CASE REPORT

"Malignant" Carotid Artery Dissection

J. Max Findlay, Robert Ashforth, Naeem Dean

ABSTRACT: ***Purpose:*** Carotid artery dissection resulting in occlusion or severe narrowing and massive intracranial embolism can result in life-threatening hemispheric ischemia. Aggressive endovascular and microsurgical measures may be necessary to salvage life and minimize stroke morbidity in this extreme situation. ***Patients and methods:*** We have treated two middle-aged women who presented within an hour of spontaneous cervical internal carotid artery (ICA) dissection causing hemiplegia, forced head and eye deviation, and declining consciousness. The first patient had a carotid occlusion through which a catheter could not be passed, so intracranial thrombolysis was achieved through a microcatheter navigated through the posterior circulation. Surgical intimectomy and thrombectomy of the dissected ICA was then carried out using an intraoperative Fogarty arterial embolectomy catheter passed up the dissected ICA, followed by endovascular stenting of the reopened cervical ICA. The second patient underwent intracranial microsurgical embolectomy and, after an unsuccessful attempt of stenting the dissected and severely narrowed cervical ICA, surgical reopening again with a Fogarty catheter. Both patients suffered basal ganglionic infarcts but most of the middle cerebral artery territories were preserved and the patients made satisfactory recoveries. ***Conclusions:*** "Malignant" carotid artery dissection causing occlusion or near occlusion with intracranial embolism is an important cause of severe and life-threatening hemispheric ischemia. Treatment should include aggressive endovascular and microsurgical interventions when the hemisphere is at risk.

RÉSUMÉ: Dissection "maligne" de la carotide. ***But:*** La dissection de la carotide produisant une occlusion ou un rétrécissement sévère et une embolie intracrânienne massive peut produire une ischémie hémisphérique menaçant la vie. Des mesures endovasculaires et microchirurgicales agressives peuvent être nécessaires pour sauver la vie et minimiser la morbidité par accident vasculaire cérébral dans cette situation extrême. ***Patients et Méthodes:*** Nous avons traité deux femmes d'âge moyen qui ont présenté une hémiplégie, une déviation forcée de la tête et des yeux et une altération de l'état de conscience moins d'une heure après une dissection spontanée de la carotide interne (CI) cervicale. La première avait une occlusion carotidienne à travers laquelle un cathéter ne pouvait passer, de telle sorte que la thrombolyse intracrânienne a été effectuée au moyen d'un microcathéter introduit par la circulation postérieure. Une intimectomie chirurgicale et une thrombectomie de la CI disséquée a ensuite été effectuée au moyen d'un cathéter de Fogerty pour embolectomie artérielle placé au delà de la CI disséquée, suivie de la mise en place d'une endoprothèse dans la CI cervicale réouverte. La deuxième patiente a subi une embolectomie microchirurgicale intracrânienne et, après une tentative infructueuse de mise en place d'une endoprothèse dans la CI cervicale disséquée et sévèrement rétrécie, elle a subi une réouverture chirurgicale avec un cathéter de Fogarthy. Les deux patientes ont subi des infarctus dans les noyaux gris centraux mais la plupart des territoires de l'artère cérébrale moyenne ont été préservés et les patientes ont eu une récupération satisfaisante. ***Conclusions:*** La dissection "maligne" de la carotide causant une occlusion totale ou quasi totale avec embolie intracrânienne est une cause importante d'ischémie hémisphérique sévère, menaçant la vie. Le traitement devrait inclure des interventions endovasculaires et microchirurgicales agressives quand l'hémisphère est à risque.

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Carotid artery dissection can, on occasion, present with life-threatening cerebral ischemia due to acute carotid occlusion or severe stenosis with massive intracranial embolism. We have recently managed two patients with this kind of "malignant" carotid dissection and evolving infarction of the whole middle cerebral artery territory, combining endovascular and surgical treatments. In this report we wish to distinguish this separate group of cervical carotid artery dissections where acute carotid revascularization is necessary to preserve life and minimize morbidity.

