

SHORT COMMUNICATION

Brain embolism, left atrial thrombi and cardiosurgery

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Patients with embolization into the brain and mobile thrombus in the left atrium (LA) are in the danger of recurrent embolization. A patient with the history of recent cerebral vascular accident (CVA) would be at higher risk of cerebral complications due to cardiopulmonary bypass and this risk may be as high as that of re-embolization.

We present a case of a 41-year old man with an acute ischemic focus (3x3 cm) in the temporoparietal lobe verified by computer tomography (CT). Transthoracic echocardiography showed severe aortic insufficiency, low ejection fraction of dilated left ventricle (LV). Transesophageal echocardiography showed a mobile thrombus (2.2x1.1 cm) in LA. The cardiovascular surgeon consultant did not recommend urgent operation. Instead, the patient was treated by low molecular heparin. CT of the brain after 10 days of treatment was normal. Patient underwent a successful aortic valve replacement. At the time of surgery there was no thrombus in the LA. Subsequently, the patient recovered normally with no neurologic sequelae.

This case illustrates the difficulty arising from the consideration of the relative risks of acute surgery vs conservative management in patient with recent CVA and a large mobile thrombus in LA. (Short communication)

The development of thrombi in a healthy heart is practically impossible. Thrombi develop in coincidence with pathologic states: atrial fibrillation, mitral stenosis, (especially atrial fibrillation), myocardial infarction, left ventricular aneurysm, dilatation cardiomyopathy, endocardial prostheses (artificial valves, electrodes) etc. Less frequently they develop in coincidence with prolapsed mitral valve, aneurysms of atrial septum, left atrial myxoma and endocarditis (1).

The clinical picture of brain embolisation depends on localisation and size of ischemic brain tissue. Vascular brain accidents can be classified as transient ischemic attacks - focal brain dysfunction recovering usually up to 10-15 minutes, or 24 hours at maximum, protracted reversible ischemic neurological deficit, or brain infarction (BI) (7). Without supplementary examinations (CT, or magnetic resonance), the assessment of BI diagnosis in coincidence with transient symptoms, can be very difficult.

Case description

A 41-year old patient was admitted into a regional internal clinic after previous TIA. Subsequently, after echocardiographic examination revealing dilated heart compartments with a suspected valvular defect, he was sent to a more specialised clinic. The

diagnosis of TIA was linked with embolisation of possible vegetation from the fibrotic aortic valve, or thrombi from dilated heart compartments. Paradoxical embolisation was taken into consideration in coincidence with the history of possible recent profound thrombophlebitis of the lower leg 4 weeks ago, as well as dysrhythmia.

Case history: a half-year lasting history of exercise-induced dyspnea with progression for the past two months (occurring during shaving, putting on shoes, or minor housework) with palpitations and night dyspnea. On the 19th Aug 2000, after a coughing attack, he collapsed. He had dysarthria and felt weakness in his left upper and lower limbs. The called physician stated his suspicion of TIA within the vascular bed of the right carotid. The patient refused hospitalisation.

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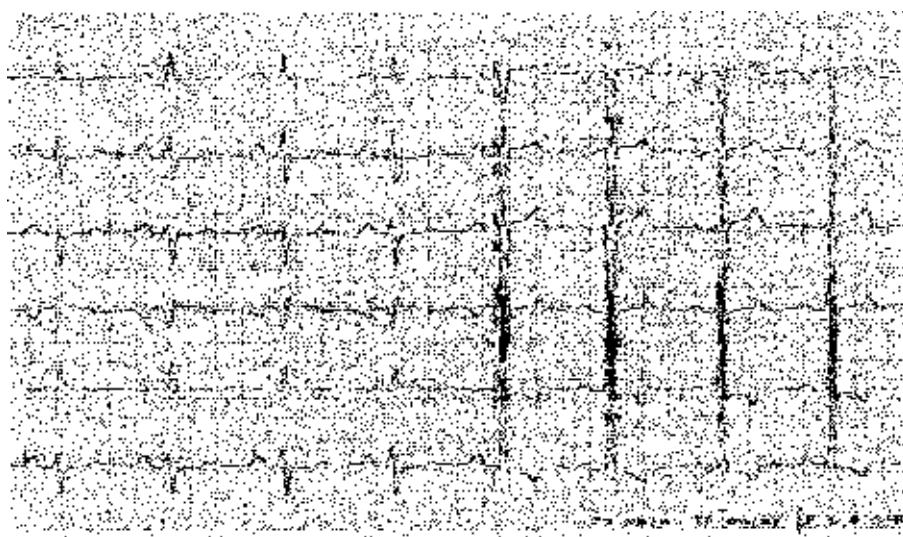


Fig. 1. Hypertrophy and overloading of left ventricle and both atria.

