

## Case Report

# Cardioembolic Stroke Revealed by Increased Hemostatic Markers Associated With Intracardiac Thrombus

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**Background and Purpose:** The presence of hemocoagulative disorders in acute ischemic stroke has been reported occasionally. However, the cause of the hemostatic derangement has not been fully elucidated.

**Case Description:** A 66-year-old woman with a history of hypertension and myocardial infarction developed pure motor hemiparesis. On admission, she was thought to have a lacunar infarction. However, computed tomography of the brain with contrast medium revealed a small infarct in the cortex of the frontal lobe. Conventional angiography showed no stenotic or occlusive lesions. Sensitive hemocoagulative tests revealed hypercoagulative and hyperfibrinolytic states. Ultrafast computed tomography of the heart with contrast enhancement demonstrated a large left ventricular mural thrombus. There were no further abnormal findings suggestive of other systemic diseases that affect blood coagulability. As a result, the patient was diagnosed as having suffered a cardioembolic stroke.

**Conclusions:** An intracardiac thrombus could be one of the causes of the hemostatic disorders of acute cardioembolic stroke. (*Stroke* 1991;22:1317-1319)

The presence of hemocoagulative disorders in acute ischemic stroke has been reported occasionally.<sup>1-5</sup> A recent report by Feinberg et al<sup>5</sup> demonstrated advanced fibrin formation and increased fibrinolysis during the acute phase of ischemic stroke by measuring plasma levels of sensitive hemostatic markers such as fibrinopeptide A, B $\beta$ 1-42, B $\beta$ 15-42, and D-dimer in patients with acute ischemic stroke, including cardiogenic cerebral embolism. However, the cause of the hemostatic derangement has not been fully elucidated. The present case is instructive in that an intracardiac thrombus could be one of the causes of the hemostatic disorders of acute cardioembolic stroke.

### Case Report

The patient was a 66-year-old right-handed woman. She had been hypertensive for approximately 20 years and had had a myocardial infarction 4 years earlier. She had not been on antiplatelet or anticoagulant drugs for the past 2 years.

On October 31, 1989, she noticed difficulty of speech on awakening in the morning. She was admit-

ted to the Stroke Care Unit of the National Cardiovascular Center on the same day. On initial examination, she had a blood pressure of 160/92 mm Hg and a regular heart rate of 70 beats/min. There were no ocular, carotid, subclavian, or femoral bruits. She showed mild dysarthria due to left lower facial weakness and mild left hemiparesis on her left extremities. She was fully conscious and had no higher brain dysfunction such as aphasia, apraxia, or agnosia. No other abnormal findings were detected at the bedside examination.

Plain computed tomography (CT) of the brain was normal, both on admission and on hospital day 8. On day 14, however, CT scan of the brain with contrast medium revealed a small area of enhancement in the frontal cortex, consistent with a recent infarction (Figure 1).

Conventional four-vessel cerebral angiography on hospital day 3 demonstrated only minor plaques, with no ulceration at the bifurcation of the carotid arteries on either side, which was also noted by duplex ultrasonographic examination. There were no other severely stenotic or occlusive vessels. A renal scintigram revealed a small renal infarction on the left side.

Routine laboratory data of urine analysis, fecal occult blood test, complete blood counts, and serum chemistry showed no abnormalities. However, considerable abnormalities of hemostatic markers were