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Case report

Patient no. 1

A 50-year-old previously healthy woman collapsed in her home and on arrival at hospital 50 minutes later was found to have right hemiplegia, global aphasia and conjugate eye and head deviation to the left. Her family was unaware of any recent head or neck trauma and she had never undergone chiropractic manipulation. A cranial CT scan done 75 minutes after the ictus showed a hyperdense left middle cerebral artery (MCA) (Figure 1a) and early ischemic changes in the adjacent insular region. Cerebral angiography immediately following demonstrated a tapered occlusion of the left cervical internal carotid artery (ICA) suggesting carotid dissection (Figure 1b). No contrast entered the left side of the circle of Willis from the right ICA injection. A catheter could not be passed through the occluded left ICA but a 0.016 inch microguide wire (Terumo Corp., Tokyo) and then 0.018 inch microcatheter (Rapid Transit, Cordis Endovascular Systems Inc., Miami) was passed through the vertebrobasilar arteries and a hypoplastic left posterior communicating artery into the left supraclinoid ICA, allowing demonstration of a partially patent left anterior cerebral artery, and confirming complete blockage of the left MCA (Figures 1c, d). Over 40 minutes a total of 31 mg of recombinant tissue plasminogen activator was infused into the left MCA via this microcatheter, resulting in partial recanalization (Figure 2a), but without a change in the patient's condition.

Since the patient's condition remained consistent with an evolving complete MCA infarction, and because poor flow into the left supraclinoid ICA threatened MCA reocclusion, an attempt to surgically reopen the left cervical ICA was made. The left common carotid bifurcation was exposed, and approximately 15 mm from its origin the ICA became slightly swollen and blue in color. The patient was given intravenous heparin (5000 IU) and the common and external carotid arteries were temporarily occluded. With the assistance of the surgical microscope, a short arteriotomy was made in the proximal and normal-appearing ICA, which had an intact lumen. There was no retrograde bleeding down the ICA. A 2F Fogarty arterial embolectomy catheter (Edwards Lifesciences LLC, Irvine, Ca) was passed up the ICA, meeting no resistance until it reached 12 cm. It was withdrawn several millimeters and the balloon was inflated with 0.2 ml of sterile saline. The catheter was then withdrawn, retrieving a column of thrombus and a ring of intimal tissue encircling the catheter beneath the balloon. This was immediately followed by weak back-bleeding from the ICA. The catheter was passed several more times but without withdrawing additional debris, so the arteriotomy was closed and the arteries were declamped. Intraoperative Doppler ultrasound recordings indicated pulsatile flow up the ICA, roughly four hours from stroke onset.

The patient was taken back to the angiography suite under anaesthesia where it was confirmed that flow had been reestablished in the cervical ICA, which now had an irregular and widened lumen (Figure 2b). There was rapid filling of the left MCA. An 8 x 14 mm Wallstent (Boston Scientific Scimed, Inc., Minneapolis) was deployed down the ICA to its origin, resulting in an improved, smoother appearance of the cervical segment of the artery (Figure 2c). The patient was kept fully heparinized following the procedure.

The following day the patient was alert, but had a severe expressive aphasia. She had antigravity power in the right leg, but no arm movement. CT scanning showed basal ganglia and frontal infarcts, but the majority of cerebral cortex supplied by the MCA was intact (Figure 3a). At discharge three weeks later she was ambulating and was beginning to move her right arm. Heparin had been replaced by clopidogrel (75 mg bid). After 18 months she was fluently conversant complaining of occasional word-finding difficulties, had a weak but useful right arm, a spastic right hand, and a normal gait. She was independent in daily living, and had resumed part-time work as a children's book writer and editor. A follow-up cerebral angiogram at that time showed intimal hyperplasia within the carotid stent, resulting in moderate stenosis, which will be monitored (Figure 3b).

Patient no. 2

This 49-year-old medical transcriptionist with no history of neurological or cardiovascular disease or any type of head or neck trauma, had a sudden onset of left-sided paralysis while eating lunch with her family. On arrival to hospital 30 minutes later she was alert and able to talk, but had a dense left hemiplegia and forced eye and head deviation to the right. A CT scan showed a hyperdense right MCA, but was otherwise normal (Figure 4a). An interventional radiologist was not immediately available so neurosurgery was consulted. Two hours and 45 minutes from stroke onset and immediately prior to surgery, she had become drowsy with little verbal output. A right fronto-temporal craniotomy and MCA embolectomy was performed, with reestablishment of brisk MCA flow four hours and 45 minutes from ictus. At surgery it was found that the MCA embolus extended from the ICA terminus to the MCA bifurcation, with clot extending into both MCA divisions, and two main-branch arteriotomies were required to remove all embolus. Flow in the MCA and all exposed branches was confirmed with an intraoperative micro-Doppler flow probe.

The patient awoke promptly from surgery and was able to move her left side against gravity, and was able to look to the right. The next day she was alert, could lift both upper and lower left limbs to command, and could grip with her left hand. A cerebral angiogram confirmed the clinical suspicion of a right cervical ICA dissection, ending in a severe stenosis just proximal to the carotid canal (Figure 4b). The right MCA was patent but receiving little flow. A CT scan showed patchy right temporal lobe and basal ganglionic infarction, but most MCA-supplied territory was preserved. A heparin infusion was continued and blood pressure maintained over 140 mm Hg systolic with the support of a norepinephrine infusion.

