Case - Bladder malakoplakia in a kidney transplant recipient

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INTRODUCTION

Malakoplakia is a rare inflammatory disorder predominantly affecting immunocompromised individuals. This condition commonly manifests in the genitourinary tract, although cases have been reported in various other systems.[1,2] Mainstays of treatment include reduction in immunosuppression, and administration of intracellular-acting antibiotics. Employing cholinergic agonists can also provide a theoretical clinical benefit.[1,3,4] Surgical intervention remains a viable treatment option if medical management fails.

KEY MESSAGES

- Malakoplakia is a rare condition characterized by a florid histiocytic inflammation.
- This condition most commonly presents in the genitourinary tract and predominantly affects immunocompromised individuals.
- Diagnosis is confirmed histologically by identifying Michaelis-Gutmann bodies, pathognomonic intracellular inclusions resulting from defective macrophage functioning.
- Due to its rarity, there are currently no standardized guidelines for the management and surveillance of this disease.
- Mainstays of treatment are primarily guided by individual case reports and multi-disciplinary consensus, and include reduction in immunosuppression, administration of intracellular-acting antibiotics, and potentially using cholinergic agonists.

In this report, we present a unique case of bladder malakoplakia in a kidney transplant recipient which, despite limited ability to reduce immunosuppression due to progressive graft failure, was successfully treated through a combination of partial surgical resection and targeted antibiotic therapy.

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CASE REPORT

We present the case of a 79-year-old male followed by urology for recurrent Escherichia coli urinary tract infections (UTIs) since 2020, and lower urinary tract symptoms (LUTS) secondary to benign prostatic hyperplasia (BPH). His medical history includes end-stage renal disease thought to be secondary to focal segmental glomerulosclerosis. He initially was on intermittent hemodialysis and subsequently underwent kidney transplantation in 2018. At the time of our evaluation, he was on tacrolimus and prednisone. He had previously been treated with mycophenolate; however, this was discontinued due to leukopenia and recurrent infections.

Renal function was noted to be deteriorating slowly over time for many reasons, including an early episode of short-lived T cell-mediated rejection, and recurrent acute kidney injury usually associated with infection and hypotension. eGFR had been fluctuating between 17 and 22 ml/min since 2021. No donor-specific antibodies were ever identified. The patient is also known for Factor V Leiden deficiency associated with prior deep vein thrombosis and pulmonary embolism for which he takes warfarin. He has a history of recurrent Clostridium difficile infections. Additional conditions include angina, hypertension, dyslipidemia, multiple thyroid nodules and a remote pituitary adenoma.

Investigations

The patient underwent a cystoscopy in 2021 for evaluation for recurrent UTIs and LUTS, both of which showed evidence of benign prostatic obstruction and no bladder lesions. The patient was put on oral alpha-blockers and a 5-alpha-reductase inhibitor, as well as cranberry supplements and probiotics for his urinary tract infections. Urodynamic studies were performed which demonstrated an elevated bladder outlet obstruction index and a borderline bladder contractility index. At the time, the patient's symptoms significantly improved with medical therapy, and they therefore declined to proceed with surgery. In 2024, a cystoscopy was repeated due to a recent increase in frequency of symptomatic UTIs, which revealed multiple new large, solid, suspicious masses distributed throughout the bladder and bladder neck. A computed tomography scan demonstrated that these lesions were localized to the bladder (Figure 1) and showed no evidence of metastatic disease. Urine cytology was negative for malignancy.

The patient underwent a transurethral resection of the bladder tumours (TURBT) to elucidate the origin of the lesions. Photographs of the lesions discovered were taken and are shown in Figure 2. The patient was originally planned for full resection, however due to the extent of disease, the patient's frailty, and the inability to visualize the transplant ureteric orifice intraoperatively, the surgical team opted for a limited resection to avoid complications. Approximately 60% of the lesions were removed and sent for pathological evaluation, after which the need for further surgery would be reassessed. Michaelis-Gutmann bodies, pathognomonic cytoplasmic inclusions,[1] can be appreciated in the H&E-stained specimen found in Figure 3A.

Histopathological examination revealed large sheets of foamy histiocytes confirmed by positive CD68-staining (Figure 3B) and negative CKAE1/AE3-staining (Figure 3C). The

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histiocytic population totally replaced the lamina propria and involved the muscularis propria. Several histiocytes exhibited intracytoplasmic round, well circumscribed inclusions with targetoid appearance, that were positive for Von Kossa staining, consistent with Michaelis-Gutmann bodies (Figure 3D). These inclusions can be visualized in Figure 3A as well. Infectious agents were absent on Gram, Grocott, and Ziehl-Neelsen stains. Similarly, there was no evidence of malignancy. Given the clinical history of kidney transplant on immunosuppression, recurrent E. Coli UTIs, and histologic findings, this clinical presentation was strongly in keeping with malakoplakia of the bladder.

