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Malakoplakia of Urinary Bladder: A Case Report

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ABSTRACT

Malakoplakia is a chronic inflammatory disease commonly affecting the urinary tract. It can affect other organs also. It generally affects middle-aged immunocompromised individuals. *E.coli* is the most common pathogen followed by proteus, mycobacteria. Macrophage dysfunction is the most common pathway in pathogenesis. Clinically this condition can mimic malignancy. Histopathological diagnosis is needed for diagnosis. Treatment includes targeted antibiotic therapy. Multiple, large lesions and those which persists needs surgical excision,

INTRODUCTION-

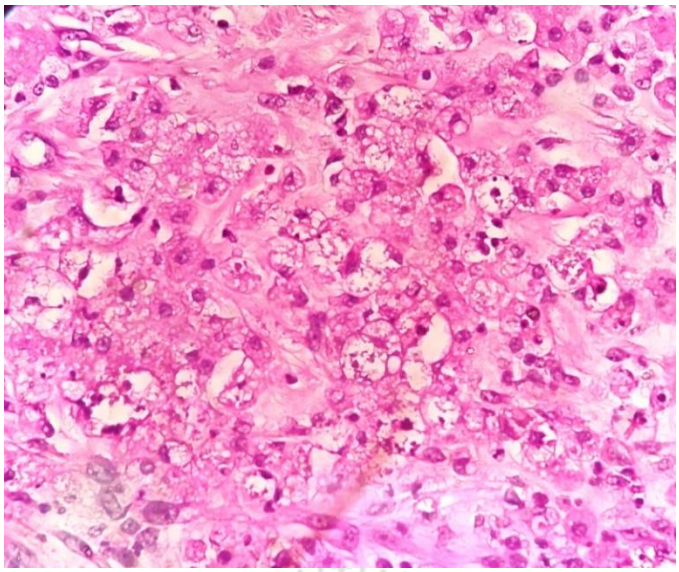
Malakoplakia is a rare chronic inflammatory disease that was first described in 1902 by Michaelis and Gutmann and is more common in females[1]. Both etiology and pathogenesis have remained unknown; however, its occurrence has been linked with macrophage malfunction. Defects in phagocytic or degenerative functions of histiocytes in response to gram-negative coli forms like *E.coli*, Proteus are believed to result in chronic inflammatory states presenting as plaque or papule formation[2]. It forms soft, yellow plaques, hence the name from Greek words malako (soft),plakia (plaque) has been derived[3]. The lesion is usually solitary but can be multiple[5]. Malakoplakia can affect all body organs; the bladder being the most frequently affected one. Patients of any age may develop malakoplakia, but the peak occurrence is in middle age. The disease is more common in immunocompromised individuals[6]. The clinical diagnosis of malakoplakia is very difficult. It is often misdiagnosed as a malignant condition and requires confirmation by histopathology. It is characterized histologically by Von Hansemann histiocytes and Michaelis Gutmann bodies. Von Hansemann cells are oval histiocytes that contain intracytoplasmic bodies called Michaelis Gutmann bodies[7,8].

CASE REPORT-

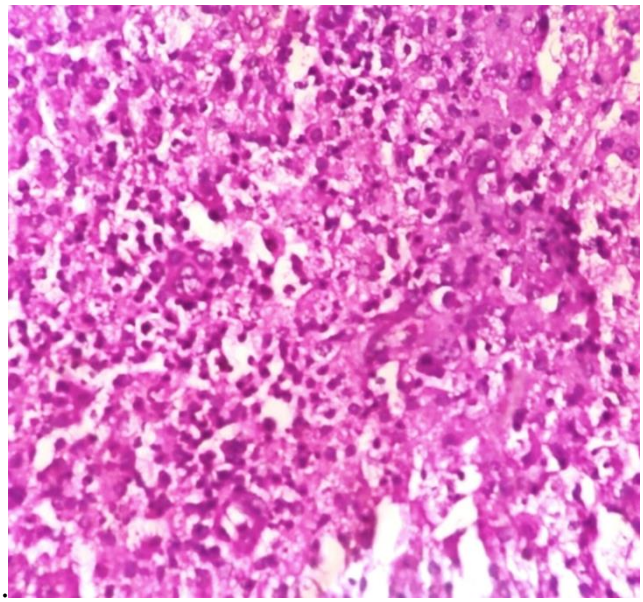
55 Years male presented with a history of fever with chills and burning micturition for 2 days. USG Abdomen and pelvis revealed outpouching of a short segment of bowel loop with oedematous wall and maintained vascularity, noted adjacent to the right-superolateral margin of urinary bladder suggesting the internal type of supracervical hernia, however, CECT was suggested for confirmation. CT scan of the abdomen and pelvis revealed-A a relatively well-defined fluid density lesion with a thick irregular wall measuring 5.1X4.3X5.2 cm seen along the superior aspect of the bladder with adjacent bladder wall thickening. The significant surrounding fat stranding was noted. Findings suggested-? An infected urachal cyst (abscess/collection). Another possibility includes an infected urinary bladder wall diverticulum.

