

MOBILE CAROTID ARTERY THROMBUS

Zoltan Bajko^a, Rodica Balasa^a, Sanda Petrutiu^a,

Lucian Toma^b, Simona Russu^a, Ion Pascu^a

^aNeurology Department, Emergency County Hospital, Mures, Romania

^bFirst Surgical Clinic, Emergency County Hospital, Mures, Romania

ABSTRACT

Mobile carotid artery thrombus is an uncommon entity that usually presents as a neurological emergency. The natural history and optimal treatment are unknown.

We present here the case of a 26-year-old male patient with acute right MCA territory infarction. Carotid ultrasound (CU) demonstrated a mobile thrombus in the right internal carotid artery. He was treated with anticoagulation and delayed surgical intervention complicated in the postoperative period with carotid occlusion. Control carotid ultrasound examination after one year revealed partial recanalization of the occluded arteries.

This case underlines the importance of performing ultrasound in the early, hyperacute phase of stroke.

Key words: mobile carotid thrombus, free-floating thrombus, carotid ultrasound, carotid occlusion, carotid recanalization

Mobile thrombus in the carotid artery is a very rare condition with a high risk of embolic cerebrovascular events. Despite the widespread use of high-resolution carotid duplex ultrasonography is rarely recognized before embolic complications. The estimated prevalence is 1 in 2000 ultrasound examinations or less. (1,2,3)

Also difficult to define, the ideal definition of mobile carotid thrombus (free floating thrombus) according to Bhatti et al. would be: "An elongated thrombus attached to the arterial wall with circumferential blood flow at its distal most aspect with cyclical motion relating to cardiac cycles". (4)

The natural history of these lesions and the optimal treatment strategies are unknown. The majority of patients reported in the literature were treated surgically. The immediate surgical intervention (≤ 7 days) occurred most frequently. (4) Carotid duplex sonography is currently the best method to demonstrate dynamic changes of vessel walls and their related structures (2, 5). The often used MRA and CT angiography as first-line diagnostic methods provide only a snapshot image of the thrombus and cannot display the floating character and the change in its position over time.

We report a case of a young stroke patient with a mobile thrombus in the internal carotid artery of unknown origin, who was managed with anticoagulation and delayed surgical intervention complicated in the postoperative period with carotid occlusion.

CASE PRESENTATION

A 26-year-old man was admitted to the emergency room with new onset left sided weakness that had started 60 minutes prior to his presentation. He reported a similar, but transient episode 5 months ago. The patient was a smoker (10 cigarettes/day), had been healthy previously and denied any history of alcohol or drug abuse. The family medical history was negative.

Clinical examination at admission revealed numerous focal dental infections; blood pressure and heart rate were normal (110/70 mmHg, 90/min).

Neurological examination indicated left sided central facial palsy, left sided hemiparesis, diminished reflexes and pyramidal signs in the left upper and lower extremity (NIH Stroke Scale: 11 points).

Author for correspondence:

Zoltan Bajko, Neurology Department, Emergency County Hospital, 50 Gh. Marinescu Street, Mures, Romania
email: bzoltan2003@yahoo.com