Treatment

After the procedure, the patient was admitted and received 2.25g Piperacillin-tazobactam intravenously for 5 days before being switched to cefixime 200mg daily (dose adjusted for renal clearance). This antibiotic regimen was decided upon as the patient's latest urine culture showed an organism that was resistant to trimethoprim/sulfamethoxazole (TMP/SMX), ciprofloxacin and fosfomycin. After histopathological analysis was obtained, a literature review was performed and a multi-disciplinary discussion with the urology, infectious disease and nephrology teams occurred to optimize the patient's management. The infectious disease consultant recommended continuation of cefixime 200mg orally daily with oral vancomycin to prevent C. difficile colitis. The nephrology transplant team decided against altering the immunosuppressant regimen since the maintenance immunosuppression was already at a minimum. Repeat TURBT was avoided at this time due to the patient's high surgical and anesthetic risk, frailty, and histopathology negative for malignancy.

Outcome and followup

A repeat cystoscopy performed two months after the TURBT showed marked improvement, with approximately 80 percent of the malakoplakia lesions resolved and a corresponding reduction in urinary symptoms, including chronic dysuria. Six weeks later, at three and a half months after the TURBT, a cystoscopy demonstrated further resolution, with about 90 percent of the lesions improved. This indicated sustained effectiveness of the antibiotic regimen despite no reduction in immunosuppression.

At six months after resection, cystoscopy showed complete resolution of all plaques (Figure 4). The patient's symptomatic improvement remained stable, with no urinary complaints, fever, or pain reported two months after the TURBT. The patient underwent regular monthly urine culture surveillance while on antibiotics. Unfortunately, his E. coli strain developed resistance to all oral antimicrobials and given his history of recurrent C. difficile infection, it was decided to stop his oral cefixime and to follow him closely clinically and with follow up cystoscopies.

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DISCUSSION

Standard treatment of malakoplakia involves three key strategies: reducing immunosuppression, administering intracellular-acting antibiotics like fluoroquinolones or TMP/SMX, and employing cholinergic agonists to enhance phagolysosome function via increased intracellular cGMP.[1,3,4] Unfortunately, the patient's E. coli isolate was resistant to both ciprofloxacin and TMP/SMX. Although beta-lactam antibiotics are not traditionally considered intracellular agents, cefixime has demonstrated partial intracellular penetration, which may help explain its clinical efficacy in this case.[5,6] Rifampin, sometimes used adjunctively in malakoplakia for its intracellular activity, was not administered because urine sterility was achieved with cefixime alone and due to its interaction with warfarin.[4,7] Bethanechol, a cholinergic agonist, has shown anecdotal efficacy however needs further exploration.[8] In this particular patient, bethanechol was contraindicated given his underlying history of coronary artery disease. There are reports of these management strategies successfully leading to stable kidney function and resolution of malakoplakia. [9–11]

Our management strategy aligns with existing literature on genitourinary malakoplakia among renal allograft recipients. Faced with our patient's progressive renal function decline, and since our patient was on minimal immunosuppression, the nephrology team opted to maintain the current immunosuppressive regimen to avoid rejection. Administering cefixime 200mg orally daily, follow-up cystoscopies two and four months post-TURBT showed respectively 80% and 90% regression of the malakoplakia lesions. It is also essential to consider the limitations of antibiotic resistance, including resistance to those commonly used for malakoplakia, while also remaining cognizant of the patient's recurrent C. difficile infections. Input from urology, infectious diseases, and nephrology was paramount in guiding our management plan.

CONCLUSIONS

This case contributes to the growing body of evidence that supports varied approaches to managing malakoplakia. Notably, it highlights that alteration of immunosuppressive therapy may not be a requisite component of baseline treatment. When the causal organism is resistant and immunosuppression must be maintained, tailored antibiotic therapy combined with close monitoring may still yield favourable outcomes.

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FIGURES AND TABLES

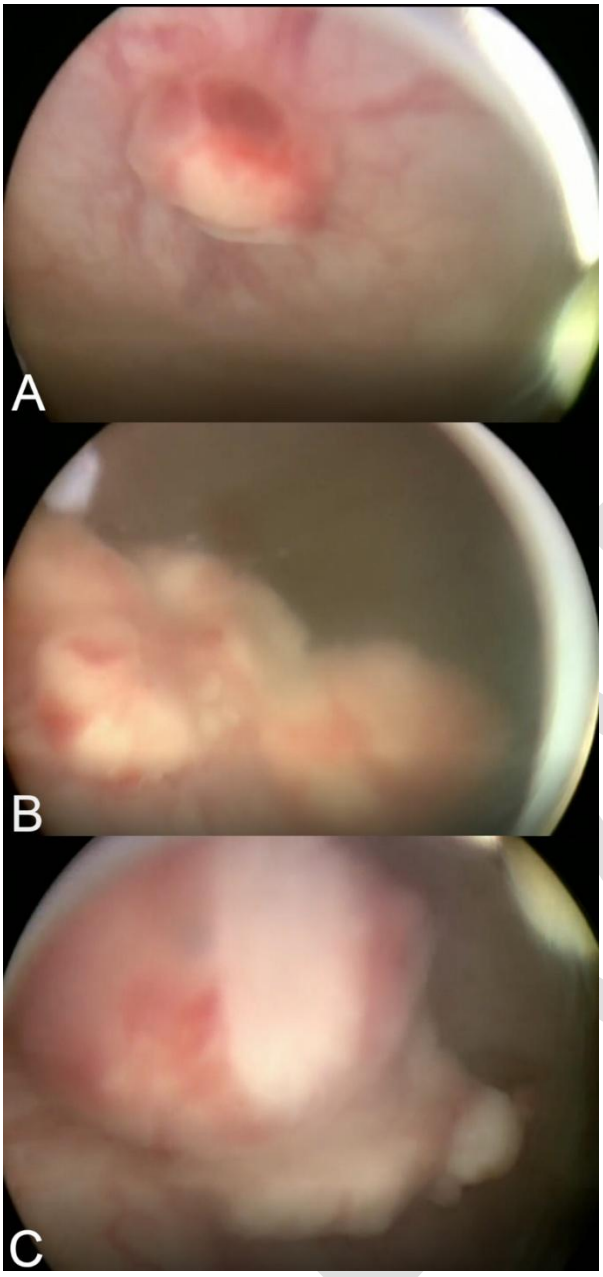
Figure 1. Malakoplakia computed tomography.



