CASE REPORT

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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[2,3,11,15,17,18]. We report a case of fatal HSV infection started from herpes labialis and esophagitis. She had 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[2,3,11,15-19]. 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 immuno-depressant patients[2,3,11,15,17,19]. 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-

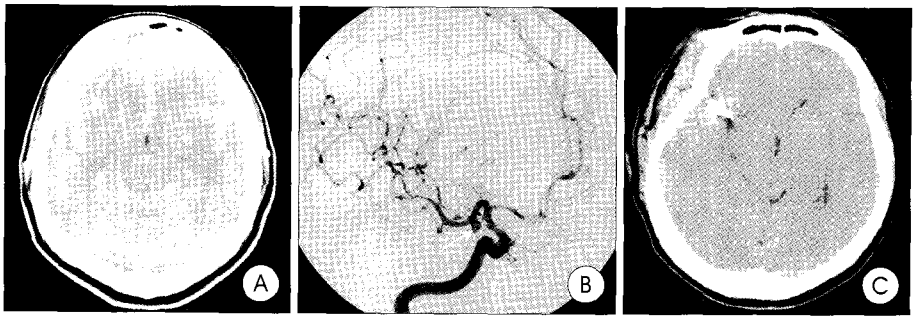

Fig. 1. A : The brain computed tomography (CT) showing diffuse subarachnoid hemorrhage (SAH) in both Sylvian fissures. B : The right internal carotid angiography revealing a middle cerebral artery aneurysm (arrow). C : The brain CT on 10th postoperative day demonstrating aneurysmal clip and resolution of SAH.

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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 esophageal ulcer 4 days later (Fig. 2D). The result of biopsy was reported as a herpetic esophagitis with ground glass nuclear change and multinucleation of squamous epithelium (Fig. 3A, B).

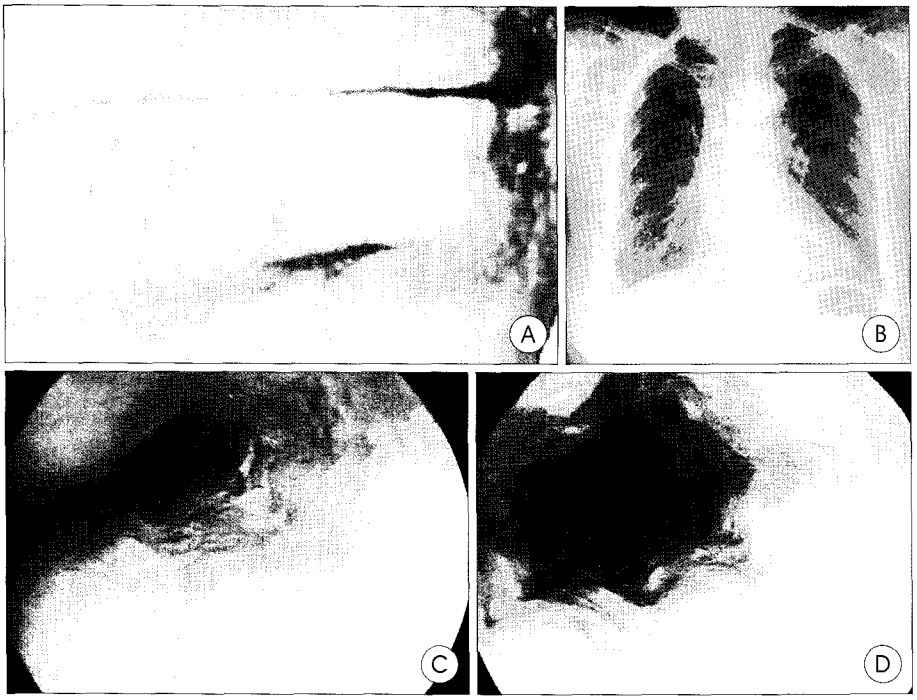

Fig. 2. A : Perioral ulcerative lesion seen on 10 days after operation. B : The plain chest film on 13th postoperative day shows pleural effusion on right lower lung. C : The endoscopic finding on 15th postoperative day shows multiple coalescent ulcerations from upper esophagus to lower esophagus. D : The endoscopic finding on 20th postoperative day showing diffuse esophageal ulcer.

