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Case Report

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Granulomatous interstitial nephritis with advanced chronic kidney disease after intravesical bacillus calmette-guérin therapy: a case report

Abstract

Background: Intravesical Bacillus Calmette-Guérin (BCG) is the standard immunotherapy for high-risk non-muscle-invasive bladder cancer. Although severe renal toxicity is rare (0.1–0.4%), clinically significant chronic kidney disease (CKD) related to BCG-associated granulomatous interstitial nephritis (GIN) is likely under-recognised.

Case Presentation: A female in her 60s with T1 high-grade papillary urothelial carcinoma received six weekly induction BCG instillations. Four months after completing therapy, she presented with oliguria, severe pedal oedema, and dyspnoea. Serum creatinine had risen from 0.9 mg/dL pre-BCG to 5.0 mg/dL at presentation. Renal biopsy showed non-caseating granulomatous interstitial nephritis with multinucleated giant cells, marked interstitial fibrosis (>40% cortical area), and tubular atrophy, on a background of bilaterally small, echogenic kidneys on ultrasonography, suggesting a component of chronicity. Autoimmune, vasculitic, and mycobacterial investigations were negative. BCG was stopped and the patient was managed with two hemodialysis sessions, high-dose prednisolone (40 mg/day tapered over 12 weeks), and a finite course of anti-tubercular therapy initiated empirically after multidisciplinary discussion. Renal function partially improved but stabilised at CKD Stage 4 (eGFR ~25 mL/min/1.73 m²) without dialysis dependence. Subsequently, she developed CKD-related cardiovascular abnormalities, including isolated systolic hypertension and complex arrhythmias on 24-hour Holter monitoring, consistent with CKD-associated cardiovascular dysfunction in keeping with cardiorenal interactions.

Conclusion: This case illustrates presumed BCG-associated GIN with superimposed chronic tubulointerstitial damage, presenting late with advanced CKD and CKD-related cardiovascular sequelae. It underscores the importance of structured renal surveillance during and after intravesical BCG, cautious causal interpretation when chronic changes are present, and early nephrology referral for unexplained creatinine elevation. Surveillance suggestions derived from this case and existing literature should be viewed as pragmatic guidance rather than formal standards of care.

Keywords: intravesical BCG, granulomatous interstitial nephritis, chronic kidney disease, cardiorenal interactions, bladder cancer, immunotherapy complications, case report

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Introduction

Intravesical Bacillus Calmette-Guérin (BCG), a live-attenuated strain of Mycobacterium bovis, is an established adjuvant immunotherapy for high-risk non-muscle-invasive bladder cancer since its introduction by Morales et al. in 1976.[1] Its antitumour efficacy is mediated by localised pro-inflammatory cytokine activation involving both innate and adaptive immune pathways.[2,3] While irritative lower urinary tract symptoms occur in 27–90% of patients,[4] severe renal toxicity is uncommon (0.1–0.4%),[4–6] most often manifesting as acute, potentially reversible granulomatous interstitial nephritis (GIN).[7] Reports describing progression to advanced or irreversible CKD are limited and often heterogeneous, suggesting this complication may be under-recognised.[6]

We describe a case of presumed BCG-associated GIN in a woman with high-risk non-muscle-invasive bladder cancer who presented four months after completing induction therapy with advanced kidney dysfunction on a background of bilaterally small echogenic kidneys and extensive interstitial fibrosis. The case highlights the challenges of causal attribution when chronic changes are present, the potential for CKD-related cardiovascular dysfunction, and the practical importance of structured renal surveillance during and after intravesical BCG.

Case presentation

Written informed consent for publication was obtained from the patient. A female in her 60s, with a background of dyslipidaemia and no documented prior history of diabetes (HbA1c 5.7%), hypertension, CKD, or tuberculosis, presented to the Emergency Department with a 5-day history of progressive oliguria, severe bilateral pitting pedal oedema extending to the thighs, and dyspnoea at rest. She was a lifelong non-smoker.