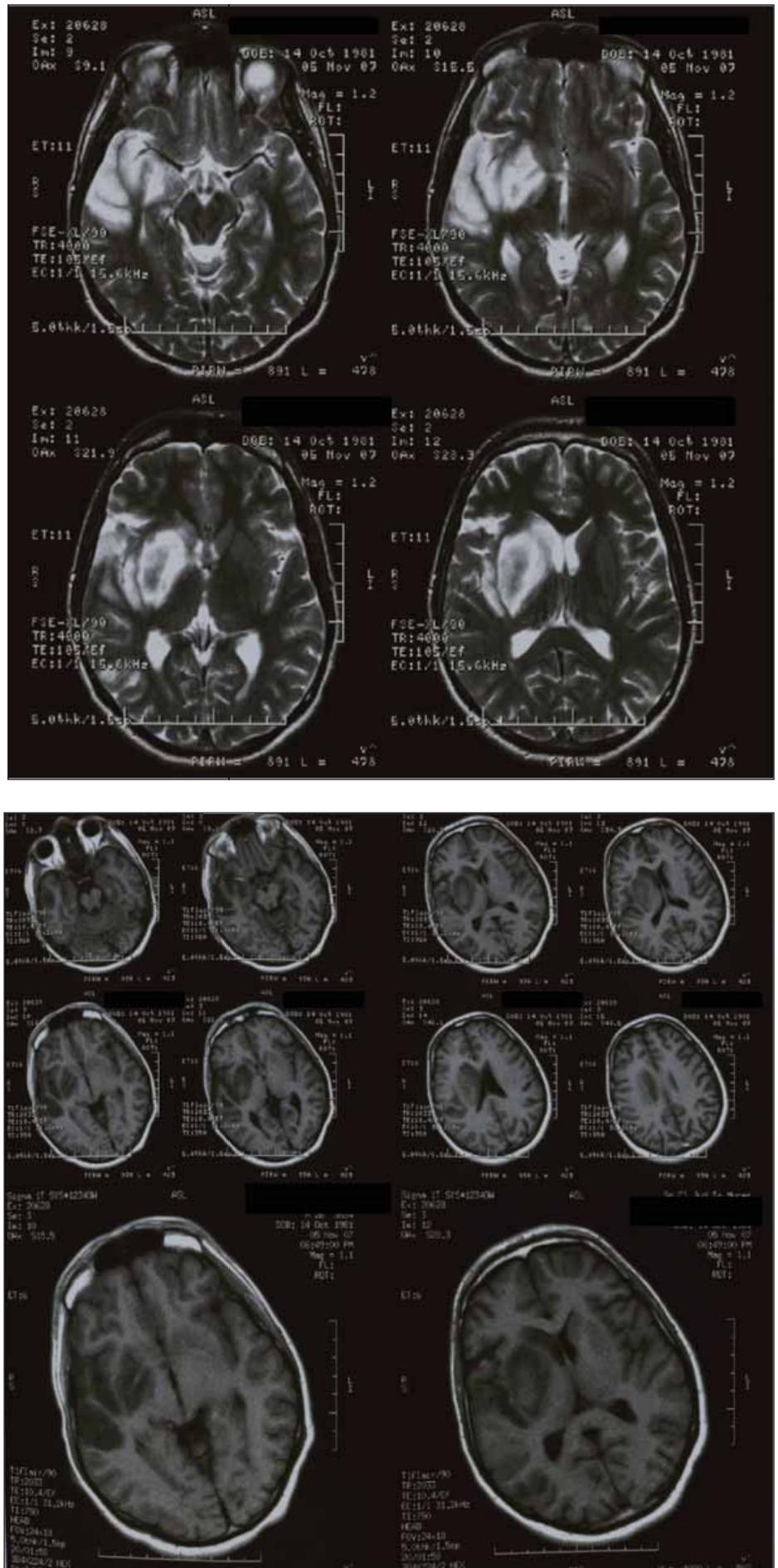


FIGURE 1. Cerebral MRI examination. Right MCA territory infarction.

