Cardioembolic Stroke Followed by Isolated Celiac Artery Thromboembolism

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Abstract

A 78-year-old man with Parkinson's disease, paroxysmal atrial fibrillation, and congestive heart failure was admitted to our hospital due to global aphasia and right-sided hemiparesis. A cardioembolic stroke from a left ventricular thrombus was diagnosed; several days later, anticoagulants were started. On the seventh day, the patient suddenly developed severe acidosis and kidney and liver dysfunction. He died the following afternoon. Autopsy revealed an isolated celiac artery embolism from the left ventricular thrombus. This is the first reported case of isolated celiac artery embolism occurring after acute ischemic stroke.

Key words: cardioembolic stroke, celiac artery embolism

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Introduction

Stroke patients can develop many complications in the acute phase, including infections, falls, and a variety of systemic disorders (1). Systemic embolism, though not common even after an embolic stroke, is one of the more serious complications. We report a rare case of acute thromboembolism of the celiac artery that occurred 7 days after a cardioembolic stroke.

Case Report

A 78-year-old man with paroxysmal atrial fibrillation and chronic congestive heart failure was admitted to hospital due to global aphasia and right-sided paralysis. His past medical history included a 6-year history of hypertension and Parkinson's disease, a 2-year history of chronic renal failure, a cervical decompression, and surgery for gastric atonia. His medications included oral aspirin 100 mg/day, anti-Parkinsonian agents, and diuretics.

On admission, his blood pressure was 160/101 mmHg, and his pulse was 103 beats/min and regular. No abnormal sounds were heard on chest auscultation. Operative scars were noted in the back of the neck and in the midline of the upper abdomen. On neurological examination, he was

drowsy and had global aphasia. The eyes deviated to the right side, and the pupils were isocoric. He had a right-sided central type facial palsy. His right arm was flaccid, while the left arm had cog-wheel type rigidity. The right leg had a severe palsy; he could only move it horizontally with difficulty. The left leg was rigo-spastic. The deep tendon reflex was brisk on the right side. Babinski's sign was present on the right side and equivocal on the left side. The admission National Institutes of Health Stroke Scale score (2) was 26.

Laboratory data showed renal failure: urea nitrogen (UN) was 42.1 mg/dl, creatinine (Cr) was 2.19 mg/dl. Aspartate transaminase (AST, 34 IU/l), alanine aminotransferase (ALT, 6 IU/l), choline esterase (246 IU/l), γ -glutamyltransferase (20 IU/l), and C-reactive protein (CRP, 0.03 mg/dl) were within normal limits. He had a mild anemia and a mild thrombocytopenia. His prothrombin time international normalized ratio (PT-INR) was 1.12. The electrocardiogram showed normal sinus rhythm with left ventricular hypertrophy and ST depression in II, aVF, V5, and V6. Chest radiography showed cardiomegaly with a cardiothoracic ratio of 71% and mild pulmonary congestion. Brain CT showed early ischemic changes in the left cerebrum and the dot sign in the left Sylvian fissure. On carotid ultrasonography, small plaques were noted in the bifurcations bilaterally. The flow velocities of the right and left carotid arteries were equal. Cardioembolic stroke was diagnosed; oral aspirin was

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Figure 1. A. Section of the origin of the celiac artery (hematoxylin-eosin stain). A fresh thrombus occludes the arterial lumen. No plaque fragments or cholesterol crystals are seen. B. Microscopic section of the thrombus in the left ventricular apex. C. Microscopic section of the thrombus in the celiac artery (the square part of Figure A). On pathology, these thrombi resemble each other, which, given the clinical history, suggests that the celiac artery thrombus originated from the left ventricular thrombus.

stopped and glycerol was given. On the 4th day, transthoracic echocardiography showed a small thrombus in the left ventricular apex. Brain MRI demonstrated fresh ischemic lesions that were located diffusely in the territory of the left MCA. Hemorrhagic changes were not seen; and MR angiography demonstrated showed the left MCA was patent. Unfractionated heparin of 10,000 units/day and warfarin of 3 mg/day, as well as enteral nutrition, were then started.