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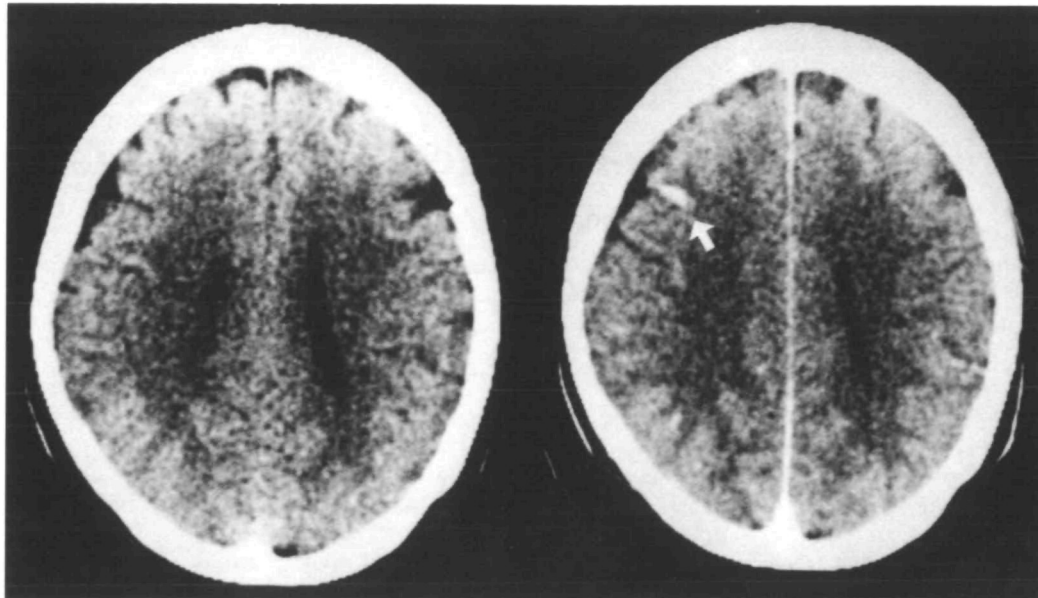


FIGURE 1. Computed tomography (CT) of the brain. Left panel: Plain CT. Right panel: Enhanced CT. Lesion was demonstrated clearly by CT with contrast medium (arrow).

present in the tests conducted on hospital day 14. They were increased plasma levels of fibrinopeptide A, 3.2 ng/ml (normal value <2.0); fibrinopeptide B $\beta$ 15-42, 7.5 ng/ml (<4.8); D-dimer, 769 ng/ml (<150); thrombin-antithrombin III complex, 8.6 ng/ml (<3.0); and  $\alpha_2$ -plasmin inhibitor-plasmin complex, 2.3 ng/ml (<0.8). Prothrombin time was 0.99 (international normalized ratio), activated partial thromboplastin time 35 seconds, plasma level of antithrombin III 80.5% (83-123), and protein C 79.6% (80-120).

The electrocardiogram was compatible with an old anterior myocardial infarction. The echocardiographic examination demonstrated that the left ventricular end-diastolic dimension was 60 mm and the anterior wall

motion of the left ventricle was severely reduced. However, further information in detail could not be obtained because of a small echo window. An ultrafast CT scan of the heart with a contrast medium demonstrated a large mural thrombus at the apex of the left ventricle on day 16 (Figure 2).

Warfarin potassium was started immediately to prevent a recurrence of systemic embolization after the detection of the intracardiac thrombus. A follow-up CT of the heart performed 1 month later showed that the large mural thrombus had disappeared. There was no recurrence of systemic embolism.

#### Discussion

Because of a long history of hypertension and the presence of pure motor hemiparesis, this patient was

