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A Rare Case of Gastrointestinal Malakoplakia with Achromobacter Sepsis

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Abstract

Background: Malakoplakia is a rare complication of E. coli infection, rarely seen with other infections in immunocompromised patients. **Aim**: This case highlights a rare case of Malakoplakia. It should be considered in immunocompromised patients with gastrointestinal/genitourinary symptoms, and abdominal lymphadenopathy. **Case**: We present a case with Achromobacter sepsis from an unknown source. Upon imaging, nonspecific mesenteric lymphadenopathy and colon changes prompted a colonoscopy and node biopsy, leading to the current diagnosis. **Conclusion**: Nonspecific gastrointestinal symptoms in immunocompromised patients should be explored for uncommon causes. Exploration includes obtaining imaging, performing colonoscopy, and taking biopsies. Malakoplakia is one such rare cause that is seldom considered and particulars of diagnosis and treatment are still unclear.

Keywords

Achromobacter Xylosoxidans, Malakoplakia

1. Introduction

Malakoplakia is a rare, chronic granulomatous inflammatory disease that results from impaired histiocyte clearance of bacteria in immune-compromised individuals. It was first reported in 1902. Pathogenesis is complex and poorly understood, involving an interplay between chronic bacterial infections, mostly E. coli, and compromised immune response [1] [2].

The urinary bladder is commonly involved but the gastrointestinal (GI) tract can also be involved in cases [1]. With a roughly 4 to 1 ratio, malakoplakia of the genitourinary tract is more common in females. Males are approximately two times more likely than females to develop cutaneous malakoplakia. Malakoplakia

can occur at any age, but it is most common in older people, with a peak incidence in those over 50 [3].

We present a case of a post-lung transplant patient who was suspected to have diffuse colonic metastatic disease based on computed tomography (CT) that was later determined to be diffuse malakoplakia related to systemic infection.

2. Case Presentation

A 62-year-old female presented with a one-month-long history of progressive fatigue, shortness of breath, abdominal distension, and diarrhea. Intake vital signs were stable, with a blood pressure of 122/77 mmHg, a pulse of 85 bpm, a temperature of 36.9˚C (98.4˚F), and a respiratory rate of 18 breaths per minute. Laboratory results showed liver enzymes (AST and ALT) within normal limits but slightly decreased albumin (2.5 g/dL). The coagulation panel revealed an elevated INR and prolonged prothrombin time. Pancreatic enzymes were within normal limits. The CBC indicated anemia, with hemoglobin levels ranging from 7.3 to 8.7 g/dL, low hematocrit, and mild thrombocytopenia. Routine chemistry was significant for mildly elevated creatinine (1.03 - 1.62 mg/dL) with stable electrolytes. Further workup revealed bilateral ground-glass opacities on CT. Abdominal X-ray was significant for large debris in the stomach, prompting an endoscopy, which was nonremarkable for any anatomical obstruction.

She had a history of bilateral lung transplant and was on tacrolimus, prophylactic Bactrim, and valganciclovir. During this hospitalization, the patient was found to have gram-negative bacteremia with *Achromobacter xylosoxidans* (found in pools and well water) and candidemia, prompting the involvement of transplant infectious disease specialists. Candidemia was deemed to be pulmonary in origin, but the source of bacteremia was unclear. The patient was started on eraxis and zosyn, which was modified to cefiderocol and then meropenem due to persistent positive cultures. An echocardiogram and magnetic resource imaging (MRI) of the spine were performed to look for possible endocarditis or epidural abscess as a source of infection, but they were unremarkable.

Given the waterborne source, GI was considered as a source leading to abdominal imaging, which revealed extensive metastatic adenopathy in the abdomen and upper pelvis highly suspicious for metastatic disease with a possible primary lesion in the hepatic flexure of the colon. Image-guided abdominal lymph node biopsy performed by interventional radiology revealed malakoplakia (**Figures 1-3**), a rare inflammatory condition that typically occurs in immunocompromised individuals and is thought to be secondary to a bactericidal defect in macrophages. Subsequent colonoscopy demonstrated nodular mucosa in ascending colon and hepatic flexure with pathology redemonstrating malakoplakia. Malakoplakia findings were not amendable to surgical interventions. After the literature review, the team concluded treating the underlying infection. A decision was made to medically treat the patient with meropenem which the bacteria was sensitive to.

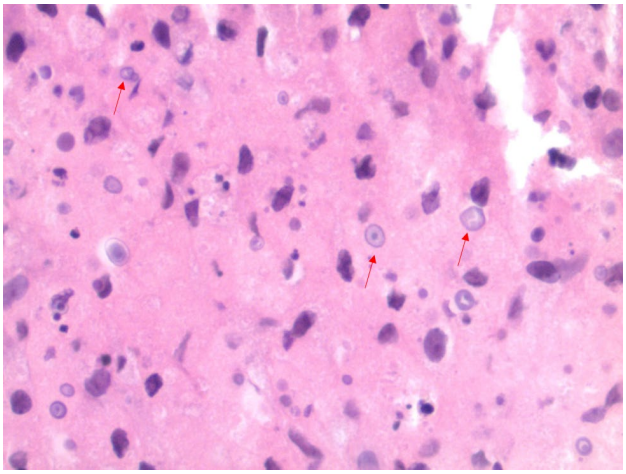

Figure 1. Ascending colon biopsy section, Hematoxylin and Eosin stain (high magnification): Characteristic Michaelis-Guttman bodies (red arrows) are identified within the submucosal histiocytic infiltrate.

