Case Report

Malakoplakia in a Transplant Kidney: A Rare Case Report

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Abstract

Malakoplakia is a rare inflammatory condition, commonly associated with immunosuppression. It usually affects the urinary bladder. Involvement of renal parenchyma is uncommon. This is a case report of a 49 year old renal transplant recipient with persistent graft dysfunction following an episode of urinary tract infection. Creatinine level was high and urine analysis showed pus cells and E.Coli organisms. Renal allograft biopsy was performed to rule out rejection and interstitial nephritis. The biopsy showed interstitial inflammation with histiocytes showing Michaelis-Gutmann bodies which were highlighted by Perls Prussian blue stain. Hence, the diagnosis of Malakoplakia was made. Treatment with prolonged antibiotics led to significant improvement in the creatinine value and graft function. This is a rare case which involves the renal parenchymal tissue following transplantation and correct diagnosis is required for graft survival and good prognosis.

Keywords: malakoplakia; renal transplant; renal biopsy; michaelis-gutmann bodies

Introduction

Malakoplakia is a chronic inflammatory condition, associated with gram-negative bacterial infection, most commonly E.Coli [1]. It commonly affects urinary bladder, though it can also involve other solid organs. However, isolated involvement of transplant kidney is rare.

We report a case of malakoplakia affecting the allograft kidney. The renal function improved significantly after the diagnosis and a prolonged course of antibiotics.

Case Report

A 49 year old female who underwent a live donor across the blood group renal transplantation one year ago. She had recurrent pyelonephritis. She had persistent renal dysfunction despite antibiotics. Serum albumin was 4 gm/dl. Urine

analysis showed 10-15 pus cells/hpf and urine culture showed E.coli organisms. Due to persistent high creatinine levels with an initial value of 3.2 mg/dl on 06.05.2025, a renal biopsy was performed to rule out rejection and interstitial nephritis.

The tissue was sent in two containers, one in formalin for light microscopy and one in Michel's solution for immunofluorescence microscopy. Multiple serial sections were stained with H&E, PAS, Jones methenamine silver and Masson trichrome stains.

Light Microscopy with special stains: Sections showed a core of renal tissue with 60% cortex and 40% medulla. Of the 8 glomeruli present in the core one showed ischaemic changes. The remaining glomeruli were normocellular with patent capillary loops lined by normal appearing basement membranes. No mesangial widening or capillary basement membrane splitting was seen. Dense diffuse inflammatory infiltrate was present in the cortex and medulla comprising of lymphocytes, neutrophils, histiocytes and few plasma cells (Figure 1). The histiocytes are predominantly present in the medulla and they

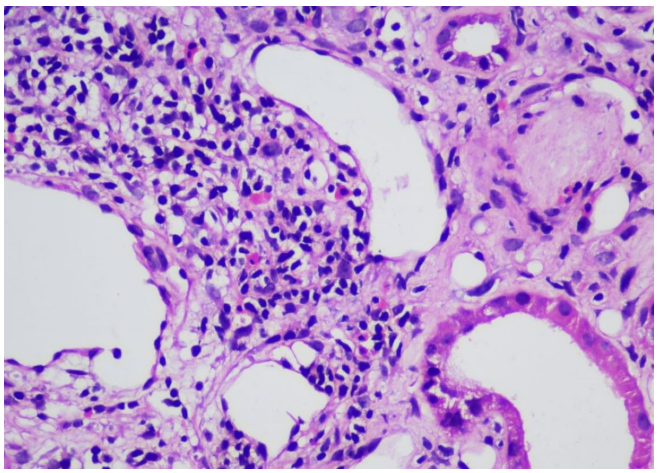

Figure 1: Renal biopsy showing interstitial inflammation composed of lymphocytes, plasma cells and histiocytes. (H&E. 40X).

had abundant granular eosinophilic cytoplasm. Basophilic cytoplasmic inclusions (Michaelis-Gutmann bodies) which are positive for Iron, highlighted by Perl's Prussian Blue stain were present in many of the histiocytes (Figure 2, Figure 3).

