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Endovascular recanalization of symptomatic flow-limiting cervical carotid dissection in an isolated hemisphere

Clemens M. Schirmer, M.D., Ph.D., Basar Atalay, M.D., and Adel M. Malek, M.D., Ph.D.

Cerebrovascular and Endovascular Division, Department of Neurosurgery, Tufts Medical Center and Tufts University School of Medicine, Boston, Massachusetts

Object. Internal carotid artery dissection (ICAD) is a common cause of stroke in young patients, which may lead to major transient or permanent disability. Internal carotid artery dissection may occur spontaneously or after trauma and may present with a rapid neurological deterioration or with hemodynamic compromise and a delayed and unstable neurological deficit. Endovascular intervention using stent angioplasty can be used as an alternative to anticoagulation and open surgical therapy in this setting to restore blood flow through the affected carotid artery.

Methods. The authors present the cases of 2 patients with flow-limiting symptomatic ICAD leading to near-complete occlusion and without sufficient collateral supply. Both patients had isolated cerebral hemispheres without significant blood flow from the anterior or posterior communicating arteries. In both cases, the patients demonstrated blood pressure–dependent subacute unstable neurological deficits as a result of the hemodynamic compromise resulting from the dissection.

Results. Both patients underwent careful microwire-based selection of the true lumen followed by confirmatory microinjection and subsequent exchange-length microwire-based recanalization using tandem telescoping endovascular stenting. In both cases the neurological state improved, and no permanent neurological deficit ensued.

Conclusions. The treatment of ICAD may be difficult in patients with subacute unstable neurological deficits related to symptomatic hypoperfusion, especially in the setting of a hemodynamically isolated hemisphere. Anticoagulation alone may be insufficient in these patients. Although there is no widely accepted guideline for the treatment of ICAD, the authors recommend stent-mediated endovascular recanalization in cases of symptomatic flow-limiting hemodynamic compromise, especially in cases of an isolated hemisphere lacking sufficient communicating artery compensatory perfusion. *(DOI: 10.3171/2011.2.FOCUS1139)*

Key Words • **carotid artery** • **dissection** • **isolated hemisphere** • **stent** • **stroke** • **trauma** • **hypoperfusion**

Internal carotid artery dissection occurs spontaneously or in the setting of trauma and can lead to ischemic and hemorrhagic stroke through thromboembolic complications or hemodynamic compromise. Internal carotid artery dissection accounts for a small proportion of strokes overall but is a more common cause of stroke in the younger population. The natural history is not very well understood. Some reports point to stroke due to ICAD that is unexpectedly benign in some cases,[15,23,29] while others have suggested possible major transient or permanent disability.[17,25,34,38,40,43] In a series of 260 patients with nontraumatic intracranial arterial dissections, Yamaura[45] reported an overall mortality rate of 26% and poor outcomes in 5%, with the mortality rate reaching 49% in patients with carotid artery lesions. Traumatic dissections have been reported to occur in 1% of patients with blunt injury mechanisms to the neck,[37] and spontaneous dissections may be triggered by seemingly innocuous events, including violent coughing, nose blowing, and forceful neck rotations.[38] In spontaneous dissections, collagen tissue abnormalities such as Marfan syndrome or fibromuscular dysplasia may be associated with ICAD.[5,6,14] Internal carotid artery dissection may present with a wide variety of neurological symptoms including headache, carotidynia, oculosympathetic palsy, hemiparesis, and hemiplegia.[21,43]

Patients with ICAD are routinely treated using anticoagulation therapy at most institutions, despite a lack of evidence from well-designed studies supporting this treatment's efficacy.[31] Anticoagulation alone has been shown to be effective in a number of case series,[16,21,42] although strokes may occur despite anticoagulation,[3] and the treatment is usually contraindicated in the setting of trauma with concomitant injuries. Endovascular stent placement has been proposed in patients with worsening neurological condition and in cases in which anticoagulation is contraindicated.[1,3,8–12,20,22,26,27,30,32]

Abbreviations used in this paper: ICA = internal carotid artery; ICAD = ICA dissection; MCA = middle cerebral artery.