She had been diagnosed six months earlier with high-grade papillary urothelial carcinoma (T1 high-grade; lamina propria invasion confirmed; detrusor muscle free of tumour) and treated with transurethral resection of bladder tumour (TURBT), followed by six weekly induction cycles of intravesical BCG (strain not documented). Pre-treatment serum creatinine was 0.9 mg/dL (eGFR >60 mL/min/1.73 m²). No interim renal monitoring (serum creatinine or urinalysis) was performed during the four-month interval between BCG completion and the acute presentation.

On admission, vital signs were: blood pressure 170/80 mm Hg (wide pulse pressure 90 mm Hg), pulse 100 beats/min, respiratory rate 20 breaths/min, temperature 99.6°F (37.6°C), SpO_2 94% on room

air. Cardiovascular examination revealed an S3 gallop with bilateral basal crepitations on auscultation. During hospitalisation, serum creatinine rose from 3.0 mg/dL on admission to a peak of 5.0 mg/dL over four days (3.0 → 3.5 → 4.0 → 4.5 → 5.0 mg/dL, Days 0–4). The key clinical milestones are summarised in Table 1 to complement the narrative description.

Table 1 Chronological clinical timeline from baseline to ongoing follow-up.

Time point	Clinical event
Baseline (pre-BCG)	Creatinine 0.9 mg/dL; eGFR >60 mL/min/1.73 m²
Month 0	TURBT; T1 high-grade urothelial carcinoma diagnosed
Month 1–2	Six weekly intravesical BCG induction instillations completed
Month 4	Acute presentation: oliguria, oedema, dyspnoea
Day 0 (admission)	Creatinine 3.0 mg/dL
Days 1–4	Creatinine 3.5 → 4.0 → 4.5 → 5.0 mg/dL
Day 2	Renal ultrasonography: bilaterally small echogenic kidneys; serology panel negative
Day 3	Chest radiograph: pulmonary oedema; echocardiography: LVEF 72%, Grade I DD, mild LVH
Day 4	ABG: pH 7.20, $PaCO_2$ 48 mm Hg, HCO_3^- 16 mEq/L; peak creatinine 5.0 mg/dL
Day 5	Renal biopsy; first hemodialysis session
Day 6	Biopsy: GIN confirmed; BCG stopped; prednisolone and ATT initiated
Day 7	Second hemodialysis; creatinine 4.5 mg/dL
Day 10–12	Discharge; creatinine 3.5 mg/dL; eGFR 15–20 mL/min/1.73 m²
Week 2–12	Outpatient steroid taper; ATT completed
Month 6	Creatinine 3.2 mg/dL; eGFR ~25 mL/min/1.73 m² (CKD Stage 4); CKD-related cardiovascular abnormalities noted
Ongoing	Conservative CKD management; monthly nephrology follow-up; no permanent dialysis

Investigations

Arterial blood gas (room air) showed pH 7.20, $PaCO_2$ 48 mm Hg, PaO_2 100 mm Hg, and HCO_3^- 16 mEq/L, consistent with severe metabolic acidosis with concurrent CO_2 retention. Serum electrolytes were: Na^+ 145 mEq/L, K^+ 4.4 mEq/L. Serum albumin was 2.8 g/dL. Complete blood count showed haemoglobin 10.0 g/dL, total leukocyte count 18,000 cells/µL, and platelets 300,000 cells/µL. Urinalysis revealed 2+ proteinuria and 10–12 RBCs/HPF with sterile culture.

An immunological panel (ANCA-C, ANCA-P, anti-dsDNA, ANA) was negative, making active vasculitis or lupus nephritis unlikely. Mycobacterial workup (Mantoux, interferon-gamma release assay, sputum CBNAAT/GeneXpert) was also negative. Key laboratory parameters at peak illness are summarised in Table 2.

Table 2 Key laboratory parameters at peak admission (Day 4, pre-dialysis).