The patient's condition remained stable for six days, at which time the vasopressor was discontinued. Twelve hours later, at which point her systolic blood pressure had fallen to 100 mm Hg, she lost all power in her left arm. Resumption of norepinephrine infusion and elevation of her systolic blood pressure over 150 mm Hg reversed the deficit over several hours. The next day repeat angiography showed worsening of the right ICA stenosis (Figure 5a), and still very poor angiographic filling of the right ICA (Figure 5b). There appeared to be no collateral flow from the left carotid via the anterior communicating artery. A guidewire could not be passed up the right ICA, precluding an attempt of stenting open this artery. Induced hypertension was continued until the next day, the ninth day from admission, at which time she underwent surgery.

At operation the right internal carotid artery had a blue discoloration 12 mm from its origin. Following an additional 2000 IU of heparin the origin of the ICA was occluded with a large aneurysm clip. The common and external carotid arteries were kept patent, since even temporarily compromise of collateral flow to the distal ICA from external carotid artery branches was considered undesirable. Under magnification an arteriotomy was made in the proximal ICA. The 2F Fogarty catheter was passed 14 cm up the ICA until slight resistance was felt, and withdrawal of the expanded balloon was accompanied by thrombus and a small amount of back-bleeding. An intraoperative angiogram was taken using a flat-plate beneath the patient's head and neck, an overhead portable

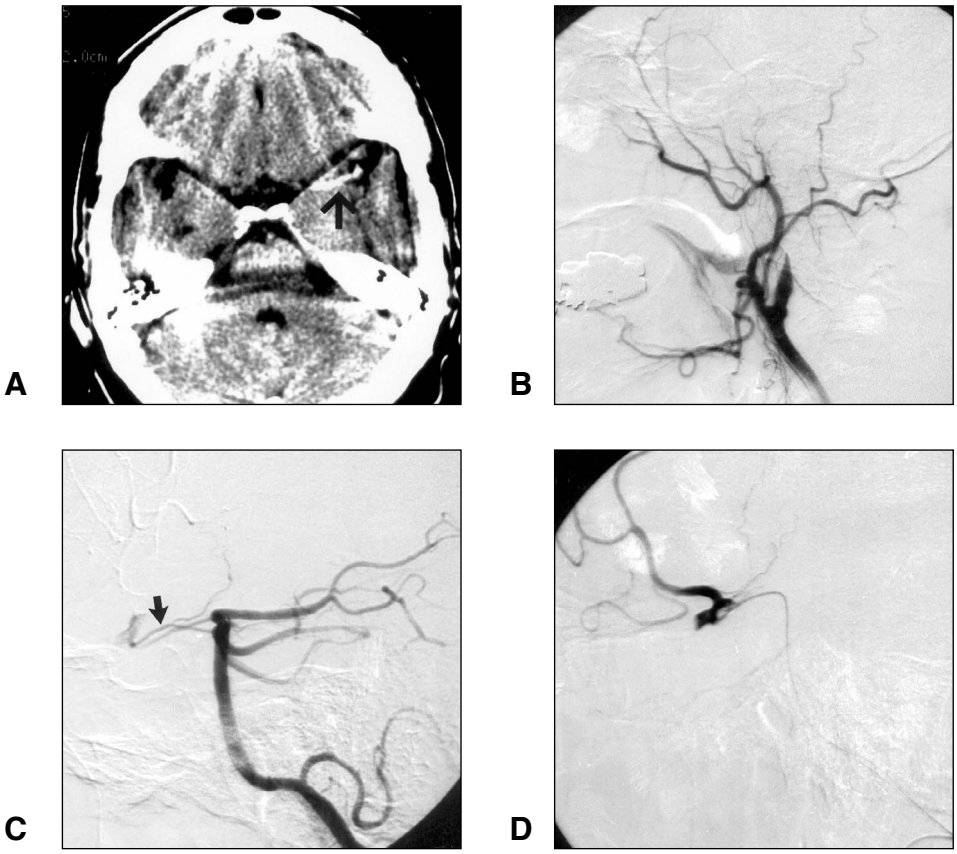

Figure 1: *A CT scan done 75 minutes after collapse of the first patient shows a "hyperdense" left middle cerebral artery (a), and cerebral angiography following demonstrated a tapered occlusion of the mid-left cervical internal carotid artery (b), suggesting spontaneous dissection. Vertebral angiography showed patency of the posterior communicating arteries (c), and a microcatheter passed through the left posterior communicating artery showed patency of the anterior cerebral artery but confirmed complete left middle cerebral artery embolic occlusion (d).*

camera, and a hand injection of 6 cc of omnipaque contrast agent directly into the right common carotid artery through a 20 gauge angiocatheter. This demonstrated patency of the ICA and rapid filling of the MCA.

