Case Report

Recurrent bladder malakoplakia: A rare bladder lesion mimicking malignancy

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Abstract

Background: Malakoplakia is a rare granulomatous disease that commonly involves the genitourinary tract with the urinary bladder being the most frequently affected site. It is characterized by histiocytes containing distinct basophilic calcified inclusions called Michaelis–Gutmann bodies. It is believed to result from abnormally functioning macrophages, with inclusions representing calcifications around incompletely digested bacteria. Although its pathogenesis remains unknown, it is well-documented that the condition is associated with chronic urinary tract infections and immunosuppression. Grossly, it can present as soft, yellow plaques, nodules, bladder mass, or even without any visible lesion. It poses a huge diagnostic challenge as it tends to mimic malignancy. **Case presentation:** Described here is an 86-year-old female with recurrent bladder malakoplakia who presented with foul-smelling urine, hematuria, and dysuria. The clinicopathological features of this rare bladder lesion are described along with a review of the literature. **Conclusion:** Early identification of malakoplakia's features by pathologists is essential for effective patient management. This condition should be considered in the differential diagnosis of bladder lesions, especially when *Escherichia coli* is present.

Keywords: Malakoplakia, Granulomatous disease, Urinary bladder, Michaelis–Gutmann bodies, Urinary tract infection, Neoplasm, Urinary tract pseudotumor, Hematuria, Rare disorder, *Escherichia coli*

1. INTRODUCTION

Malakoplakia is an unusual chronic inflammatory disease first described in 1902 by Michaelis and Gutmann [1]. The term malakoplakia was introduced in the following year by Von Hansemann [2]. It is derived from the Greek word "malakos" (soft) and "plakos" (plaque). Although it is generally confined to the urinary tract, it has also been reported at other body sites, such as prostate [3], testis and epididymis [4], gastrointestinal tract [5,6], adrenal gland [7], vagina [8], skin [9], lung [10], and bone [11]. It typically occurs in a middle-aged female in association with a coliform urinary tract infection. The disease is more common in patients with diabetes mellitus or immunocompromised individuals, such as those with autoimmune disease, acquired immunodeficiency syndrome, or recent transplant recipients [12]. The main presenting symptoms are hematuria, recurrent urinary infection, and urinary obstruction. Grossly, malakoplakia can present as a soft yellow plaque, nodules, ulcer, or even as a bladder mass. Although it is a benign condition, it often mimics a malignant neoplasm when a mass-like lesion is present [13]. The lesion is usually single but can be multiple or may even present without any visible lesion. Histological examination of the lesion shows large mononuclear histiocytes (Von Hanseman cells), lymphocytes, and Michaelis–Gutmann bodies. Michaelis–Gutmann bodies composed of calcium and calcospherites are pathognomonic of this condition. These are believed to be calcifications around incompletely digested bacteria and there seemingly exists a strong relationship with the coliform infection [14]. The exact etiology is unknown, but it is believed to be caused by a defective degradative function of histiocytes in response to coliform or proteus infection [15]. The underlying abnormality is a low cGMP/cAMP ratio leading to malfunctioning intracellular microtubules and therefore defective phagocytosis [14].

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2. CASE PRESENTATION

2.1. The case

The 86-year-old female was admitted to the hospital with a 3-day history of nausea, diarrhea, foul-smelling urine, dysuria, and hematuria. Her history was significant for atrial fibrillation on anticoagulation, gastroesophageal reflux disease, hypertension, hypothyroidism, hyperlipidemia, neuralgia, and previous knee replacement. At admission, her blood pressure was quite low, being at 79/49. There was no chest pain or shortness of breath. The ECG showed low voltage but no ST-segment changes. The investigations showed low hemoglobin at 94 (reference range: 118 – 158 g/L), and elevated white blood cell count at 15.4 (reference range: 4.0 – 11.0 × 10^9/L). Her creatinine was elevated at 252 (reference range: 49 – 90 μmol/L). Her troponin was markedly elevated at 2695 (reference range: 0 – 39 ng/L). The clinical impression was that of a septic shock and given her septic presentation her myocardial infarction (MI) was believed to be a type 2 MI from which she slowly recovered.

