Cervical Mycotic Aneurysm in a Patient with Alcoholic Cirrhosis

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Abstract

We report a case of 65-year-old man with alcoholic cirrhosis and diabetes mellitus, in whom a cervical mycotic aneurysm suddenly developed after sepsis with methicillin-resistant Staphylococcus aureus. Severe infection associated with alcoholic cirrhosis may cause a typical mycotic aneurysm.

Key words: mycotic aneurysm, cirrhosis

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Introduction

A patient with liver cirrhosis from alcoholism is readily susceptible to septic shock and endotoxemia with abnormal Kupffer cells. Septic emboli may cause mycotic aneurysms. The term mycotic aneurysm refers to any aneurysm that results from an infectious process involving the arterial wall. There are few reports of patients with alcoholic cirrhosis and a cervical mycotic aneurysm. Here, we report the rare case of a 65-year-old man with alcoholic cirrhosis and diabetes mellitus, in whom a cervical mycotic aneurysm suddenly developed.

Case Report

A 65-year-old man was admitted to our hospital because he had lost both short-term and long-term memory. Three days earlier, he had broken his left clavicle without an open fracture. Before admission, he had been treated with insulin for type 2 diabetes mellitus and with diuretics for ascites. The patient’s blood pressure on admission was 162/98 mmHg and his temperature was 38.3°C. Physical examination on admission showed no anemia or jaundice, but did show enlargement of the midclavicle on the left side. The patient had a flapping tremor and seemed slightly confused. The results of the laboratory examination were as follows: hemoglobin, 11.5 g/dL (normal, 11.3-17.4); white blood counts, 13,700/μL (normal, 5,000-8,500); blood platelet counts, 22.5×10⁴/mm³ (normal, 12.5-37.5×10⁴); albumin, 2.4 g/dL (normal, 3.5-5.0); alanine aminotransferase, 21 IU/L (normal, 5-37); aspartate aminotransferase, 20 IU/L (normal, 10-27); lactate dehydrogenase, 185 IU/L (normal, 90-210); alkaline phosphatase, 518 IU/L (normal, 104-338); gamma-glutaryltranspeptidase, 71 IU/L (normal, 12-50); total bilirubin, 1.3 mg/dL (normal, 0.3-1.2); glucose, 518 mg/dL (normal, 65-110); ammonia, 118 μmol/L (normal, 0-70); and CRP, 20.7 mg/dL (normal, 0.0-0.3). Hepatitis B surface (HBs) antigen and hepatitis B core (HBc) antibody were negative. Serum HCV antibody and HCV-RNA were negative. Antimitochondrial and antinuclear antibodies were negative. However, the patient was found to have sepsis.

Initially, the patient received empiric antibiotics (carbapenems) with insulin therapy. Later, his blood culture showed a methicillin-resistant Staphylococcus aureus (MRSA) infection, and also MRSA was isolated from soft tissue near his broken left clavicle, so he received anti-MRSA antibiotics (vancomycin). His inflammatory sign disappeared promptly. Two months after admission, the right side of the patient’s neck was enlarged despite the use of anti-MRSA antibiotics. His swollen neck mass was palpable and pulsatile. He had a thorough throat neurological examination including lingual and velar movement. He showed no neurological abnormality in the throat and showed no bulbar palsy. He also complained of slight difficulty in swallowing due to compression caused by the swollen neck mass. A neck ultrasound and a computed tomography scan showed a tumor, 16.7×34.5 mm, in the right side of the neck. That tumor was found to be a
pseudoaneurysm at his left common carotid artery just beneath the bifurcation. In spite of the swelling of his neck, there were no signs of hemorrhage or petechiae on the eyes or nails and no skin rashes. And there was no dental abnormality. His extremities showed no edema or cyanosis. Transthoracic echocardiography showed normal left ventricular functions, normal operation of valves, and revealed no vegetations. We continued performing medical treatment with appropriate antibiotics for more than the usual 2 months, but his neck continued to be enlarged and we feared rupture of the aneurysm. A neurosurgeon was consulted and with the patient’s consent, he surgically removed the aneurysm in the neck. Conventional angiography performed in preparation for surgery also confirmed the diagnosis of a pseudoaneurysm (Fig. 1-A, B).

This neck aneurysm was coated with hyaline and infected cells; it was not lacerated or crushed. Figure 2 shows a section of this aneurysm around the blood vessel. This aneurysm was surrounded by a thrombus filled with inflammatory cells (shown in Fig. 3). This aneurysm did not present atheromatous aspects. The symptom disappeared gradually. However, he died of multiple organ failure from sepsis of bedsore at the lower end of the sacrum nine months after the operation.

Discussion

There are three types of extra-cranial aneurysm: traumatic aneurysm, mycotic aneurysm, and oncotic or neoplastic aneurysm (2). Traumatic aneurysm is a pseudo-aneurysm or dissected aneurysm. Mycotic aneurysm results from septic emboli. Bacterial endocarditis is the most common cause. Oncotic or neoplastic aneurysm results from a myxoma or choriocarcinoma and this type of aneurysm may cause tumor emboli and sometimes brain abscess.

The present patient had no history of trauma and the wall of his aneurysm was neither lacerated nor crushed, although it was filled with inflammatory cells. There were no atherosclerotic plaques or calcium structures (crystals) around the blood vessels. Furthermore, MRSA was isolated from two blood cultures and from soft tissue near his broken left clavicle. Therefore, his aneurysm was diagnosed as mycotic. We hypothesize that the patient had bacteremia from MRSA that produced septic emboli that in turn produced a mycotic aneurysm.

This patient already had liver cirrhosis and diabetes mellitus, so he was readily susceptible to sepsis and endotoxemia because of the porto-systemic shunt in his liver. In addition, liver cirrhosis is known to cause abnormal activation of Kupffer cells (1). Kupffer cells, which are the resident macrophages of the liver, produce oxidants and cytokines for the purpose of host defense. Chronic alcohol consumption causes abnormal activation of Kupffer cells. Cytokines and oxidants produced by these cells inflict damage on the liver itself.

A few reports have shown a connection between liver cirrhosis and aneurysm; splenic aneurysms are seen in 3.2% of all patients with liver cirrhosis (3-5). The widespread use of antibiotics has decreased the incidence of mycotic aneurysm, which accounts for 2.6% of all abdominal aneurysms. Of
cerebral aneurysms, iatrogenic or traumatic aneurysm accounts for 1%, mycotic aneurysm accounts for 2-3%, and atherosclerotic or arteriovenous malformation accounts for about 30% (3, 4). Therefore, the incidence of mycotic aneurysm is not especially high in patients without liver cirrhosis. In Japan, 2.3% of patients with liver cirrhosis experience endotoxemia; in blood cultures from these patients with endotoxemia, 69% show Gram-positive staining. Especially in mycotic intracranial aneurysms, Gram-positive cocci are commonly isolated; streptococci are the most commonly isolated (30-44%) and second most common are Staphylococci (14-18%) (6-8). In this case, Staphylococcus aureus was isolated from the patient. Some reports have shown the effectiveness of medical treatment alone (9, 10), but most patients who also have neurological dysfunction benefit from surgical treatment, such as aneurysm removal (7).

Most cases of mycotic aneurysm are treated with antibiotics for 4-6 weeks. In this case, although anti-MRSA antibiotics were used for 2 months, neurological dysfunction occurred. Antibiotics may be given for more than 8 weeks to patients with liver cirrhosis and mycotic aneurysm. However, if neurological dysfunction is occurs or the aneurysm becomes enlarged, we recommend removal of the mycotic aneurysm to reduce the risk of rupture.

References