Postoperative angiography confirmed good ICA caliber (Figure 6), and while it was rough in several places, stenting was not felt necessary. Heparin and vasopressor support were discontinued and clopidogrel (75 mg bid) started the following day. She was ambulatory within several days and was discharged home with mild hemiparesis eighteen days from surgery.

Discussion

Reports from Ehrenfeld and Wylie,[1] Fisher et al[2] and Mokri et al[3] in the 1970s indicated that spontaneous, nontraumatic

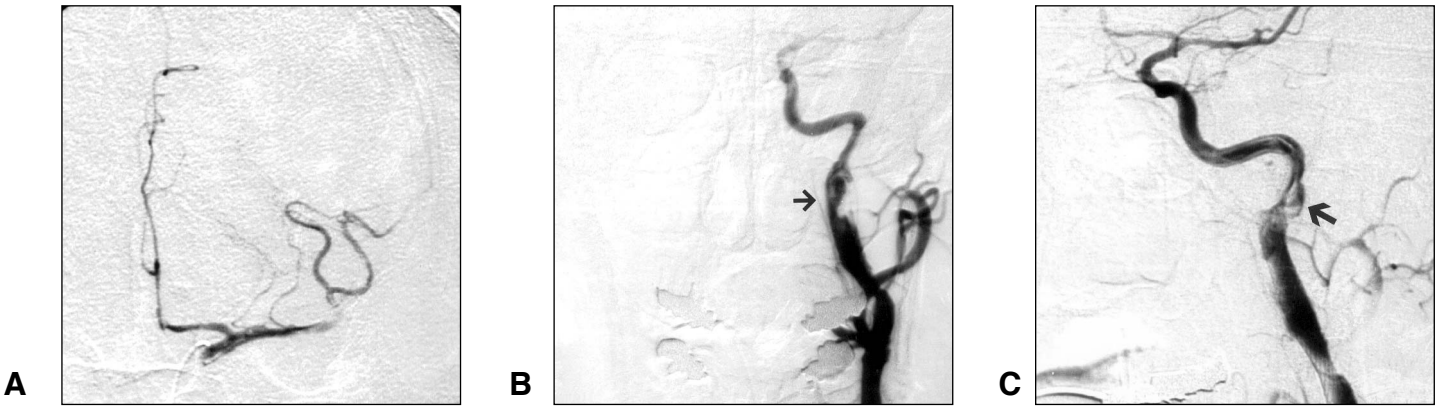

Figure 2: *Following the administration of rt-PA into the left middle cerebral artery there is partial recanalization (a). Angiography immediately following surgical thrombectomy and reopening of the left cervical internal carotid artery shows irregularity of the distal extra-cranial artery, with a probable intimal flap (arrow, b). Deployment of a stent improved the appearance of the cervical segment of the internal carotid artery, but irregularity and either a persistent double lumen or flow laminar artifact in the proximal petrous segment (arrow, c).*

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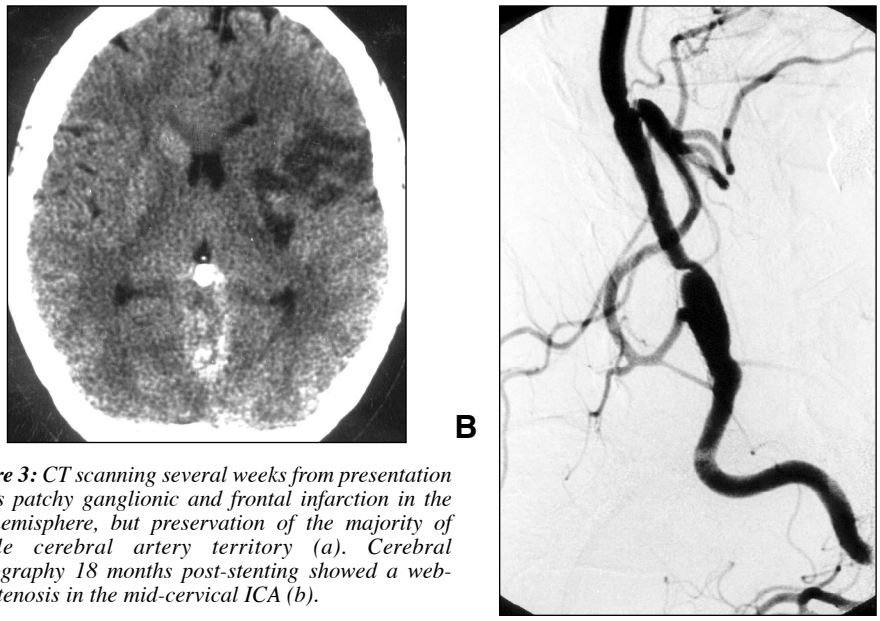