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Patients with poorly developed or absent communicating arteries and a cerebral hemisphere supplied solely by the index ICA, a configuration found in 18%–44% of the population,[24,35,44] are in a high-risk group for the imminent stroke after ICAD due to hemodynamic compromise during the subacute period. In this report, we present the cases of 2 patients with isolated hemispheres in whom symptomatic dissection of the ICA led to near-complete occlusion of the vessel. In both cases, the dissection resulted in blood pressure–dependent transient neurological deficit, which was treated by endovascular revascularization. We reviewed the relevant literature, and we discuss the rationale of endovascular treatment of ICAD in the setting of hypoperfusion in the affected hemisphere.

Case Reports

Endovascular Technique

Both patients underwent catheter angiography of the cervical and intracranial vasculature. Intravenous heparin was administered to achieve an activated clotting time of longer than 250 seconds. An 8 Fr guide catheter (Brite Tip) coaxially over a 5 Fr vertebral 125-cm catheter was placed in the common carotid artery via a femoral vascular sheath (Avanti, Cordis Corp.). A microcatheter was used coaxially over a 0.014-in microguidewire to gently probe, identify, and enter the true arterial lumen under real-time high-resolution digital roadmap angiography. This was then navigated past the site of dissection into the petrous intracranial distal portion. A microinjection was performed to ensure continued presence in the true lumen and good outflow. At this point, a 300-cm exchange-length microguidewire was passed through the microcatheter with its tip positioned in the petrous segment and was used for advancing the stent delivery catheter. A Nitinol shape-memory alloy self-expanding stent (Precise Stent, Cordis Corp.) was used in this study and was advanced over the microwire until its tip reached the distal portion of the dissection at the junction of the cervical and petrous portions of the ICA. The first stent was deployed from distal to proximal. A subsequent stent was deployed in tandem overlapping fashion until the proximal inflow zone was covered and recanalized. Patients were maintained on a regimen of clopidogrel (75 mg orally per day) for 6 weeks and aspirin (325 mg orally per day) indefinitely.

Case 1

History and Examination. This 49-year-old man skated inadvertently into a wall and suffered blunt trauma. He did not notice any problems at the time, such as neck pain, external abrasions, or cervical fractures. Three days later, he noted sudden onset of right arm weakness, along with difficulty in speaking and finding words. He was hospitalized with these complaints at an outside institution, and MR imaging/MR angiography demonstrated dissection of the left ICA with good flow to intracranial vessels. His symptoms resolved spontaneously after systemic anticoagulation with intravenous heparin was initiated. He was discharged from the hospital 3 days later and was placed on a regimen oral warfarin together with enoxaparin. Again, 3 days later he suddenly felt dizzy while sitting, noted an unsteady gait, paresthesias to bilateral lower extremities, and return of speech difficulties. He presented to the hospital with dysphasia and right hemiplegia. Additional MR imaging/MR angiography findings were compared with those obtained 5 days earlier and revealed extension of the dissection with virtually no intracranial blood flow visualized through the left MCA. There was no sign of a lesion or stroke on diffusion-weighted MR imaging sequences.

Treatment. Catheter-based digital-subtraction angiographic studies demonstrated the previously suspected left carotid artery dissection (Fig. 1). Persistent flow was noted through the dissected segment of the left ICA, although it was sluggish (compared with the filling of branches of the external carotid artery) due to the dissection flap, which was under pressure and consequently compressing the true lumen. In addition, injection of the right ICA did not reveal anterior communicating flow or perfusion to the left MCA and no retrograde flow into the left A_1 segment of the anterior cerebral artery after injection of the right ICA. An SL-10 microcatheter (Boston Scientific Corp.) was advanced into the high petrous segment of the ICA after the true lu-