Parameter	Patient value	Reference range
Serum creatinine	5.0 mg/dL	0.6–1.2 mg/dL
eGFR (CKD-EPI)	<15 mL/min/1.73 m²	>60 mL/min/1.73 m²
Serum sodium	145 mEq/L	135–145 mEq/L
Serum potassium	4.4 mEq/L	3.5–5.0 mEq/L
Serum albumin	2.8 g/dL	3.5–5.0 g/dL
Haemoglobin	10.0 g/dL	12.0–16.0 g/dL
Total leukocyte count	18,000 cells/µL	4,000–11,000 cells/µL
Platelet count	300,000 cells/µL	150,000–450,000 cells/µL
HbA1c	5.7%	<5.7%
pH (ABG)	7.20	7.35–7.45
$PaCO_2$	48 mm Hg	35–45 mm Hg
HCO_3^-	16 mEq/L	22–26 mEq/L
ANCA-C / ANCA-P / ANA	Negative	Negative
Anti-dsDNA	Negative	Negative
Mantoux / IGRA / CBNAAT	Negative	Negative

Renal ultrasonography demonstrated bilaterally small kidneys (right 7.8 cm, left 7.5 cm) with increased echogenicity and loss of corticomedullary differentiation, indicating chronic structural change. Chest radiograph showed bilateral pulmonary consolidations consistent with pulmonary oedema, without cavitary or miliary lesions. Transthoracic echocardiography revealed a left ventricular ejection fraction of 72%, mild concentric left ventricular hypertrophy, Grade I diastolic dysfunction, no significant valvular pathology, and a dilated inferior vena cava with reduced respiratory variation.

Percutaneous ultrasound-guided renal biopsy (Day 5) confirmed granulomatous interstitial nephritis characterised by non-caseating epithelioid granulomas with multinucleated Langhans-type giant cells, extensive interstitial fibrosis (>40% cortical area), tubular atrophy, and a mononuclear interstitial infiltrate as in Figure 1. Immunofluorescence was negative, arguing against immune-complex glomerulonephritis. Ziehl–Neelsen staining demonstrated no acid-fast bacilli, and tissue GeneXpert MTB/RIF (CBNAAT) did not detect Mycobacterium tuberculosis complex. The combination of non-caseating granulomas without demonstrable organisms, negative systemic mycobacterial workup, and temporal association with BCG therapy supported a diagnosis of presumed BCG-associated hypersensitivity-mediated GIN on a background of chronic tubulointerstitial damage.

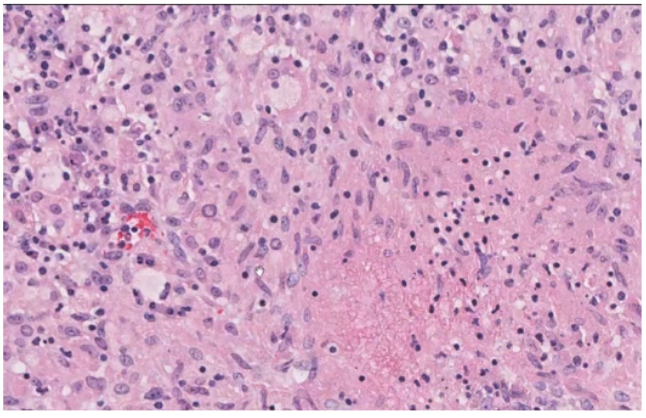

Figure 1 Renal biopsy showing non-caseating granulomatous interstitial nephritis with epithelioid histiocytes and multinucleated giant cells in the interstitium, associated with dense inflammatory infiltrate (H&E stain, high-power view).

To visually complement the tabulated values, the trajectory of serum creatinine from baseline through peak illness and subsequent partial recovery is depicted in Figure 2, highlighting the unmonitored interval between BCG completion and presentation.