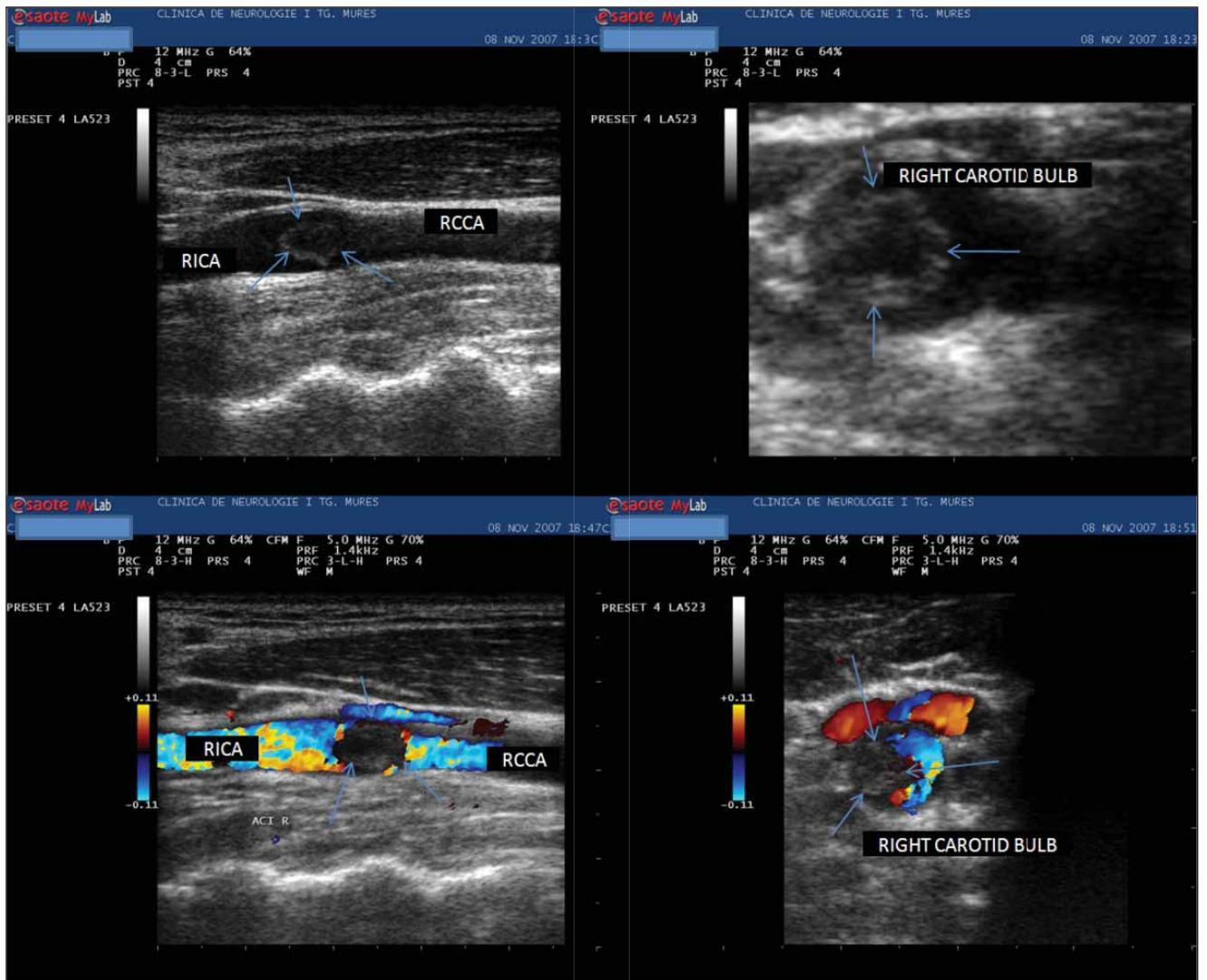


FIGURE 2. Carotid duplex ultrasonography. Mobile thrombus in the right carotid bulb.

Admission CT examination showed no signs of acute ischemia. Cerebral MRI examination the following day revealed an infarction in the right MCA territory (Figure 1). Carotid ultrasound demonstrated a mobile thrombus in the right internal carotid artery (Figure 2). Cardiac work up revealed patent foramen ovale and no other cardiac abnormalities.

Routine laboratory examinations (complete blood count, serum biochemistry, lipid profile) were normal excepting leukocyte count, which indicated leukocytosis ($23,600/\text{mm}^3$). Infectious diseases serology (Anti HIV antibody, VDRL) and autoimmune markers (ANA, ANCA, anticardiolipin ab., anti-dsDNA ab.) were negative. Protein C, protein S and antithrombin III, factor V Leiden mutation, homocysteine level were not performed. Hematological work up and consultation excluded a myeloproliferative disease.

He was treated with LMWH during the first two weeks followed by surgical intervention for thrombus removal. Histopathology confirmed free floating thrombus. The neurological status was stable after surgery.

One week after intervention the carotid ultrasound examination revealed complete occlusion of the right CCA and ICA (Figure 3). The etiology of the hypercoagulability state was not identified after diagnostic work-up. Oral anticoagulation was initiated. The patient was discharged with improved neurological status (Modified Rankin Scale 3). Carotid ultrasound examination after 1 year demonstrated recanalization of right CCA and ICA (Figure 4). The neurological status of the patient was significantly improved (Modified Rankin Scale 1).

