Emergent Middle Cerebral Artery Embolectomy: A Useful Technique for Cranial Base Surgery

Abstract—Given the poor natural history of untreated symptomatic acute middle cerebral artery occlusion, we have attempted emergent reperfusion in all three cases of acute embolic middle cerebral artery occlusion seen on our cranial base service over the last 10 years. One patient developed a massive stroke requiring a life-saving “strokectomy” within 48 hours, which left him permanently hemiplegic, hemianopic, and hemihypesthetic after a failed attempt at reperfusion by superselective endovascular injection of urokinase. The other two patients, who were aphasic and densely hemiparetic, underwent successful emergent embolectomy with reperfusion established within 5 and 12 hours, respectively. One of the two is now neurologically normal, and the second is left with a subtle monoparesis but is independent in activities of daily living. Since middle cerebral artery embolism in cranial base patients usually occurs in a closely monitored hospital setting, we are presented with a unique opportunity for early successful operative intervention. Principles for optimizing outcome include: early recognition and diagnosis, maximization of medical therapy during the diagnostic workup prior to embolectomy (induced hypertension, intravascular volume expansion, and pharmacologic cerebral metabolic demand reduction), confirmation that the involved region does not have absent blood flow by xenon/computed tomography, early operative intervention, and careful surgical technique. (*Skull Base Surgery*, 3(2):80–86, 1993)

Cranial base surgery often involves extensive manipulation of the great vessels at the base of the brain, which increases the risk of vessel injury and subsequent distal embolization. In over 900 surgeries performed on our cranial base service over the last 10 years, we have experienced three cases of acute embolic middle cerebral artery (MCA) occlusion. Given the poor natural history outcome of untreated acute MCA occlusion in patients with significant neurologic deficits,^{1–7} we attempted emergent reperfusion in all three cases. Since this complication usually occurs in a closely monitored hospital setting in this group of patients, cranial base surgeons have a unique opportunity for early and successful operative intervention. The purpose of this article is to present three cases of symptomatic acute MCA embolization, treatment, and outcome.

CASE SUMMARIES

Case 1

A 47-year-old right-handed man presented with recurrent nasopharyngeal squamous carcinoma involving the right internal carotid artery (ICA) in the infratemporal fossa. After passing a 15-minute clinical right ICA balloon test occlusion as well as an ICA-occluded stable xenon/computed tomography (Xe/CT) cerebral blood flow (CBF) evaluation,^{8–10} he underwent endovascular sacrifice of his right ICA by detachable balloon 24 hours prior to planned surgical tumor resection. The artery was occluded distally by a detachable balloon at the petrocavernous junction and proximally by a detachable balloon at the high cervical

ICA as well as two proximal endovascular coils. Twelve hours later, he suddenly developed left-sided hemiplegia and a right gaze preference. Portable skull films did not demonstrate balloon migration. An emergent angiogram by contralateral ICA injection revealed abrupt dye cutoff at the contralateral ICA bifurcation, with no filling of the contralateral M₁ segment (Fig. 1B). A Xe/CT CBF study revealed markedly reduced flow in the right MCA territory (Fig. 1A). A microcatheter was successfully negotiated across the anterior communicating artery to the contralateral M₁ segment. Selective urokinase injection was able temporarily to open the MCA system with the catheter still in place (Fig. 2A). However, with catheter withdrawal, the MCA again occluded despite continued injection of up to 235,000 U of urokinase (Fig. 2B). Within 48 hours of his ictus, he had developed a large right MCA infarction exactly corresponding to the region of decreased CBF with significant midline shift (Fig. 1C). On the third day, he became comatose with further brain swelling and midline shift. An emergent craniotomy was performed to resect the region of decreased CBF demonstrated by Xe/CT (Fig. 1D). After a stormy postoperative course, he was discharged 2 months later to rehabilitation. At the time of discharge, he was alert and conversant but had dense left hemiplegia, a left hemianopsia, and left-sided hemihypesthesia.

