A Case of Postoperative Herpes Simplex Pneumonia

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Herpes simplex virus (HSV) esophagitis is a rare disease and most of cases are reported in the immunocompromised patients. We report a case of fatal HSV infection started from herpes labialis and esophagitis. She initially suffered from perioral ulcer, esophagitis, and interstitial pneumonitis later and eventually died of respiratory insufficiency one month later after the aneurysmal surgery. She did not have any immunocompromised medical history and also had no evidence of herpes encephalitis in laboratory study and magnetic resonance image (MRI). With the availability of effective agents for the treatment of HSV infection, early recognition of HSV esophagitis is important because it may be clinically confused with Cushing ulcer and may be complicated with a fatal pneumonitis.

KEY WORDS: Herpes simplex virus infection · Esophagitis · Pneumonitis.

Introduction

Herpes simplex virus (HSV) esophagitis and pneumonitis can rarely occur in immunocompetent patients. They are difficult to diagnose with non-invasive method and have high mortality rate. Otherwise, several cases of them have been reported as a self-limiting illness in immunodepressant patients. We report a case of herpes simplex esophagitis progressed a fatal herpes simplex pneumonitis after the subarachnoid hemorrhage (SAH).

Case Report

A sixty-two-year-old woman underwent aneurysmal surgery of middle cerebral artery, after admission to our hospital due to severe headache and drowsy consciousness. There were no specific medical problems. The brain computed tomography (CT) revealed SAH with diffuse high density in the both Sylvian fissures and basal cistern. Because of right middle cerebral artery (MCA) vasospasm on 10th days after operation, angioplasty was performed with papaverine. General as well as neurological conditions were improved afterward (Fig. 1A, B, C).

On 10th postoperative day, she developed perioral ulcer (Fig. 2A). On 13th postoperative day, high fever and leukocytosis were noted. Chest X-ray showed minimally pleural effusion on right lower lung field (Fig. 2B). Enterococci and Candida were cultured in urine and Methicillin-resistant coagulase-negative staphylococci were cultured in sputum, and sputum smears for AFB were negative. Because of urinary tract infection and pneumonia, antibiotic treatments were initiated. On 15th postoperative day, she showed odynophagia and he-

![Fig. 1. A: The brain computed tomography (CT) showing diffuse subarachnoid hemorrhage (SAH) in both Sylvian fissures. B: The right internal carotid angiography revealing a middle cerebral artery aneurysm (arrow). C: The brain CT on 10th postoperative day demonstrating aneurysmal clip and resolution of SAH.](image-url)
matemesis with burning substernal discomfort. There was
diffusely spread ulceration from upper esophagus to lower
esophagus on the gastric endoscopy and biopsy (Fig. 2C).
These symptoms were aggravated with diffuse spread esopha-
geal ulcer 4 days later (Fig. 2D). The result of biopsy was re-
ported as a herpetic esophagitis with ground glass nuclear change and mul-
tinucleation of squamous epithelium (Fig. 3A, B).

On 34 weeks after operation, her condition was aggravated and br-
anchoscopy with multi-lobar bronchial alveolar lavage (BAL) was
performed to determine causative organism. At the cytologic study
of bronchial washing, no typical findings of HSV infection were
observed but viral culture by the inoculation of BAL fluid was re-
sulted in the growth of HSV type 1. Although intravenous acyclovir
was given, she died on 32nd post-
operative day due to acute respira-
tory distress syndrome by HSV pneumonia (Fig. 4A). There was
no evidence of herpetic encephalitis
on the brain magnetic resonance
image (MRI) and viral culture of ceb Robbins fluid (Fig. 4B, C).

Discussion

HSV is enveloped, linear, dou-
able-stranded DNA viruses,
whose only known hosts are human beings. HSV is common patho-
gen invading the oropharyngeal mucosa, gastrointestinal (GI) tract,
genitalia and the skin. HSV may produce a wide range of disease
from unapparent infection to fatal illness. Immuno compromised
individuals often develop more severe HSV infections, similar to other
opportunist infections. Recurrent outbreaks occur with greater fre-
quency, and lesions may be extensive or persistent. Multiple large, chro-
ic, necrotic, or hyperkeratotic ulcers can form in atypical locations.
It can cause fatal respiratory and
esophageal infection in patients with burns, organ transplantation, malignancy, immunosuppressive therapy, and prolonged intubation. However, HSV pneumonia and esophagitis rarely occurs in immunocompetent patients. Herpes esophagitis was first reported by Johnson in 1940. The incidence of herpes esophagitis has been reported to be 1.8% in autopsy patients. Almost all cases were immunocompromised hosts. Chief complaints of these patients were odynophagia, dysphagia, retrosternal pain, herpetic skin and oral lesions as well. Although herpes esophagitis in an immunocompetent host is self-limiting, it is occasionally a source of disseminated infection in the susceptible host. The diagnosis of HSV esophagitis is usually established with an esophagoscope. It also permits acquisition of samples for histology, culture, and polymerase chain reaction (PCR). Endoscopic appearance of HSV esophagitis depends on how long the infection has been present. Vesicles are the earliest lesions, seen predominantly in the mid-to-distal esophagus. Characteristic sharply demarcated ulcers that may have surrounding erythema may also be present. Inflammatory exudates may develop. The endoscopic appearance can be confused with esophagitis secondary to candida or peptic disease. Effective diagnostic methods are histological study and culture of esophageal biopsies. Ground-glass appearance in the nuclei, Cowdry type A nuclear inclusion bodies and multinuclear cells are distinctive features of herpes esophagitis. Herpes esophagitis may be a early sign of disseminated visceral HSV infection. It is easy to confuse the simple and common condition like HSV type I and Cushing. For this reason, the treatment of our case was delayed. Antiviral treatment with acyclovir for herpes esophagitis in immunocompetent hosts has been reported to be effective. Pamuk et al. reported that antiviral therapy for stomatitis and herpes esophagitis is necessary. Cushing ulcer (so-called stress ulcer) is often defined as acute gastroduodenal mucosal lesions following shock, trauma, ingestion of alcohol and in the course of severe, chronic medical illness as well as in neurosurgical patients.

Morgan, who cultured HSV from the lung tissue of the patient with severe burns, reported HSV pneumonia in 1949. HSV pneumonia is difficult to diagnose with non-invasive method because the diagnosis of HSV pneumonia must depend on histologic evidence of pneumonia associated with viral isolation from the lung. Ramsey et al. reported that the localized HSV pneumonia was usually preceded by oral mucosal HSV infection, often associated with herpetic tracheitis or esophagitis. We considered that a fatal herpes pneumonia was complicated by herpes simplex esophagitis and labialis, and thus the early diagnosis and treatment might have been helpful to avoid mortality.

**Conclusion**

Early recognition of HSV esophagitis is important because it might confuse with more benign Cushing ulcer but may progress a fatal condition in neurosurgical patients.

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**References**