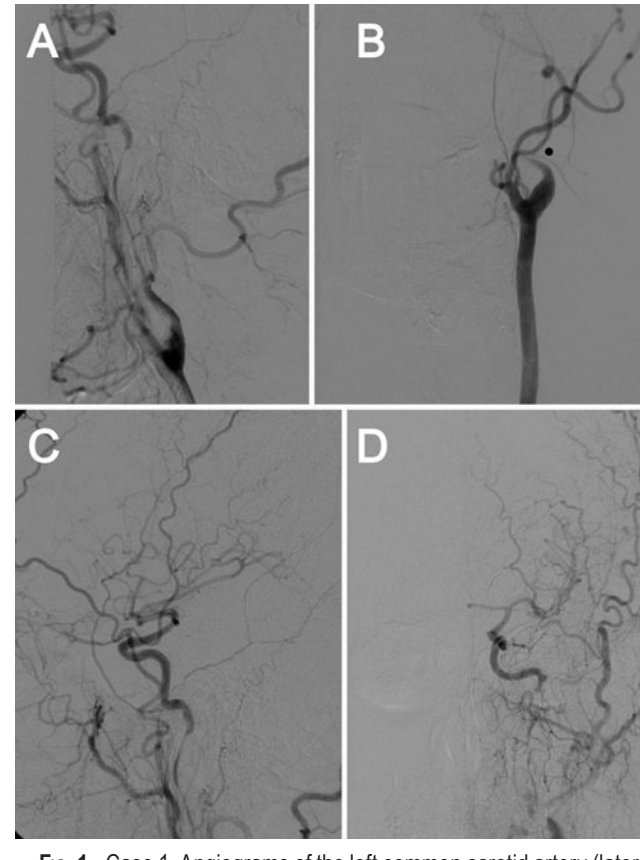


Fig. 1. Case 1. Angiograms of the left common carotid artery (lateral **[A]** and anteroposterior **[B]** views) demonstrating an almost complete occlusion of the ICA with poor, sluggish intracranial filling through the compressed true lumen and minor collateral vessels of the external carotid artery (lateral **[C]** and anteroposterior **[D]** views).

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men of the dissection had been selected with an Agility 14 microwire (Cordis Corp.). Patency and antegrade flow in the petrous ICA was confirmed by microcatheter contrast injection (Fig. 2). The microwire was exchanged for a supersoft 300-cm-long wire (Stabilizer, Cordis Corp.) inserted through the microcatheter and advanced into the supraclinoid segment, followed by 5 × 40–mm Precise Stent deployment from the proximal portion to the junction of the cervical and petrous ICAs. The stent delivery microcatheter was withdrawn, and an angiographic run revealed very poor flow through the stent and worsening of the proximal component of the inflow zone of the dissection. There was concern that perhaps the first stent had been deployed in the false lumen, thus sealing the carotid artery shut. Nonetheless, since there was constant access through the true lumen with the exchange-length microwire, and since confirmation was made initially of being within the true lumen via microcatheter injections, the decision was made to deploy a second proximal overlapping stent to connect the true lumen to its proximal portion. Accordingly, a second 6 × 40–mm stent was guided in overlapping fashion over the stabilizer wire that was kept in the supraclinoid segment and was delivered with a small overlap with the proximal portion of the stent ending in the distal portion of the left ICA bulb without crossing into the bifurcation. This second stent successfully tacked the inflow zone of the dissection, which had been inflated in an accordion-like manner and thus had worsened the lumen, and this resulted in full restoration of left ICA flow (Fig. 2).