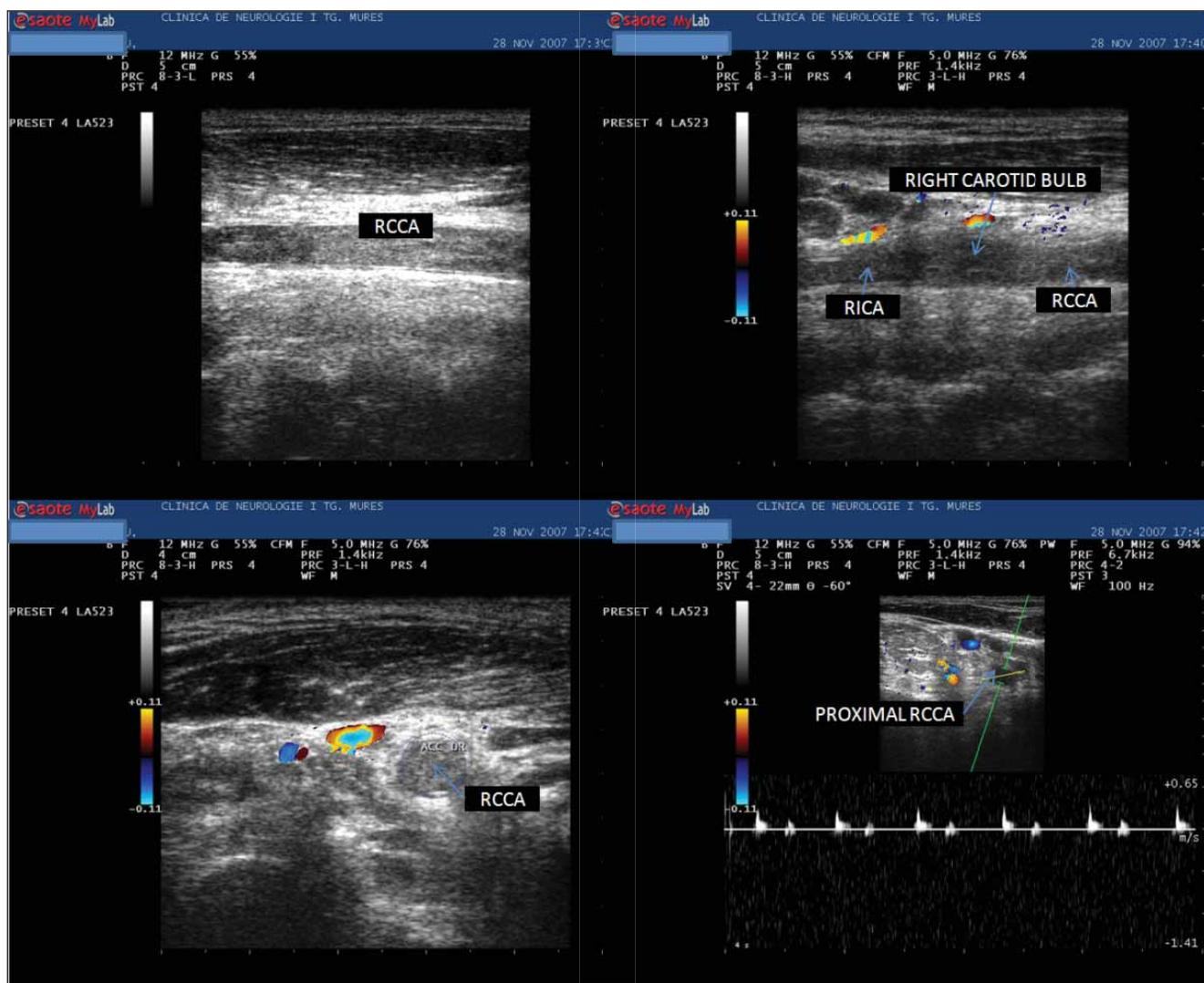


FIGURE 3. Carotid duplex ultrasonography. Complete thrombotic occlusion of the right common and internal carotid artery.

DISCUSSION

Atheromatous plaque complications (plaque ulceration, intraplaque hemorrhage) seem to be the most frequent cause of mobile carotid thrombus. However, spontaneous thrombus formation has been reported in a variety of conditions as autoimmune disorders (6, 7) and different hypercoagulable states. (4) Bhatti AF et al. in their literature review found hypercoagulability in 47% of patients with free floating carotid thrombus tested serologically for this condition (4).

Our patient was not tested for primary hypercoagulability, but leukocytosis could be an explication for acquired hypercoagulable state. There are no extensive literature data dealing with leukocytosis and cerebrovascular thromboembolic events. The role of leukocytosis in ischemic cardiac events is lots better documented (8).

In the former mentioned literature review (4) almost all of the patients were symptomatic, most of them presented transient neurological symptoms, were younger than typically seen with atherosclerotic carotid disease and were predominantly male. Usually the etiology was not clear. The main diagnostic method in this review was angiography because many of the papers predate the era of ultrasound imaging.

High resolution carotid sonography is currently the best diagnostic method for this pathology. Despite the widespread availability of duplex sonography the detection of mobile carotid lesions before embolisation is extremely rare. The explication could be the younger age of these patients placing them outside of high risk population underventing screening examinations.

