Esophageal Perforation from a Fish Bone was Complicated by Pericarditis and Recurrent Stroke in an Acute Stroke Patient

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Abstract- Early diagnosis of esophageal perforation by foreign body impalement in acute stroke is difficult because of the accompanying masking neurological symptoms. Delayed detection of esophageal perforation results in devastating complications such as mediastinitis and pericarditis. Furthermore, atrial fibrillation, due to acute pericarditis, may aggravate the clinical condition as a result of increased incidence of recurrent stroke. Here we present a 59-year-old male patient who sustained an acute stroke followed by fish bone impalement in the esophagus. This was complicated by esophageal perforation with subsequent acute mediastinitis, pericarditis, and recurrent stroke with multiple cerebral infarcts. Even with thoracotomy, mediastinal drainage and broad-spectrum antibiotics, the patient passed away. This case highlights the importance of alertness and early diagnosis in the acute stroke patient with a history of fish bone impalement.

Key Words: Stroke, Esophageal perforation, Pericarditis, Mediastinitis, Atrial fibrillation

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INTRODUCTION

Stroke patients have a greater propensity toward swallowing foreign bodies, and are therefore at increase risk of esophageal perforation[(1)]. Although esophagus perforation caused by foreign body impalement is rare, the associated morbidity and mortality are very high[(2-4)]. Because the incidence of complications and mortality is closely related to the interval between perforation and the initiation of treatment, prompt diagnosis and intervention is mandatory[(5)]. However, early diagnosis of esophageal perforation in acute stroke patients is difficult because of masking neurological symptoms such as dysphagia, pneumonia, and altered consciousness. Here we present a case of acute stroke complicated with esophageal perforation due to fish bone impalement, followed by subsequent pericarditis and mediastinitis. During the admission, the patient sustained recurrent strokes with infarction in the territories of multiple cerebral vessels. These strokes were ascribed to embolisms resulting from the active pericarditis induced atrial fibrillation, which aggravated the clinical picture and led to mortality.

CASE REPORT

A 59-year-old man with poorly controlled diabetes mellitus and hypertension sustained left hemiparesis and

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upper limb spasticity as sequelae of an old stroke nine years ago. On June 4, 2002, he felt more weakness in the left limbs and experienced easy choking. On June 5, he choked again at lunch, with a fish bone stuck in the esophagus. After the meal he noticed a sore throat and discomfort in the left neck. On June 6, he had decreased coordination and even more weakness in the left limbs. There are also slurred speech, general malaise, and persistent laryngeal or pharyngeal discomfort.

In the emergency room, the patient had clear consciousness but appeared unwell. Physical examination was unremarkable except for the irregular heart beat. Neurologic examination demonstrated left hemiplegia, tendency of falling down forwardly, and slurred speech. He denied previous history of heart or lung diseases. Also, there was no history of recent stress or mood change. A computed tomography (CT) scan of the brain on June 6 showed an old infarction in the territory of the right middle cerebral artery with encephalomalacic changes (Figs. A-C, left). Chest and neck (soft tissue) X-ray examination showed no evidence of active lung lesions, retropharyngeal soft tissue swelling, or a radioopaque foreign body. Rigid fiberscopy did not reveal any foreign body in the larynx. An esophagogram showed no definite extravasation of contrast medium. The hemogram showed leucocytosis (WBC 13000 cells/mm^3, segment 98%), although no fever was noted. He was admitted to the neurological ward with a diagnosis of recurrent stroke.

During the first two days of hospitalization, his general condition was stable. On June 9, he complained of abdominal discomfort, chest pain, and shortness of breath. Mild fever (up to 38.5°C) was noted. The electrocardiogram (EKG) showed an elevated S-T segment in the V3-V4 leads. Blood tests showed normal CK, CK-MB, and troponin I levels. On June 10, a repeated EKG showed atrial fibrillation and S-T segment elevation in all of the V1-V6 leads, suggesting the possibility of pericarditis. The clinical condition deteriorated with new-onset right hemiparesis and cloudiness of consciousness. Tachypnea, tachycardia, and bilateral diffuse rales as well as crackles were noted. Chest X-ray showed bilateral severe pleural effusions with mediastinal widening (Fig. D). C-reactive-protein (CRP) was 378 mg/L. Thoracic CT showed low attenuation collection and infiltration in the mediastinum with thickening of the pericardium. There was also low attenuation fluid collection in the pleural space bilaterally (Fig. E). Emergency intubation was performed due to severe dyspnea. Consciousness level of the patient deteriorated. Under the diagnosis of mediastinitis with abscess formation, immediate thoracotomy and mediastinal drainage was performed. During the operation, examination of the esophageal wall revealed a transmural laceration with impalement by a fishbone-like foreign body at the level