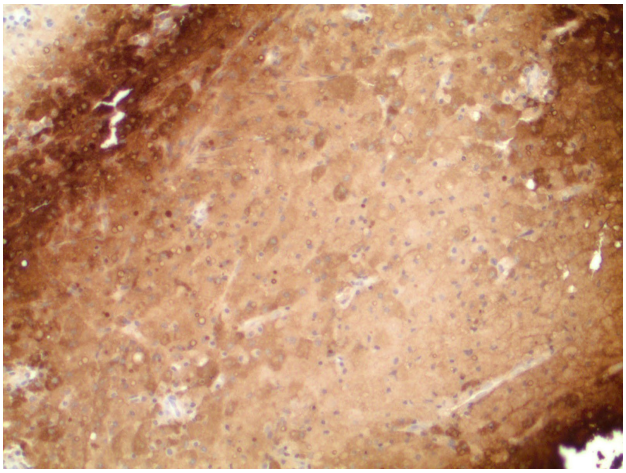

Figure 2. Ascending colon biopsy section, CD68 immunohistochemical stain (high magnification): CD68 highlights the histiocytic infiltrate. Multiple Michaelis-Gutmann bodies are seen.

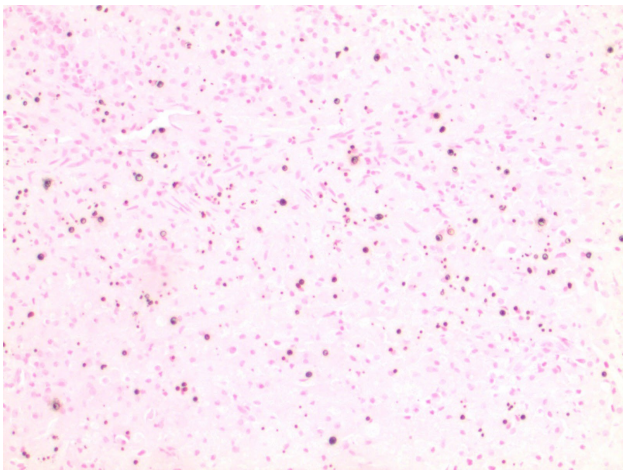

Figure 3. Ascending the colon biopsy section, Von Kossa Stain highlights calcium deposits within Michaelis-Guttman bodies.

The patient then developed increased work of breathing of breathing and tachypnea, requiring supplemental oxygen at 6 L/min via nasal cannula. The patient was started on dialysis and transferred to ICU. The patient tested positive for COVID-19 and was started on Remdesivir therapy, but ultimately expired due to worsening respiratory distress.

3. Discussion

Malakoplakia results from bactericidal defects of histiocytes, resulting in an accumulation of phagolysosomes in immunocompromised populations. It produces granulomatous inflammation. Gram-negative rod organisms, especially *E. coli* have been known to be a culprit. Malakoplakia is most seen within the urinary and gastrointestinal tracts [4] [5].

Literature in urinary tract manifests as diffusely increased renal parenchymal changes, corticomedullary differentiation loss, enlarged kidneys, and masses [6] [7].

In GI, the presentation ranges from flat lesions to multiple ulcerated polyps. In a recent study, the rectum was the most frequent site followed by the sigmoid colon, cecum, and ascending and transverse colon [4].

Diagnosis requires histology, characterized by clumps of histiocytes with eosinophilic cytoplasm known as Hansemann cells. Michaelis-Gutmann bodies with concentric laminations are diagnostic. These bodies imply calcium-covered phagosomes, which stain with kossa calcium stains and CD68 highlights the histiocytic infiltrate [5].

The diagnosis, as in this case, can often be delayed and result in unnecessary interventions until a true diagnosis is achieved. Once diagnosed, antibiotic therapy is tailored to the culprit organism. Treatment possibilities include administering antibiotics that can enter macrophages and aid in the intracellular death of bacteria since monocytes and macrophages have a diminished capacity to undertake intracellular bacteriocidal activity. Ciprofloxacin, rifampin, and trimethoprim-sulfamethoxazole are effective treatments for malakoplakia [8]. Perhaps as a result of their ability to reach high intracellular concentrations in macrophages, fluoroquinolones are particularly beneficial in treating malakoplakia. In a study, patients on antibiotics treatment including daptomycin, mikamycin, vancomycin, cephalexin, levofloxacin, azithromycin, ciprofloxacin, resection of the mass/plaque-like lesions, with resolution in 60% patients [9] [10].

4. Conclusion

Malakoplakia is a rare complication seen with gram-negative infections in patients with impaired immunity. Nonspecific imaging findings make diagnosis challenging. It should be considered and actively sought in immunocompromised patients with infections of unknown origin, gastrointestinal/genitourinary symptoms, and abdominal lymphadenopathy. Treatment involves treating the underlying infection with antibiotics like fluroquinolones, and bactrim commonly used.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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