

CLINICAL VIGNETTE

Kaposi's Sarcoma of GI Tract

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Case Report

A 30-year-old male with sickle cell trait and AIDS presented with abdominal pain and 7-pound unintentional weight loss in one month. He was diagnosed with HIV six years prior and was noncompliant with HARRT therapy. HIV viral load was >3000 copies/mL and CD4 count was 144 cells/mm³. Physical exam showed diffuse erythematous, violaceous lesions. Laboratory data included hemoglobin 5.7, platelet 80K, albumin 1.8, elevated alkaline phosphatase over 1K, and elevated ALT/AST. Punch skin biopsy confirmed Kaposi's sarcoma (KS). CT scan of abdomen and pelvis showed non-specific gallbladder wall thickening, and rectal wall thickening. EGD showed a large fungating mass described as polypoid, sessile, submucosal and ulcerated as well as partially circumferential oozing mass lesions throughout the stomach and duodenum. He was started on liposomal doxorubicin and rituximab given concern of KS cytokine syndrome. His diffuse body rash resolved and anemia slowly improved with each cycle of treatment. Unfortunately, he relapsed shortly after 6 cycles of treatment and was transitioned to hospice.

Discussion

Kaposi's sarcoma (KS) was first seen and described in 1872 by Dr. Moritz Kaposi involving skin and GI tract.¹ It is caused by HHV8 that initially affects the skin, but it can extend to mucous membranes and internal organs. At the beginning of the HIV epidemic, KS was one of the most frequent manifestations in up to 50% of immunocompromised patients.² The incidence of KS has dramatically decreased in both US and Europe with HAART.

KS has a variable clinical course ranging from very indolent to rapidly progressive disease. Gastrointestinal involvement is less frequent and can affect any part of the gastrointestinal tract. It grows from the submucosa, initially without any clinical symptoms. In untreated AIDS, the GI tract is involved in approximately 40%-51%, with only 20% of affected individuals having GI symptoms.³ With continued tumor growth, considerable variation in clinical presentation occurs including abdominal pain, nausea, vomiting, malabsorption, iron deficiency anemia, frank gastrointestinal bleeding, and rarely mechanical obstruction. The main risk factors that predict visceral involvement by KS include CD4 < 100 cells/mm³, viral load > 100,000 copies/mL, no previous use of HAART, and presence of skin involvement.⁴ Identification of HHV8 is

required by means of HHV8 DNA with sensitivity and specificity of almost 100%.

First-line treatment of KS of GI tract is administration of liposomal doxorubicin, with second line treatment with paclitaxel. In a retrospective study by Martin et al,⁵ the long-term prognosis of HIV-positive patients with KS treated with liposomal doxorubicin showed a complete response rate of 49%, partial response rate of 28%, and inadequate response of 14.5%. Despite favorable outcomes with dual HAART and liposomal doxorubicin treatment, the incidence of disease relapse is reported to be 13% per year with the majority in the first year after completing chemotherapy.⁵

KS is the most common GI tumor in patients with HIV. Even though HAART has reduced its incidence, clinicians should identify high-risk patients, when to screen for HHV8 infection for timely diagnosis, treatment, and avoid future complications.

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