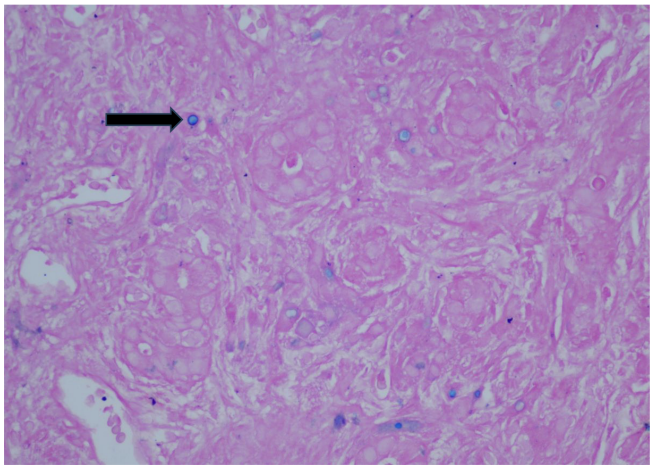

Figure 2: Perl's stain for iron demonstrating Michaelis-Gutmann bodies (arrow). (Perls Prussian Blue stain. 20X).

Severe tubular atrophy and interstitial fibrosis were present involving more than 90% of the cortex. Many cortical tubules showed neutrophilic infiltrate with granular and neutrophilic casts within the cortical tubules. No nucleomegaly or viral inclusion was seen. Interstitial fibrosis appeared storiform at places. Artery was not included in the biopsy and occasional arterioles showed thickened wall. The peritubular capillaries were unremarkable.

Immunofluorescence: Core for immunofluorescence microscopy comprised of cortex with four glomeruli. Diffuse inflammatory infiltrate was present in the interstitial tissue. On a scale of 0-4+, diffuse patchy 2+ granular staining for IgA and 1+ lambda light chain were present in the glomerular mesangium. The glomeruli were negative for fibrinogen, IgG, IgM, C3c, C1q and kappa light chain. The tubular protein casts showed 4+ staining for IgA, kappa and lambda light chains. Diffuse 2+ linear continuous staining for C4d was present along the wall of 70% peritubular capillaries.

A diagnosis of renal parenchymal malakoplakia was made. Appropriate prolonged course of fluoroquinolones was given. The creatinine value after treatment was significantly lowered to 0.9 mg/dl on 16.10.2025, which indicates the improved renal function and graft function.

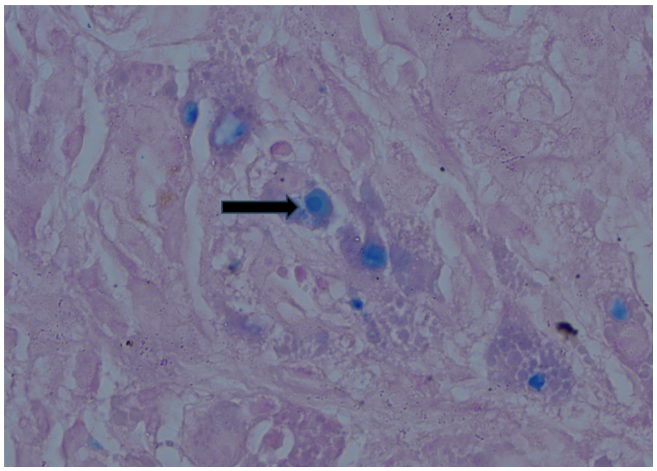

Figure 3: Macrophages highlighting Michaelis-Gutmann bodies (arrow). (Perls Prussian Blue stain. 40X).

Discussion

Malakoplakia is a rare inflammatory condition that most commonly involves urinary tract, but it can also affect colon, rectum and less frequently the skin and lungs. The disease is caused due to impaired phagocytic function of macrophages, thus causing accumulation of incompletely destroyed bacteria within the cytoplasm of histiocytes.

The disease is more common in transplant patients since immunodeficient state favours infection and impairs immune response to infection [2, 3]. Urinary bladder is the frequently involved site leading to obstructive uropathy.

Biopsy of the involved tissue shows accumulation of histiocytes with dense granular cytoplasm. Periodic Acid-Schiff (PAS) stain show positive granules, and highlight the Michaelis-Gutmann bodies, which are pathognomonic of the disease [4]. These macrophages are called Von Hansemann cells. Encrusted iron and calcium are present in the Michaelis-Guttmann bodies which are stained with Perls Prussian Blue stain and Von Kossa stain and help to confirm the diagnosis.

Malakoplakia affecting the renal allograft parenchymal tissue is rare [5]. It can mimic a tumor on imaging. Biopsy helps to establish the diagnosis. Malakoplakia of the graft kidney is associated with poor graft outcome. It can present with renal dysfunction, pain, dysuria or lower urinary tract symptoms. Graft biopsy is essential for diagnosis. Prompt diagnosis and prolonged antibiotics can help in better graft outcome.

Conclusion

This case is reported due to its rarity with regards to location of the disease. Malakoplakia should be considered in the differential diagnosis of unexplained renal allograft dysfunction. Biopsy and histopathological examination are crucial for establishing the diagnosis and recognizing the need for prolonged treatment with antibiotics.

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