

DELAYED THROMBUS PROPAGATION AFTER PARENT ARTERY CLIPPING FOR GIANT FUSIFORM ANEURYSMS OF THE CIRCLE OF WILLIS

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BACKGROUND

Obliteration of a giant fusiform aneurysm without significant therapeutic morbidity is extremely difficult. Ischemic complications have been often reported.

METHODS

Two patients with giant fusiform aneurysms of the circle of Willis are presented. Both patients underwent proximal parent artery clipping after a bypass procedure. Balloon occlusion tests confirmed both patients' ability to tolerate flow reduction after proximal clipping.

RESULTS

Although both patients awoke from anesthesia without neurologic deficit, they developed contralateral hemiparesis several hours after the operation as a result of a small infarct in the basal ganglia. These ischemic events might be attributed to the delayed thrombosis involving the orifice of the distal perforating arteries.

CONCLUSIONS

In the treatment for giant fusiform aneurysms of the circle of Willis, special attention should be paid not only to flow reduction, but also to delayed thrombus propagation that may not be predicted by preoperative balloon occlusion testing. © 1999 by Elsevier Science Inc.

KEY WORDS

Giant aneurysm, fusiform aneurysm, balloon occlusion test, revascularization.

Treatment of giant fusiform aneurysms is among the most challenging neurosurgical problems [1-8,10,11,13-16]. We present two patients with giant fusiform aneurysms of the circle of

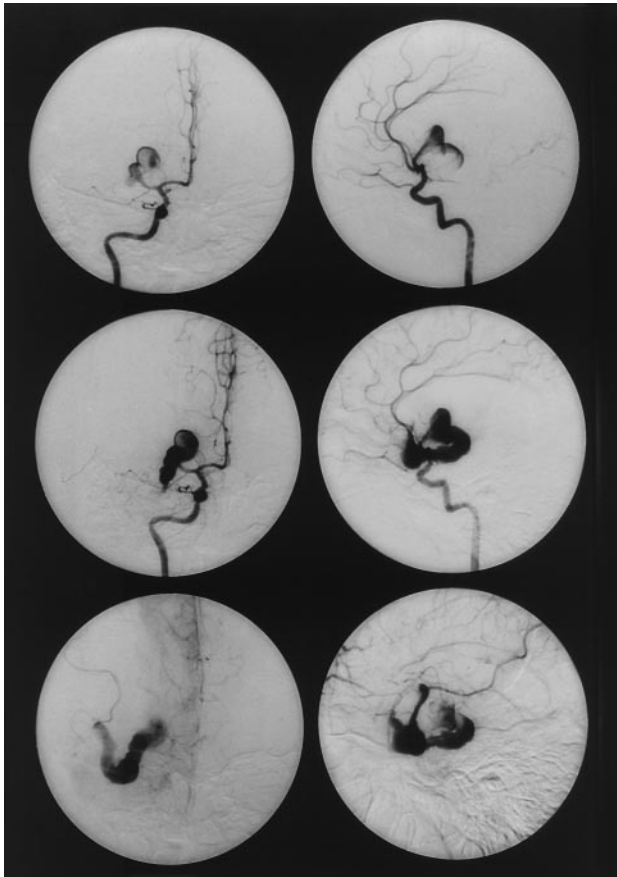
Willis who underwent proximal parent artery clipping and subsequently developed ischemia of the basal ganglia as a result of delayed thrombotic occlusion of the perforating arteries.

CASE REPORTS

CASE 1

A 32-year-old woman was admitted for treatment of a giant fusiform aneurysm of the right middle cerebral artery (MCA) on June 28, 1994. She had been treated with superficial temporal artery (STA)-MCA double anastomoses at another hospital before admission to our clinic. She had suffered from incapacitating right-sided headache and generalized convulsive seizures. Angiography revealed a giant fusiform aneurysm of the right MCA (Figure 1). The horizontal segment of the MCA distal to the origin of the temporopolar artery showed fusiform dilatation and marked tortuosity. The distal end of the fusiform aneurysm involved the inferior trunk of the MCA beyond the MCA bifurcation. The superior trunk of the MCA was not visible on a right internal carotid angiogram and no lenticulostriate arteries were seen originating from the aneurysm. The right STA supplied both the supra- and infra-Sylvian regions. To evaluate the patient's ability to tolerate flow reduction induced by proximal MCA clipping, a balloon occlusion test was performed. A mini-balloon was introduced into the horizontal segment of the right MCA just at the origin of the temporopolar artery. The balloon inflation resulted in temporary occlusion of both the horizontal segment of the MCA and of the temporopolar artery (Figure 2). During a test occlusion of 15 min, the patient re-