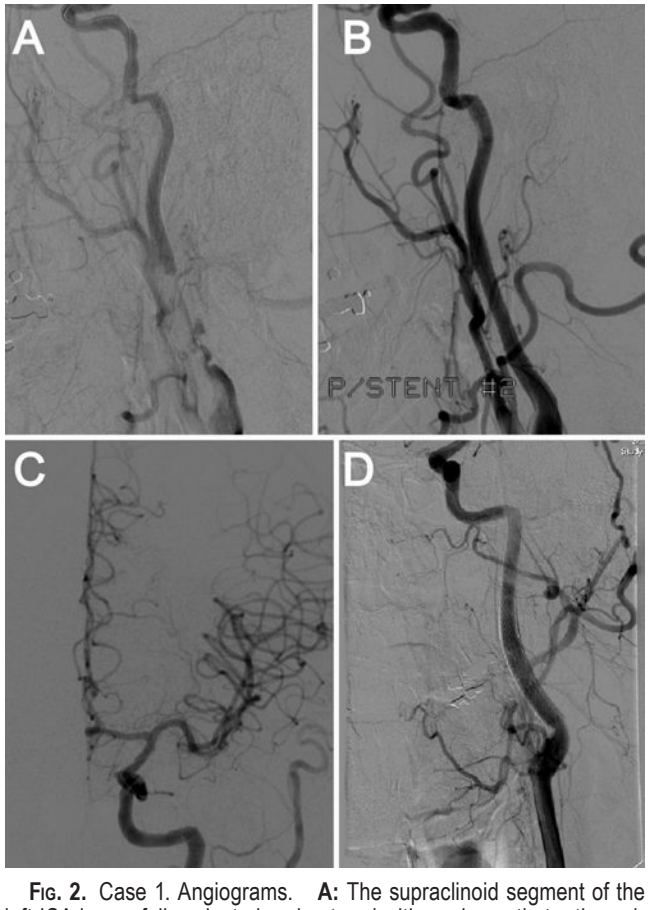


Fig. 2. Case 1. Angiograms. **A:** The supraclinoid segment of the left ICA is carefully selected and entered with a microcatheter through the true lumen of the ICAD, and a self-expanding Precise stent is deployed. Very poor flow is noted through the stent, with worsening of the proximal component of the inflow zone of the dissection. **B:** A second stent is delivered in tandem with a small overlap enabling the tacking down of the ICAD inflow zone. **C:** Resumption of flow is seen in the distal left ICA and improved left-sided intracranial perfusion. **D:** Follow-up angiogram obtained 6 months later demonstrating a good and stable stent position with preservation of the vessel lumen.

Posttreatment Course. The patient tolerated the procedure without deficit and became insensitive to blood pressure variation. He was discharged from the hospital on the 3rd postintervention day and was placed on a regimen of dual antiplatelet therapy. Follow-up angiography at 6 months demonstrated persistent patency of the stented left ICA segment with no evidence of stenosis. The patient remains asymptomatic at 6 years postintervention with patent stents on CT angiography (Fig. 2).

Case 2

History and Examination. This 64-year-old man complained of a severe headache that had persisted for the past 3 weeks. Magnetic resonance imaging of the brain revealed a left frontal deep-seated hemorrhage. Subsequently, diagnostic catheter angiography was performed at an outside institution, after which the patient became aphasic and hemiplegic. The patient was started on a heparin drip. Additional MR imaging/MR angiography revealed multiple left cerebral emboli and decreased flow in the anterior cerebral artery and MCA on the left and no flow in the proximal left ICA with minimal distal reconstitution. The patient was transferred for further management and underwent microcatheter-based recanalization and stenting of the left ICA (Fig. 3).

Treatment. An 8 Fr guide catheter was placed in the left common carotid artery, and an S-10 microcatheter used over a Transend EX microwire (Boston Scientific) to identify the true lumen of the left ICA dissection. The wire was then removed, and the SL-10 microcatheter was gently advanced without microwire support until it reached the supraclinoid segment. From this location, a contrast microinjection revealed that the microcatheter remained in the true lumen as evidenced by good distal flow in the petrous and supraclinoid segments (Fig. 4). An exchange-length microwire was advanced into the intracranial ICA over the microcatheter, with the latter being exchanged for a Precise 5 × 40–mm stent, which was positioned with its distal end in the transition zone of the petrous and cervical segments. After stent deployment, a test injection through the guide catheter revealed excellent resumption of flow into the left ICA. Examination of the transition zone between the stent and the native vessel and the lower portion revealed a persistent area of irregularity in the anterior portion suggestive of incomplete coverage of the inflow zone of the dissection. Accordingly, the decision was made to deploy a second 5 × 40–mm tandem proximal overlapping stent. This led to coverage of irregularity at the transition zone, and there was now excellent flow through the left ICA in the cervical portion.