Case 2

A 57-year-old right-handed woman with bilateral intracavernous carotid artery aneurysms, which was giant on the left, presented with a 2-month history of progressively severe left retro-orbital pain and left-sided headaches. During a preoperative left ICA balloon test occlusion, she developed a high cervical ICA dissection with complete vessel occlusion without any change in her neurologic condition.^{11,12} She was aggressively intravenously hydrated, maintained with a systolic blood pressure 170 mmHg or greater, and transported to the CT scanner. During her Xe/CT CBF study, she suddenly became aphasic and contralaterally hemiplegic despite a normal sensorium. Her Xe/CT showed reduced blood flow in her left MCA distribution (10 to 18 cc/100gm/min; Fig. 3A). Suspecting distal MCA embolization from her ICA dissection/occlusion, she was rushed to the operating room and placed in etomidate coma with burst suppression confirmed by electroencephalogram.

A left frontotemporal craniotomy with orbitozygomatic osteotomy was performed. After splitting the sylvian fissure, the supraclinoid carotid artery, the carotid bifurcation, and the proximal anterior cerebral artery were found to be pulsatile. An embolus was found occluding the main branch of the MCA, and a small clot was seen at the

Figure 1. Case 1. A: A xenon computed tomography (Xe/CT) cerebral blood flow (CBF) study immediately after the onset of neurologic deficits revealed markedly reduced flow in the right middle cerebral artery (MCA) territory (arrow). B: A cerebral angiogram by contralateral internal carotid artery (ICA) injection revealed abrupt dye cutoff at the contralateral ICA bifurcation, with no filling of the contralateral M₁ segment. (Figure continued on next page)

Figure 3. Case 2. A: A xenon computed tomography (Xe/CT) cerebral blood flow (CBF) study immediately after the onset of neurologic deficits showed reduced blood flow in the left middle cerebral artery (MCA) distribution (arrow). B: A repeat Xe/CT study after emergent MCA embolectomy revealed normal symmetrical MCA flow.

origin of an M_1 perforating artery. After placing proximal and distal temporary clips, a transverse arteriotomy one-quarter the circumference of the involved vessel was performed over the embolus. After removing the distal clip, the embolus was “milked” free from distal to proximal with good back bleeding. The same procedure was repeated for the proximal portion of the embolus. Throughout the procedure, the vessel lumen was intermittently irrigated with heparinized saline. The arteriotomy was closed with interrupted 10-0 nylon sutures. The total time from her ictus in the CT scanner until reestablishment of MCA flow was 4.5 to 5 hours. After repairing her ICA dissection and resecting her giant left intracavernous aneurysm, an intraoperative left ICA angiogram was obtained that showed good ipsilateral MCA filling via a patent left ICA.

Immediately postoperatively her speech was normal and her right-sided hemiplegia had improved, leaving residual right arm weakness. She also had a partial left oculomotor nerve paresis as a result of her cavernous sinus exploration. A follow-up Xe/CT CBF study revealed normal symmetric MCA flow (Fig. 3B). Twenty-one months after surgery, she has returned to homemaking, has normal speech, and has no demonstrable cranial nerve deficit or motor weakness on neurologic examination.

Case 3

A 47-year-old right-handed woman presented with a left cavernous sinus mass lesion. After passing her preoperative left ICA balloon test occlusion as well as an ICA-occluded stable Xe/CT CBF evaluation, she underwent gross total resection of her tumor via a left frontotemporal, infratemporal approach with orbitozygomatic osteotomy. She awoke from surgery with a left cavernous sinus syndrome, related to her cavernous sinus exploration, but without lateralizing neurologic signs or altered sensorium. Three days after surgery, she suddenly developed global aphasia, dense right-sided hemiparesis, and altered sen-

sorium. A Xe/CT CBF study revealed reduced flow in her left MCA distribution (15 to 20 cc/100gm/min; Fig. 4B) and an emergent angiogram revealed occlusion of the main superior branch of the MCA (Fig. 5). She was aggressively intravenously hydrated, maintained with systolic blood pressure 170 mmHg or higher, and taken to the operating room.