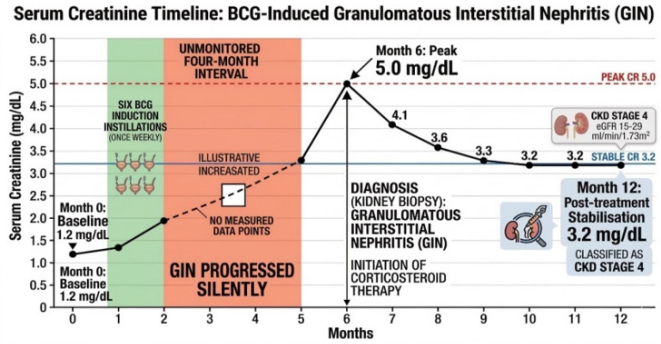

Figure 2 Timeline of serum creatinine (mg/dl) from baseline through peak (5.0 mg/dl) and post-treatment stabilisation (3.2 mg/dl; CKD stage 4). Six BCG induction instillations are indicated at months 1–2. The unmonitored four-month interval (months 2–4) during which GIN progressed silently is highlighted.

Therapeutic interventions

Intravesical BCG therapy was permanently discontinued following the diagnosis of GIN. Two emergency hemodialysis sessions (4 hours each on Days 5 and 7) via a temporary internal jugular catheter achieved a net ultrafiltration of approximately 3.5 L, corrected metabolic acidosis, and improved respiratory status.

High-dose corticosteroid therapy was commenced after histological confirmation of GIN, with oral prednisolone 40 mg/day initiated on Day 6 and tapered over 12 weeks (40 → 30 → 20 → 10 → 5 mg/day in approximately fortnightly steps). This regimen was selected in line with published experience suggesting benefit of corticosteroids in severe interstitial nephritis, particularly where significant inflammatory activity coexists with fibrosis.[6,8,9,10]

Pyridoxine 25 mg/day was co-administered.

Despite the absence of microbiological confirmation of active mycobacterial infection, a finite course of anti-tubercular therapy (ATT) was started after multidisciplinary discussion with nephrology, urology, and infectious disease specialists. The rationale was to provide coverage for possible occult BCG-related infection in the context of granulomatous lesions, while recognising the limitations of available diagnostics. The regimen comprised Rcinex (Rifampicin 600 mg + Isoniazid 300 mg once daily) and Ethambutol, dose-adjusted for CKD, for 12 weeks; pyrazinamide was omitted because of intrinsic Mycobacterium bovis resistance.[6,7]

Antihypertensive therapy was streamlined to focus on blood pressure control and renoprotection without overemphasis on specific agents. The patient was managed with a calcium channel blocker (Cilnidipine) and an alpha-blocker (Prazosin), titrated according to blood pressure, with dose de-escalation as haemodynamic stability improved. Additional supportive measures included dietary protein restriction, loop diuretics, and anaemia management as per CKD guidelines.

Outcome and follow-up

Following two hemodialysis sessions, serum creatinine decreased from a peak of 5.0 mg/dL to 4.5 mg/dL (Day 7) and 3.5 mg/dL at discharge (Day 10–12), with eGFR improving to approximately 15–20 mL/min/1.73 m². Renal function did not return to the pre-BCG baseline. At six-month follow-up, creatinine stabilised at 3.2 mg/dL (eGFR ~25 mL/min/1.73 m²), consistent with CKD Stage 4, without ongoing dialysis requirements. Follow-up cystoscopy showed no tumour recurrence.

Over time, the patient developed features of CKD-related cardiovascular involvement. Ambulatory blood pressure monitoring demonstrated persistent isolated systolic hypertension (around 170/80 mm Hg, pulse pressure ~90 mm Hg), in keeping with arterial stiffening frequently observed in CKD.[11,12] Twenty-four-hour Holter monitoring revealed episodes of sinus tachycardia and bradycardia, frequent premature ventricular contractions, and premature atrial contractions, consistent with electrophysiological instability in the context of advanced CKD and structural cardiac changes.[11,12,14] These findings were interpreted as CKD-associated cardiovascular dysfunction within the spectrum of chronic cardiorenal interactions rather than as proof of a distinct cardiorenal syndrome subtype in isolation.

