Recurrent cerebral embolism secondary to esophageal and atrial foreign body complicated by infective endocarditis

Hui Liang, MD, a Ziqi Xu, MD, a Qiang Feng, MD, b and Liang Ma, MD, b Hangzhou, China

Ingestion leading to an esophageal foreign body (FB) is not uncommon in clinical practice. Although many are retrieved without event, FBs can lead to devastating complications such as esophageal perforation, tracheoesophageal fistula, and respiratory distress. Neurologic complications of FBs are rare. Here we describe the case of a patient with recurrent stroke secondary to an esophageal and atrial FB complicated by infective endocarditis (IE), a complication that has not been reported before.

CLINICAL SUMMARY

A 43-year-old Chinese woman was referred to our hospital with sudden right-sided weakness. She had a 4-day history of fever and slight left upper abdominal pain and a 3-day history of vertigo. A cranial computed tomographic scan appeared normal. There were no vascular risk factors. On admission, the patient’s temperature was 39.6°C, and a few moist rales had been audible over both lung bases. The heart sounds were normal, with no murmurs. The abdomen was soft without tenderness. On neurologic examination, the patient was noted to have a right central facial palsy and right hemiparesis. A blood test showed elevations in white blood cell count (17,000 cells/μL) and C-reactive protein level (39.8 mg·dL−1). Brain magnetic resonance imaging showed multifocal, acute infarcts in the cerebellum and bilateral cerebral hemisphere (Figure 1, A and B). An abdominal computed tomographic scan revealed acute splenic infarction. Echocardiography revealed a moderately sized, highly mobile, nonhomogeneous mass in the left atrium (LA) measuring 3.0 × 1 cm (Figure 1, C). The diagnosis of IE was suspected, and meropenem was prescribed for antibiotic therapy. The patient remained febrile, and her family members mentioned that she had reported symptoms of painful swallowing and transit difficulty swallowing 1 day before the occurrence of fever. An emergency thoracic computed tomographic scan was then performed for a suspected esophageal disorder and revealed a 2.5-cm FB located primarily in the thoracic esophagus piercing the LA (Figure 2). The patient underwent heart surgery under extracorporeal circulation, and a 1.5-cm fish bone was discovered in the LA, surrounded by an irregular mass. The mass and fish bone were removed, followed by atrial repair. The esophagus left came in close contact with LA wall, and an obvious perforation was found in the esophagus. A T-tube method was used to treat this perforation. Cultures from the removed masses grew Candida albicans. Histopathologic examination revealed the masses to be composed mainly of mixed thrombus interlaced with

FIGURE 1. A and B, Brain magnetic resonance images with diffusion-weighted imaging (A) and apparent diffusion coefficient (B) demonstrated acute infarcts in bilateral cerebral hemisphere. C, Echocardiography revealed a moderately sized, nonhomogeneous mass in the left atrium.

J Thorac Cardiovasc Surg 2014;148:e213-4
0022-5223/836.00
Copyright © 2014 by The American Association for Thoracic Surgery
http://dx.doi.org/10.1016/j.jtcvs.2014.08.012
massive neutrophilic infiltration. After antimicrobial therapy for 5 weeks, the patient recovered, and a postoperative barium swallow revealed no anastomotic leak. She was discharged uneventfully.

DISCUSSION

FBs in the heart are rare occurrences and can result from intravenous drug abuse, trauma, or iatrogenic causes. The esophageal wall is thin, and there is no serosal membrane enclosing the outer layer. The lodging of FBs in the esophagus may cause pressure changes in the wall and cause perforation. Because the esophagus and heart are closely collocated, FBs in the esophagus may penetrate the heart just as in our case. Among 121 patients with esophageal FBs, induced perforation, complicated cervical abscesses, mediastinitis and mediastinal abscesses were found. In our case, the fish bone penetrated the LA and created a lesion on it, which served as an anatomic substrate for infection. Vegetations with signs of active infection were formed as a result of C albicans endocarditis associated with the retained fish bone and consequently led to multiple embolism events. Although there are no guidelines for the treatment of FBs localized in the heart, surgical removal of this FB was necessary to cut off the infectious source and alleviate the heart injury.

At admission, we struggled with the appropriateness of anticoagulation administration because of the risk of recurrent stroke and intracerebral hemorrhage. Initiating anticoagulation may seem reasonable to prevent embolization of infected or noninfected platelet fibrin valvular vegetations. A large number of patients with IE who subsequently had a stroke, however, had early intracerebral hemorrhage and hemorrhagic transformation. Risk stratification and decision making should be based on the individual risk balance of potentially devastating hemorrhagic transformation and further embolic ischemic stroke. The risk of hemorrhagic transformation depends mainly on the size of the infarcted tissue. It therefore seemed reasonable not to use anticoagulation to treat our patient with multiple and large territorial infarctions. Rapid institution of effective antibiotic therapy and surgery together represent the cornerstone of treatment of IE to reduce the mortality and morbidity from embolic complications and heart failure. Some findings provide a basis for the new concept that statin therapy is associated with a reduced risk of in-hospital and subsequent mortality from IE. A continued evaluation of these drugs and their potential impact on subsequent embolism among patients with IE is warranted.

CONCLUSIONS

We should be aware of the rare neurologic complications of esophageal FB. Prompt correct diagnosis and treatment as soon as possible are key to avoiding these complications.

References