Figure 3: *CT scanning several weeks from presentation shows patchy ganglionic and frontal infarction in the left hemisphere, but preservation of the majority of middle cerebral artery territory (a). Cerebral angiography 18 months post-stenting showed a web-like stenosis in the mid-cervical ICA (b).*

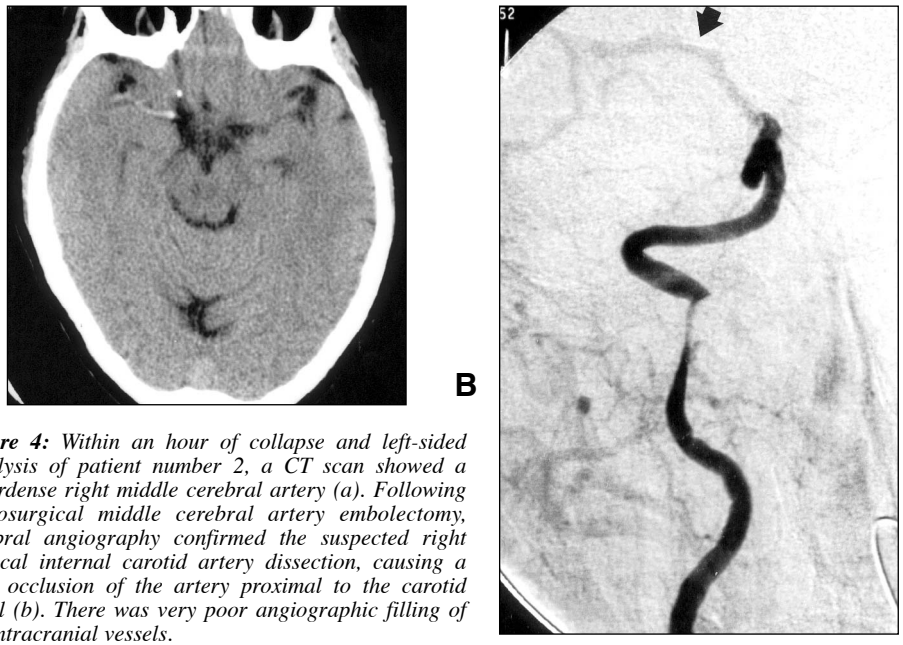

Figure 4: *Within an hour of collapse and left-sided paralysis of patient number 2, a CT scan showed a hyperdense right middle cerebral artery (a). Following microsurgical middle cerebral artery embolectomy, cerebral angiography confirmed the suspected right cervical internal carotid artery dissection, causing a near occlusion of the artery proximal to the carotid canal (b). There was very poor angiographic filling of the intracranial vessels.*

dissection of the extracranial carotid artery was not as rare as thought prior to that time.[4] Describing patients without any notable precipitating head or neck trauma, these authors found the condition commonest in mid-life, presenting with combinations of hemispheric or retinal ischemia or infarction, ipsilateral head or neck pain, and incomplete Horner's syndrome (oculosympathetic palsy, consisting of miosis and ptosis). The commonest finding at angiography was shown to be long, irregular narrowing of the ICA beginning beyond the common carotid bifurcation (the "string sign"), usually ending at the petrous bone and carotid canal and sometimes associated with an aneurysmal outpouching somewhere along the dissected segment. Early occlusion from carotid dissection usually appears as a tapering occlusion beyond the carotid sinus more distal than generally seen with atherothrombotic occlusions.

