular collection at the lower pole.

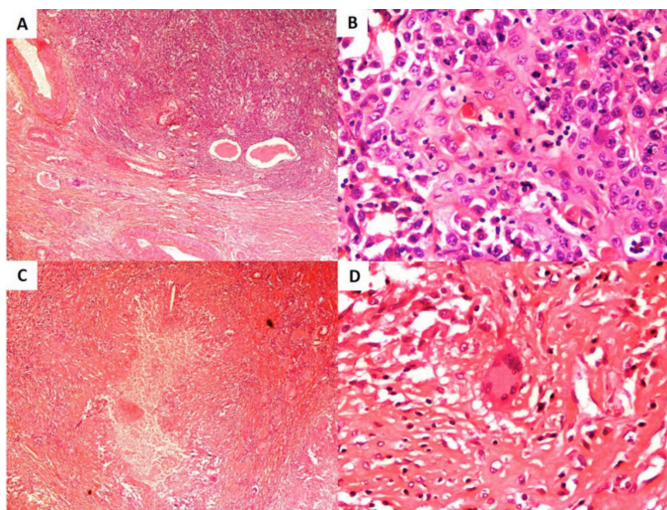


Figure 2: The histopathological features of the nephrectomy specimen. (A): Low-power photomicrograph showing fibrotic kidney parenchyma in the lower part and a malignant tumour in the upper part of the field (H and E, x40). (B): High-power photomicrograph showing a high-grade tumour with areas of squamous differentiation (H and E, x400). (C): Low-power view showing a large epithelioid granuloma with necrotic centre (H and E, x40). (D): High-power photomicrograph showing part of one epithelioid granuloma with a Langhan's type giant cell (H and E, x400).



Figure 3: Axial plane of the contrast-enhanced CT scan showing multiple enhancing nodules in the right renal bed (white arrow) and in the abdominal wall along the scar of previous surgery.

Peroperatively, the kidney was found distorted and densely adherent to the peritoneum and the posterior abdominal wall. During dissection, there was a gross spillage of pus, and the peritoneal cavity was also breached. The ureter, single renal artery, and vein were ligated and cut. After taking out the specimen, the renal bed was washed with hydrogen peroxide, and a 16 Fr drain was placed. In the postoperative period, the patient was kept in a high-density unit for 48 hours and then shifted to the ward. The postoperative period went uneventful and he was discharged on the 5th postoperative day.

After two weeks, the histopathological analysis was finalised. It revealed moderately differentiated SCC of the pelvicalyceal system infiltrating into renal parenchyma (Figure 2 A, B) with a pathological stage of pT3, N0, and Mx. There was also chronic caseating granulomatous inflammation, consistent with TB in kidney parenchyma and ureter (Figure 2 C, D).

On the follow-up visit at four weeks, anti-tuberculosis treatment (ATT) was initiated first, followed by a repeat CT chest, abdomen, and pelvis with contrast, advised for re-staging purposes and to look for any residual disease. There was interval development of multiple enhancing nodules in the right renal bed and the abdominal wall along the scar. Multiple para-aortic lymph nodes were also noted (Figure 3). Overall findings were suggestive of the progression of the local disease process. No distant metastasis was seen.

After nine weeks of surgery, the patient revisited the clinic with a fungating lesion over the nephrectomy scar mark. An excisional biopsy was performed and histopathological analysis showed an infiltrating neoplastic lesion with features of moderately differentiated SCC.

The case was discussed in a tumour board meeting, and adjuvant chemotherapy was planned. However, the poor functional status of the patient prevented the administration of adjuvant chemoradiation. Two months later, the patient

presented to the emergency department with generalised weakness, altered mental status, and respiratory distress, necessitating admission to the intensive care unit. Unfortunately, the patient succumbed to acute respiratory distress syndrome (ARDS).

DISCUSSION

Primary SCC of the kidney is a rare, highly aggressive malignancy characterised by a poor prognosis and elevated mortality, primarily due to its significant metastatic potential. Typically, patients present with flank pain, weight loss, and haematuria, often in association with chronic nephrolithiasis. Carcinogenesis is thought to result from chronic irritation, inflammation, or infection, leading to squamous metaplasia. The clinical presentation of SCC can be diverse, ranging from a grossly hydronephrotic obstructed kidney to XGP, cystic forms, or even cutaneous lesions if diagnosis is delayed.⁴⁻⁷ Imaging often fails to provide precise diagnostic clues, and histopathology is required for confirmation after nephrectomy. In this case, the initial clinical diagnosis was XGP, but histopathology later revealed SCC alongside renal TB.

Because primary renal SCC is so rare, no specific treatment guidelines exist. Radical nephrectomy, followed by adjuvant chemotherapy, remains the cornerstone of treatment. Radical nephrectomy is the preferred treatment for localised SCC that has not metastasised, as partial nephrectomy is associated with higher recurrence rates due to the invasive nature of SCC, which tends to permeate both renal parenchyma and surrounding tissues.

There are only two documented cases of concurrent urothelial cancer and renal TB in the literature,⁸ and primary SCC with renal TB has never been reported before. Some researchers have suggested a possible link between TB and certain cancers, such as colonic cancer,⁹ potentially due to chronic inflammatory injury leading to metaplasia and dysplasia. Patients with lung cancer, head and neck malignancies, breast cancer, lymphoproliferative disorders, leukaemia, stomach cancer, and other neoplasm have all been recorded to have TB.¹⁰

In the present case, the tumour metastasised rapidly to the skin within eight weeks of surgery, likely due to delays in starting adjuvant chemotherapy. The presence of renal TB, as well as the patient's poor functional status, worsened the prognosis. Initially, we initiated ATT, followed by the scheduling of adjuvant cisplatin-based chemotherapy. However, even after eight weeks of intensive ATT, the patient's deteriorating condition prevented the administration of adjuvant chemotherapy. The patient passed away two months after beginning ATT.

This case highlights the diagnostic complexity and therapeutic challenges of managing a patient with renal SCC and renal TB. Primary renal SCC in conjunction with TB has a poor

prognosis, particularly when ATT and adjuvant chemotherapy are delayed. Prompt and aggressive treatment, coupled with strict follow-up, is therefore essential.

PATIENT'S CONSENT:

Informed consent was taken from the patient.

COMPETING INTEREST:

The authors declared no conflict of interest.

AUTHORS' CONTRIBUTION:

NM: Conception of work, literature review, and collection of data.

HB: Literature research and drafting of the manuscript.

NAM: Critical revision of the manuscript for important intellectual content.

ASS: Final approval and agreement to be accountable for all aspects of work.

All authors approved the final version of the manuscript to be published.

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