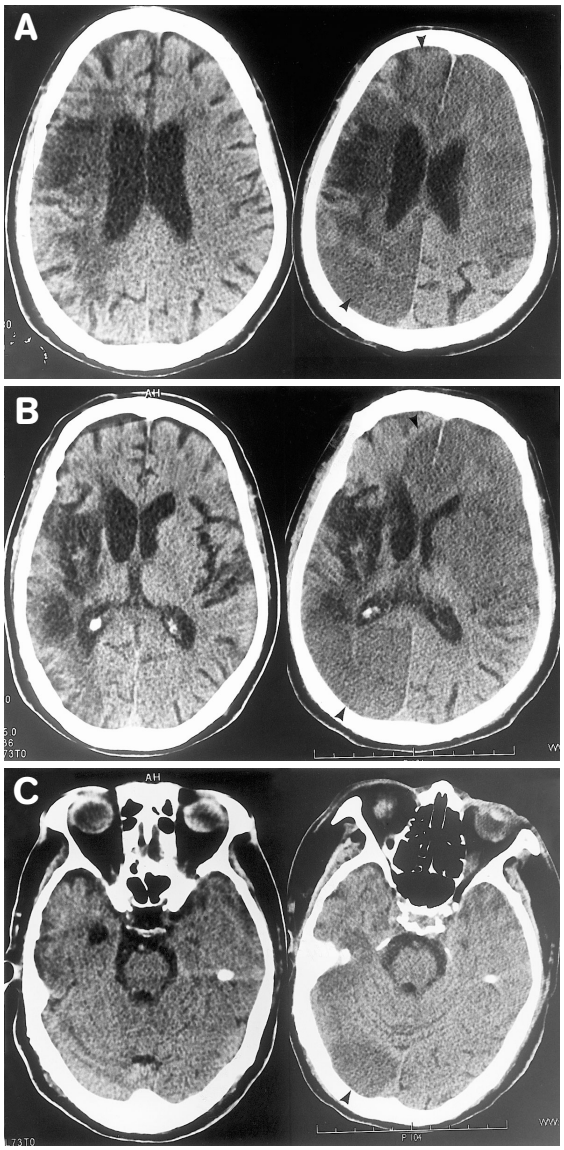

Figure A-C. (Left) Axial sections of brain CT from the emergency room show an old infarction with encephalomalacic changes in the territory of the right middle cerebral artery. (Right) Postoperative follow-up brain CT at the same levels shows new low attenuation lesions in the territories of bilateral anterior cerebral arteries, left middle cerebral artery, right posterior cerebral artery, and right middle cerebral artery (arrowheads).

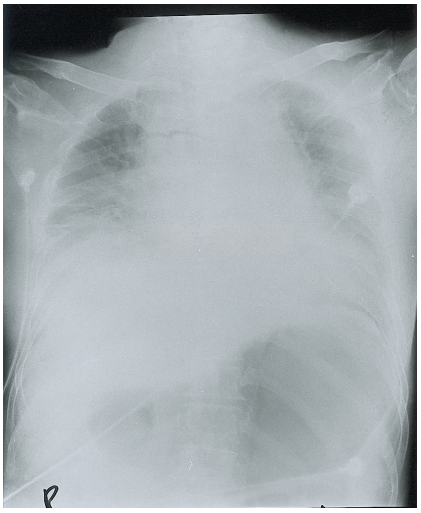

Figure D. Chest X-ray (anteroposterior view) shows bilateral pleural effusions with cardiomegaly and mediastinal widening.

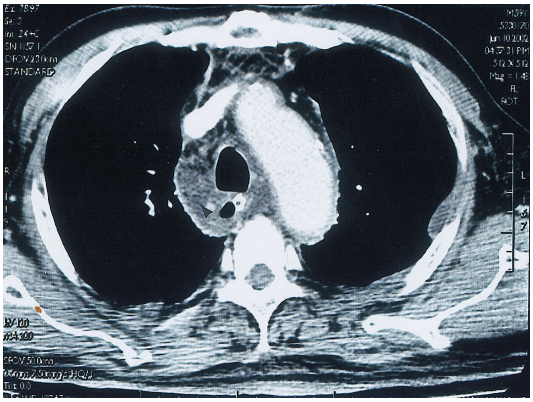

Figure E. Chest CT shows low attenuation collection and infiltration in the left neck region with extension into the mediastinum adjacent to the esophagus and trachea (arrowhead).