The subject of carotid dissection has been reviewed by Hart and Easton,[5] Anson and Crowell[6] and most recently by Schievink.[7] Dissection of the vertebral artery is considered an analagous process, and is at least as common as carotid artery dissection along its extradural segment, but occurs intradurally (usually resulting in subarachnoid hemorrhage) much more commonly than intradural dissection of the carotid artery.[8,9] Beginning with an intimal tear, blood penetrates the vessel wall and dissects within or between the vessel wall layers, resulting in

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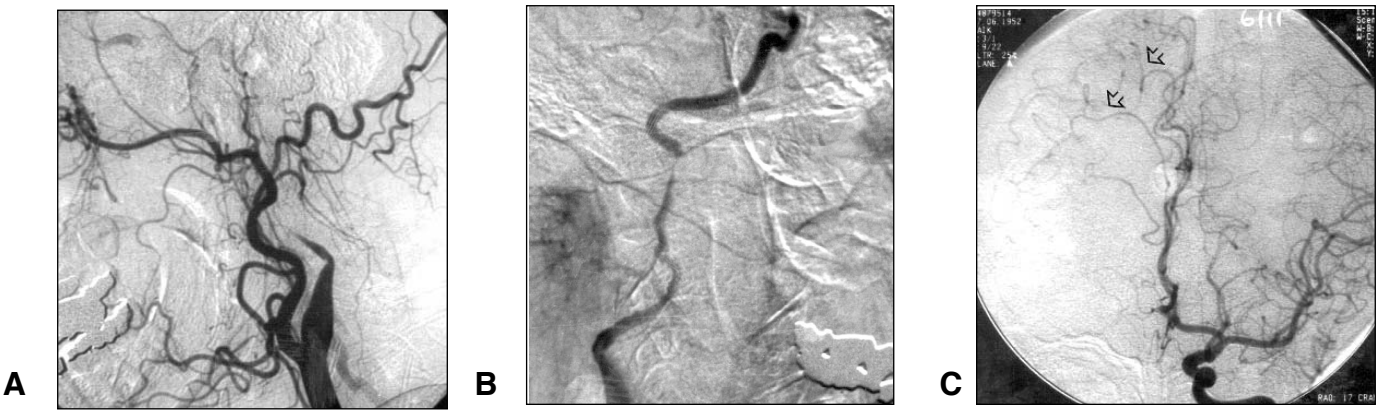

Figure 5: *Seven days after carotid dissection, and following clinical failure of vasopressor and induced hypertension withdrawal, repeat cerebral angiography showed worsening of the right carotid dissection (a), with very sluggish filling of the petrous and cavernous segments (b). Injection of contrast into the left internal carotid showed that both anterior cerebral arteries filled from the left, with some collateral flow reaching the distal right middle cerebral artery territory (arrows, c). The right proximal anterior cerebral artery A1 segment did not opacify.*