Her previous craniotomy was opened and the sylvian fissure opened under the microscope. The MCA bifurcated into two main divisions. The inferior division was pulsatile but the superior division was nonpulsatile, as were two distal main branches arising from this trunk of the major subdivision. A perpendicular arteriotomy was made over the main superior division extending approximately one-quarter the vessel circumference. The distal clot was “milked” free in a distal-to-proximal direction from both secondary vessel branches. Good back bleeding was encountered and temporary clips were applied. This process was then repeated for the proximal portion of the thrombus. Throughout the procedure, the vessel lumen was intermittently irrigated with heparinized saline. The arteriotomy was closed with interrupted 10-0 nylon sutures.

Postoperatively, she awoke with normal speech, a normal level of consciousness, and a mild contralateral hemiparesis. A follow-up Xe/CT CBF study demonstrated normal, symmetrical MCA flows (Fig. 4C). Eight months postoperatively, her hemiparesis had improved to a subtle contralateral monoparesis. She is currently independent in activities of daily living.

DISCUSSION

Natural History of Acute Middle Cerebral Artery Occlusion

Case 1 is a good example of what can happen if reperfusion is not reestablished rapidly after symptomatic acute MCA occlusion. This patient would likely have died

Figure 4. Case 3. A: A preoperative left internal carotid artery (ICA)-occluded xenon computed tomography (Xe/CT) cerebral blood flow (CBF) study demonstrated normal symmetrical middle cerebral artery (MCA) blood flow. B: A Xe/CT CBF study immediately after the onset of neurologic deficits showed reduced blood flow in the left MCA distribution (arrow). C: A repeat Xe/CT CBF study after emergent embolectomy confirmed the resumption of normal symmetrical MCA blood flow.

if heroic surgery had not been performed, and even though his life was spared, he was left with a severe residual neurologic deficit. Table 1 summarizes the published data concerning the natural history of acute MCA occlusion. From these data, it is clear that case 1 was not unusual in the severity of his insult. If we restrict our attention to only those patients with significant neurologic deficits after MCA occlusion (significant hemiparesis, aphasia, or depressed level of consciousness), symptomatic acute MCA occlusion carries an initial mortality rate of 14 to 33%.^{2,4,7} Of those patients who survive, 46 to 50% do poorly (no improvement or worsening of their initial deficit), whereas only 50 to 54% have spontaneous improvement in their deficits.^{2,4,7} Given the poor natural history

outcome for symptomatic acute MCA occlusion, attempts to alter the natural course of acute symptomatic MCA occlusion would seem to be warranted.

Emergent Reestablishment of Middle Cerebral Artery Flow

The first report of emergent embolectomy for acute MCA occlusion was by Welch in 1956.¹³ A review in 1985 found 24 cases in the literature and presented 19 cases from the Mayo Clinic who were operated on within 24 hours.^{14,15} Since that time, we have identified ten additional cases in the literature treated within 24 hours.^{11,16-18}

Figure 5. Case 3. A: Preoperative left internal carotid artery (ICA) cerebral angiogram. B: An emergent left ICA cerebral angiogram after the onset of neurologic deficits demonstrated occlusion of the main superior branch of the middle cerebral artery (arrow).

Table 1. Natural History of Acute Middle Cerebral Artery Occlusion

Reference	Year	No.	Initial Mortality (%)	Survivor Outcome	
				Poor (%)	Fair-Excellent (%)
Bladin ²	1964	20	25	?	?
Krayenbuhl and Yasargil ³	1964*	100	10	46	54
Burrows and Lascelles ⁴	1965	59	33	50	50
Allcock ⁵	1967*	29 [†]	5	35	65
Kaste and Waltimo ⁶	1976*	83	5	27	72
Yoshimoto et al. ⁷	1986	188	14	46	54

*These three series included a significant number of patients with minimal or no neurologic deficits.

[†]Eleven additional patients with middle cerebral artery stenosis without occlusion were excluded.

Other ways to reestablish emergently MCA blood flow include intravascular fibrinolytic therapy^{19,20} and superficial temporal artery (STA)-to-MCA bypass surgery.²¹ While these latter two possibilities can be quite effective, intravascular fibrinolysis by superselective catheterization can be technically difficult to perform if the ipsilateral ICA has already been sacrificed (as in case 1), and STA-MCA bypass surgery requires a preserved STA of adequate size. Since many of the cranial base surgery patients who experience this complication will do so in the immediate postoperative period, it will often be faster and technically simpler to reopen the preexisting craniotomy to reexplore the MCA.