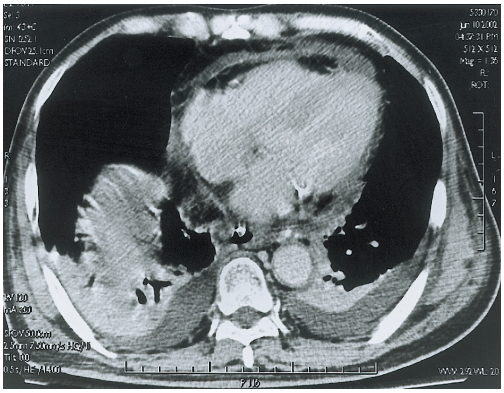

Figure F. Chest CT shows fluid collection and infiltration in the mediastinum with bilateral pleural fluid collection and thickening of the pericardium.

of azygus vein-superior vena cava junction. 150 ml of pus and 500 ml of turbid pleural effusion were drained from the mediastinum and from the mediastinal wound. Severe pleural adhesions with some necrotic pleura were also found. The patient became comatose postoperatively. Follow-up brain CT on June 11 showed new low attenuation lesions in the territories of bilateral anterior cerebral arteries, left middle cerebral artery, right posterior cerebral artery, and right middle cerebral artery (Figs. A-C, right). Despite local wound drainage and intravenous broad-spectrum antibiotics, systemic sepsis with multiple organ failure occurred, and the patient passed away on June 23.

DISCUSSION

Esophageal perforation due to foreign body ingestion is rare, accounting for 1% to 4% of total reported esophageal perforation cases[2]. Stroke patients are prone to choking and have a greater risk of swallowing foreign bodies, such as fish or chicken bones. Once the foreign body is impacted in the esophagus, mucosal edema secondary to local trauma develops[6] and the inflammatory reaction with subsequent infection may occur in 24 hours[7]. Esophageal perforation therefore tends to occur in 24 hours after foreign body impaction in the majority of cases[6]. The associated morbidity and mortality are generally high and is closely related to the interval between the perforation and the initiation of treatment[2-4]. In patients treated within 24 hours of the onset of symptoms, the survival rate is 92%. However, the mortality rate is 40% to 50% in patients who were not treated until 24 hours after the injury[6]. Because the classic clinical findings of chest pain, fever, subcutaneous emphysema, and pneumothorax are often absent in the early stage[8], the accurate diagnosis could be delayed[2-4], particularly in stroke patients with accompanying neurological symptoms of dysphagia, pneumonia and disturbed consciousness.

Esophageal perforations can be subdivided into cervical, thoracic and abdominal perforations[5]. Perforation of the thoracic esophagus results directly in mediastinal contamination, leading to a rapid development of pneumomediastinum and mediastinitis. The thin mediastinal pleura is usually ruptured by the inflammatory process, producing effusion and contamination of the pleural space[5]. Gastric contents and fluids are then

drawn into the pleural space by the negative intrathoracic pressure[5], leading to a more complicated and devastating clinical course.

Pericarditis with effusion is a life-threatening complication with a reported incidence of about 13% after esophageal perforation[9]. Constrictive pericarditis frequently induces diastolic dysfunction and results in impaired ventricular filling[10]. Subsequent tachycardia appears to improve the hemodynamic profile[10], as the case in our patient. Acute pericarditis also induces a higher incidence of atrial fibrillation[11]. Patients with chronic atrial fibrillation are much more liable to stroke than subjects from an age-matched population with a normal cardiac rhythm[12]. Embolism may occur during paroxysmal atrial fibrillation and further complicate the clinical picture. Actually stroke might be the most important factor determining the mortality and morbidity associated with atrial fibrillation[13]. In our patient, recurrent strokes with infarctions in the territories of multiple vessels are most likely related to embolism associated with atrial fibrillation and may contribute to his mortality. Although repeated strokes could also be caused by septic shock and systemic hypotension, the patient had sustained multiple cerebral infarctions prior to the development of septic shock. To our knowledge no similar cases have been reported before.

A contrast esophagogram is the standard diagnostic procedure in cases of suspected esophageal perforation. However, the false-negative rate can exceed 10%[14] and it is contraindicated in stroke patients because of repeated positional changes required by the examination. Flexible esophagoscopy is another useful diagnostic adjuvant because it provides highly accurate localization and direct visualization of perforations[5,15]. It had been very helpful in the early diagnosis of this patient reported here.

This case report highlights the need for a high degree of alertness when caring stroke patients with a history of fish bone impalement. Particular attention should be given to those who have neck or chest pain and leucocytosis, so that the complications of possible esophageal perforation can be reduced. In addition, it is important for stroke patients with swallowing disturbance to avoid those potentially hazardous food such as bone-containing fish or chicken.

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