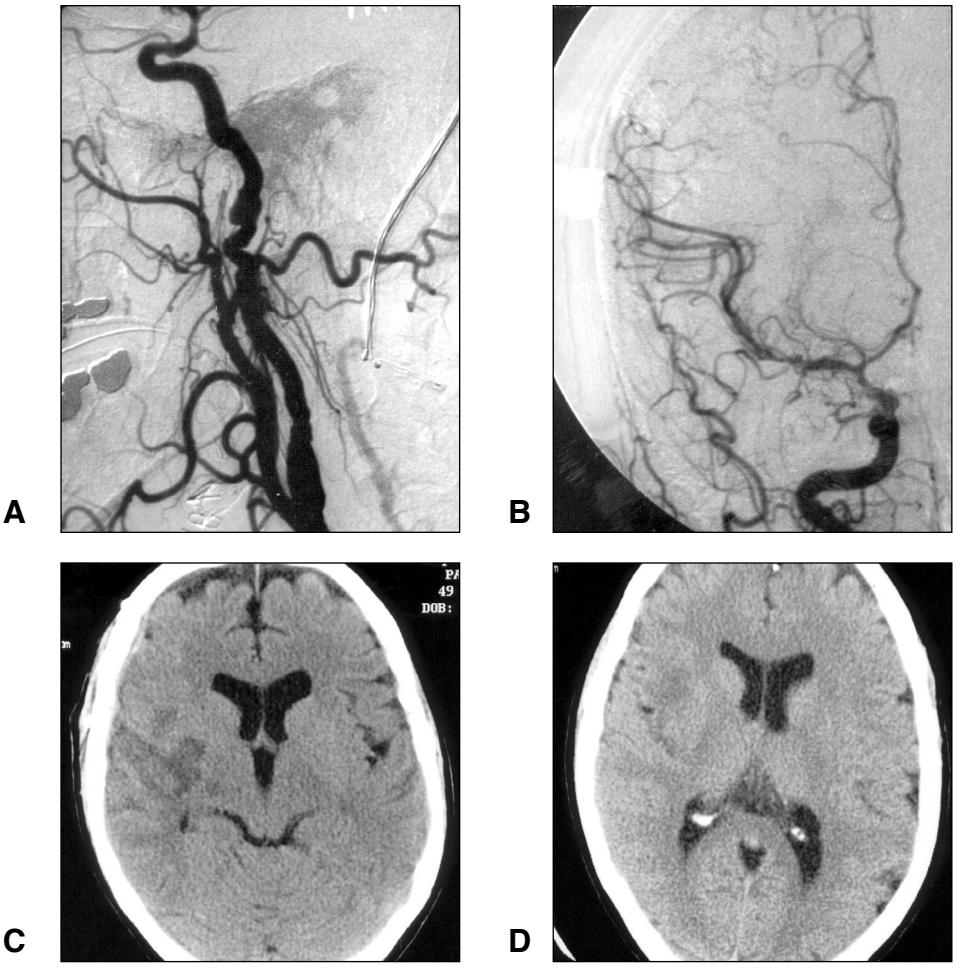

Figure 6: *The right extracranial internal carotid showed mild to moderate irregularity throughout its length following surgical balloon-catheter intimectomy and thrombectomy (a), and there was brisk filling of the right-sided intracranial vessels (b). Follow-up CT scanning showed patchy right ganglionic and fronto-temporal infarction, but most of the middle cerebral artery territory remained intact (c,d).*

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narrowing or occlusion of the true lumen and, if the intramural hematoma breaks back into the normal lumen distally, embolization can occur. Rarely a second "false" lumen can form. Aneurysmal dilatations result from subadentitial dissections (a dissecting aneurysm), and since the aneurysm wall is made up of elements of the normal arterial wall, they are not false- or pseudoaneurysms, although they are frequently referred to as such. Most patients suffering spontaneous craniocerebral dissections do not have any recognizable underlying connective tissue disorder or heritable arteriopathy that might render them susceptible to injury from normal arterial stresses or stretches.

The diagnosis of carotid artery dissection should be suspected in patients presenting with acute carotid distribution ischemia, especially when younger or middle aged and lacking obvious risk factors for atherosclerosis. The majority of strokes in patients with dissection of the carotid artery are embolic in the territory of the MCA.[10,11] Additional diagnostic clues, not present in all patients, are the presence of head or face pain and a partial Horner's syndrome. Carotid duplex ultrasonography may suggest the diagnosis,[12,13] as can magnetic resonance imaging,[14,15] but conventional angiography remains the most accurate diagnostic test.

The treatment for most carotid dissections causing transient ischemia or minor thromboembolic strokes has been either anticoagulation or platelet antagonists, although neither has been tested in randomized trials for this condition.[16]. Follow-up imaging has indicated that the majority of carotid dissections "heal" spontaneously, with improvement or resolution of up to three-quarters of stenoses.[17-19]

More aggressive interventions have generally been reserved for patients with recurrent cerebral ischemia despite medical therapy, or for patients with aneurysmal forms of carotid dissection considered potential sources of thromboemboli or causing compressive symptoms, since it is well-recognized that these aneurysms do not rupture.[20] Surgical options have included aneurysm resection and insertion of an interposition graft,[21] ligation of the ICA with or without a carotid bypass graft, such as a superficial temporal to MCA anastomosis,[3,7,22] an extracranial to supraclinoid ICA saphenous vein bypass,[23] or an extracranial to petrous ICA saphenous vein bypass.[7,24] The majority of these procedures described in the literature have been intended to eliminate large dissecting aneurysms. Endovascular treatments described recently have consisted of stent placement over the dissected carotid segment, sometimes combined with coil-embolization of aneurysmal outpouchings through the stent struts.[25-27]

The two patients with carotid dissections we have described are unique in that they presented soon after the onset of complete MCA-territory ischemia due to carotid occlusion or near occlusion and intracranial embolism. This type of hemispheric stroke, characterized by hemiplegia, forced eye and head deviation, and declining consciousness is associated with either death or severe disability in up to 80% of patients.[28] It is unlikely that the patients described in our report would have either survived or recovered as fully without early revascularization. The percentage of all spontaneous extracranial carotid dissections that present with this "malignant" MCA ischemic syndrome is not known with certainty, but is probably less than 10%.[5-7,29] Yet, in a series of 818 MCA infarcts analyzed in the Lausanne Stroke Registry, 25% of the 208 large MCA infarcts described were due to dissection,[30] and 19 of 63 patients (30%) who underwent hemicraniectomy for complete MCA infarction in the Hedielberg series had carotid dissections as the underlying etiology.[31] When only large MCA infarcts are considered, particularly in young people, carotid dissection appears to be a leading cause. The management of this "malignant" variant of carotid dissection should be considered separately from carotid dissections in general.