Posttreatment Course. After the intervention, the pa-

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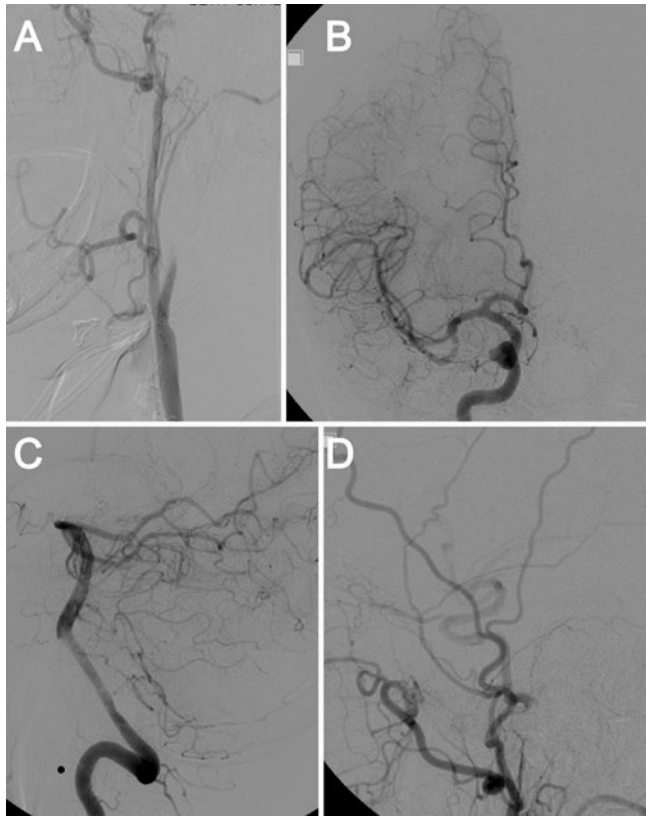


Fig. 3. Case 2. **A:** Angiogram of the left common carotid artery demonstrating a complete occlusion of the ICA. **B:** Injection of the right carotid artery (anteroposterior view) demonstrating no cross-flow through the anterior communicating artery. **C:** The left vertebral artery (lateral view) showing only minimal flow through the left posterior communicating artery. **D:** Additional blood supply to the distal left supraclinoid ICA (lateral view) stems from collateral flow through the left ophthalmic artery.

tient was awake, alert, and aphasic but following simple commands. He had some movement in the right upper and lower extremities on the bed. His neurological examination improved to moderate weakness on the right side. He continued to be intermittently confused and disoriented. He was discharged home and was placed on a regimen of clopidogrel and aspirin. Follow-up at 6 months showed improvement in the dysphasia and hemiparesis, and a CT angiogram (the patient refused catheter angiography) demonstrated persistent patency of the stented left ICA segment and no evidence of stenosis (Fig. 4).

Discussion

Carotid artery dissection may result in occlusion or severe narrowing of the affected vessel and subsequent hypoperfusion of the distal territory or thromboembolism causing transient ischemic attacks or stroke. In patients with an incomplete circle of Willis and vascular supply to one hemisphere that is entirely dependent on the dissected ICA, the resultant ischemic territory may encompass the entire hemisphere. Aggressive endovascular and microsurgical measures may be necessary to salvage neurological function and minimize stroke morbidity in



Fig. 4. Case 2. **A:** The true lumen of the ICAD is successfully selected as shown by microcatheter injection in the left ICA (lateral view). **B and C:** A self-expanding Precise stent immediately restores flow in the left ICA (anteroposterior **[B]** and lateral **[C]** views). **D:** A flap at the inflow zone persists after deployment of the stent *(arrowhead).* **E and F:** A second tandem overlapping stent is deployed, restoring the vessel profile completely (anteroposterior **[E]** and lateral **[F]** views). **G:** Injection of the left ICA (lateral view) showing resumption of a normal flow pattern intracranially. **H:** A CT angiogram obtained at the 6-month follow-up reveals good contrast material flow in the petrous portion of the left ICA *(arrow).* **I:** A 3D reconstruction confirms stable placement of both stents with flow in the proximal cervical left ICA.