The urine culture on multiple occasions showed growth of *E. coli* and *Pseudomonas aeruginosa*. The antibiotic sensitivity testing for *E. coli* demonstrated susceptibility for amoxicillin, cefazolin, nitrofurantoin, amoxicillin/clavulanic acid, and resistance for trimethoprim/sulfamethoxazole. The *P. aeruginosa* showed sensitivity for ceftazidime, gentamycin, tobramycin, piperacillin/tazobactam, and resistance for ciprofloxacin. Blood culture revealed growth of *P. aeruginosa* sensitive to ceftazidime, tobramycin, and piperacillin/tazobactam. The organisms were resistant to ciprofloxacin.

A computed tomography (CT) scan showed atrophy of the left kidney with thinning of the renal cortex. There was chronic moderate left-sided hydronephrosis and dilation of the left renal pelvis. The proximal left ureter was dilated. A cyst was found in the mid-pole of the left kidney, measuring 2.4 cm. No stones were seen. There was moderate right-sided hydronephrosis. No renal calculi were identified. The proximal and mid-right ureter was also moderately dilated. The distal right ureter was difficult to visualize. The etiology of hydronephrosis was uncertain on the CT scan examination. There existed diffuse bladder wall thickening, measuring up to 1.2 cm. The differential diagnosis included infectious/inflammatory as well as neoplastic etiology.

Cystoscopy exhibited multiple well-defined raised plaque-like whitened lesions, measuring 0.2 to 1.0 cm in size. The lesions displayed a smooth rounded dome-like appearance. The left ureteric orifice was narrowed but the right ureteric orifice was not. The ureteric mucosa in some areas resembled bladder mucosal lesions. Some of the bladder lesions were biopsied for histological examination.

A review of the patient's chart indicated that the patient had presented with recurrent urinary tract infection 1½ years back. Cystoscopic examination during that time showed multiple, well-defined raised plaque-like whitened lesions, in several areas of the bladder. The biopsies from the lesion showed features of malakoplakia. Six months later, an ascending colon mass was noted and was suspected to be colonic adenocarcinoma. The mass measured 4.1 × 2.1 × 1.2 cm. The endoscopic biopsy of the mass showed extensive ulceration with granulation tissue with no evidence of malignancy. However, in view of the endoscopic appearance and size of the mass with strong suspicion of malignancy, and likelihood of sampling error, right hemicolectomy was carried out. The pathological examination of the mass revealed features of malakoplakia with no evidence of malignancy.

2.2. Pathology

Histological examination of the bladder biopsy showed sheets of foamy epithelioid histiocytes with PAS-positive granular eosinophilic cytoplasm. Scattered lymphocytes were present. Numerous calcified Michaelis–Gutmann bodies were observed which were further highlighted with Von Kossa staining. Morphological features were consistent with malakoplakia. The overlying urothelium was markedly attenuated and showed reactive changes (Figures 1-4). There was no evidence of malignancy.

2.3. Immunohistochemistry

The immunohistochemistry demonstrated positivity for vimentin and CD 68 in the histiocytic cells. The pan-cytokeratin (AE1/AE3) immunostaining was negative (Figure 4). The immunohistochemical staining pattern was in keeping with the morphological diagnosis of malakoplakia.

2.4. Treatment and follow-up

The patient was treated with parenteral antibiotics (piperacillin/tazobactam) for a prolonged period of time (3 weeks). The patient was followed up on the outpatient basis and was admitted to the hospital a few times with a urinary tract infection. The abdominal-pelvic ultrasound revealed an atrophic left kidney and severe right-sided hydronephrosis. The urine culture was positive for Enterobacter and Enterococcus. The cystoscopy showed a bladder filled with cloudy urine. There was increasing hydronephrosis and poor appearance of the right and left kidney. She has been treated with antibiotics and is followed on an outpatient basis.

