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## ENDOSCOPIC IMAGE OF INTEREST

### COLORECTAL INVOLVEMENT OF KAPOSI'S SARCOMA IN A HIV-NEGATIVE CASE

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A case of Kaposi's sarcoma (KS) in a 70-year-old man who was using corticosteroid for the treatment of asthma is presented. KS lesions occurred in the skin, colon, and rectum. Macroscopic appearances of the lesions varied from polypoid, hemorrhagic mucosal nodules and ulcers to red macules in the mucosal plane to plaque-like indurations of the wall. As the case was HIV negative, it is believed that KS developed due to corticosteroid-induced immunosuppression.

**Key words:** colorectal involvement, HIV negativity, Kaposi's sarcoma.

#### INTRODUCTION

Kaposi's sarcoma (KS) is an angioproliferative disease which originates from endothelial cells, myofibroblasts, and monocyte-macrophage cells.<sup>1,2</sup> Iatrogenic KS can be seen in patients under immunosuppressive treatment. Most iatrogenic KS patients are human herpesvirus 8 (HHV-8) positive prior to transplantation, suggesting reactivation of latent viral infection leading to disease.<sup>3–6</sup> We present a case of KS in a HIV-negative patient which occurred in skin, colon, and rectum following long-term immunosuppressive therapy for asthma.

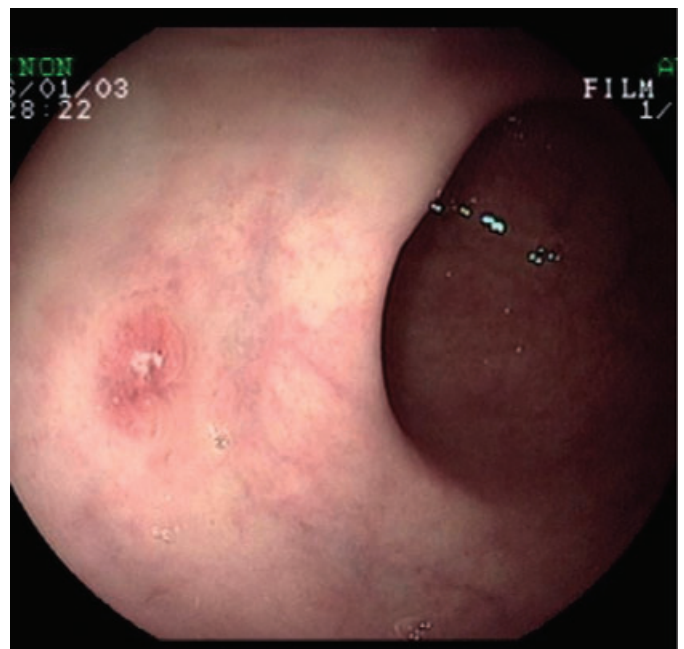
#### CASE REPORT

A 70-year-old man suffering from abdominal pain and rectal bleeding of 3 months duration was admitted to our hospital. Rectal bleeding after defecation which increased for the past month was remarkable. He had been treated for asthma for over 10 years. He used 20–40 mg/day methylprednisolone (at least six times/day) for 2–3 months due to asthma exacerbation. Upon physical examination, hyperemic macular and nodular lesions on his face, hands, back, and abdomen were detected. Colonoscopy revealed multiple polypoid lesions with or without central ulceration, varying 5–20 mm, from the rectum (Fig. 1) to the cecum (Fig. 2). Upper gastrointestinal system endoscopy was normal. Histopathologically, colon, rectum (Fig. 3), and skin biopsies showed that thin-walled, anatomizing vascular spaces were intermingled with spindle cells. Immunohistochemically, spindle cells were reactive for HHV-8 (clone: 13B10; 1:30; Novacastra, Newcastle, UK), CD34 (clone: QBend10; 1:200; Neomarkers, Fremont, CA, USA), and FVIII-RA (clone: F<sub>8-86</sub>; 1:150; Neomarkers) in skin and rectum. HIV serology was negative on enzyme-linked

immunosorbent assay examinations, and HIV negativity was confirmed with western blot. After medical oncology consultation, epirubicine 45 mg/week was added to his treatment. The patient died due to KS 4 months after the diagnosis.