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this extreme situation.[25] Internal carotid artery dissection has been held accountable for up to 20% of strokes in a younger population; its incidence has been reported to be 2.6–2.9 per 100,000 population.[19,39] Internal carotid artery dissection results from an intimal tear with spread of the circulating blood into the vessel wall that progresses into a subintimal dissection and subsequently to a narrowing of the lumen or pseudoaneurysm formation.[21] The extracranial portion of the ICA is affected much more commonly; 90% of dissections occur in the cervical segment of the ICA, and the dissection usually terminates at the level of entry into the carotid canal.[21,27] As was the case in Case 1, hyperextension and lateral rotation of the neck is thought to lead to stretch of the carotid artery over the upper cervical vertebral bodies.[42] Our experience with Case 2 demonstrates that endovascular procedures, including diagnostic angiography and interventional procedures, may lead to ICAD.[7,18]

Although periorbital headache is the most common presenting symptom of ICAD, ischemic or thromboembolic infarction is the most severe clinical sequela. In 80% of the cases, retinal and/or cerebral ischemia has been reported (20%–30% of transient ischemic attacks and 40%–60% of complete stroke).[21] Transcranial Doppler ultrasonography studies have demonstrated frequent microemboli[41] in accordance with imaging studies, revealing that strokes due to ICAD are predominantly thromboembolic in origin.[23,41] Strokes following ICAD are embolic in the majority of cases, but hemodynamic compromise is also another considerable proportion.[29,43] In our Case 1, the deficit was not fixed but rather fluctuated directly in relation to cerebral perfusion pressure. A decrease in the systolic blood pressure below 120 mm Hg dynamically prompted the development of a reversible neurological deficit. Cohen et al.[9] reported that angiographic parenchymography or perfusion MR imaging can be used to determine the extent of salvageable ischemic penumbra to select the patients who would potentially benefit from a revascularization. Hemodynamically significant stenosis and pseudoaneurysm formation develops in a number of patients despite anticoagulation, increasing the risk of flow-related infarction and distal embolization.[33,36]

Internal carotid artery dissection is not a fixed lesion and should be viewed as a dynamic entity. Clinical and radiological findings may change within hours, as occurred in our patients. The timing and type of treatment in patients with ICAD remain controversial, and there are no controlled studies for the treatment comparing anticoagulation or other invasive procedures. Liu et al.[27] reported the indications for stent placement for ICAD as follows: 1) dissection-induced stenosis that has not responded to a course of medical therapy; 2) ischemic or thromboembolic symptoms; 3) dissection in a patient with contraindications for anticoagulation therapy; and 4) iatrogenic dissection with severe stenosis. Contraindications for anticoagulation have to be considered on a case-by-case basis as the interventional therapy usually involves anticoagulation and dual antiplatelet therapy.

The development of hemorrhagic transformation or progression of the dissection has been reported in patients who have undergone anticoagulation therapy, but there are also other reports of good results in patients treated with anticoagulation.[5] Although there are no accepted guidelines for the ICAD treatment, whether anticoagulation or stent deployment, we believe that stenting would be very appropriate in patients with a rapidly deteriorating neurological status in the acute, clinically dynamic phase of dissection with an underlying hemodynamic rather than simple thromboembolic etiology. In a previous study, we treated 10 cases of ICAD with endovascular stenting and reported good neurological outcomes.[32] In 7 of these patients, there were intracranial infarctions, and none of them developed hemorrhagic transformation after treatment. The major difference of this study from the previous one is the treatment of ICAD in the isolated hemisphere that has the main blood supply from the side affected by ICAD. Neither case demonstrated hemorrhagic transformation likely because the cerebral tissue remained viable and not infarcted.

Timing of the reperfusion is also very important. Salvage of the penumbra at risk might not be feasible without early reperfusion. Ischemic cerebral tissue may potentially benefit from thrombolysis between 3 and 6 hours, and there is increasing evidence that noninfarcted but hypoperfused portions of ischemic brain tissue might remain viable and regain function even after 12 hours.[28] The timing of endovascular revascularization and stent placement depends on the clinical condition of the patients, and guidelines regarding this facet of the management of these patients are lacking. The literature contains reports of stent deployment over time periods ranging from emergency procedures to interventions performed 3 months after the onset of symptoms.[2,4,9,13,22,27] We do not advocate primary carotid artery stenting for all cases of carotid artery dissection, but we believe that early stenting may provide a better chance for reperfusion of the critical penumbra. We also believe that stenting could be combined with antithrombolytic therapy in select cases demonstrating hypoperfusion with poor reserve, or in cases of bilateral carotid or carotid and vertebral dissections, given the unpredictable dynamic nature of ICAD progression to occlusion in certain cases. Nonetheless, the clinical decision process must be individualized for each patient, and its risks and benefits need to be clearly explained to the patient and the patient's family or health care proxy.