On the 21st Aug 2000 he was admitted to a regional hospital with the symptoms of large vascular circuit stasis. For two days he was treated with cardiotonic, vasodilating and diuretic therapy. On 23rd Aug 2000 he was sent to our department for consultation examination.

Objective examination: The patient with asthenic habitus, 186 cm tall, weight of 74 kg, with no signs of neurologic deficit or heart insufficiency. Blood pressure 135/50 mmHg, pulse 66/min, diastolic murmur over aorta, parasternal murmur on the left 3/6, varices in both lower legs with no signs of inflammation.

The electrocardiogram showed sinus rhythm, hypertrophy, and left ventricular and atrial overloading (Fig. 1). Normal hematologic, blood clotting and biochemical parameters. Echocardiogram showed dilatation of the left ventricle with decreased global systolic function and ejection fraction of 35 % (Fig. 2). Other selected echocardiographic parameters are given in Table 1.

Transesophageal examination has confirmed severe regurgitation on the fibrotic aortic valve 3/4 (Fig. 3), the valves were with no vegetation. There was no shunt on the atrial level (Fig. 4), the left atrial auricle contained mobile thrombi (Fig. 5).

The patient at an assumed high risk of new embolisation was admitted at the Coronary Care Unit of SÜSCH. CT confirmed TIA by revealing a large temporoparietal ischemic malacic focus on the right within the vascular bed of the medial cerebral artery

(Fig 6). On the day of admittance, assuming a need of cardiosurgical intervention, the patient was dentally defocused. On the 24th Aug 2000, we consulted the patient's cardiosurgical state as to the possibility of acute extirpation of thrombi from the left atrial auricle, and valve replacement.

Discussion

1. The patient after embolisation into the brain, with left atrial thrombi and subsequent high embolisation potential, is endangered by the possibility of recurrent embolisation.

2. The shortness of time elapsing from embolisation and a fresh ischemic malacic focus represent high risk arising from surgery using extracorporeal circulation:

- a) spreading of ischemia due to change in perfusion pressures during intervention,

- b) bleeding into the focus due to hypocoagulation state during heparinisation.

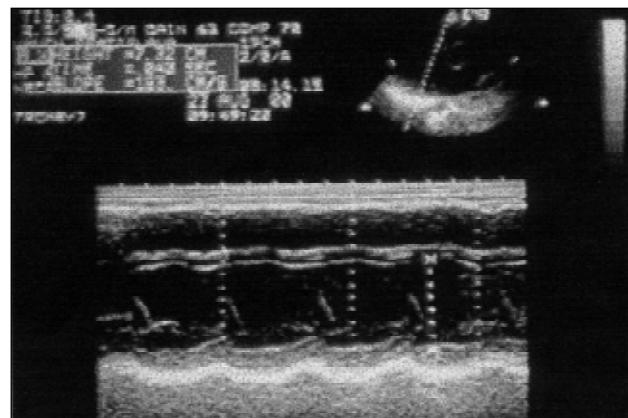


Fig. 2. Dilated left ventricle, EF 35 %.

Tab. 1. Some TTE parameters.

