Local Intraarterial Fibrinolytic Therapy for Embolic Stroke Associated With Vascular Anomalies

—Two Case Reports—

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Abstract

Two cases of embolic stroke associated with vascular anomalies were treated by intraarterial fibrinolytic therapy. A 78-year-old male had embolic stroke associated with fenestration of the middle cerebral artery (MCA). The embolus lodged in the terminal portion of the right internal carotid artery (ICA) and in the M₂ portion of the right MCA. The fenestration was not detected before local fibrinolytic therapy, but was identified by postinterventional angiography. An 82-year-old female had a left-sided persistent primitive trigeminal artery (PPTA), and the embolus lodged in the terminal portion of the left ICA. Left internal carotid angiography also showed rich collateral circulation via the abundant leptomeningeal anastomosis. The PPTA involved the left ICA, so the anomalous anatomy had to be considered in performing local thrombolysis. However, the rich collateral circulation allowed recanalization of the occluded ICA without inducing bleeding. Dissolution of persistently adherent emboli must be performed with great care because of the possibility of such rare vascular anomalies.

Key words: persistent primitive trigeminal artery, fenestration of middle cerebral artery, embolic stroke, fibrinolysis

Introduction

Recently, local intraarterial fibrinolytic therapy has become widely used for the treatment of embolic occlusion in cerebral arteries with a recanalization rate of 60%.¹¹⁾ Advanced methods such as central intraarterial fibrinolysis and percutaneous transluminal angioplasty can be used to treat high-risk patients with occluded vessels, including the middle cerebral artery (MCA) and the internal carotid artery (ICA).^{7,10)} However, these techniques may involve great risk if the occluded artery has a malformation, since the guide wire and the microcatheter must be blindly passed beyond the embolus. We describe two cases of embolic stroke with an associated cerebral arterial anomaly, fenestration of the MCA in one case, and persistent primitive trigeminal artery (PPTA) in another.

Case Reports

Case 1: A 78-year-old male was admitted to our

hospital approximately 10 minutes after onset of stroke. He presented with left hemiplegia and coma. Computed tomography (CT) showed no abnormalities. Right carotid angiography revealed thrombus in the most distal portion of ICA, but the right anterior cerebral artery (ACA) and MCA were filled with contrast medium via a crevice between the embolus and the arterial wall. Occlusive embolus at the trifurcation of the right MCA was also visualized (Fig. 1A).

We performed emergent dissolution of the embolus by urokinase infusion directly into the right MCA and the ACA beyond the embolus at the tip of the ICA. After administration of 840000 IU of urokinase, the right MCA trifurcation became free of thromboemboli. Some residual embolus remained in the proximal portion of the MCA (Fig. 1B), but fibrinolytic therapy was terminated to avoid urokinase-induced bleeding.

After therapy, his level of consciousness improved, and the hemiplegia resolved. On the next morning, no paresis had recurred. CT performed 24 hours after thrombolysis showed a small hypodense area in the right putamen (Fig. 2). Seven days later,

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Fig. 1 Case 1. **A:** Right carotid angiogram before intervention revealing a thrombus in the distal internal carotid artery, and filling of the right anterior cerebral artery and middle cerebral artery (MCA) via a crevice between the embolus and the vascular wall. An occlusive embolus at the trifurcation of the right MCA is also demonstrated. **B:** Right carotid angiogram after local intraarterial administration of urokinase showing the right MCA trifurcation is free of thromboemboli, but residual embolus is still present in the proximal portion of the MCA. **C:** Cerebral angiogram 1 week after intervention disclosing absence of emboli and the fenestration of the MCA.

Fig. 2 Case 1. Postintervention computed tomography scan showing a small low-density lesion in the right putamen (arrow).

postinterventional angiography showed no residual embolism in the MCA, but a fenestration was noted in the proximal portion of the right MCA (Fig. 1C). The patient was discharged 14 days later with no neurological deficits.

Case 2: An 82-year-old female was admitted in a somnolent state 1 hour after sudden onset of com-

plete right hemiplegia. CT showed no abnormalities. Left carotid angiography revealed complete occlusion beginning in the terminal portion of ICA, as well as a left-sided PPTA (Fig. 3A, B). The left posterior cerebral artery (PCA) was supplied mainly by the left ICA via the dilated left posterior communicating artery. A rich leptomeningeal anastomosis between the left PCA and the left MCA was also revealed (Fig. 3C). The basilar artery, the bilateral superior cerebellar arteries, and the right PCA were supplied mainly by the left ICA via the PPTA.