Figure 2 illustrates the temporal evolution of serum creatinine from pre-BCG baseline through peak acute injury and subsequent partial recovery, together with the timing of BCG instillations and major clinical interventions.

Discussion

This case contributes to the evolving spectrum of renal complications associated with intravesical BCG. BCG-related GIN typically presents during or shortly after intravesical therapy as an acute tubulointerstitial nephritis that may be reversible with prompt recognition and treatment.[4–7,15,9,17–19]] In contrast, the present case manifested four months after completion of induction therapy, with advanced kidney dysfunction, bilaterally small echogenic kidneys, and extensive interstitial fibrosis and tubular atrophy on biopsy. These features indicate that while BCG exposure likely acted as an important trigger for granulomatous inflammation, a degree of chronic tubulointerstitial damage may have preceded the acute presentation.

Causality must therefore be interpreted cautiously. The temporal relationship to BCG, compatible renal histology, exclusion of alternative systemic causes, and concordance with published reports support a diagnosis of presumed BCG-associated GIN.[6,7,15] However, the chronic radiological and histological changes suggest either previously unrecognised CKD or a prolonged subclinical phase of tubulointerstitial injury that evolved during the unmonitored interval after BCG completion. Acknowledging this uncertainty strengthens the clinical interpretation and avoids overstating the extent to which BCG exposure alone explains the irreversible CKD.

The differential diagnosis of granulomatous interstitial nephritis is broad and includes drug-induced tubulointerstitial nephritis (e.g., non-steroidal anti-inflammatory drugs, antibiotics, proton pump inhibitors), sarcoidosis, systemic infections (mycobacterial, fungal), autoimmune diseases, and idiopathic forms.[9,10,19] In this patient, there was no history of prolonged NSAID use, recent high-risk antibiotic exposure, or proton pump inhibitor overuse. There were no clinical, radiological, or laboratory features suggestive of sarcoidosis, systemic vasculitis, or connective tissue disease. Mycobacterial investigations were repeatedly negative, and there was no evidence of disseminated infection. Against this background, the temporal association with intravesical BCG and the biopsy pattern favoured a hypersensitivity-type BCG-associated GIN superimposed on chronic scarring.

Mohammed and Arastu (2017) described an "emerging spectrum" of renal injury following intravesical BCG, including both acute and chronic presentations, and proposed that repeated immune-mediated insults may lead to cumulative nephron loss and progressive CKD in susceptible individuals.[6] In the present case, the absence of interval

renal surveillance may have coincided with a period during which a potentially reversible inflammatory phase evolved into established fibrosis (>40% of cortical area), although this sequence cannot be demonstrated directly from a single case, aligning with data that delayed recognition is associated with incomplete recovery.[6,8] Studies of interstitial nephritis more broadly suggest that the extent of fibrosis and tubular atrophy at diagnosis is a key determinant of long-term renal outcome.[8–10]

The cardiovascular findings in this patient illustrate the close interplay between CKD and cardiac structure and function. Chronic kidney disease is strongly associated with left ventricular hypertrophy, diastolic dysfunction, arterial stiffness, and increased arrhythmia risk, all of which contribute to the high burden of cardiovascular mortality in CKD populations.[8–10,15] In keeping with this, our patient developed isolated systolic hypertension with wide pulse pressure, likely reflecting arterial stiffness, and complex arrhythmias on Holter monitoring. These features are consistent with CKD-related cardiovascular dysfunction within the spectrum of chronic cardiorenal interactions and support heightened vigilance for cardiovascular complications in patients with advanced CKD after BCG-associated nephrotoxicity.

