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(Citation)
The Kobe journal of the medical sciences,41(3):63-70

(Issue Date)
1995-06

(Resource Type)
departmental bulletin paper

(Version)
Version of Record

(URL)
https://hdl.handle.net/20.500.14094/E0000929

Kobe J. Med. Sci. 41, 63/70, June 1995

FOLLOW-UP OBSERVATION OF A PATIENT WITH LEFT VENTRICULAR THROMBUS BY ECHOCARDIOGRAPHY

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INDEXING WORDS

thrombosis; warfarin; embolism; echocardiography; myocardial infarction

SYNOPSIS

We report a case of left ventricular thrombus which caused systemic embolism during warfarin therapy. A 66-year-old man admitted to our hospital with intermittent loss of consciousness and incomplete palsy. The patient had a past history of cerebral infarction, hypertension and diabetes mellitus. Electrocardiography showed abnormal Q waves at II, III, and aVF. Echocardiography showed a mobile protruding thrombus at the left ventricular apex. Left ventricular cineangiography showed a filling defect at the left ventricle. Coronary angiography confirmed total occlusion of the right coronary artery and significant stenosis of the left coronary artery. After administration of warfarin, the patient suddenly fell down to the comatose state. His left arm became pale without pulsation. The thrombus in the left ventricle disappeared by echocardiography and systemic embolism was suspected. Thromboembolectomy from his left arm was performed. Two weeks after the day brain computed tomography showed low density area at the posterior lobe of the cerebrum. Although the eyesight of the patient was lost, he could stand and walk with some help two months later. This is a rare case of systemic embolism during warfarin therapy due to left ventricular thrombus whose process was constantly observed by echocardiography.

Received for publication: June 30, 1995

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INTRODUCTION

Left ventricular thrombosis is one of the causes of systemic embolism including cerebral embolism. Anticoagulant therapy with warfarin is often performed to regress the thrombi. However, systemic embolism is reported in few cases even during warfarin therapy. Two dimensional echocardiography can detect left ventricular thrombi but it is rare that the process of cerebral embolism from intracardiac thrombi is monitored by two dimensional echocardiography during warfarin therapy. We report here such a case of left ventricular thrombus which caused cerebral and left brachial arterial embolism during warfarin therapy.

CASE REPORT

A 66-year-old man was admitted to our hospital with loss of consciousness and weakness of the right arm on Mar. 2, 1993. The patient had a past history of cerebral infarction and its operation in 1979. He was treated for diabetes mellitus and hypertension since 1984 but their control was poor because of his irregular visit to hospital. The weakness of his right arm appeared on Feb. 24, 1993 and he fell down twice because of fainting on Mar. 2. On the same day, loss of consciousness for a few minutes appeared and he arrived at the hospital. Brain computed tomography (CT) showed a low density area at his right lobe which showed no change compared with the previous one. The blood pressure was 226/110 mmHg, and the heart rate was 72/min with regular sinus rhythm. Serum total cholesterol was 305 mg/dl and blood glucose was 203 mg/dl. The chest X-ray film showed the protrusion of left fourth arch and cardiothoracic ratio was 56%. The electrocardiogram showed mild left ventricular hypertrophy and abnormal Q waves at II, III, and aVF. As old myocardial infarction was suspected, echocardiography was performed. Two dimensional echocardiography revealed akinesis of left ventricular apex and about 2 cm-sized mass attaching to the apex (Fig. 1, A and B). The mass was strawberry-shaped and had mosaic pattern. The mass had no stem and the diameter of the attaching site was 8 mm. The mass was 24 x 13 mm measured by two dimensional echocardiography. The blood flow around the mass was very slow assessed by color doppler echocardiography. The mass attached to the akinetic wall but was mobile according to the heart beat. The patient was diagnosed to have an old myocardial infarction and left ventricular thrombus. Left ventriculography and coronary angiography was performed (Fig. 2). Left ventriculography revealed mild dyskinesis of the left ventricular apical portion (from the anterior wall to the inferior wall) and the ejection fraction was 40% (Fig. 2, A and B). A mobile thrombus attached to the dyskinetic apex . Coronary angiography showed total occlusion of the proximal portion of right coronary artery, Ninety nine % stenosis with delayed image of mid left

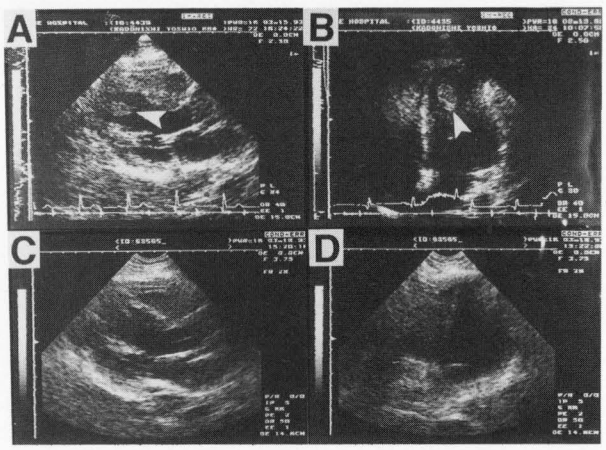

Fig.1. Two-dimensional echocardiography. A and B, before warfarin therapy. C and D, after the embolic event. A, in parasternal long axisis view of left ventricle, left ventricular apex is akinetic and a strawberry-like mass attaches to the apical wall (arrow head). It is mobile according to the heart beat. B, in four chamber view, protruding thrombus is shown more clearly (arrow head). C and D, in both parasternal long axisis and four chamber view, the thrombus has disappeared.

