A Case of Colonic Malakoplakia

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Introduction

Malakoplakia is a rare histiocytic inflammatory response that is usually found in the urinary tract. Gastrointestinal involvement is the second most common location affected and is a result of dysfunctional macrophages. Malakoplakia is associated with immunosuppressive therapy, colorectal carcinoma, and infectious diseases. Here we present a case of a patient with alcohol abuse and hepatitis B who was found to have a 2 mm sessile polyp in the cecum on screening colonoscopy. Histopathology revealed malakoplakia.

Case Presentation

A 54 year old man with past medical history of alcohol use disorder was referred to clinic for acute, resolved, asymptomatic liver enzyme abnormalities. He endorses drinking six cans of beer and a fifth of liquor daily for many years. He does not use intravenous drugs. He denies diarrhea, rectal bleeding, or abdominal pain. Physical exam was significant for a soft, nontender abdomen and no stigmata of chronic liver disease. Laboratory findings were significant for AST of 270 U/L, ALT of 63 U/L, ALP of 65 U/L, and total bilirubin of 0.3 mg/dL. Further workup showed positive hepatitis B surface antigen, reactive hepatitis B core antibody and hepatitis B DNA by PCR was 172K IU/mL. Patient underwent screening colonoscopy which revealed a 2 mm sessile polyp in the cecum (shown in Figure A.). Histological examination of the polyp showed aggregates of granular histiocytes containing Michaelis- Gutmann bodies, histochemically confirmed by periodic acid-Schiff and von Kossa stains which is pathognomonic for malakoplakia (Figure B). Upon further chart review, patient was found to have indeterminant HIV testing 1 year prior and was subsequently referred to infectious disease with follow up surveillance colonoscopy in 6 months. He was subsequently lost to follow up due to alcoholism.

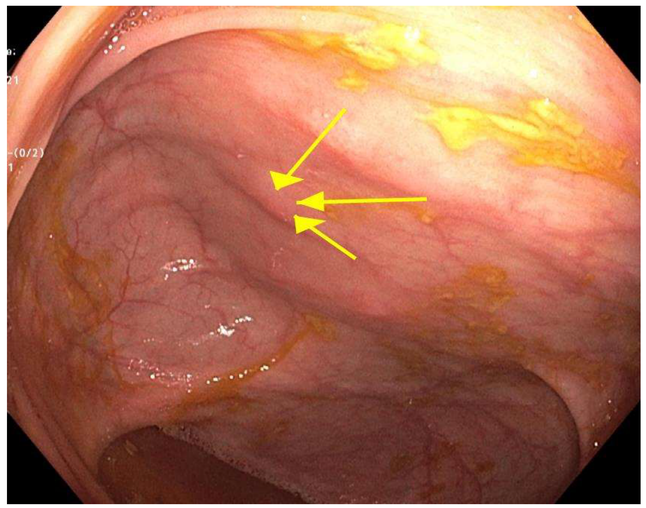
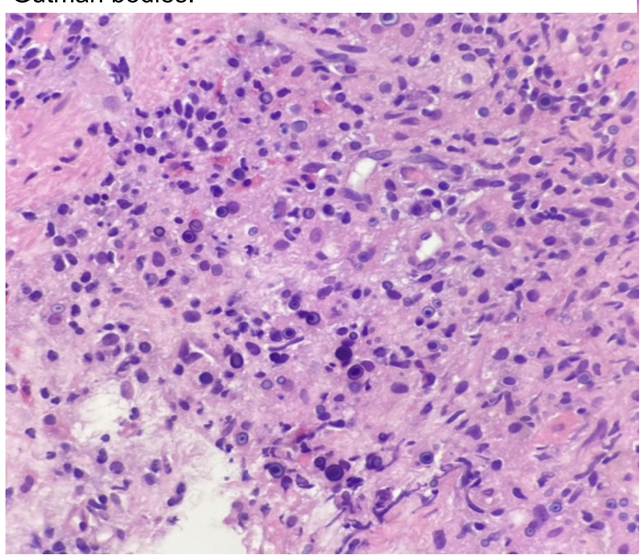


Figure A. (above) Two mm cecum polyp denoted by yellow arrows. Figure B. (below) High-power view of malakoplakia. Macrophages contain basophilic targetoid structures, which are called Michaelis-Gutman bodies.



Discussion

The first report of colonic malakoplakia was described by Terner and Lattes in 1965. The gastrointestinal tract is the second most common site usually involving the ascending colon, sigmoid and rectum. Clinical manifestations range from asymptomatic to abdominal pain, fever, diarrhea, and rectal bleeding. However, diagnosis is often incidental as was the case with this patient. Early lesions appear yellow-tan in color, flat, and soft when manipulated. More developed lesions become gray-tan in color and raised with a central depressed area and peripheral hyperemia.

Diagnosis is made on biopsy with characteristic von Hansemann cells which are aggregates of histiocytes with eosinophilic cytoplasm. Intracytoplasmic Michaelis-Gutmann bodies are pathognomonic and described as laminated inclusion bodies with phagosomes encrusted by calcium and iron salts. Further, when stained with periodic acid-Schiff and von Kossa calcium stains, they have a targetoid appearance.

The pathogenesis of malakoplakia is unclear, however possible pathogenic mechanisms include a causative organism, altered immune response, and phagocyte disfunction. The most susceptible include immunosuppressed patients who are post-transplant or have an untreated systemic disease such as AIDS and chronic hepatitis. Therefore, treatment includes antibiotic therapy, immunotherapy modification, and surgical resection. Trimethoprim-sulfamethoxazole and quinolones are preferred due to their high macrophage concentration and ability to penetrate host macrophages.

While most cases of malakoplakia are diagnosed incidentally on screening colonoscopy, there does exist an association with colorectal adenocarcinoma. Zhang et al. published one of the largest case series of 26 patients with 19% being simultaneously diagnosed with colorectal cancer. Therefore, the finding of malakoplakia should prompt a workup for systemic infections, immune compromising diseases, and malignancy.

References

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