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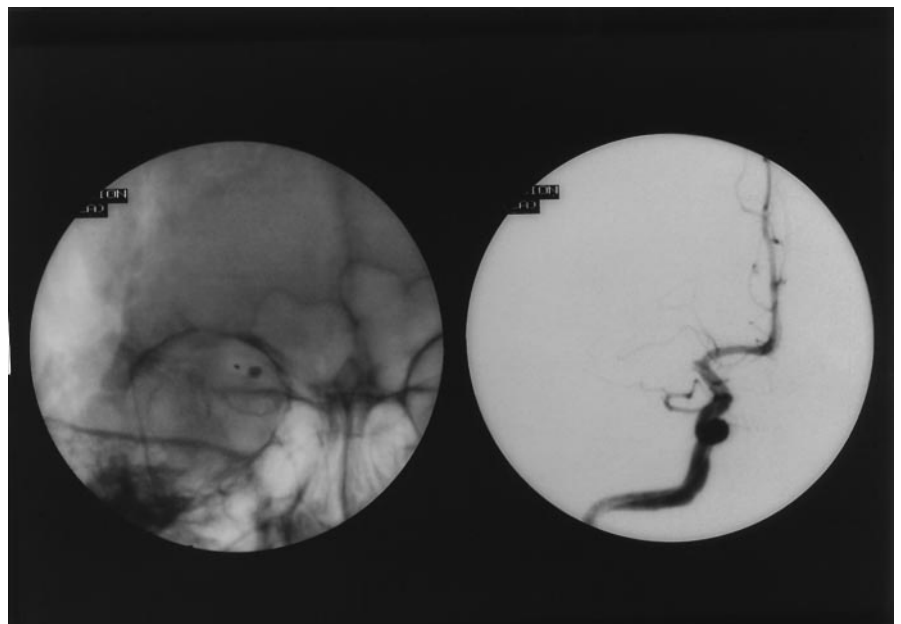
1 Right internal carotid angiograms (Case 1). **Left:** A-P view; **right:** lateral view; **(upper)** Early arterial phase; **(middle)** later arterial phase; **(bottom)** early venous phase.

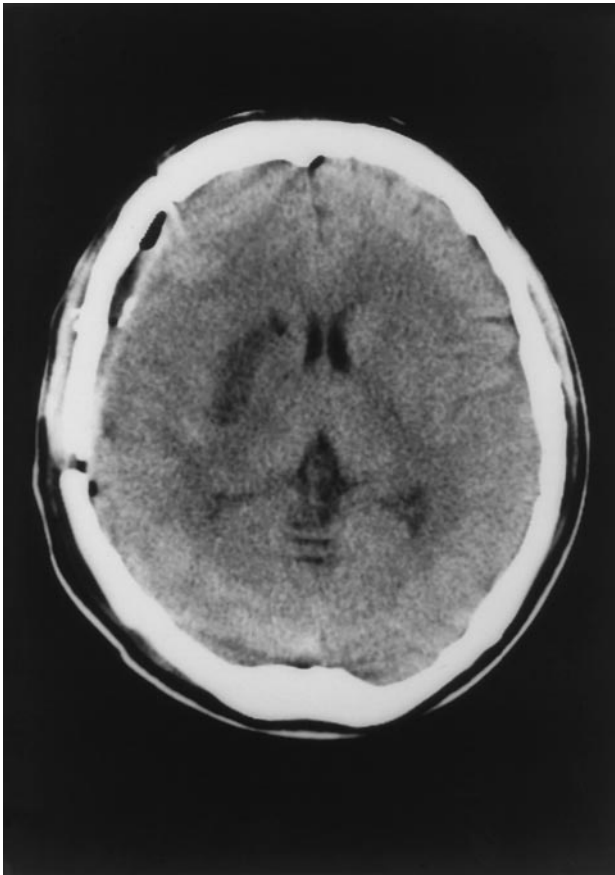
mained free of symptoms without any change in electroencephalographic monitoring. The right MCA distribution was sufficiently supplied through STA-MCA anastomoses and leptomeningeal collaterals.

On July 27, 1994, proximal clipping of the aneurysm was performed using a trans-Sylvian approach. The MCA was clipped just after the origin of the tiny perforating artery and the temporopolar artery. The perforating artery from the inferior trunk was noted to run a recurrent course to the anterior perforated substance.