In the evening of the 7th day, the patient suddenly vomited a large amount of stomach contents; he aspirated some of the contents and developed respiratory failure. On examination, the abdomen was rigid, the bowel sounds were weak, and the extremities were cyanotic. After intubation,

mechanical ventilation was started. Laboratory data showed: mild transaminase elevation (AST 96 IU/l, ALT 73 IU/l), renal failure as noted on admission (BUN 72.9 mg/dl, Cr 1.72 mg/dl), CRP elevation (14.29 mg/dl), PT-INR of 2.38, and a partial thromboplastin time of 57.6 seconds. Heparin-induced thrombocytopenia was ruled out since the platelet count ($162 \times 10^3/\text{mm}^3$) had not decreased since admission. On arterial blood gas analysis, a mild metabolic acidosis and a compensatory respiratory alkalosis were noted with oxygen at 5 l/min by face mask (pH 7.402, pCO_2 24.2 mmHg, pO_2 107.8 mmHg, HCO_3^- 14.7 mg/dl, base excess -8.1 mEq/l, SaO_2 98.0%). Despite treatment with antibiotics and bicarbonate, the metabolic acidosis caused by kidney and liver

dysfunction progressed rapidly; the patient died on the following afternoon.

An autopsy was conducted, and a celiac artery thromboembolism was found; there was no evidence of thromboembolism in any other arteries. A partly organized thrombus was also seen in the left ventricle. The pathological findings of the thrombus in the left ventricle and the thrombus in the celiac artery were similar (Fig. 1). Ischemic change was observed in the liver and in the gastric and duodenal mucosa. Although the intima-media complex of the aorta and arteries showed severe atherosclerotic change, no atheromatous plaques were seen at the origin of the celiac artery. The renal arteries were patent on both sides, and both kidneys showed benign sclerotic changes.

Discussion

The present patient is a rare example of cardioembolic stroke that was followed by isolated thromboembolism of the celiac artery in the subacute phase. On autopsy, a thrombus that had occluded the celiac artery and that had originated from a left ventricular thrombus secondary to reduced systolic function was found. A previous paper reported the case of a 23-year-old man who developed isolated thromboembolism of the celiac artery due to intracardiac thrombus that occurred after an acute myocardial infarction (3). Other authors described a case with gangrene of the gall-bladder due to celiac artery embolus (4). In another case report, a patient had developed hepatic infarction after 8 days after stroke onset due to diffuse atherosclerotic ulceration at the right coronary sinus (5). Thromboembolism of the celiac artery as part of a systemic shower of emboli has been reported by many authors (6). Several cases of hepatic infarction due to embolism from heart or aorta have also been reported in the previous literature (7, 8). Another important cause of celiac artery occlusion is arterial dissection (9-12). However, there have been no previous reports of an isolated celiac artery embolism as a complication following soon after a stroke.

Emboli that cause intestinal ischemia originate from the heart in >75% of cases; they lodge preferentially just distal to the origin of the middle colic artery from the superior mesenteric artery (13). One of the reasons for the rarity of isolated celiac artery occlusion is the fact that the celiac artery often branches vertically at the ascending aorta. How-

ever, in our patient, the autopsy showed that the angle between the celiac artery and the descending aorta was not particularly sharp. Thus, the reason why the thrombus from the left ventricle had occluded only the celiac artery is unclear.

The timing of when anticoagulant therapy to prevent a second embolism after a cardioembolic stroke should be started remains controversial (14). The early initiation of anticoagulant therapy may cause hemorrhagic transformation with recanalization of the occluded cerebral artery. Therefore, we usually start low dose unfractionated heparin after excluding hemorrhagic change on brain CT scan more than 24 hours after stroke onset. In patients with a massive brain infarction, such as one that involves almost all of the MCA area on one side, sufficient time must pass before anticoagulant therapy can be started. From our patient's neurological signs and symptoms, it was highly suspected that the proximal side of the left MCA had been occluded at the stroke onset. Therefore, we started anticoagulation after we had confirmed the findings of MRI and MR angiography on the 4th day.

The best strategy to deal with intracardiac thrombus, particularly in the acute phase of a massive cerebral infarction, has not yet been established (15). Although low dose unfractionated heparin was started in this case to prevent thromboembolism due to cardiac thrombus, this might have accelerated the separation of the thrombus that was attached to the endocardium.

Acute thromboembolism of the mesenteric artery is often treated by surgery, embolectomy, or thrombolysis (13, 16, 17). In the present case, thrombolysis was problematic due to the patient's generally poor condition with brain, lung, and kidney dysfunction. Therefore, only expectant treatment was appropriate.

In conclusion, we reported a cardioembolic stroke patient who developed isolated celiac artery embolism during the subacute phase. Further studies are needed to establish the proper anticoagulant therapy for patients during the acute phase of embolic stroke in whom a cardiac thrombus is found.

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