

CLINICAL NOTE

Herpetic esophagitis: a case report on an immunocompetent adolescent

Susana de-la-Riva¹, Miguel Muñoz-Navas¹, Iago Rodríguez-Lago¹, Juan Carrascosa¹, Miguel Ángel Idoate² and Rafael Carias²

¹Department of Gastroenterology. Endoscopy Unit. ²Department of Pathology. Clínica Universidad de Navarra. Pamplona, Spain

ABSTRACT

Herpetic esophagitis in immunocompetent individuals is a rare entity that should be suspected clinically by an acute onset of symptoms, and without apparent cause of a symptomatic triad consisting on odynophagia, heartburn and fever.

Its occurrence may be due to reactivation of a previous infection or less often a primary infection. Herpes simplex type 1 is the most common cause.

Upper endoscopy establishes the diagnosis of suspicion of herpetic esophagitis. It also allows to take multiple biopsy samples and viral culture, leading to a definitive diagnosis.

The severity of symptoms is related to the degree of oesophageal involvement. In immunocompromised patients treatment is indicated with acyclovir, but the indication in immunocompetent patients is controversial because the process is time, limited with a low probability of complications.

We present a case of acute herpetic esophagitis in an immunocompetent host that debuted acutely with severe upper gastrointestinal tract symptoms, associated with an insidious and non-specific onset of flu-like symptoms. Endoscopic findings showed a severe involvement in the lower third of the oesophageal mucosa.

Key words: Herpetic esophagitis. Immunocompetent host. Adolescent. Aciclovir.

De la Riva S, Muñoz-Navas M, Rodríguez-Lago I, Carrascosa J, Idoate M, Carias R. Herpetic esophagitis: a case report on an immunocompetent adolescent. *Rev Esp Enferm Dig* 2012; 104: 214-217.

Received: 03-10-11.
Accepted: 06-10-11.

Correspondence: Susana de la Riva Onandia. Department of Gastroenterology. Clínica Universidad de Navarra. Avenida Pío XII, 36. 31008 Pamplona, Spain. e-mail: Sdelariva@unav.es

INTRODUCTION

Herpetic esophagitis caused by the herpes simplex virus (HSV) is a common opportunistic infection in immunocompromised patients (as HIV), immunosuppressed or affected by severe systemic diseases such as cancers and large burns. However, the infection in immunocompetent patients is an uncommon condition rarely reported in the literature and most often affecting young adults (1).

It usually presents with the development of unexplained acute and intense odynophagia, dysphagia, chest pain, fever, and, in 23% of all the cases, respiratory symptoms (cough) are present (2). Prodromal symptoms are often nonspecific and associated with malaise. Sometimes orolabial herpetic lesions are observed at the same time (3,4).

The diagnosis of herpetic esophagitis is established by compatible histological findings and a positive culture of the samples obtained by upper endoscopy. In the case of primary infection, a seroconversion is also diagnostic (1).

The initial clinical suspicion allows for a prompt diagnosis and the establishment of the correct therapeutic measures to prevent an insidious course of the disease and its complications (2,3).

We report a case of herpetic esophagitis in a young immunocompetent patient, who debuted acutely with severe symptoms from the upper gastrointestinal tract associated at the start with flu-like symptoms.

Upper endoscopy showed a significant and striking involvement of the mucosa in the lower third of the esophagus with the final diagnosis of herpetic esophagitis.

CASE REPORT

A 16-year-old male patient referred severe odynophagia and chest pain during the last 15 days that worsened with deep inspiration. Anorexia was also present. He had suffered multiple episodes of food impaction at medium-low thoracic

level that required the induction of emesis for resolution. About 72 hours before the onset of esophageal symptoms, he developed fever until 38.9 °C and myalgias, attributed to a flu-like syndrome, that was absent at the time of consultation. No previous history of typical symptoms of gastroesophageal reflux disease (GERD), food, environmental or drug allergies were reported. He recovered in childhood from chickenpox and herpes zoster at 15 years of age.

On physical examination there were no remarkable findings. Biochemical blood analyses were normal, including inflammatory parameters (ESR and CRP). Only a leukocyte count of 7,100 10⁹/L was remarkable on complete blood count.

An upper endoscopy was performed because of the severity of the symptoms. It showed multiple ulcerations in the middle and distal third of the esophagus with well defined borders, moderate depth and longitudinal orientation that coalesce and became deeper in the distal esophagus (Fig. 1). Brushing cytology, biopsy and viral culture were performed endoscopically.

Cytology findings were compatible with acute ulcer. Histological examination was suggestive of herpetic ulcerative esophagitis with visualization of cytomegalic, multinucleated keratinocytes with nuclear membrane reinforcement and presence of inclusion bodies (Fig. 2). Cultures were positive for HSV type 1 (Shell-Vial technique).