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Figure 2. Photographs of the lesions.



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Figure 3. Pathology.

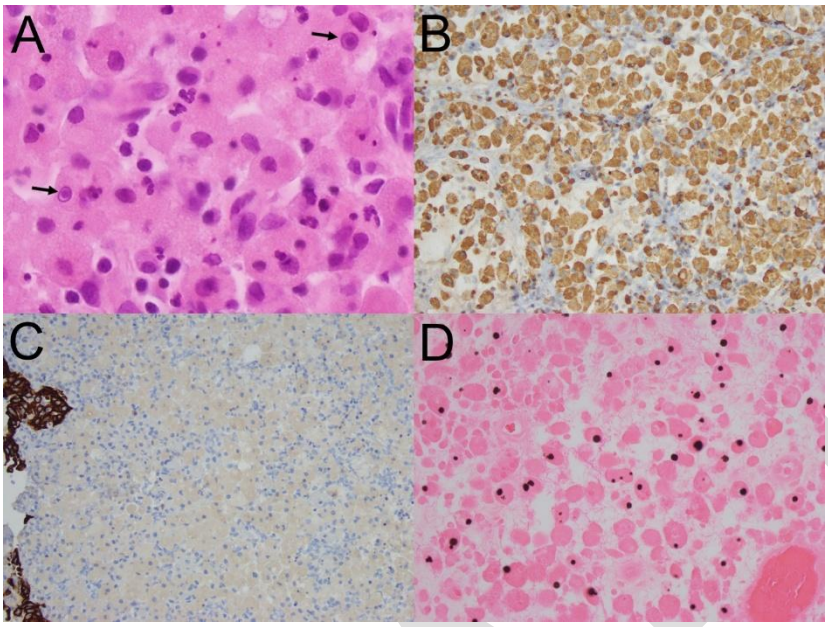
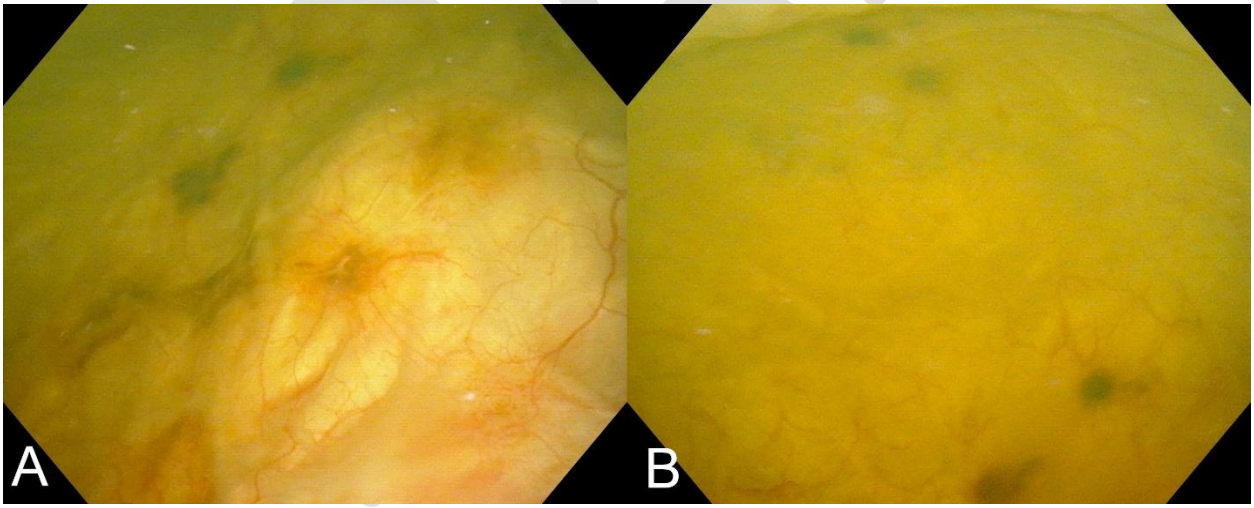


Figure 4. Cystoscopy showing complete resolution of all plaques at 6 months.



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