Endovascular stenting has some readily apparent advantages over surgical treatment, including immediate restoration of flow in the artery leading to early reperfusion of the ischemic brain areas, and the patient may be kept only on a regimen of antiplatelet agents. Recent studies have reported only low rates of complications including distal embolization, perioperative stroke, or new deficits attributable to stent deployment despite performing angioplasty of the deployed stent.[30,32] Atherosclerotic stenoses require angioplasty with higher balloon pressures to treat the underlying often calcified atherosclerotic lesion. The absence of such underlying atherosclerosis in most ICAD cases makes balloon angioplasty very seldom needed; instead, the radial force of the appropriately sized self-expanding stent can be relied on to restore the native vessel caliber.[32]

The most common disadvantages and limitations of

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endovascular therapy include the apparent difficulty to correctly identify, microcatheterize, and canalize the true lumen of the ICA and the potential need for deployment of more than one stent in a telescoping fashion to cross the often long distance of the dissection. A note of caution must be stated with respect to the often-worsening angiographic appearance after deployment of the distal stent. Provided that the true lumen was initially confirmed and an exchange-length microwire was kept in place throughout in the true lumen, the endovascular operator must not panic or withdraw the exchange microwire, but rather forge ahead and deploy additional tandem overlapping proximal stents until the proximal dissection inflow zone is tacked down, access to the false lumen is sealed off, and the healthy segments of the affected carotid artery are reconnected. The transient worsening of the flow after distal stent deployment is usually the result of the windsock-like dissection flap becoming bunched up proximally as it is squeezed back like a tube of toothpaste, leading to further compression and a worsening angiographic appearance compared with that seen before intervention. Our long-term follow-up imaging suggests that these long constructs in nonatherosclerotic vessels remain widely patent at follow-up and do not seem to share the same risk of restenosis seen in stent therapy for atherosclerotic disease.

Conclusions

The 2 patients presented in this report underwent endovascular stenting after a thorough discussion about other noninvasive options had been conducted and the failure of medical therapy had been determined. They underwent recanalization in the setting of minimal diffusion-weighted imaging changes on MR imaging and dense hemodynamically dependent deficits. The hemodynamic isolation of the hemisphere supplied by the affected ICA warranted a rather aggressive therapy. In both cases, restoration of carotid artery flow resulted in the return of neurological function with little residual deficit, and the treated ICA was seen to remain patent after a 6-month follow-up period.

Internal carotid artery dissection causing occlusion or near-occlusion with intracranial embolism is an important cause of severe and potentially life-threatening hemispheric ischemia. Stenting may be an appropriate method to alter the clinical course in select patients during the acute, clinically dynamic phase of dissection. We believe that endovascular stent deployment is a feasible and effective method to treat subacute ICAD with complete or near-complete occlusion, especially in the absence of adequate collateral flow. Stenting should be considered in patients in whom radiographic evidence of an imminent irreversible stroke is evident, such as in those with isolated cerebral hemisphere ipsilateral to the dissection.

Disclosure

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Author contributions to the study and manuscript preparation include the following. Conception and design: Malek. Acquisition of data: Malek. Analysis and interpretation of data: Malek, Atalay. Drafting the article: Schirmer, Atalay. Critically revising the article: all authors. Reviewed final version of the manuscript and approved it for submission: all authors.

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Address correspondence to: Adel M. Malek, M.D., Ph.D., Department of Neurosurgery, Tufts Medical Center, 800 Washington Street #178, Boston, Massachusetts 02111. email: amalek@tuftmedical center.org.

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