3. DISCUSSION

Malakoplakia most commonly affects the urinary tract, followed, in order of decreasing frequency, by the genital

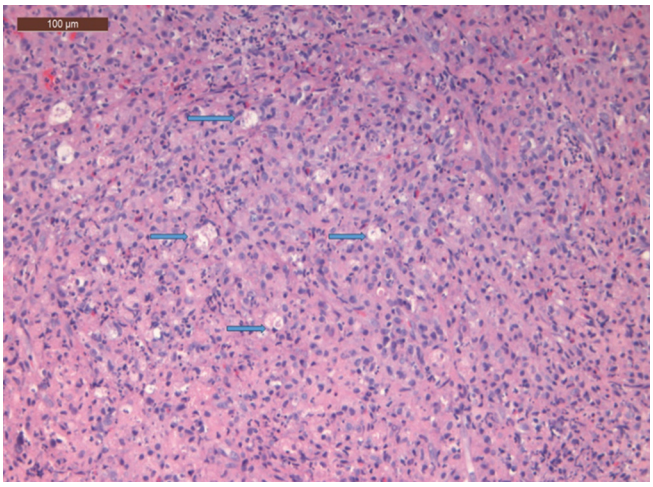

Figure 1. Histologic features of malakoplakia. The lesion showed mixed acute and chronic inflammatory infiltrate with numerous scattered histiocytic cells known as Hansemann cells. The histiocytic cells (indicated with blue arrow) showed abundant vacuolated cytoplasm (H and E, ×25).

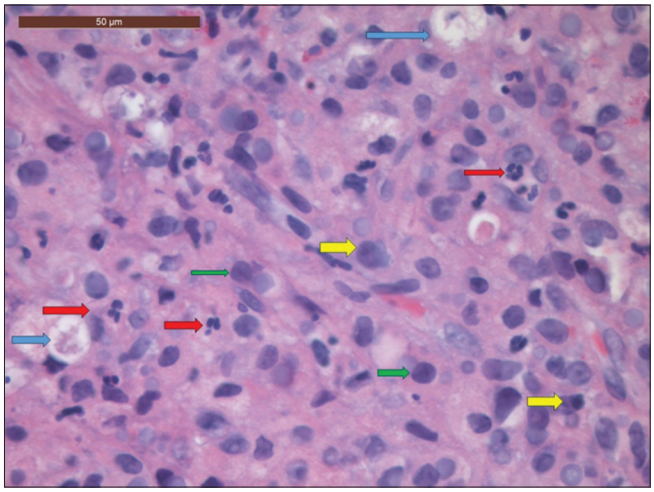

Figure 2. Higher magnification showed mixed acute and chronic inflammation by neutrophils (red arrow), lymphocytes (green arrow), plasma cells (yellow arrow), and histiocytes (blue arrow) (H and E, ×50).

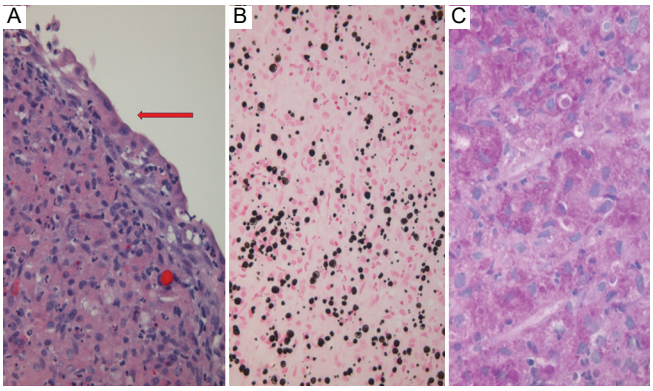

Figure 3. (A) Attenuated urothelial lining was noted in some areas (arrow) (H and E, ×50). (B) Numerous intracytoplasmic calcified bodies known as Michaelis–Gutmann bodies were present throughout the lesion (Von Kossa stain, ×100). (C) Epithelioid histiocytes known as Hansemann cells showed PAS-positive granular eosinophilic cytoplasm (PAS stain, ×100).

