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P422 -Non-infective malakoplakia developing in a renal transplant and associated with poor outcome

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Introduction: Malakoplakia is a rare, chronic inflammatory disorder which results in the formation of granulomatous plaques or nodules. The exact aetiology remains uncertain, however likely develops due to defective bacteriocidal function in macrophages following phagocytosis of a pathogen. The majority of cases are seen in immunosuppressed individuals, and up to 80% of cases are associated with gram negative, mostly e. coli infection, with the genitourinary tract most frequently affected. Renal transplant patients are therefore at notable risk. The diagnosis is made histologically, with large, eosinophilic histiocytes (von Hansemann cells) containing pathognomonic Michaelis-Gutmann bodies seen on biopsy: the manifestation of the residual, abnormal phagolysosome containing iron and calcium deposits. Due to the strong association with infection, in transplant patients the mainstay of treatment is reduction of immunosuppression and prolonged antibiotics, with surgical resection in refractory cases. We present a unique case of malakoplakia developing in a renal transplant patient despite no evidence of bacterial or mycobacterial infection, and instead speculated to be due to an abnormal immune response.

Case: A 19 year old female with end stage renal failure secondary to renal dysplasia underwent renal transplantation and subsequent immunosuppression with oral prednisolone, mycophenolate and tacrolimus. She was treated for an episode of acute vascular rejection with graft dysfunction, and continued to have poor compliance and erratic levels of immunosuppression. Despite repeatedly negative urine cultures and no clinical episodes of infection, she developed acute graft failure with malakoplakia confirmed on biopsy. Due to the lack of infective trigger it was speculated that malakoplakia had developed due to an abnormal immune response, therefore her prednisolone was increased and mycophenolate and tacrolimus left unchanged. Repeat biopsy confirmed established malakoplakia with significant interstitial fibrosis and tubular atrophy, with no microbial growth on tissue culture. There were no episodes of infection associated with increased corticosteroid. Despite this, she remained dialysis dependent with a poor clinical outcome.

Discussion/Learning points: We present a case of biopsy proven malakoplakia in a renal transplant patient, which was not thought to be triggered by infection, but rather an abnormal immune response. Despite an increase in immunosuppression there was no improvement either clinically or histopathologically. Current literature reports an overall cure rate of 53% in malakoplakia, however it is likely that those cases with no infective association have a worse clinical outcome. Malakoplakia remains a rare condition, however one to remain vigilant for in renal transplant patients.