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Case Report

CASE REPORT

Herpes simplex esophagitis in an immunocompetent host: a case report

Binit Upadhaya Regmi[1], Ram Chandra Subedi[1,*], Sujit Kumar Mandal[1], Shova Kunwar[2] and Binod Karki[1]

[1]Department of Medicine, Shree Birendra Hospital, Nepalese Army Institute of Health Sciences, Kathmandu, Nepal, [2]Department of Pathology, Shree Birendra Hospital, Nepalese Army of Institute of Health Sciences, Kathmandu, Nepal

*Correspondence address. Ram Chandra, R.C., Subedi, Department of Medicine, Shree Birendra Hospital, Nepalese Army Institute of Health Sciences, Chhauni, Kathmandu, Nepal. Tel: +9779851279838; E-mail: manohar.rcs@gmail.com

Abstract

Esophagitis caused by Herpes Simplex virus is a well-recognized opportunistic infection in the immunocompromised or severely ill host. However, it is uncommon in otherwise immunocompetent host. It usually responds well to a course of acyclovir. We report a case of young female without any other immunocompromised state who presented with severe dysphagia. She was diagnosed endoscopically and later with histopathology and recovered well after a course of acyclovir.

INTRODUCTION

Esophagitis caused by Herpes Simplex virus (HSV) is a well-recognized opportunistic infection in the immunocompromised or severely ill host [1–3]. As an acute, self-limiting illness herpes simplex esophagitis (HSE) can occur occasionally in otherwise immunocompetent host with no other underlying immunological problems [1, 4]. In the past most of the HSE cases were diagnosed at post-mortem examination of immunocompromised or severely ill persons [5, 6]. Due to its self-limiting nature and coexistence with reflux symptoms HSE in immunocompetent host may go undiagnosed [1, 4]. However, due to recent advancement in diagnostic techniques many cases of HSE in healthy individuals has been reported [4]. Here, we present an immunocompetent female with HSV esophagitis who had a dramatic response to acyclovir therapy.

CASE REPORT

A previously healthy 55-year-old female presented with dysphagia of acute onset along with marked odynophagia. She had history of fever and sore throat a week before for which she was treated with oral co-amoxiclav and paracetamol at the local health facility. Dysphagia was present both for solid and liquids. There was no history of use of any steroids or immunocompromising drugs. She had no history of smoking or alcohol use. Also, there was no personal or family history of antecedent HSV exposure. On examination, her vitals were normal. Her general and systemic examination findings were within normal limits. Her oral examination revealed no ulcer or blisters. During the initial evaluation of fever, her investigations for etiologies of fever including Malaria, Dengue, Typhoid, Leptospira, Scrub typhus were unremarkable. Reverse-transcription polymerase chain reaction (RT-PCR) for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) was done which was negative. Sonological examination of abdomen was unremarkable.

Serology for Human Immunodeficiency virus I and II (HIV I/II), Hepatitis B surface antigen (HBsAg) and antibody to Hepatitis C virus (anti-HCV) were nonreactive. Upper gastrointestinal (UGI) endoscopy was done which revealed multiple ulcers with hemorrhages especially in the middle and distal third of esophagus (Fig. 1). The gastroesophageal junction, stomach

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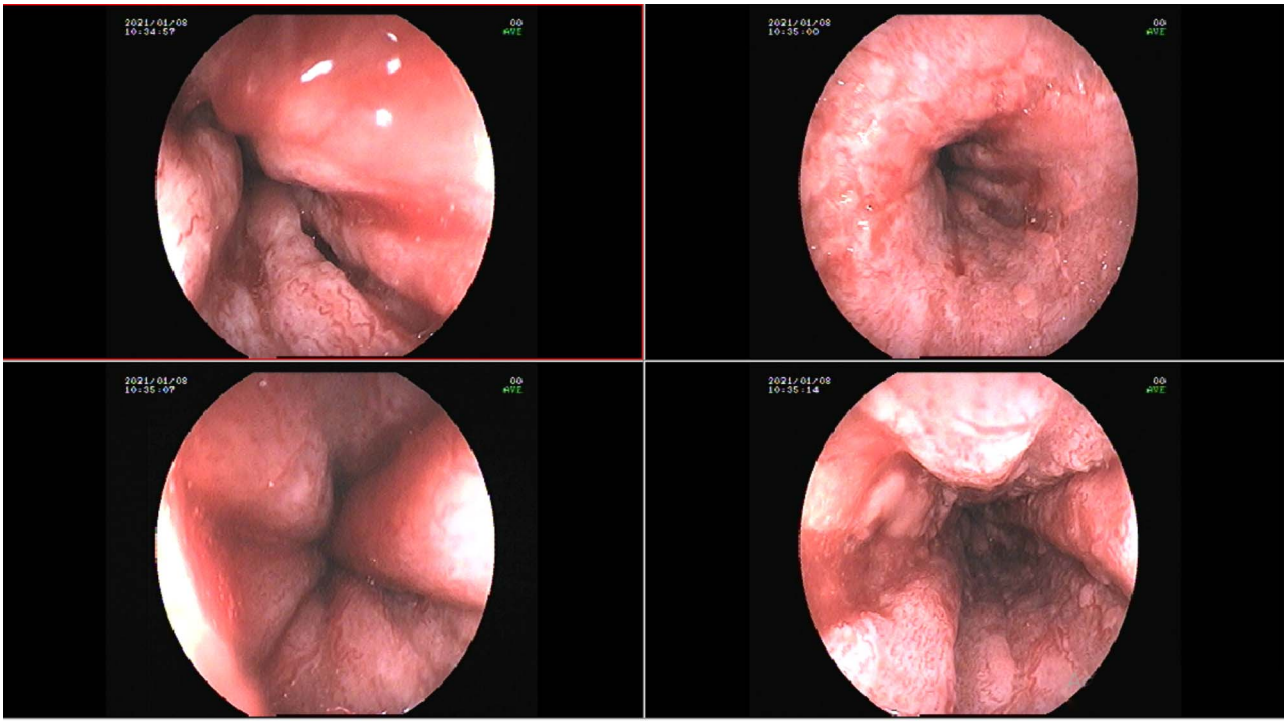