Szendro G. et al published two asymptomatic cases managed without surgery. The diagnosis was

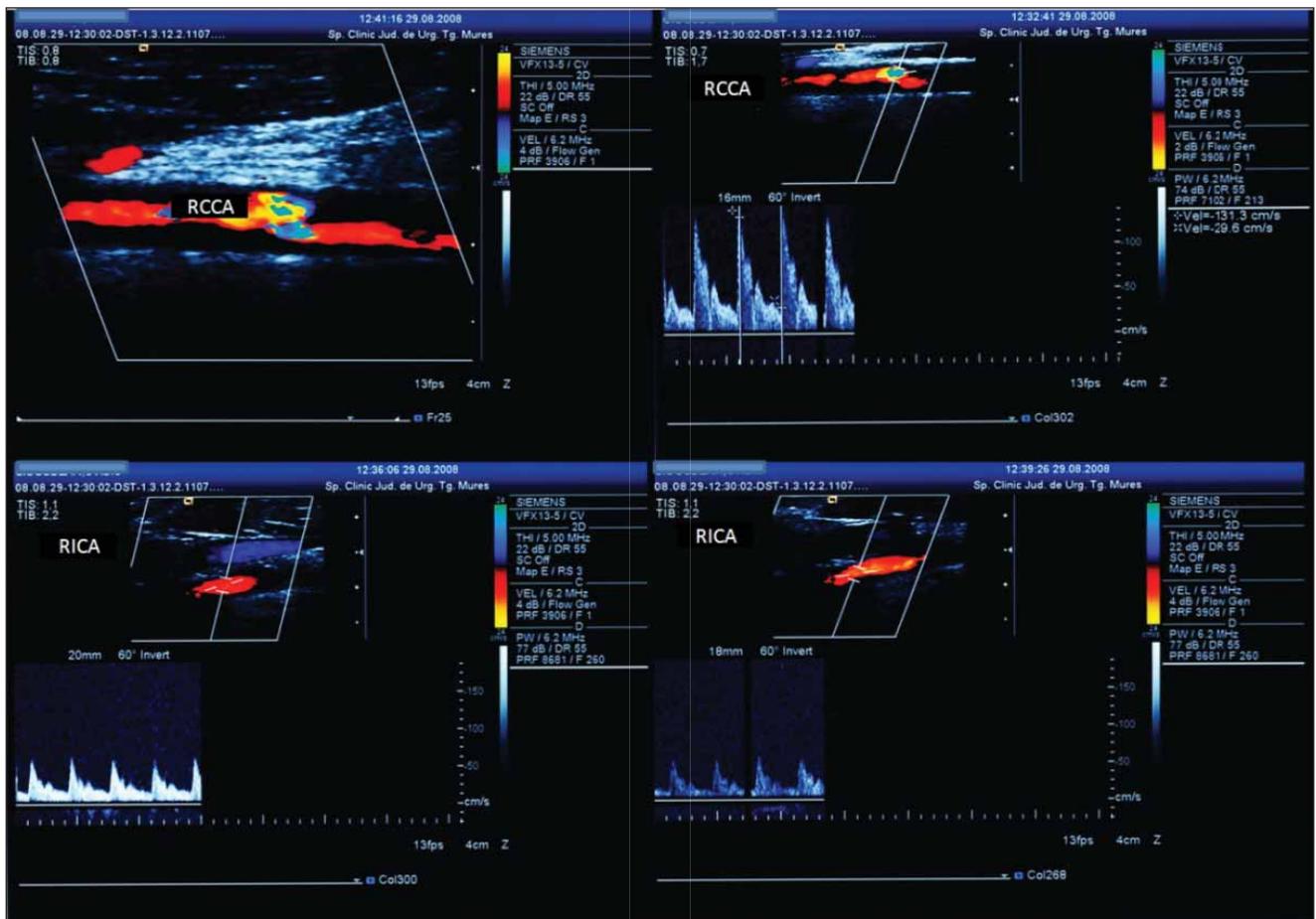


FIGURE 4. Carotid duplex ultrasonography. Partially recanalized right common and internal carotid artery.

established by carotid ultrasound. The examination was performed because of the underlying vascular pathology (coronary heart disease and peripheral artery disease) and the cause of floating thrombus was a complicated atherosclerotic plaque in both cases.

Due to its very low incidence the treatment of mobile carotid thrombus has not been standardized. Some of the authors recommend anticoagulation, others consider this lesions a surgical emergency needing immediate thrombectomy or endarterectomy. (6)

Because of large MCA territory infarction in our case the therapeutic option was delayed surgical intervention after anticoagulation treatment. The occurrence of postoperative CCA and ICA occlusion

in the affected side underlined the hypercoagulability state of the patient.

Spontaneous recanalization of the occluded internal carotid artery has been previously described. (9) The mechanism of the recanalization and the role of antiplatelet and anticoagulation therapy in this process are unknown. Several mechanisms, including spontaneous clot lysis, vasospasm, and distal embolization of occlusive clot have been proposed to explain recanalization. (9)

In summary, we have presented a case of a young stroke patient with two rare phenomenons: free floating carotid thrombus and spontaneous recanalization of the occluded common and internal carotid artery.

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