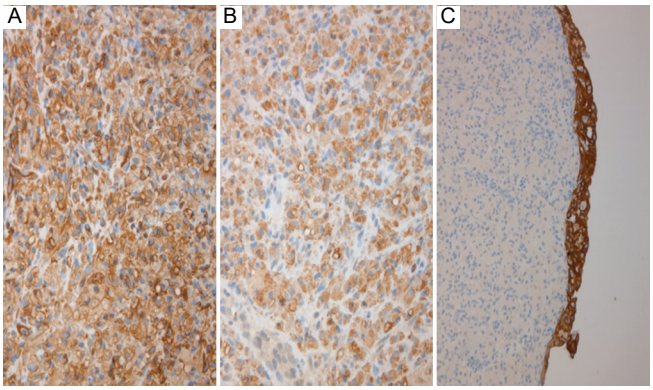

Figure 4. Immunohistochemical studies. The histiocytic cells showed positivity for vimentin (A ×100) and CD68 (B ×100). The overlying urothelium showed positivity for pan-cytokeratin (AE1/AE3) (C ×50). The lymphohistiocytic inflammatory infiltrate was negative for pan-cytokeratin (AE1/AE3).

tract, gastrointestinal tract, and retroperitoneum. The bladder is involved in 40% of all patients and 75% of patients with urinary tract malakoplakia. Nonetheless, the renal pelvis, ureter, and urethra may also be the primary site of disease [16]. Urinary tract malakoplakia afflicts females 4 times as frequently as males, but extra-urinary tract malakoplakia is more common in males [17]. The disease occurs at any age but there is a peak incidence at 50 years and children are rarely affected. The youngest case was reportedly a 6-week-old child with malakoplakia of the adrenal gland and colon who died with miliary tuberculosis [18]. The clinical presentation depends on its site of origin. Bladder malakoplakia usually presents with hematuria and symptoms of bladder irritability in the form of frequency, urgency, or dysuria. Patients with bladder malakoplakia usually have a history of chronic urinary tract infection, usually, with *E. coli* as well as other Gram-negative coliform organisms, such as Proteus or Klebsiella species. Cases of malakoplakia without demonstrable concurrent infection have been reported. When it is associated with malignancy, malakoplakia is identified before, concurrently, or after the diagnosis of malignancy [14,19-21]. Polisini *et al*. reviewed the literature on malakoplakia of the bladder and analyzed the data from 35 articles about malakoplakia to describe the clinicopathological features, comorbidities, urine culture findings, serum creatinine levels, and presence of hydronephrosis on imaging. 47.22% of the patients from the reviewed cases suffered from recurrent urinary tract infections, and 19.44% from immune disorders. Urine culture was positive in 69.44% of cases, with *E. coli* being isolated in 92% of the cases. Hydronephrosis was present in 44.44% of the cases, with the majority of patients having bilateral hydronephrosis [22]. Cystoscopy demonstrates single or multiple polypoid masses resembling urothelial carcinoma or chronic cystitis.

Grossly, the typical lesion is a soft yellow-brown plaque. There may be central ulceration and peripheral hyperemia.