anterior descending artery and 90% stenosis of distal left circumflex artery (Fig. 2, C and D). Treatment with warfarin therapy or thrombectomy together with coronary artery bypass graft was discussed. Because the patient had past history of cerebral infarction and poorly controlled diabetes mellitus with minimum activity of daily living, warfarin therapy was selected. On the 9th and 12th hospital days, the values of thrombotest were 58 and 105%, respectively. On the 12th hospital day, warfarin potassium (3 mg per day) was begun to be administered. On the 14th and 16th hospital days, the values of thrombotest decreased to 74 and 20.5%, respectively. On the 17th hospital day, the patient was sitting on the bed and suddenly lost his consciousness and became in the comatose state. His left upper arm became pale and his left radial artery was not palpable. The pulsation of the arteries of his lower legs was weak and the ankle pressure index was about 10%. The brain CT of the day showed no new findings. The echocardiography clarified the complete disappearance of the left ventricular thrombus (Fig. 1, C and D). Therefore, we concluded that systemic multiple embolism occurred from left ventricular thrombus to his brain and extremities. Thromboembolectomy with Forgaty balloon catheters was performed from his left upper arm and about 5 mm-sized two white thrombi were removed from the left brachial artery. The pulsation of

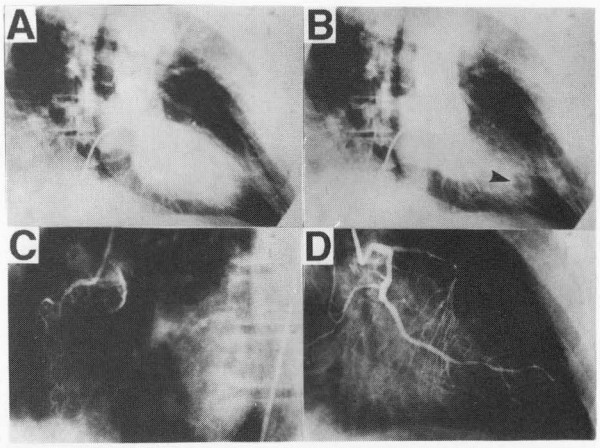

Fig.2. Left ventriculogram and coronary angiogram. A and B, left ventriculogram. A, enddiastolic. B, endsystolic. The apical wall is akinetic and the left ventricular wall around apex is hypokinetic. About 2 cm-sized round defect is mobile according to the heart beat (arrow head). Part of the mass attaches to the wall. C and D, coronary angiogram. C, right coronary artery. Total occlusion of the proximal portion is observed. D, left coronary artery. 99% stenosis with delayed image of mid anterior descending artery and 90% stenosis of distal circumflex artery were observed.

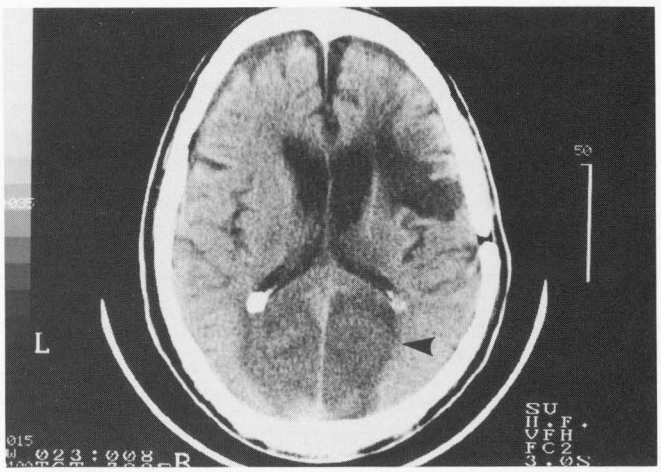

Fig.3. Brain CT. Two weeks after the onset of the embolism, plain brain CT shows the low density area with isodensity area in the posterior lobe of the cerebrum (arrow head). The low density area of right mid lobe corresponds to the area of old infarction.

the arteries of his left arm became well. From the next day, heparin (4,000U per day) was administered for four days. A few days after the administration of heparin, the pulsation of the arteries of his lower legs also became well. The brain CT two weeks after the event showed low density area with partial isodensity area at the posterior lobe (Fig. 3). The electrocardiogram showed no remarkable change except the heart rate during this process. His eyesight was lost but after the rehabilitation for two months, he became alert and was able to walk with some help.