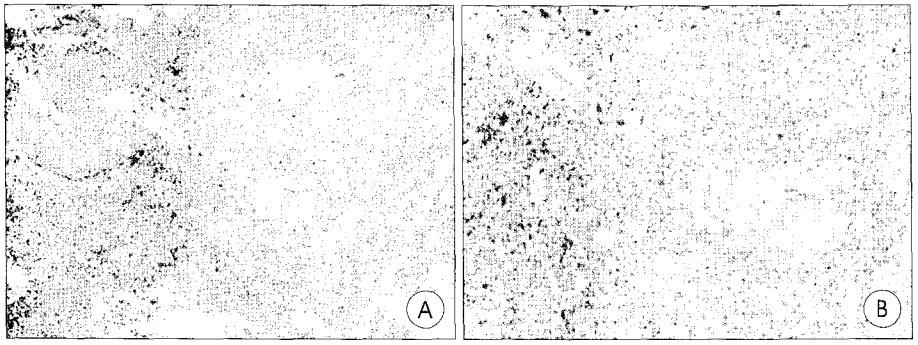

Fig. 3. A : Esophageal tissue histopathologic findings revealing rich in lymphocytes, epitheloid histiocytes and showing loss of overlying squamous epithelium with necrotic debris remaining on the ulcer base at the left (Micro, H&E, ×100). B : The high power view demonstrating ground-glass nuclear change and multinucleation of squamous epithelium (Micro, H&E, ×400).

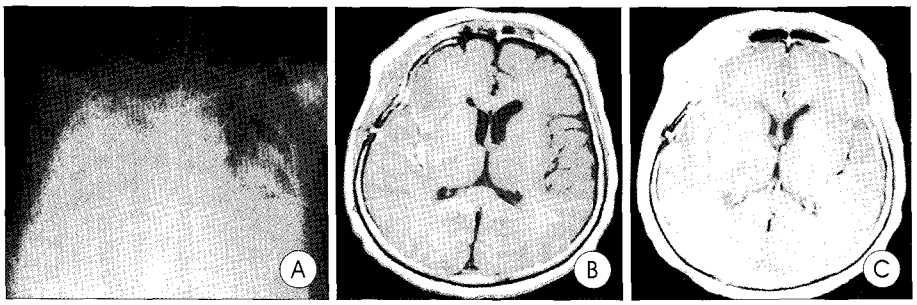

Fig. 4. A : The plain chest film at 28th postoperative day shows marked haziness in both lung zones. B, C : The brain MRI shows the high signal intensity with enhancement around Sylvian fissure but do not coincide typical herpes encephalitis.

On 3rd weeks after operation, her condition was aggravated and bronchoscopy 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 resulted in the growth of HSV type I. Although intravenous acyclovir was given, she died on 32nd postoperative day due to acute respiratory distress syndrome by HSV pneumonitis (Fig. 4A). There was no evidence of herpetic encephalitis on the brain magnetic resonance image (MRI) and viral culture of cerebrospinal fluid (Fig. 4B, C).

Discussion

HSV is enveloped, linear, double-stranded DNA viruses, whose only known hosts are human beings[22]. HSV is common pathogen invading the oropharyngeal mucosa, gastrointestinal (GI) tract, genitalia and the skin[8,12,17,22]. HSV may produce a wide range of disease from unapparent infection to fatal illness. Immunocompromised individuals often develop more severe HSV infections, similar to other opportunistic infections. Recurrent outbreaks occur with greater frequency, and lesions may be extensive or persistent. Multiple large, chronic, 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[7,8,12,14]. However, HSV pneumonia and esophagitis rarely occurs in immunocompetent patients[2,3,11,15-17,19].

Herpes esophagitis was first reported by Johnson[10,11] in 1940. The incidence of herpes esophagitis has been reported to be 1.8% in autopsy patients[9,11]. Almost all cases were immunocompromised hosts[2,3,11,15,17]. Chief complaints of these patients were odynophagia, dysphagia, retrosternal pain, herpetic skin and oral lesions as well[4,11,17]. Although herpes esophagitis in an immunocompetent host is self-limiting, it is occasionally a source of disseminated infection in the susceptible host[4,12]. The diagnosis of HSV esophagitis is usually established with an esophagoscopy. It also permits acquisition of samples for histology, culture, and polymerase chain reaction (PCR)[11,12,15,17,22]. 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[4,11,12,17]. The endoscopic appearance can be confused with esophagitis secondary to candida or peptic disease[2,17]. 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[2,11,12]. Herpes esophagitis may be a early sign of disseminated visceral HSV infection[2,5,6,20]. It is easy to confuse the simple and common condition like HSV type1 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[4,12,17]. Pamuk et al.[15] 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[21].

Morgan[13,16], 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[1,8]. Ramsey et al.[16] 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 motality[2,5,6,15].

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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