A diagnosis of herpetic esophagitis secondary to HSV type 1 in an immunocompetent patient was established. Associated immunodeficient processes were ruled out by normal immunoglobulin levels, negative HIV serology and normal CD4/CD8 count.

Symptoms persisted, so it was decided to initiate intravenous antiviral therapy with acyclovir 5 mg/kg for 5 days and subsequent oral valaciclovir. The patient recovered well and was discharged after 5 days.

DISCUSSION

Herpetic esophagitis in immunocompetent individuals is a rare entity that should be suspected clinically by the acute onset, and without an apparent cause, of a triad consisting on odynophagia, heartburn and fever.

As it is a rare entity, the diagnosis should be made after carefully exclude other pathologies that involve similar endoscopic images (mainly peptic esophagitis and cytomegalovirus esophagitis) and immune disorders.

The primary source of infection is discussed and seems to differ depending on the state of the patient's immune system. While in immunocompromised patients it is usually secondary to virus reactivation, in immunocompetent subjects is probably due to local extension of the virus from an orolabial or pharyngeal source (1).

About its pathogenesis in immunocompetent patients it has been postulated a theory of the contact of an esophageal eroded mucosa with contaminated saliva by the virus. The mucosal erosion mechanism can be associated with GERD (5) or esophageal instrumentation (6). This theory has been sustained over time but has not been confirmed in most clinical cases reported in the literature. Another hypothesis is the transmission by direct contact with a family member with herpes simplex skin lesions. Previous reported case series have found this risk factor in approximately 20% of affected patients (2-4,7).

The symptoms previously published in the literature describe an acute onset of esophageal complaints as chest pain (46.4%), odynophagia (60.7%), dysphagia for both solids and liquids (37.5%), heartburn and/or vomiting (8). Prior to the upper digestive symptoms described, a clinical course that ranges from non-specific flu-like symptoms to a temperature of 39 °C with malaise, anorexia and weight loss can be seen.

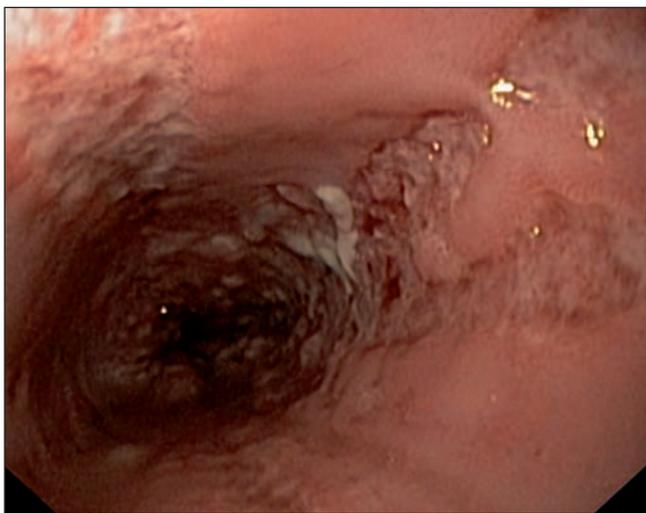


Fig. 1. Multiple ulcerations in the middle and distal third of the esophagus with well defined borders, moderate depth and longitudinal orientation that coalesce and became deeper in the distal esophagus.

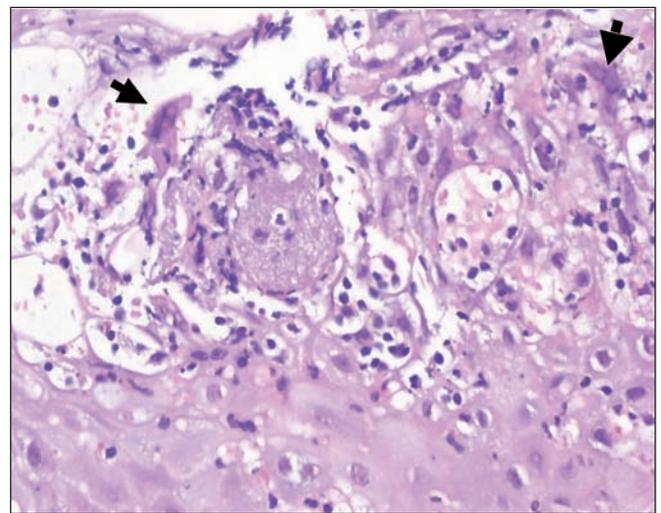


Fig. 2. Histological examination with visualization of cytomegalic, multinucleated keratinocytes with nuclear membrane reinforcement and presence of inclusion bodies (H&E, x200).

