

CASE REPORTS

Gastric varicella: two cases in cancer patients

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ABSTRACT

Gastric involvement with the varicella-zoster virus is an uncommon clinical condition where early suspicion and diagnosis are important to prevent the consequences deriving from its high morbidity and mortality, which in immunocompromised patients oscillate between 9% and 41% according to the various series. Two cases of gastric involvement with the varicella-zoster virus (VZV) in two patients with blood cancer are reported below. Gastric lesions are usually preceded by typical papulovesicular skin lesions. When gastric involvement is the first symptom of the disease its diagnosis and management may be delayed, which may entail severe consequences for immunocompromised patients. It is therefore that we suggest its inclusion in the algorithm for immunocompromised patients with abdominal pain and ulcer-like endoscopic lesions.

Key words: Gastritis. Varicela. Inmunosupresión.

INTRODUCTION

Gastric involvement with VZV is uncommon (1). The highest number of cases reported in the literature corresponds to immunocompromised patients after hematopoietic stem cell transplantation. Isolated VZV reactivation cases with gastric involvement have been reported in apparently immunocompetent patients (2) and in children, where a higher risk for VZV visceral spread has been described and immunocompromise is a common finding (3).

We report two cases of gastric involvement with VZV. The first one corresponds to an adult female with blood cancer (no transplant) and primary infection with VZV including the stomach, where typical skin manifestations developed later (2,4). This clinical picture has never been reported before.

CASE REPORTS

The first case refers to a 60-year-old woman who presented at the emergency room with epigastralgia and vomit-

ing for 5 days without fever or intestinal habit changes. Her history was remarkable for chronic lymphocytic leukemia, diagnosed in 2009 and initially treated with chemotherapy in 2011 (6 cycles of rituximab, fludarabine and cyclophosphamide until 2012), which resulted in complete remission. Physical examination only revealed deep epigastric tenderness without peritoneal irritation signs. Notable laboratory results include: normal renal function, Na 138 mEq/L, K 2.8 mEq/L, total bilirubin 1.3 mg/dL, ALT 138 IU/L, AST 91 IU/L, GGT 517 IU/L, normal AP and amylase, CRP 5.7 mg/L, CBC and coagulation panel without changes. Given the elevated transaminase levels a sonogram was performed on admission, which revealed a small amount of ascitic fluid around the gallbladder and at the flanks, the rest being normal. A gastroscopy was ordered because of persistent vomiting despite absolute dieting and fluid therapy, and multiple round ulcers with necrotic bottom, up to 1 cm in diameter, were found in the gastric antrum and body (Fig. 1). Biopsy samples were taken, revealing nonspecific changes in the absence of *H. pylori*, and a polymerase chain reaction (PCR) was ordered to rule out a viral etiology. On the fourth day after admission the patient developed a pruriginous papulovesicular skin rash in the face and trunk that was associated with improved abdominal pain and resolved vomiting. At this time a VZV serology was performed, which showed results consistent with primary VZV infection (IgM+, IgG+). An echoendoscopic procedure was performed on the 7th day after admission in order to control lesions and rule out extraluminal disease, which revealed ulcers in resolution with a fibrin bottom and no other abnormal findings. Finally, PCR was positive for VZV, confirming the condition's etiology.

The absence of skin lesions at disease onset and their late development delayed diagnostic suspicion and treatment initiation with acyclovir (10 mg/kg IV every 8 hours for 10 days), which was eventually dismissed since the clinical and endoscopic picture was nearly solved after symptomatic therapy at the time of diagnosis (Fig. 2).

Received: 30-07-2016
Accepted: 18-08-2016

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Sastre-Lozano VM, Martínez-García P, Torregrosa-Lloret M, Sánchez-Sánchez C, Sevilla-Cáceres L, Romero-Cara P, Morán-Sánchez S. Gastric varicella: two cases in cancer patients. *Rev Esp Enferm Dig* 2016;108(10):670-672.