#### DISCUSSION

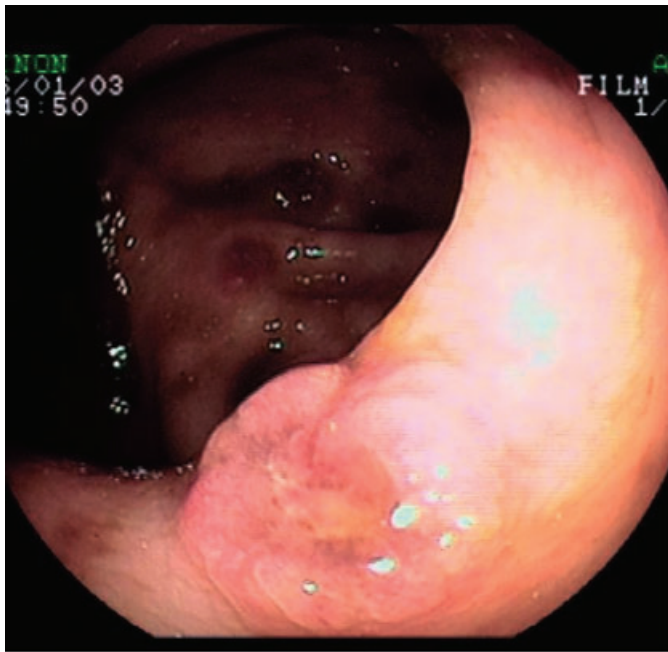
Gastrointestinal involvement of KS has been reported up to 40% of patients with AIDS.<sup>7</sup> Lower gastrointestinal tract involvement of KS is rare and is usually asymptomatic. In our case, colon and rectum involvement were remarkable with rectal bleeding during or after defecation.



**Fig. 1.** Nodular lesion in the rectum showing central erosion with surrounding hyperemia.

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**Fig. 2.** Cecum showing an ulceration of 1 cm in diameter with slightly raised reddish nodular lesions in the surrounding mucosa.

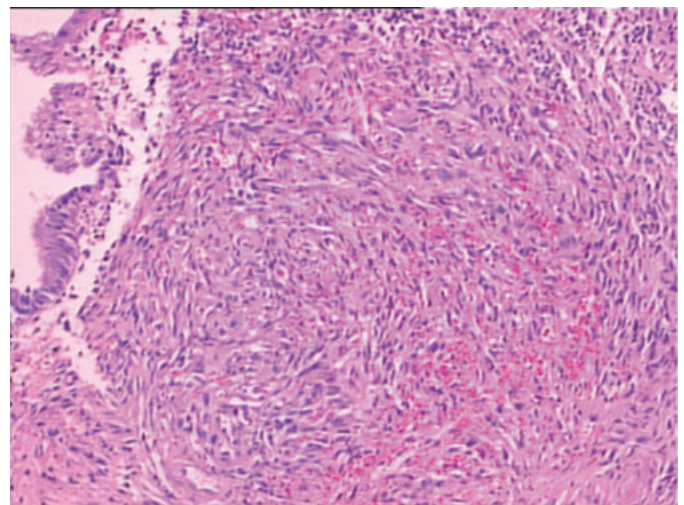
Endoscopic appearances can vary from red macules in the mucosal plane to plaque-like indurations of the wall to polypoid, hemorrhagic mucosal nodules and ulcers in KS.<sup>8,9</sup>

No evidence of correlation between the development of KS and dose or duration of steroid therapy has been detected.<sup>6</sup> Bursics *et al.* reported a case of ulcerative colitis who developed KS after steroid treatment with a dose of 12–125 mg/day for 4 months.<sup>10</sup>

In conclusion, immunosuppressive therapy can cause KS. In HIV-negative patients, latent HHV-8 virus can be reactivated and may lead to KS.

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**Fig. 3.** Rectum mucosa showing hemorrhagic vascular channels intermingled with spindle cell proliferation (H&E, ×200).

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