McClure reviewed 34 cases described in the literature and found that the lesion was variably described as flat (seventeen cases), nodular (nine cases), papillary (two cases), polypoid, hemorrhagic, trabeculated, and tumor-like (one case each). The size of the lesion ranged from 1 cm to 20 cm [23]. Microscopically, malakoplakia presents as a submucosal lesion in early stages under intact urothelia, with ulceration of the urothelium occurring at a later stage. The lesion is characterized by dense aggregates of large mononuclear cells known as Hansemann cells, with pale finely-vacuolated cytoplasm. Within the cytoplasm of these cells are oval calcified inclusions, *that is*, Michaelis–Gutmann bodies. The stroma is infiltrated by lymphocytes and plasma cells. Smith reviewed 24 cases from the files of the Armed Forces Institute of Pathology and postulated that malakoplakia goes through three histological phases: an early (prediagnostic) phase, which is characterized by the presence of plasma cells and macrophages, a classic phase with Michalis– Gutmann bodies, and a fibrosing phase with only occasional Michalis–Gutmann bodies. Therefore, Michalis–Gutmann bodies may not be seen in early- and late-stage malakoplakia [24]. Standard staining may not reveal Michaelis and Gutmann bodies and special staining with PAS or Von Kossa may be required [23]. The immunohistochemistry demonstrates positivity for CD68 and alpha chymotrypsin in the macrophages. Histologically, it is important to differentiate malakoplakia from chronic xanthogranulomatous cystitis and urothelial carcinoma. Xanthogranulomatous cystitis, such as malakoplakia, may present with hematuria and cystoscopic examination shows single or multiple polypoid bladder masses. Microscopic examination reveals inflammatory infiltration with lipid-laden macrophages, lymphocytes, and plasma cells. Scattered Touton-type giant cells may be present. The overall histological picture vaguely resembles malakoplakia except that Michaelis–Gutmann bodies are not seen. Urothelial carcinoma can be easily differentiated from malakoplakia on microscopic examination; however, the diagnosis of malakoplakia does not preclude a concomitant diagnosis of urothelial carcinoma as it has been recorded occasionally [25].

The clinical differential diagnoses include benign lesions such as polypoid cystitis or other infections. It may also resemble primary or metastatic bladder neoplasm on cystoscopic examination. Clinically, there are no distinguishing features to make a definite diagnosis of malakoplakia and a definite diagnosis requires histopathological examination. Pathologically, differential diagnoses involve poorly differentiated urothelial carcinoma, including plasmacytoid and lymphoepithelioma, like urothelial carcinoma. The urothelial carcinoma may show features of urothelial carcinoma *in situ* in the overlying urothelium. The urothelial carcinoma also exhibits positivity for cytokeratin staining, which is negative in malakoplakia. Gleason pattern 5 prostatic adenocarcinoma can also mimic malakoplakia on histological examination. Again, prostatic adenocarcinoma is positive for pan-cytokeratin and other prostate-specific markers, such as prostate-specific antigen and prostatic-specific acid phosphatase.

On electron microscopy, Michalis–Gutmann bodies are seen as crystalline central cores with peripheral lamellar rings of mineral deposits. Although the lack of Michaelis–Gutmann bodies on histological examination may render the diagnosis difficult, ultrastructural examination may be a useful tool when malakoplakia is suspected. Jung *et al*. examined the full process of Michaelis–Gutmann body formation at the ultrastructural level using both scanning electron microscopy and transmission electron microscopy and realigned the ultrastructural findings according to the sequence of events as pre-phagosomal, phagosomal, and post-phagosomal stages [26]. They recognized, for the first time, the *E. coli* captured by phagosomes or partially damaged by lysosomal attack within the cell [26].

The exact etiology of malakoplakia is unknown, but it is thought to be caused by a defect in the degradative function of histiocytes in response to *E. coli* or proteus infection [15]. Von Hansemann cells arise because of abnormal phagocytosis and the Michaelis–Gutmann bodies within them may represent mineralized fragments of bacteria [27,28]. Lewin *et al*. postulated that bacteria phagocytosed by malakoplakic macrophages are incompletely digested and persist as dense amorphous aggregates within the phagolysosomes [28]. These later become encrusted with calcium phosphate aggregates from laminated Michaelis–Gutmann bodies. The cause of defective phagocytosis is most probably related to a low CGMP/cAMP ratio leading to malfunctioning intracellular microtubules, which are essential for phagocytosis [28].

