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Cardiac Rupture Complicating Cerebral Intraarterial Thrombolytic Therapy

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Summary: We report a case of fatal cardiac rupture occurring during intraarterial thrombolytic therapy for acute embolic stroke in a patient with recent myocardial infarction.

Index terms: Interventional neuroradiology, complications; Thrombolysis

Early intraarterial thrombolytic therapy is recognized as a valuable treatment option in the setting of acute stroke. Cardiac rupture is a catastrophic complication of myocardial infarction. The risk of this complication has been shown to increase with late coronary thrombolytic therapy (more than 7 hours after onset of myocardial infarction) (1) and has also been reported as a complication of cerebral intraarterial thrombolytic therapy (2).

Case Report

A 71-year-old woman was admitted to the coronary care unit approximately 24 hours after the onset of acute myocardial infarction confirmed by an electrocardiogram and enzyme parameters. Coronary thrombolytic therapy was not pursued owing to the delayed presentation. Her progress was uneventful until day 7, when she experienced acute onset of left-sided hemiparesis.

A computed tomographic (CT) scan at this time revealed subtle sulcal effacement throughout the territory of the right middle cerebral artery (MCA) but no evidence of intracranial hemorrhage (Fig 1A). Cerebral angiography was performed 4.5 hours after the onset of hemiparesis as a prelude to intraarterial thrombolytic therapy. The right MCA was found to be occluded 4 mm distal to the internal carotid bifurcation, with a well-defined cap deformity, characteristic of an embolus (Fig 1B).

A Tracker-18 Unibody catheter (Target Therapeutics, Fremont, Calif) was navigated into the MCA thrombus over a 0.014-inch Dasher guidewire (Target Therapeutics). Intraarterial thrombolysis was begun with urokinase, using the standard pulse-spray technique. A total of 1 million units of urokinase was delivered over a period of 1 hour. After lysis, a right internal carotid angiogram revealed a patent M1 segment with only minimal narrowing, consistent with a small volume of residual embolus (Fig 1C). The superior division of the right MCA and lenticulostriate branches were patent, with no evidence of distal emboli. The inferior MCA division remained occluded. Minor neurologic improvement was observed 1 hour after the start of thrombolysis.

Within 5 minutes of the postlysis angiogram, the patient became apneic and profoundly hypotensive consequent to complete electromechanical dissociation. Her hemodynamic status continued to decline, and attempts at resuscitation were unsuccessful.

Postmortem examination revealed rupture of the anterior myocardial wall through a large zone of subacute transmural infarction with massive hemopericardium. The right cerebral hemisphere showed changes of early infarction in the MCA territory with only minute foci of petechial hemorrhage and no parenchymal hematoma.

Discussion

Intraarterial thrombolytic therapy is a recognized treatment option in the setting of acute thromboembolic stroke. Early techniques included intraarterial urokinase infusion into the carotid or vertebral artery (3, 4). More recent studies have shown the efficacy of direct delivery of urokinase into thrombi through a microcatheter system. This method allows delivery of highly concentrated thrombolytic agent directly into the thrombus as well as mechanically disrupts the thrombotic mass (2). The major complication associated with both the early and more recent techniques is hemorrhagic conversion of the zone of infarction, which has a reported frequency of 14% to 25%. The majority of