Although we achieved good results in two cases of emergent MCA embolectomy, it is important to base future therapeutic decisions on a larger patient experience. The largest surgical experience for acute MCA embolectomy comes from the Mayo Clinic.^{14,15} In 1985 they reported on 19 cases with significant neurologic deficits, all of whom were operated on within 24 hours of onset of their deficit (median time to reperfusion, 6.5 hours; range, 3 to 18). Postoperative angiographic MCA patency was achieved in 16 of 19 patients (84%). Interestingly, two of the three who remained occluded died [the only two deaths (10.5%) in the series]. Of the 16 patent cases, only two developed hemorrhagic infarction from reperfusion. Overall, 73.6% of patients had a fair (meaning independent in activities of living) to excellent (meaning no residual deficits) outcome.

Optimizing Patient Outcome

At least two important factors contribute to a potentially improved outcome for cranial base surgery patients who undergo emergent MCA embolectomy for acute MCA embolic occlusion. First, in cranial base surgery patients this complication will usually occur in a closely monitored hospital setting. This factor allows for early recognition of the onset of the neurologic deficit, early maximization of medical therapy, early radiologic confirmation of the diagnosis, and early intervention for revascularization. Second, most MCA emboli in cranial base surgery patients will be fibrin-platelet clots resulting

from vessel injury rather than atheromatous emboli. Meyer and colleagues^{14,15} have shown that fibrin-platelet clots are technically easier to remove, and are more likely to lead to a patent MCA after embolectomy, than atheromatous emboli.

Optimal medical management during the acute period includes intravascular volume expansion and induced hypertension²²⁻²⁷ in order to maximize collateral blood flow to the ischemic region. If the patient does not significantly improve with this therapy, serious consideration should be given to an emergent revascularization procedure, particularly embolectomy. If the surgeon decides to proceed with operative intervention, additional measures that may lengthen the safe time period for reperfusion or improved clinical outcome include barbiturate- or etomidate-induced coma to the point of burst suppression by electroencephalogram,²⁸⁻³⁴ as well as moderate hypothermia (32 to 34° C).

It is well known that the likelihood of infarction after ischemia is dependent on both the degree of reduction of blood flow and the duration of the ischemia.³⁵ Empiric experimental evidence for severe reductions in MCA blood flow in dogs and nonhuman primates suggests that embolectomy after acute MCA occlusion must be performed within 3 to 6 hours in order to have a fair likelihood of success.³⁶⁻³⁸ However, if sufficient leptomeningeal collaterals are present to maintain a marginal (although ultimately inadequate) blood flow in the effected distribution, the therapeutic window for intervention may be extended for several additional hours. The stable Xe/CT CBF study allows us to measure accurately the degree of CBF reduction in any given patient prior to deciding the necessity and the timing of operative intervention. A finding of flows below the threshold for cellular membrane ionic homeostasis failure³⁹⁻⁴¹ in the affected region several hours after MCA occlusion may mean that reperfusion runs a high risk of reperfusion hemorrhage into an area of established infarction and has a very low probability of improving the clinical situation.¹⁰ Flows between 10 and 20 cc/100gm/min in the very acute period suggest a region of ischemia rather than infarction; whether or not infarction will occur depends on how long this low flow is allowed to continue. Given enough time, permanent CBF reductions below 15 to 18 cc/100 gm/min lead uniformly to

infarction.^{35,42} However, when the patient is seen within 2 to 3 hours after acute MCA occlusion, reperfusion should be attempted regardless of the blood flow in an attempt to improve perfusion to the "ischemic penumbra," and potentially reduce the size of cerebral infarction.

No surgeon likes to have complications. However, the steps taken during the immediate acute period after the onset of a complication can often mean the difference between clinical disaster and successful recovery. In very few circumstances is this adage more true than in cases of acute symptomatic MCA embolic occlusion. Due to the usual nature and timing of this complication when it occurs on the cranial base service, we are in a unique position to optimize patient outcome. Emergent MCA embolectomy should be kept in mind as a useful technique for cranial base surgeons.

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