Many causes of malakoplakia have been proposed, including tuberculosis, sarcoidosis, neoplasia, fungal and viral infection. Smith *et al*. excluded all these causes in each of his 24 patients [24]. Stanton and Maxted reviewed 153 cases of malakoplakia in literature, 93 of which had microbiological culture on urine, blood, or malakoplakic plaques, 89.4% had a coliform infection, 4.3% had a non-coliform infection, and 6.4% had negative culture [16]. Of those with coliform infection, 72% were *E. coli*, 18% were unspecified coliform organisms, and 15% were coliform organisms other than *E. coli* like Klebsiella, Proteus, and Pseudomonas [16]. McClure found that *E. coli* was cultured from the urine of all 11 patients with malakoplakia of the prostate [23]. Deridder *et al*. reported positive cultures for *E. coli* in 13 out of 14 patients with malakoplakia of the renal parenchyma [29].

The relationship between malakoplakia and *E. coli* infection is not straightforward. *E. coli* infection is very common, but malakoplakia is very rare. Hence, some other factors may be

involved in the pathogenesis of malakoplakia. Malakoplakia is also seen at body sites where coliform organisms should not exist, such as the brain and skeleton. This raises the possibility of unusual strains of *E. coli* or abnormal immune response. Out of 183 patients reviewed by Stanton and Maxted, 41% had an intercurrent systemic illness, carcinoma, immune deficiency, or autoimmune disease [16]. Its occurrence has also been linked to immunosuppression and transplant recipients [16,24,30-32]. According to Curran, a combination of impaired host defense and defective phagocytosis leads to the accumulation of bacterial degradation products and results in a granulomatous reaction [14].

The treatment of malakoplakia is usually medical and local excision of the mass is indicated if the standard conservative management fails. There are no widely established guidelines for the medical management of malakoplakia, but most approaches involve antibiotics that concentrate intracellularly in macrophages, such as quinolones, trimethoprim, and rifampicin [15]. The optimal duration of antibiotic treatment is not clear. Where successful outcomes have been achieved, antibiotics have been given for more than 2 months. Bethanechol, a choline agonist, and ascorbic acid are also given to correct the decreased cGMP levels in monocytes that interfere with complete bacterial killing. Antibiotics are recommended for patients with bilateral or multifocal disease, whereas surgical excision is recommended for unifocal disease [33]. Surgical treatment such as transurethral resection may also be necessary for bladder malakoplakia if the lesion is large and obstructs the ureter [13]. As malakoplakia is associated with immunosuppression, discontinuation of immunosuppressive drug is usually needed depending on the risk–to–benefit ratio [13,16,34].

Malakoplakia is a benign condition usually associated with a good prognosis; however, if not identified early, it can be fatal [35]. Stamatiou *et al*. described a 72-year-old male whose bladder malakoplakia resulted in renal failure and death. Apparently, 3 years before the admission, the patient had symptoms of malakoplakia which went undiagnosed. The bladder biopsy diagnosed the lesion as non-specific inflammation. The case emphasizes the importance of early diagnosis to determine the best course of treatment [36]. Long-term follow-up is recommended as malakoplakia tends to persist or recur.

4. CONCLUSION

Prompt evaluation and recognition of the characteristic features of malakoplakia by a pathologist is important for prompt and appropriate management of the patient. The condition should be considered in the differential diagnosis of inflammatory or mass lesions involving the bladder, particularly when *E. coli* is identified.

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CONFLICT OF INTEREST

The author declares no conflict of interest.

AUTHOR CONTRIBUTIONS

This is a single-authored article.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Not applicable.

CONSENT FOR PUBLICATION

This case report has been completely anonymized, and all tissue was obtained as part of the standard of care for the patient; hence, no consent was required.

AVAILABILITY OF DATA

All data generated or analyzed during this study are included in this published article.

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