Three hours had passed since the onset of symptoms, so emergent local intraarterial fibrinolytic therapy was performed via a coaxial catheter with the tip positioned in the middle segment of the ICA. Over 60 minutes, a total of 480000 IU urokinase was administered. Subsequent angiography showed that the occluded arteries were completely recanalized (Fig. 3D, E). Total elapsed time from the onset was approximately 5 hours.

Her level of consciousness improved immediately after treatment. Postoperative CT showed a small hypodense area in the anterior limb of the internal capsule (Fig. 4). The patient was discharged 30 days later with no neurological symptoms.

Discussion

Fenestration of the MCA is a rare malformation with a prevalence ranging from 0.26% to 0.44%.^{4,8)} The pathogenesis of MCA fenestration is not completely understood, but this anomaly is thought to

Fig. 3 Case 2. A, B: Left internal carotid angiograms, anteroposterior (A) and lateral views (B), showing the left posterior cerebral artery (PCA) via the posterior communicating artery, and filling of the right PCA and bilateral superior cerebellar arteries via the persistent primitive trigeminal artery. The left anterior cerebral artery and left middle cerebral artery (MCA) are not visualized, indicating embolic occlusion of the left internal carotid artery. C: Left internal carotid angiogram, anteroposterior view in the venous phase, showing rich collateral circulation in the left MCA area via the leptomeningeal anastomosis. D, E: Left internal carotid angiograms, anteroposterior (D) and lateral views (E), showing the occluded arteries have completely recanalized after intervention.

Fig. 4 Case 2. Postintervention computed tomography scan showing a small low-density lesion involving the anterior limb of the left internal capsule (arrow).

represent persistence of the plexiform network, which the primitive MCA forms with the ICA on the 35th day of embryonic development.⁹⁾ The specific site of the fenestration was the M₁ portion in 19 cases, the M₂ portion in one, and not stated in one.^{1,6)} Fenestration of the M₁ portion can be classified into three types: proximal, intermediate, and distal.⁸⁾ The proximal type of M₁ fenestration is most common.

Association with MCA aneurysm is the principal clinical significance of fenestration of the MCA. About half of the cases of fenestration of the MCA are associated with aneurysm.⁶⁾ Proposed causal

links to aneurysm formation include altered hemodynamics due to the fenestration as well as fragility of vascular endothelium in the region of the fenestration.⁶⁾ MCA fenestration has been linked to cerebral infarction in two cases.⁷⁾ Abnormal hemodynamics from the fenestration were considered to be the cause of infarction of the penetrating branch region. In our first case, a residual portion of embolus in the MCA was fixed to the arterial wall, possibly because of trapping by the fenestration. Postinterventional angiography showed spontaneous resolution of the residual embolus. Had fibrinolysis not been performed, the MCA embolus would probably have enlarged to cause more extensive ischemic damage. Percutaneous transluminal angioplasty has been advocated for treating resistant emboli,¹¹⁾ but this technique could have damaged the vessel wall because of the undetected fenestration. Conventional angiography could not easily visualize the coexisting fenestration in such a case. More accurate information concerning vascular configuration might have been obtained by spiral CT angiography in this patient.²⁾ Dissolution of persistent adherent emboli, especially those located in the proximal portion of M₁, must be performed with great care because of the possibility of such rare vascular anomalies.

PPTA is the most common of the persistent primitive arteries, with a prevalence ranging from 0.06% to 0.6%.^{3,5)} Persistent primitive arteries are usually found incidentally, but are often associated with vascular malformations and cerebral aneurysms, and sometimes may produce ischemia or trigeminal neuralgia.³⁾ In our case, the PPTA was not directly related to occurrence of the embolism, but the anomaly posed an anatomic challenge during emergent treatment of the occluded ICA. The PPTA may have been partially responsible for the rich collateral circulation via the leptomeningeal anastomosis, which allowed us to accomplish recanalization of the occluded ICA without inducing bleeding.

Local intraarterial fibrinolysis could be successfully performed in two cases in which a vascular anomaly was combined with cerebral embolism. We would advise that particular care be taken in selecting and implementing therapy in such cases.

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