Postoperatively, the patient awoke from anesthesia without any neurologic deficit. Normotensive normovolemic state was maintained during the perioperative period. Three hours later, she developed sudden severe left hemiparesis. Computed tomography (CT) showed thrombosis of the aneurysm. Subsequent imaging showed a localized infarct in the right caudate nucleus (Figure 3). Postoperative angiography showed complete obliteration of the aneurysm. The ischemic event might be attributed to progression of thrombosis, affecting the origin of the recurrent perforating artery from the inferior trunk of the MCA. The patient showed remarkable improvement in her left-sided motor weakness during the following weeks. She complained only of slight clumsiness in her left finger movements at discharge and remains able to play the piano fluently.

2 Balloon occlusion test (Case 1). **(Left)** Arrow indicates the inflated balloon. **(Right)** Right internal carotid angiogram during balloon occlusion test.



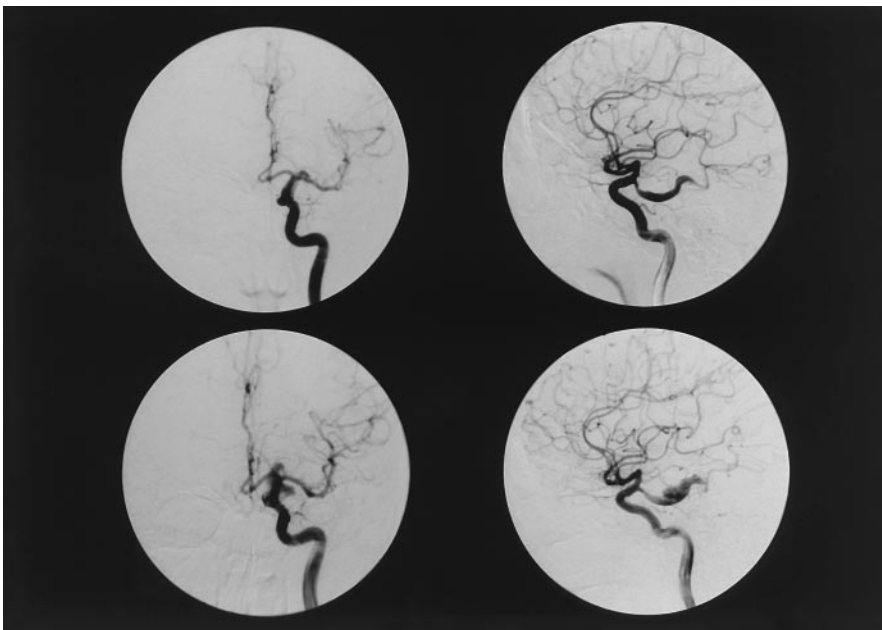


3 CT image at discharge (Case 1). Small arrow indicates the infarct in the right caudate nucleus. Arrows indicate the thrombosed giant fusiform aneurysm.

CASE 2

A 43-year-old man suffered from left-sided headache and hemifacial pain. A giant aneurysm was suspected based on a CT scan at another clinic, and he was admitted to our hospital for further evaluation. Examination on admission revealed no neurologic deficit. The patient's facial pain was in the region of the first division of the left trigeminal nerve. Angiography demonstrated a giant fusiform aneurysm of the left posterior communicating (Pcom) artery (Figure 4, **upper**). The lesion extended to the ambient segment of the posterior cerebral artery (PCA). The proximal segment of the left PCA was hypoplastic, thus the left PCA did not have a vertebrobasilar origin. The PCA distribution was supplied from the left internal carotid artery via the Pcom-PCA giant fusiform aneurysm. To evaluate whether the patient could tolerate proximal clipping of the Pcom artery, a mini-balloon was introduced into the Pcom artery and a balloon occlusion test was performed just proximal to the aneurysmal dilatation. After inflating the balloon, there was only mild constriction of the patient's right visual field, which subsided promptly after deflating the balloon.

The usual revascularization procedures for the PCA were not feasible because the ambient segment of the PCA, the recipient site of bypass procedures, was involved by the lesion. Thus, an STA interposition graft was performed between the left occipital artery and the parietooccipital artery of the PCA



4 (**Upper**) Left internal carotid angiograms (Case 2). (**Left**, A-P view; **right**, lateral view). (**Lower**) Left internal carotid angiograms (Case 2) after the bypass operation. (**Left**, A-P view; **right**, lateral view).

using a posterior interhemispheric approach on March 29, 1995. The patient's postoperative course was uneventful for the initial few days. However, 5 days after the operation he developed left blepharoptosis and marked dizziness. Within 1 day, there was complete left oculomotor palsy. CT with contrast enhancement revealed enlargement of the aneurysm. Angiography showed that the aneurysm had enlarged and the bypass was patent (Figure 4, **lower**). Because of severe dizziness, the patient could not maintain a sitting position, and he gradually became somnolent. A second balloon occlusion test was not performed because there was considerable risk of morbidity for balloon inflation just proximal to the enlarged aneurysm.