We received a specimen of excised Dome of the Urinary bladder with mass. Grossly, it was a globular mass measuring 10 x 7 x 5 cm. Cut surface showed a roughly oval greyish white lesion with irregular edges, merging into surrounding muscle, measuring 3 cm in diameter. Multiple sections revealed lamina propria containing syncytial sheets of large foamy histiocytes having

abundant eosinophilic cytoplasm and central & eccentric nuclei, few containing phagocytosed inflammatory cells. They are surrounded by cystic spaces. Michaelis Gutman's bodies were seen inside the histiocytic cells. Occasional giant cells were also evident. The lesion was irregularly infiltrating into surrounding muscle and fat. Based on these findings, it was reported as the Classic stage of Malakoplakia of the bladder.



Photomicrograph A; high power view (45X) showing scattered foamy histiocytes.



Photomicrograph 2 (High power view-45X) showing Michaelis Gutman bodies along with scattered mixed inflammatory cells.

DISCUSSION:

Malakoplakia is a benign self-limiting condition generally associated with a good prognosis if treated promptly. The initial approach includes treatment of UTI and surgical removal of the lesions. Very few cases of renal failure associated with multifocal malakoplakia have been described in the literature[1,2]. Malakoplakiacan mimics malignancy macroscopically and clinically. The diagnosis is to be confirmed by histopathologicalexamination[3].

Nearly 90% of the patients with malakoplakia have urine infections by coliforms and 40% have an autoimmune disease or some type of immunodeficiency. *E coli* is the most common gram-negative bacteria isolated, followed by proteus and mycobacterium tuberculosis[5,6].

It has been proposed that abnormal macrophage response because of defective lysosomal function plays the main role in pathogenesis. Macrophages in malakoplakiaare capable of phagocytosis but unable to digest the bacteria and thus these partially digested bacteria accumulate in monocytes or macrophages and lead to the deposition of calcium and iron on residual bacterial glycolipid. The resulting basophilic structures, the Michaelis Gutmann bodies, are considered pathognomonic for malakoplakia. Thus, these bodies contain calcium, phosphorous, and iron[5].

The clinical presentation of malakoplakia depends on the organ involved. Malakoplakia of the urinary bladder presents with irritative lower urinary tract symptoms and hematuria mimicking malignancy. Ureteralmalakoplakia can present with obstruction and formation of stricture. Renal parenchymal malakoplakia can cause fever, flank pain, and mass. Prostatic malakomlakia may mimic carcinoma of the prostate as it may present as hard induration on digital rectal examination, testicular malakoplakia may present with symptoms of epididymoorchitis[7].

Based on microscopic features, Malakoplakia can be divided into three stages:

1. Initial inflammatory stage.

2. Classic stage with abundant Michaelis-Gutmann bodies

3. Third stage with progressive fibrous tissue and scarring.

Treatment is generally based on the eradication of *E.coli* infections that are believed to cause malakoplakia manifestations. However other causes must be ruled out. The best antibiotics are those which achieve high intracellular levels such as fluoroquinolones and trimethoprim-sulfamethoxazole. In addition to these agents, bethanechol and vitamin C are believed to enhance phagocytic bactericidal activity by increasing cyclic guanosine monophosphate levels[8]. The large lesions, multiple, persists longer, need surgical intervention. Considering chances of recurrence, follow-up of cases is necessary[8].

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