Regarding treatment, published experience supports a combination of BCG cessation, corticosteroids, and, in selected cases, anti-tubercular therapy for severe BCG-associated GIN.[6,7,15,17–20] In this case, high-dose prednisolone was initiated promptly after biopsy confirmation, targeting the inflammatory component, while ATT was added empirically following multidisciplinary discussion in light of granulomatous histology and potential diagnostic limitations, with the regimen tailored to Mycobacterium bovis susceptibility patterns. The partial renal recovery observed, with stabilisation at CKD Stage 4, is concordant with reports that a substantial proportion of patients with delayed presentation or advanced fibrosis achieve only partial improvement despite appropriate therapy.[6,8]

From a practical standpoint, this case reinforces three key clinical messages. First, intravesical BCG can be followed by delayed-onset renal dysfunction, which may progress insidiously to advanced CKD without prominent urinary symptoms. Second, structured renal surveillance—such as serum creatinine and urinalysis before each instillation and at predetermined intervals (for example, 1, 3, 6, and 12 months) after completion of BCG—appears reasonable as a pragmatic approach informed by this case and the existing literature, although it is not yet codified as a formal standard of care.[6,7,18] Third, early nephrology referral for any unexplained rise in creatinine, new-onset proteinuria, or suggestive urinary abnormalities during or after BCG therapy may permit earlier intervention and limit permanent nephron loss.

Patient perspective

"I underwent bladder treatment hoping to cure my cancer, but never imagined my kidneys would be so severely affected. I hope my experience helps doctors recognise this complication earlier in other patients." The patient remains compliant with medications and monthly follow-up and is relieved to have avoided permanent dialysis so far.

Limitation(s) of the study

This report describes a single-patient experience from a single centre, which limits the generalisability of the observations. The specific Bacillus Calmette-Guérin (BCG) strain used for intravesical therapy was not documented, and vesicoureteric reflux was not formally assessed, precluding strain-specific or anatomical risk stratification. Interval renal function monitoring was not performed between BCG completion and acute presentation, restricting precise characterisation of the temporal evolution of renal injury. Although histology demonstrated extensive fibrosis and tubular atrophy, the exact duration of disease cannot be reliably inferred from biopsy appearances alone. Tissue culture for Mycobacterium bovis was not available, and only molecular testing for Mycobacterium tuberculosis complex was performed, which may have limited microbiological characterisation of the lesion.

Conclusion

This case illustrates presumed BCG-associated granulomatous interstitial nephritis occurring in the context of chronic tubulointerstitial damage and presenting late with advanced CKD and CKD-related cardiovascular sequelae. The chronic radiological and histological changes highlight the difficulty of attributing irreversible CKD solely to BCG exposure, yet the temporal relationship and compatible biopsy findings underline BCG as an important contributory factor. Clinicians should maintain a high index of suspicion for renal complications during and after intravesical BCG, ensure systematic renal surveillance, and seek early nephrology input for unexplained renal impairment.

Clinical significance

Intravesical BCG, while highly effective for high-risk non-muscle-invasive bladder cancer, can be complicated by granulomatous interstitial nephritis that may not always be acutely reversible. Delayed recognition in the absence of structured surveillance may permit progression to advanced CKD with attendant cardiovascular complications. Incorporating routine serum creatinine and urinalysis monitoring during and after BCG therapy, explicitly discussing potential renal risks during pre-treatment counselling, and documenting these risks in informed consent forms are pragmatic steps that may help identify nephrotoxicity earlier and mitigate long-term renal and cardiovascular consequences.

Ethical approval

This case report was conducted in accordance with the ethical standards of the institutional research committee and the Declaration of Helsinki. Formal ethical approval was obtained from the Institutional Ethics Committee. Written informed consent was obtained from the patient for publication of this case report and any accompanying images or clinical data. A copy of the written consent is available for review by the Editor-in-Chief of the journal upon request.

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Author's contribution

Dr. Girin Ray: Conceptualisation of the case report, clinical management of the patient, drafting of the initial manuscript.

Dr. Chandrapaul Gupta: Histopathological evaluation and interpretation, preparation of pathology figures, critical revision of the manuscript for important intellectual content.

Dr. Satyaki Basu: Literature review, data curation, drafting and editing of the discussion and tables.

All authors contributed to manuscript revision, read, and approved the final version of the manuscript. No other person fulfils the criteria for authorship.

Conflicts of interest

The authors declare no conflicts of interest related to this case report.

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