Figure 1: Endoscopic examination of herpes esophagitis showing multiple ulcers with hemorrhages especially in the middle and distal third of esophagus.

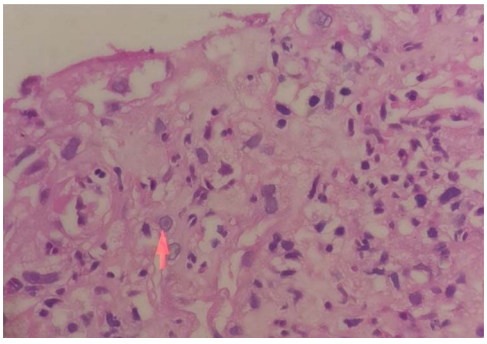

Figure 2: Histological features of herpes esophagitis showing Cowdry type A intranuclear inclusion bodies in infected epithelial cells (H&E, ×200, closed arrow).

and duodenum were normal. Multiple biopsies were taken from the ulcerated area for histopathology. Her histopathology report revealed viable squamous epithelium cells around ulcerative margin showing eosinophilic round intranuclear inclusion bodies (Cowdry type A inclusion bodies) separated by clear zone from thickened nuclear membrane features. There was no evidence of atypia or malignancy (Fig. 2). These findings were suggestive of HSE. Serology done for HSV I/II IgG level was 6.30 AU/ml (reference range: < 2 AU/ml) i.e. raised above normal range. With these, the diagnosis of HSE was made and she was started on intravenous acyclovir for initial 5 days. Later acyclovir was continued orally for total 10 days. Upper GI endoscopy was repeated after 7 days showed marked improvement in the ulcers with most of them already healing. She was discharged on oral acyclovir and sucralfate suspension. At the time of discharge, she could take orally well and her symptoms of dysphagia and odynophagia have almost disappeared. On follow-up after two weeks she was free from all symptoms and tolerating orally.

DISCUSSION

HSV is a DNA virus which can cause wide range of disease from inapparent to fatal illness. The site of involvement, immune status of host and whether the disease is primary or recurrent are the determinant of severity of infection [3]. The most common visceral organ infected by HSV is esophagus [3]. HSE usually results from reactivation of HSV and spread of virus to esophageal mucosa via vagus nerve or direct extension of oropharyngeal infection into esophagus [7]. A study conducted by Canalejo et al. [1] showed 44% patient had primary infection on the basis of serology.

HSE commonly occurs in persons with clinical evidence of immunosuppression but several cases of HSE in immunocompetent host have been described in literature [3, 8]. The common presentation in HSE is odynophagia, dysphagia and fever [9]. Reported cases of HSE were diagnosed by various methods such as endoscopy, blood test of IgM and IgG for HSV I/II, viral tissue culture and viral DNA PCR. The gold standard for diagnosing the infection is obtaining biopsy from esophageal mucosa with microscopic evaluation [10]. Acyclovir 400 mg orally five times a day for 14 to 21 days is standard treatment for immunocompromised host in HSE. Spontaneous resolution usually occurs in immunocompetent hosts after 1–2 weeks even without treatment. However, the patient may respond quickly if treated with a short course of oral acyclovir.

In this case, the patient was immunocompetent without any history of immunosuppressive drugs. There was neither history of previous herpetic infection nor evidence of herpes labialis or oropharyngeal ulcer on examination. The diagnosis was based on endoscopic finding and histopathological

examination of sample obtained from lesion in esophageal mucosa during endoscopy. The typical findings of upper GI endoscopy are multiple mucosal ulcers which are well circumscribed and have a 'volcano-like' appearance. Yield is better if biopsies or brushing are taken from the edge of an ulcer where viral cytopathic effects are most likely to be present. In our patient, the lesions of esophagus were not typical as described however the histopathology and serology for HSV supported the diagnosis. Viral culture, and DNA PCR for HSV could not be performed because of nonavailability. There was quick response to the treatment with acyclovir.

HSE should be suspected in a healthy patient with acute onset of dysphagia or odynophagia with typical ulcerations in the upper Gi endoscopy. Endoscopy should be performed in these patients with symptoms of esophagitis and biopsy should be taken to confirm the diagnosis. With the confirmation of HSE careful history taking should be obtained to rule out any immune disorder such as HIV. Although HSE in immunocompetent host is self-limiting use of acyclovir may hasten the recovery.

ETHICAL APPROVAL

No ethical approval was needed for this case report.

CONSENT

Written informed consent was obtained from the patient for the publication of this case report and accompanying images. The consent is available for the review.

GUARANTOR

Binod Karki, DM Gastroenterology.

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CONFLICTS OF INTEREST

No conflict of interest.

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