LVEDD	- left ventricular end-diastolic diameter	73 mm
LVESD	- left ventricular end-systolic diameter	49 mm
EF	- ejection fraction	35 %
AoI	- aortic insufficiency	3/4
Asc.Ao	- ascending aorta	37 mm
MI	- mitral insufficiency	2/4
LA	- left atrium	45 mm

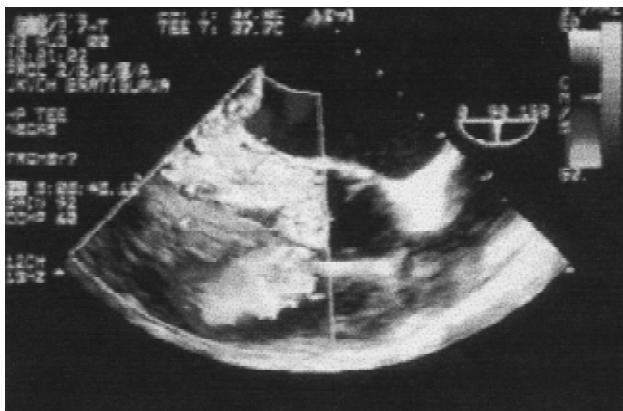


Fig. 3. Aortic regurgitation 3/4.



Fig. 5. Mobile thrombus within the left atrial auricle.

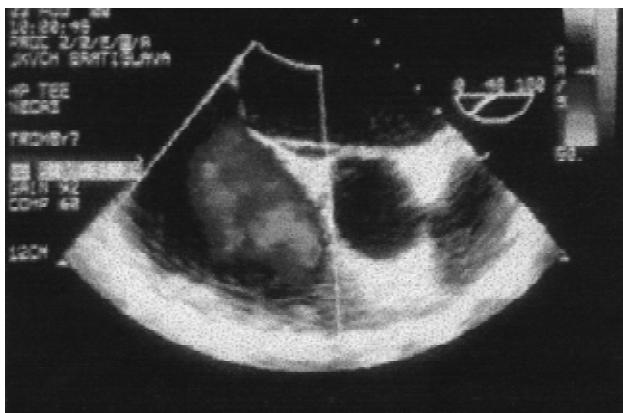


Fig. 4. No right-left shunt.

3. The risk of bleeding into the brain infarction, arising from fibrinolytic therapy for the purpose of lysis of left atrial thrombi in a patient with fresh brain infarction is as high as that arising from anticoagulation therapy after the replacement of aortic valve by valvular prosthesis in the same patient.

The presence of mobile thrombi within a regular sized atrium in coincidence with insignificant mitral regurgitation and sinus rhythm, as well as with a decreased function of dilated left ventricle, and insignificant aortic regurgitation, is rare. A CT-verified brain infarction, and the presence of a thrombus within the left atrial auricle in a young patient without the history of hypertension and thromboembolism in the past, with normal blood clotting conditions, nearly certainly excludes any other cause of embolisation. In this case, dysrhythmia is also merely hypothetical.

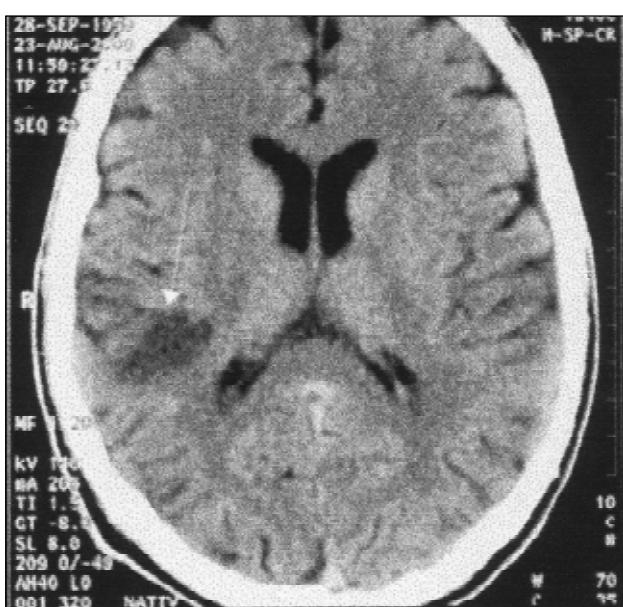


Fig. 6. Temporoparietal ischemic malacic focus — on the right.