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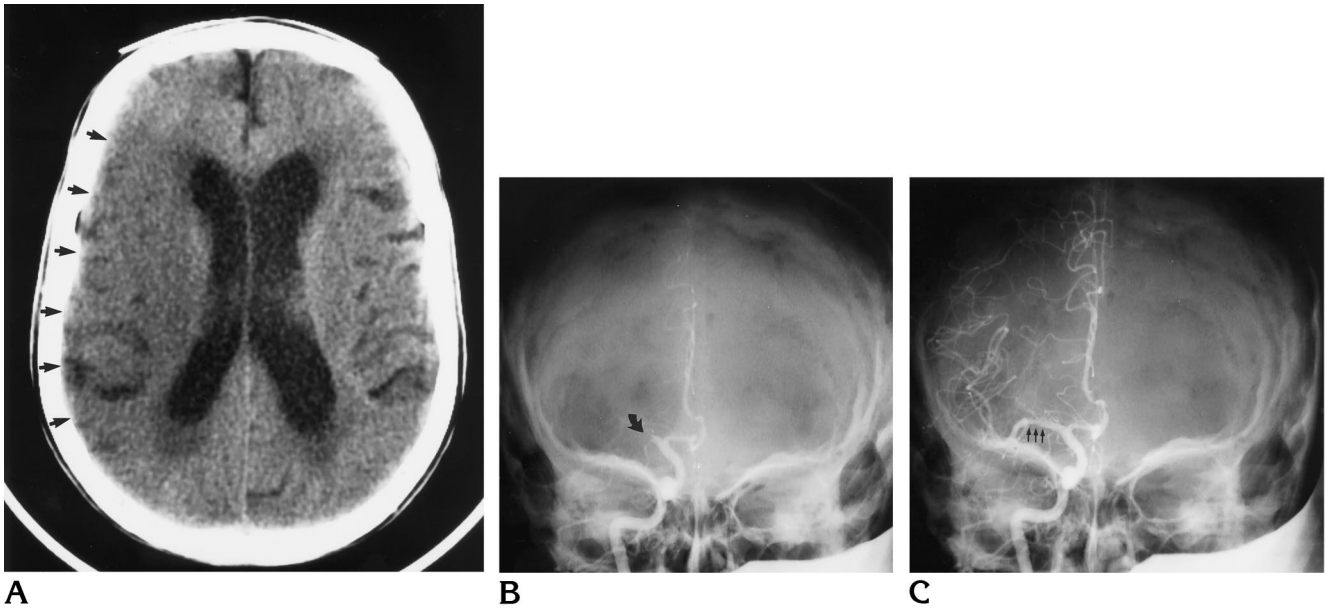

Fig 1. A 71-year-old woman with acute myocardial infarction.

A, Noncontrast CT scan shows subtle sulcal effacement throughout the right MCA territory (*arrows*).

B, Right internal carotid artery angiogram, anteroposterior projection, shows the right MCA is occluded 4 mm distal to its origin, with a cap deformity characteristic of an embolus (*arrow*).

C, After thrombolysis, right internal carotid artery angiogram, anteroposterior projection, shows the M1 segment of the right MCA is patent, with only minimal narrowing, compatible with only a small volume of residual thrombus (*arrows*).

patients with documented hemorrhagic conversion suffer no clinical deterioration (2).

Cardiac rupture is caused by dissection of blood through infarcted myocardium; it occurs in approximately 4% of patients admitted to the hospital with acute myocardial infarction. Risk factors include lack of previous angina or myocardial infarction, hypertension during the acute stage of infarction, and female sex (5).

Coronary thrombolytic therapy is widely used in the management of acute myocardial infarction. This form of treatment has been demonstrated to improve survival if administered during the first few hours of myocardial infarction (6, 7). A potentially serious complication of coronary thrombolytic therapy is conversion of bland infarction to hemorrhagic infarction. This phenomenon may lead to cardiac rupture by dissection of blood through zones of full-thickness infarction (8—10). Cardiac rupture may occur as a complication of selective cerebral thrombolytic therapy (2).

In a study of 1638 patients treated with thrombolytic therapy for acute myocardial infarction, Honan et al (1) reported an increased risk of myocardial rupture with late administration of coronary thrombolytic therapy, with a direct correlation between time to thrombolytic therapy and risk of myocardial rupture (1). This relationship was independently confirmed by the GISSI trial (11). In both studies, the odds ratio of cardiac rupture (treatment/control) increased significantly with time to treatment. Beyond 11 hours after onset of myocardial infarction, the risk of myocardial rupture became significantly higher in the group treated with thrombolytic therapy as compared with the control group. The upper limit of this period of increased risk of cardiac rupture remains unclear (1).

In another, more recent, review of 350 755 patients with acute myocardial infarction, of whom 122 243 received thrombolytic therapy, Becker et al (12) confirmed an increased risk of cardiac rupture in the group managed with early thrombolytic therapy (mean time to treatment from onset of chest pain was 4.7 hours). This study suggests that thrombolytic therapy promotes cardiac rupture within 24 to 48 hours of treatment. Cardiac rupture was the cause of 12% of in-hospital deaths in the treated group compared with 6% of in-hospital deaths in the group not receiving thrombolytic therapy. Despite the increased risk of cardiac rupture, the overall mortality remained significantly lower in the group managed with thrombolytic therapy.

Careful clinical evaluation should be exercised on a case-by-case basis with regard to cerebral thrombolytic therapy in the setting of recent myocardial infarction. As yet, the data are insufficient to predict accurately the risks of cardiac rupture from cerebral thrombolytic therapy in this group of patients or to compare these risks with the outcome of untreated MCA embolic stroke.

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