DOI: 10.17235/reed.2016.3925/2015

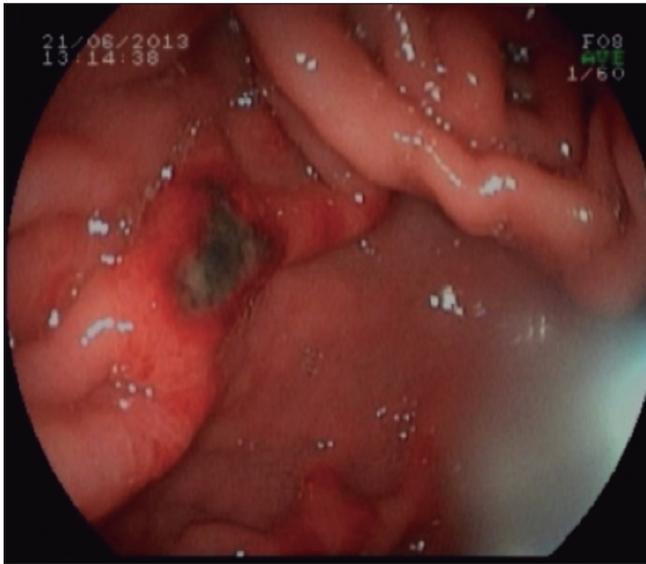


Fig. 1. Gastric body ulcer with a necrotic bottom and friable erythematous edges. The etiology was confirmed with a polymerase chain reaction positive for VZV.



Fig. 2. Echoendoscopy image obtained 7 days after symptom onset where healing ulcers that coincide with the resolution of the characteristic skin lesions may be seen.

The second case refers to a 52-year-old woman who presented at the ER in July 2013 with epigastralgia and vomiting of 3 days standing. Her history was notable for hypertension, right auricular herpes with VZV serology (IgG+, IgM-) in 2008, and peripheral non-Hodgkin T-cell Lennert's lymphoma (stage IVA) in 2010, which required specific chemotherapy with 8 CHOP (cyclophosphamide, doxorubicin, vincristin and prednisolone) cycles, rescue ESHAP (etoposide, methylprednisolone, cytarabine and cisplatin) for persistence, and then autologous peripheral blood transfusion with BEAM (carmustine, cytarabine,

etoposide and melphalan) conditioning prior to autologous hematopoietic stem-cell transplantation in November 2012. She received prophylaxis with acyclovir for VZV on the third month after transplantation. Physical examination revealed epigastric tenderness radiating to both flanks and papulous non-pruriginous lesions on her chest. Laboratory tests revealed elevated transaminases (ALT 244 IU/L, AST 185 IU/L, GGT 230 IU/L, AP 184 IU/L), with normal renal function, ions, bilirubin and amylase. CRP was 7 mg/L. CBC showed a pre-existing pancytopenia under follow-up by the Hematology service (Hb 11 mg/dL, Htc 31%, WBCs $1.76 \times 10^9/L$, platelets $22,000/mm^3$). An abdominal sonogram revealed no abnormalities, and a subsequent gastroscopy unveiled several erosions up to 7 mm in size with fibrin bottoms and no active bleeding in either the fundus or upper body, whence biopsy samples were taken (Fig. 3). Given a high suspicion for active VZV infection, therapy was immediately initiated with intravenous acyclovir for 10 days. Biopsy findings were also nonspecific and *H. pylori* was negative. The etiology of gastric lesions was confirmed as VZV using PCR.

DISCUSSION

VZV reactivation in adults usually takes place in immunocompromised settings. Late after hematopoietic stem-cell transplantation it involves 17%-50% of patients (4), more frequently in allogenic than in autologous transplants (5). Despite this, gastric involvement with VZV is an uncommon condition.

Gastric lesions are usually preceded by typical papulovesicular skin lesions (1). However, in the first patient reported gastric involvement and manifestations occurred first, and it was only later that skin lesions developed. This fact leads to a delayed diagnosis and treatment of



Fig. 3. Multiple ulcers with a fibrin bottom in the gastric body, secondary to VZV as confirmed by histology.

VZV infection, which entails high mortality rates among immunocompromised subjects (9%-41%) (6,7). Therefore, it is desirable that such infection be borne in mind and excluded in the presence of acute abdominal pain associated with gastric ulcers of unclear etiology.

VZV reactivation is relatively common 3-6 or more months after hematopoietic stem-cell transplantation (up to 70%) (6), hence prophylaxis with acyclovir is recommended in these patients. In spite of this, reactivation is no rare finding after prophylaxis completion (5).

As for the histology of gastric lesions, specific VZV infection signs may be observed, including eosinophilic inclusion bodies, cytoplasmic edema, and giant multinucleated cells (1). The condition should not be ruled out in the presence of nonspecific inflammation findings, and the study should be completed with a PCR, which will confirm the diagnosis.

Early diagnosis and treatment with intravenous acyclovir is essential because of the high morbidity and mortality of the condition, particularly in immunocompromised individuals. In the first case the absence of initial skin lesions, together with the patient's clinical and endoscopic improvement, prompted the decision to not administer antiviral therapy after VZV was confirmed by PCR. In the second instance acyclovir was initiated at diagnostic suspicion in the presence of skin lesions, the diagnosis being subsequently provided by PCR.

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