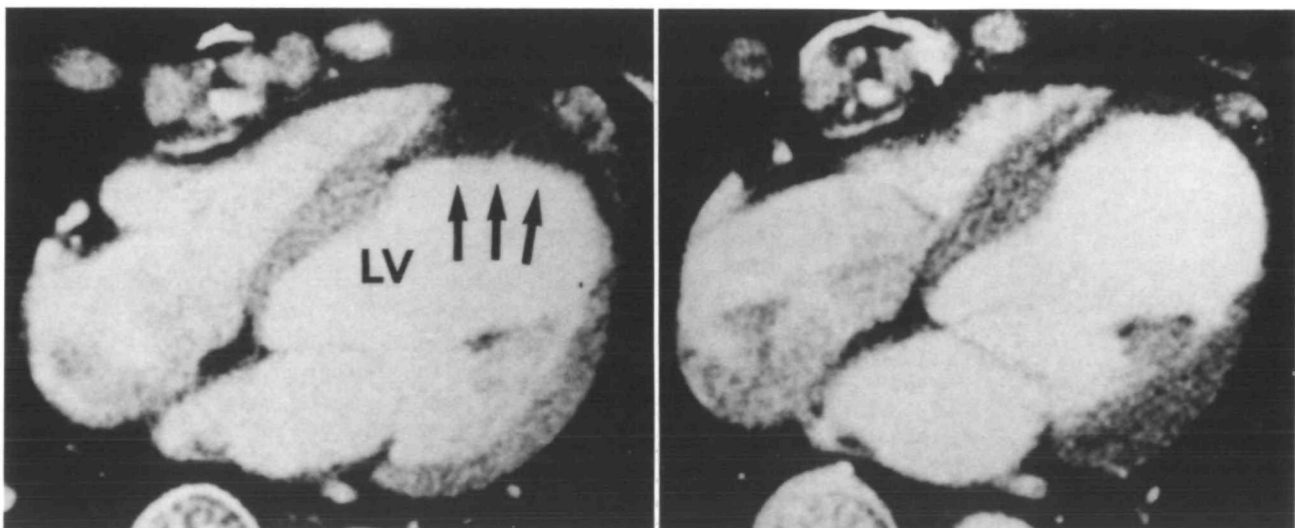


FIGURE 2. Ultrafast computed tomography of the heart with contrast medium before (left panel) and after (right panel) anticoagulation. Arrow indicates intracardiac thrombus. LV, left ventricle.

thought on admission to have a lacunar infarction in the right internal capsule or pontine base. However, brain CT with contrast enhancement revealed a lesion responsible for the recent episode in the right frontal cortex. This lesion has been reported to be responsible for pure motor hemiparesis.<sup>6,7</sup> The presence of an intracardiac mural thrombus at the apex of the left ventricle and an asymptomatic infarct in the kidney strongly supported the diagnosis of cardioembolic stroke in this patient. Further, the carotid plaques were not ulcerated enough to cause artery-to-artery embolism. There were no occluded vessels, suggesting a reopening of the previously occluded vessels. Therefore, her cortical lesion of the frontal lobe was thought to be cardioembolic in nature.

Blood coagulative studies showed enhanced thrombin and fibrinolytic activities, which were inconsistent with the reported blood coagulability study in patients with acute lacunar infarction. Feinberg et al<sup>5</sup> reported that blood coagulability in patients with acute lacunar infarction was normal and that plasma levels of fibrinopeptide A, fibrinopeptide B $\beta$ 15-42, and D-dimer were increased in patients with an acute cardiogenic cerebral embolism or atherothrombotic infarction. However, they have not elucidated the cause of the hemocoagulative abnormalities in these conditions.

On the other hand, it has been reported that hemostatic markers increased and antithrombin III levels decreased in patients with an intracardiac thrombus.<sup>8-10</sup> Therefore, elevated hemostatic markers (fibrinopeptide A, fibrinopeptide B $\beta$ 15-42, D-dimer,  $\alpha_2$ -plasmin inhibitor-plasmin complex, and thrombin-antithrombin III complex) and low levels of antithrombin III and protein C in this case were considered to be responsible for the consumption coagulopathy related to large intracardiac mural thrombus.

We speculate that the sequence of the events in this case was as follows. The left ventricle was dilated and its function reduced due to anterior myocardial infarction. The reduced cardiac function may then have produced blood stagnation in the heart, which was a major factor in producing the hypercoagulable state. Elevated blood coagulation activity was followed by a secondary enhancement of fibrinolytic activity, reflected by derangement of the hemostatic markers. When factors such as dehydration<sup>8</sup> elevated the blood coagulation activity over that of fibrinolysis,

the intracardiac thrombus could then be formed, leading to a direct cause of the embolism.

Because blood samples were taken from an antecubital vein, the result of blood coagulation tests could have been influenced by an embolus itself located in the cerebral artery. In this case, however, the embolus had already been resolved, so that we believe the embolus itself did not influence our results in the blood coagulation test.

Tohgi et al<sup>11</sup> have reported that the size of infarct correlates with hemostatic markers. However, the size of infarct in this case was so small that there seems to be no relationship between its size and the increased hemostatic markers.

Our data suggest that an intracardiac thrombus could be one of the causes of increased hemostatic markers in patients with an acute cardioembolic stroke. We recommend investigating an intracardiac thrombus, even in minor stroke patients, if the patient has any heart disease and increased hemostatic markers.

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KEY WORDS • blood coagulation disorders • cardioembolic stroke