Endoscopic findings allow us to make a differential diagnosis. In addition it also allows taking multiple samples for histology and viral culture, leading to a definitive diagnosis (9). Gastroduodenoscopy findings are similar in all the cases described in the literature regardless of the immunity of the affected patient. The most common images are the presence of a friable esophageal mucosa with multiple coalescent ulcerations of variable size and depth with a volcano-like appearance (3). The most common site of herpetic esophageal lesions is the middle and distal third of the esophagus (83%). In 15% of all cases it may affect the entire esophagus. In the remaining 2% the stomach is involved concurrently (10). Possible complications of esophageal involvement are rare but severe: upper gastrointestinal bleeding (5.3%) (3,9,10) and esophageal perforation (1.8%) (9,12).

Occasionally the coalescence of ulcers in the lower third of the esophagus may resemble a severe peptic esophagitis. This is the reason why it is important to have an adequate clinical suspicion to guide the biopsy and cultures of the esophageal mucosa (3).

The diagnosis of herpetic esophagitis is made by histological examination (62%) and virologic culture (58%) of esophageal biopsies. Histology is characterized by the presence of multinucleated giant cells with eosinophilic intranuclear inclusions called Cowdry type A inclusions (3,9). Another supplementary diagnostic method is serology testing, which allows in some cases to determine the primary infection detected after seroconversion.

Once diagnosed, the indicated treatment is acyclovir. In the immunosuppressed patient administration is accepted and established. The use of acyclovir in the immunocompetent patient is controversial since it is often a self-limited process that usually last for one to two weeks and only exceptionally may be complicated by upper gastrointestinal bleeding and esophageal perforation (4,9). The treatment experience in this population is limited, as it is a rare entity and there are no randomized controlled trials published. How-

ever, clinical experience suggests that early initiation of acyclovir treatment also promotes the prompt resolution of symptoms and helps to prevent further complications (13).

REFERENCES

1. Genereau T, Rozenberg F, Bouchaud O, Marche C, Lortholary O. Herpes esophagitis: a comprehensive review. *Clin Microbiol Infect* 1997;3:397-407.
2. Geraci G, Pisello F, Modica G, Li Volsi F, Cajozzo M, Sciumè C. Herpes simplex esophagitis in immunocompetent host: a case report. *Diagn Ther Endosc* 2009;2009:717183.
3. Kato S, Yamamoto R, Yoshimitsu S, Shimazaki K, Ogawa S, Itoh K, et al. Herpes simplex esophagitis in the immunocompetent host. *Dis Esophagus* 2005;18:340-4.
4. Ramanathan J, Rammouni M, Baran J, Jr., Khatib R. Herpes simplex virus esophagitis in the immunocompetent host: an overview. *Am J Gastroenterol* 2000;95:2171-6.
5. DiPalma JA, Brady CE. Herpes simplex esophagitis in a nonimmunosuppressed host with gastroesophageal reflux. *Gastrointestinal Endoscopy* 1984;30:24-5.
6. Pazin GJ. Herpes simplex esophagitis after trigeminal nerve surgery. *Gastroenterology* 1978;74:741-3.
7. Galbraith JC, Shafran SD. Herpes simplex esophagitis in the immunocompetent patient: report of four cases and review. *Clin Infect Dis* 1992;14:894-901.
8. Canalejo Castrillero E, Garcia Duran F, Cabello N, Garcia Martinez J. Herpes esophagitis in healthy adults and adolescents: report of 3 cases and review of the literature. *Medicine* 2010;89:204-10.
9. Domínguez Alcón L, Pita Fernández L, Carretero L. Herpes simple esophagitis in immunocompetent adult. *Rev Esp Enferm Dig* 2009;101:368-9.
10. Al-Samman M, Zuckerman MJ, Verghese A, Boman D. Gastric ulcers associated with herpes simplex esophagitis in a nonimmunocompromised patient. *J Clin Gastroenterol* 1994;18:160.
11. Nagri S, Hwang R, Anand S, Kurz J. Herpes simplex esophagitis presenting as acute necrotizing esophagitis ("black esophagus") in an immunocompetent patient. *Endoscopy* 2007;39(Supl.1):E169.
12. Cronstedt JL, Bouchama A, Hainau B, et al. Spontaneous esophageal perforation in herpes simplex esophagitis. *Am J Gastroenterol* 1992;87:124-7.
13. Kurahara K, Aoyagi K, Nakamura S, Kuwano Y, Yamamoto C, Iida M, et al. Treatment of herpes simplex esophagitis in an immunocompetent patient with intravenous acyclovir: a case report and review of the literature. *Am J Gastroenterol* 1998;93:2239-40.