DISCUSSION

Intracardiac thrombosis is sometimes detected in patients with myocardial infarction, chronic atrial fibrillation, cardiac valvular diseases and cardiomyopathy.[1,11,18] About 12.6 to 48% patient of old myocardial infarction were reported to have left ventricular thrombi.[1,6] However, direct observation of the embolic episode is few.[1] Echocardiography especially two-dimensional echocardiography has demonstrated visualization of intracardiac thrombi with a higher degree of accuracy than ventricular angiography.[8,13] Echocardiography also has usefulness on the follow up of treatment. In this case we could detect left ventricular thrombus and diagnosed embolism to the cerebrum and the left arm immediately after detection of disappearance of it . Therefore, it is evident that echocardiography is very useful to detect intracardiac thrombi and pursue the process of the treatment of thrombi.

Concerning warfarin therapy, it is almost accepted that warfarin can prevent embolism from intracardiac fresh mural thrombi especially in myocardial infarction.[4,6] However, there are some contradictory reports that warfarin has no influence on the incidence of embolism in patients awaiting cardiac transplantation, and that warfarin administration without heparin has adverse effect on thrombosis in acute myocardial infarction.[10,12] The proper treatment for patients with chronic left ventricular thrombi remains unknown.[4] The reports about the indication of warfarin therapy with mobile thrombi have been also rare. There are some discussing points getting from echocardiography on the indication of warfarin therapy——mobility, shape and appearance of the thrombi. It is said that mobile protruding thrombi tend to detach from cardiac wall and become emboli.[1,8] The patients who received anticoagulation therapy had lower incidence of embolization than those who did not receive anticoagulation therapy whether the thrombi were layered or pedunculated.[9] The rate of embolic events with warfarin therapy was 0% and that without warfarin therapy was 86%.[9] In 17 acute myocardial infarction patients with left ventricular thrombus, 88% of the thrombus was completely resolved without embolic episodes and the rest of them persisted

unchanged after one-year warfarin therapy.[15] This group contained 6 patients with protruding thrombus. These reports show that warfarin therapy is effective for the resolution of intracardiac thrombi even they are protruding. In our case, it was very difficult to decide to administer warfarin potassium or to operate (thrombectomy with coronary artery bypass graft). The reasons are as follows: (1) the thrombus was protruding with mobility but it was without a stem, (2) the patient has the past history of cerebral infarction, diabetes mellitus and old myocardial infarction with ejection fraction of 40%, and (3) he has rather low activity of daily living. From these findings, we judged that the patient had rather high operative risk. Furthermore, there remains the risk of thrombus formation even after thrombectomy as far as left ventricular aneurysm is not removed. Therefore, we selected warfarin therapy without emergent operation. As a result, embolism to the cerebrum and the left arm occurred.

When the thrombus was born in this case is unknown. However, we suspect the mechanism of thrombus formation in this case as follows: high viscosity and stagnation of blood induced the thrombosis. High viscosity of plasma is related to the poor control of cholesterol and diabetes mellitus in this patient. Both the high total cholesterol level and diabetes mellitus increase plasma viscosity.[2,3,5,20] Serum total cholesterol was 305 mg/dl and blood glucose was 203 mg/dl in this patient. Dehydration might be related to increase the viscosity in this patient. Stagnation of blood at the left ventricular apex was clear by color doppler echocardiography in this case.

Concerning coagulation and thrombolysis, we do not have enough data because of the rather short-term warfarin therapy. However, it is intriguing that as soon as the value of thrombotest became about 20%, embolic episode occurred. This phenomenon indicates that when the coagulation is blocked and the predominance of thrombolysis was built, embolic episode tends to occur. Therefore, it seems that thrombus formation and embolism depends on the balance between coagulation and thrombolysis. As a result, the warfarin therapy promotes the thrombolysis despite of its own inability in thrombolysis.

In our case, the thrombus attached to the cardiac wall directly and was classified as a protruding thrombus with rather free mobility.[9] Yasaka et al. reported 9 patients with intracardiac thrombus treated with warfarin potassium and they described that the thrombus of 8 patients regressed but that in one patient systemic embolization occurred.[19] Five of 9 thrombi were mobile.[12] Therefore, mobile thrombi do not always become emboli. Protruding thrombi with free mobility tend to be emboli more frequently than nonprotruding thrombi with or without warfarin therapy.[8,17] When left ventricular thrombi were observed, it seems prudent to administer anticoagulants to all patients if no contraindications exist.[4,14,16] However, it is not clear whether warfarin therapy decreases the incidence of embolization for protruding or mobile thrombi without

prospective randomized and possibly blinded two-dimensional echocardiographic study.[17]

From these studies, warfarin therapy is effective for resolution of thrombi as a whole but it is very difficult to foresee the embolic episodes during warfarin therapy. Further investigation containing echocardiographic qualitative analysis of intracardiac thrombi is necessary to determine the appropriate indication for warfarin therapy.

ACKNOWLEDGMENT

We are grateful to Dr. Katsuakira Kohno, Department of Anesthesia, Chibune Hospital, for his review and comments on this manuscript.

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