Proximal clipping of the aneurysm was performed on April 13 to reduce hemodynamic stress on the aneurysm. Using a pterional approach, the Pcom artery was clipped just proximal to the aneurysm. The preamillary artery and a few tiny perforating arteries from the Pcom artery were spared. Postoperatively, the patient awoke from anesthesia with only left oculomotor palsy. No other neurologic deficits were noted, and normotensive normovolemic state was maintained during the perioperative period. Approximately 20 h later, he developed right-sided motor weakness and speech disturbance. The symptoms progressed during the following hours. CT showed high density around the brainstem suggesting thrombosis of the fusiform aneurysm. Fortunately, these ischemic symptoms and the oculomotor palsy showed gradual but remarkable improvement during the following weeks. A subsequent CT scan showed an infarct in the thalamus and in the posterior temporal region (Figure 5). Angiography confirmed complete obliteration of the aneurysm. At the time of discharge the patient suffered only from a right upper quadrantanopsia and slight numbness in the right extremities.

DISCUSSION

Giant fusiform aneurysms constitute a small percentage (far less than 1%) of intracranial aneurysms [6]. According to Little et al, giant fusiform aneurysms of the cerebral arteries are defined as marked concentric dilatation, 2.5 cm or more in length [11]. Several therapeutic procedures have been reported including wrapping, direct clip reconstruction, resection with revascularization, and parent artery occlusion with or without bypass [1-16]. In most patients with giant fusiform aneurysms of the circle of Willis, perforating arteries



5 Postoperative CT image (Case 2). Small arrow indicates the infarct in the left thalamocapsular area.

prevent a direct reconstructive clipping procedure. In addition, direct clip reconstruction of an arteriosclerotic vessel poses a risk of vessel kinking or thromboembolic complications. Wrapping procedures act only to prevent subarachnoid hemorrhage, but these procedures cannot reduce mass effect, the most frequent symptom of giant fusiform aneurysms. Thus, parent artery occlusion with or without revascularization is usually recommended. Ischemic complications associated with parent artery occlusion have been frequently reported [1-4, 6,7,10-12]. Several factors have been proposed as the cause of ischemic events after parent artery occlusion: poor collateral circulation, bypass obstruction, reduced bypass flow, or flow stasis. Mostly, these factors have not been evaluated by balloon occlusion tests. Recent advances in interventional neuroradiology permit a highly selective balloon occlusion test just proximal to the lesion. Flow reduction can be accurately evaluated by this procedure [10]. In our two patients, smooth arousal from anesthesia and normotensive normovolemic control during the perioperative period suggests

that operative factors are negligible as a cause of ischemia. Delayed ischemic complications occurred after proximal parent artery occlusion, despite the fact that preoperative balloon occlusion tests showed good tolerance. Our patients illustrate the difference between ischemia induced by flow reduction and that induced by thrombotic occlusion of the distal branches. The former can be evaluated clinically and electroencephalographically. Although sufficient distal flow is maintained through bypass or collateral circulation, progressive thrombus formation after proximal clipping may carry the risk of affecting the origin of perforating branches around the distal end of the giant fusiform aneurysm. It is the purpose of this article to draw attention not only to the importance of balloon occlusion test just proximal to the giant fusiform aneurysm, but also to the potential risk in the overestimation of the tolerance of occlusion.

In conclusion, for patients who are undergoing proximal clipping of a parent artery of a giant fusiform aneurysm of the circle of Willis, we favor the use of systemic heparinization during and after the operation to reduce the risk of a delayed ischemic thrombotic complication. Intraoperative angiographic confirmation of the aneurysmal disappearance might be useful before heparinization.

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COMMENTARY

This is an interesting article presenting two giant aneurysms of the circle of Willis that were treated by permanent occlusion of the parent vessel after a negative balloon occlusion test. A delayed thrombus propagated in spite of a functioning EC-IC bypass. These two cases confirm that we may anticipate and control the ischemic complications, but we have little control of embolic or thrombotic complications, even with anticoagulation.

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