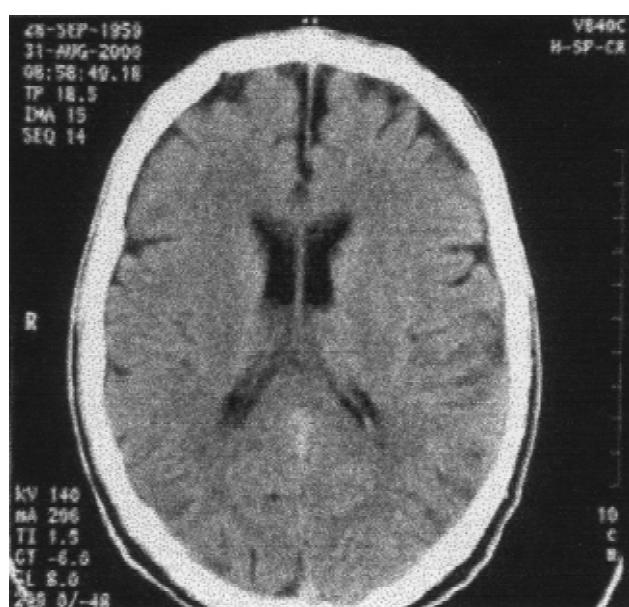


Fig. 7. Normal CT picture.

Cardiologic and cardiosurgical monographies do not provide a general consent as to the precise instruction of how and when to operate on a patient with vascular accident. It is generally recommended to perform the surgery on a stabilised patient, i.e. to delay the cardiosurgical intervention. In case of an inevitability of cardiosurgical intervention during recent cerebral vascular accident, some authors recommend to decrease the risk of operation by means of „minimal invasiveness“, i.e. so-called minimum invasive cardiosurgery without extracorporeal circulation (5). The experience from early solutions of embolic complications of infectious endocarditis favours urgent surgical proceeding. The authors (6,3,4,2) indicate that surgical results improve especially in groups of patients at high risk of re-embolisation. The question as to what formation (vegetation, or thrombus, as in our case) endangers the patient by recurrent embolisation, stays open. Its mobility and size play the crucial role. However, no one can guarantee that any formation, which has once caused embolisation, will not cause any recurrent embolisation in the future. During such early surgeries with extracorporeal circulation, the authors recommend higher perfusion pressures, more thorough observation of blood clotting and protection of the brain by means of hypothermia.

In our case, the consulting surgeon has not recommended an acute surgical solution. Full anticoagulation therapy with low molecular heparin (deltaparin) represented a compromise opposed to the conservative fibrinolytic therapy by streptokinase, or t-PA. The control CT examination performed on 31st Aug 2000 proved that the brain ischemia had healed entirely. (Fig 7). Subsequently the patient underwent surgery. The aortic valve was replaced by an artificial valvular prosthesis St. Jude No 23. No thrombi were found in the left atrial auricle during operation.

References

1. Gregor P, Widimský P et al: Kardiologie. Praha, Galén 1999, p. 477—478.
2. Horstkotte D, Piper C, Wiemer M, Arendt GI, Steinmetz H, Bergmann R, Schutte HD, Schuftheiss HP: Emergency heart valve replacement after acute cerebral embolism during florid endocarditis. Med Klin 1998; 93 (5): 284—293.
3. Nakamura K, Onitsuka T, Kuwabara M, Nakasima S, Araki K, Yano Y, Mayaset T, Matzusaki Y: A case of successful surgical treatment for active endocarditis 2 days after the onset of cerebral infarction. Japan Kyobu-Geka 1997; 50 (4): 304—307.
4. Piper C, Arendt G, Schulte HD, Korfer R, Hetzer R, Horstkotte D: Acute stroke in infective endocarditis is not a contraindication for urgent valve replacement. Cor europaeum 1999; 8 (1): 38.
5. Sakakibara Y, Nakata M, Sasaki A, Emonoto Y, Osaka M, Mitsui T: Minimally invasive direct coronary artery bypass grafting in a patient with brainstem infarction. Jap J Thorac Cardiovasc Surg 2000; 48: 229—232.
6. Schunemann S, Werner GS, Schultz R, Bitsch A, Prange HW, Kreuzer H: Embolic complications in bacterial endocarditis. Z Kardiol 1997; 86 (12): 1017—1025.
7. Štejfa M et al: Kardiologie. Praha, Grada Publishing 1998, p. 398—399.

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