Simulated Myocardial Infarction and Slow Atrial Flutter Due to Cerebral Embolism from a Free Left Atrial Thrombus

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SUMMARY

The case of a 38-year-old female who suffered a cerebral embolism from a free left atrial thrombus is reported. The clinical picture, including serum enzyme elevations, was consistent with a nontransmural myocardial infarction. Atrial flutter exhibiting very slow (162/min) and tall waves was transiently recorded indicating development of an intratral conduction disturbance.

Additional Indexing Words: Atrial flutter Cerebral embolism Acute myocardial infarction

It has long been known that acute cerebral injury can provoke cardiovascular abnormalities, especially disturbances in rhythm and conduction and repolarization abnormalities. However, the recognition of possible damage to the myocardium in response to cerebral injury, notably severe brain injury due to craniocerebral trauma\textsuperscript{1,2} is recent. Although most reports in this context have dealt with the development of various arrhythmias and ST-T abnormalities as well as a prolonged QT-interval\textsuperscript{3,4} evidence for myocardial damage, manifested by a rise in serum cardiac enzymes (CK-MB) and subendocardial hemorrhage\textsuperscript{1,3,5} has also been reported.

This communication further supports this concept by describing a young female patient with mitral stenosis in whom criteria for myocardial infarction were met in the course of cerebral embolism originating from a free-floating left atrial thrombus. The atrial flutter observed at onset of the event was of further interest because of its extremely slow rate suggesting the association of an intratral conduction disturbance.

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CASE REPORT

A 38-year-old housewife was admitted to the Neurology Department of the Cerrahpasa Medical Faculty on June 16, 1989 because of the development on the same day of transient loss of consciousness followed by aphasia and lack of balance. Dilation of the left pupil and right facial paralysis with minimal right hemiparesis were the major neurologic findings. Computed axial tomography scan failed to show a process causing the clinical picture other than cerebral infarction. Impure atrial flutter (at a rate of 320/min) with 2:1 AV conduction was observed on the ECG. Due to recurrence of sudden loss of consciousness and low blood pressure (55/mmHg) on the morning of June 21st, she was transferred to the coronary care unit of the Medical Department with the suspicion of acute myocardial ischemia secondary to coronary embolism from preexisting atrial fibrillation and mitral stenosis.

The past history revealed that she had undergone mitral commissurotomy in 1977. Her exercise tolerance had diminished in the last few months. For years, she had been on oral digoxin, 0.25 mg daily until June 16th. She complained of dizziness, a sensation of heat in the chest and of sweating on admission, when dilation of the left pupil, mild right facial paresis and dulled sensorium persisted. The blood pressure ranged around 95/75 mmHg and pulse 90/min. An opening snap and diastolic rumble were audible at the cardiac apex. The ECG showed an unusually slow (162/min) atrial flutter with tall flutter waves in lead III accompanied by 2:1 AV conduction; in addition, 3-5 mm depressions of the ST segment were noted in leads V₃ through V₅ and QTₐ was prolonged to 0.46 sec (Fig. 1). Serum enzymes were elevated moderately that morning: SGOT to 65 U, CPK to 245 U/ml (n≤80), and creatine kinase-MB to 31 U/L (n=9.6). M-mode and 2-D echocardiography demonstrated enlargement of the left atrium in which a thrombus (4×4.5 cm in size) moved freely, and mitral stenosis (2.1 cm² valve area estimated by Doppler echo).

It was thought that cerebral embolism, in the absence of coronary embolic occlusion, accounted for all the clinical and laboratory findings. While the patient was anticoagulated with heparin and preparations for cardiac surgery were undertaken in the next days, her rhythm varied between atrial fibrillation and impure atrial flutter (rate around 380/min) and the ST-depressions regressed to 1.5–2 mm. She underwent open heart surgery on June 29th during which the free atrial thrombus was removed and the mitral valve was excised and replaced by a St Jude mechanical valve. The pathological examination of the mass (6×5×3 cm) showed a fresh
thrombus whereas that of the mitral valve revealed diffuse hyalinization, focal areas of vascularization and inflammatory reaction. The patient was subsequently discharged in a satisfactory condition with the recommendation to continue treatment with coumadin.

**DISCUSSION**

Of interest are two features of the presented case of cerebral embolism secondary to a huge free thrombus in the left atrium: a) suggestive evidence of associated acute myocardial infarction, b) the unusually slow rate of atrial flutter transiently developing in the course of atrial fibrillation.

Cases of cerebrovascular accident\(^4\),\(^5\) or of acute craniocerebral injury\(^1\),\(^2\),\(^6\) which displayed a clinical picture suggesting or simulating an AMI, including a rise of serum enzymes have rarely been reported. Some of these were in older patients so that isoenzyme rises might have represented a true accompanying AMI caused by coexisting coronary artery disease.\(^7\) Another group of these patients comprises those who exhibit electrocardiographic changes and elevation of serum enzymes, in the absence of a rise in CK-MB, as a direct consequence of cerebral insult.\(^4\),\(^8\) Finally, the third subset of patients, including our case, demonstrates evolutionary ECG changes associated with a rise in serum cardiac enzymes indicating the development of myocardial damage, yet, without any evidence of myocardial infarction.\(^7\),\(^9\) It is thought that the “catecholamine storm” coupled with release of norepinephrine at cardiac beta\(_1\) receptor sites is responsible for the myocardial damage.\(^2\),\(^10\)

Atrioventricular and intraatrial conduction disturbances were evidenced in our case by 2:1 AV conduction accompanying a very slow atrial flutter (162/min) immediately following the cerebrovascular accident and persisting for several hours before reverting to atrial fibrillation. In a retrospective
study correlating the site of cerebrovascular hemorrhage in 65 patients with ECG abnormalities, Yamour et al\(^3\) found first-degree AV block in only 3 patients having basal ganglia or occipital hemorrhage. Mobitz type II AV block has been reported in a patient with subarachnoid hemorrhage simulating acute inferior myocardial infarction.\(^4\)

Common atrial flutter, type I, has a spontaneous rate which may be as low as 230/min, although antiarrhythmic drugs such as quinidine and amiodarone may reduce the rate to the range of 200/min.\(^11\) Yet, the herein described patient was receiving no antiarrhythmic drug at the time of the cerebral embolism or the arrhythmia. Thus, the extreme slowness of atrial flutter here must have been a result of the cerebrovascular accident presumably leading via neurohumoral effects to a prolongation of the refractory period within the atrial circuit. The atrial activation during flutter proceeded very slowly forwards and up toward the left arm, and rapidly backwards and down to the right.

**REFERENCES**