

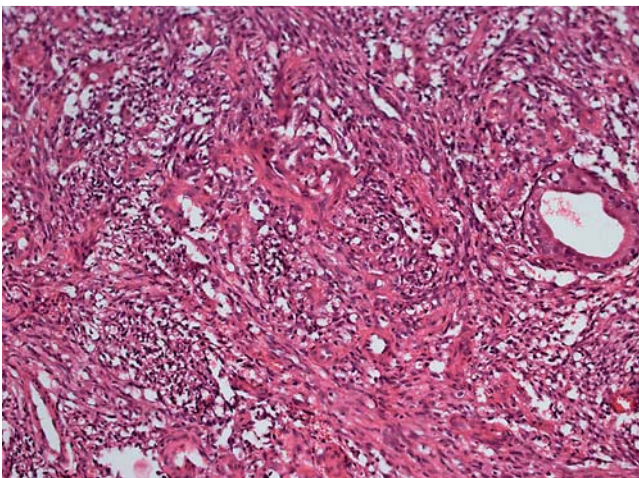
## Infectious colon polyp in patient with human immunodeficiency virus



**Fig. 1** High-definition white-light endoscopy revealed a 7-mm erythematous polypoid lesion at the level of the appendiceal orifice.



**Fig. 2** By using digital chromoendoscopy, the lesion was shown with a clear demarcation to the surrounding tissue and a small fibrinous erosion on the surface.



**Fig. 3** Histology revealed proliferating spindle cells infiltrating the lamina propria and slit-like vascular channels according to the diagnosis of Kaposi's sarcoma.

A 46-year-old Caucasian man was referred to our hospital because of weight loss and fatigue. He had been diagnosed with human immunodeficiency virus (HIV) infection 18 months earlier, and was under highly active antiretroviral therapy (HAART) with darunavir+ritonavir, emtricitabine, tenofovir disoproxil fumarate, and efavirenz.

Laboratory test data were within the normal ranges, and the viral load was under the detection limit. The total CD4 count was  $672 \text{ cells} \times 10^6/\text{L}$  (normal  $500 - 1200 \times 10^6/\text{L}$ ). Physical examination was unremarkable. High-definition white-light colonoscopy was unremarkable, except for a 7-mm erythematous polypoid lesion located in the cecum at the level of the appendiceal orifice (● Fig. 1). By using digital filter technology (i-scan; Pentax, Tokyo, Japan) during the endoscopic examination, the lesion became more obvious, and showed a clear demarcation to the surrounding tissue (● Fig. 2). Moreover, a small fibrinous erosion on the surface of the lesion became evident. Biopsies were taken. Histology revealed proliferating spindle cells infiltrating the lamina propria and slit-like vascular channels containing erythrocytes (● Fig. 3).

This patient suffered from colonic Kaposi's sarcoma. Antiretroviral therapy was changed to emtricitabine, tenofovir disoproxil fumarate, and lopinavir. However, because the patient developed severe diarrhea under lopinavir, the drug was changed to atazanavir+ritonavir. Follow-up colonoscopy 5 months later revealed a slight reduction in the size of the lesion. The patient remained clinically asymptomatic.

Kaposi's sarcoma is a vascular tumor that is caused by an opportunistic infection with human herpesvirus 8. Other potential factors for the development of Kaposi's sarcoma include altered expression and response to various growth factors and cytokines, and modulation of Kaposi's sarcoma growth by the Tat protein, which is an HIV gene product [1]. Whereas cutaneous and visceral Kaposi's sarcoma is quite frequent in patients with acquired immunodeficiency syndrome (AIDS), isolated colonic Kaposi's sarcoma is rare. Usually, Kaposi's sarcoma presents with skin lesions, but oral, nodal or, as in the present case, gastrointestinal Kaposi's sarcoma may precede cutaneous involvement.

Diagnosis should be secured by biopsies, as the differential diagnostic spectrum of lesions mimicking Kaposi's sarcoma is

wide, including non-Hodgkin lymphoma, bacillary angiomatosis, and fungal or bacterial infections. Other infectious causes of gastrointestinal pseudopolyps include *Mycobacterium tuberculosis*, histoplasmosis, blastomycosis (“Gilchrist’s disease”), and bartonellosis [2]. Patients with Kaposi’s sarcoma can present with a wide spectrum of symptoms, which could vary from an indolent process to a disseminated disease with a poor outcome.

Although Kaposi’s sarcoma is an opportunistic infection, it could still manifest in patients on HAART with a normal number of CD4 T-lymphocytes [3]. Therefore, the gastroenterologist evaluating patients with HIV/AIDS should carefully inspect the mucosa and should also not exclude the possibility that the gastrointestinal symptoms may be secondary to an opportunistic infection or tumor, even when the CD4 count is within the normal range and the viral load is low.

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**Competing interests:** None

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