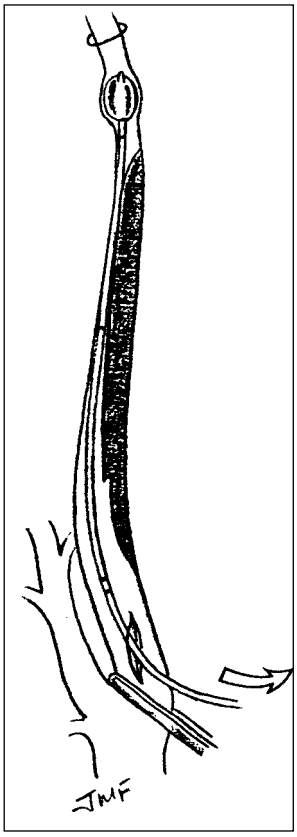

Figure 7: *Schematic drawing of balloon-catheter carotid intemectomy and thrombectomy through an arteriotomy proximal to the dissection.*

The first priority in treatment is elimination of intracranial thromboembolus. Intravenous fibrinolysis is associated with relatively low rates of recanalization of large intracranial (ICA, MCA) arteries.[32,33] Intra-arterial fibrinolysis is more effective if a catheter can be passed through the dissected segment.[34,35] In the patient treated successfully by Nesbit et al[36] in this manner, rather than attempt to repair the proximal dissected cervical ICA, the artery was deliberately occluded with coils in order to prevent further embolic events. If it isn't possible to pass a guidewire through the dissection, then either navigation of a microcatheter through collateral vessels (as in our first patient) or MCA embolectomy (as in our second)[34] are options. The time window for this type of intervention varies among individuals

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(depending on the availability of collateral blood flow), but is generally a matter of hours.[34]

The second priority is an attempt at restoring flow in the ipsilateral ICA, since lengthy bypass revascularization procedures around the dissected artery are impractical in an emergency setting when the brain is hemodynamically compromised. Liu et al[26] and Malek et al[27] described series of patients with carotid dissections that were treated primarily with carotid artery stenting. Three of the patients in these two series were stented acutely following carotid dissection, all three were iatrogenic dissections caused by angiography, and only one was symptomatic. We were unable to pass a guide-wire through either of the two dissected carotid arteries in our patients. If one is successful and able to deploy a stent in this situation there is a theoretical risk of expressing intramural clot contained within the dissected segment into the cerebral circulation. There is currently very limited experience with carotid stents in the specific circumstance of acute carotid dissection causing brain ischemia.

When stenting is not feasible, as was the case in the two patients we describe, surgical reopening of the dissected ICAs can be considered. The method we describe in this report is similar to that used by Ojemann[4] in 1972 in a patient with a high cervical carotid dissection, and consists of a Fogarty arterial embolectomy catheter intimectomy and thrombectomy, but without the arterial resection and interposition grafting used in Ojemann's patient (Figure 7). The advantages of this method are that it is simple, fast and, at least in theory, poses a lower risk of embolization into the cerebral circulation because there is only retrograde blood flow down the ICA during the procedure. Its disadvantage is that the catheter passed up the carotid artery could enter the dissection and further disrupt the vessel resulting in either occlusion or hemorrhage into the neck. To reduce these risks, it is recommended that care be taken to introduce the catheter only into true lumen (we have found magnification using the surgical microscope helpful), not to advance the catheter against resistance or further than the estimated length of the cervical carotid artery. The length of the cervical ICA is between 10 to 15cm, but is best ascertained from studying the angiogram. An intraoperative or immediate postoperative angiogram (with the patient still intubated and under anaesthesia) is also recommended. If flow is reestablished but the ICA lumen appears quite irregular, with hemodynamic compromise threatening reocclusion, then postoperative stenting is a consideration, along with anticoagulation or antiplatelet therapy. If flow has not been reestablished it is unclear how best to proceed in the face of critical hemispheric ischemia. Consideration might be given to an emergency cerebral bypass, or to decompressive hemicraniectomy if infarction cannot be prevented.

In summary, spontaneous, occlusive carotid dissection with intracranial embolism occasionally presents with life-threatening hemispheric ischemia. It is likely that this type of malignant ICA dissection will be more commonly discovered in the future with the introduction of more invasive clinical approaches to acute stroke. Aggressive endovascular and/or surgical revascularization is necessary to save the cerebral hemisphere in these situations, although some, and especially deep, brain infarction is inevitable. These types of deep infarcts do not preclude an eventual satisfactory recovery, as seen in both of the patients we have presented. In this report we describe potential roles for surgery in the management of these patients, including intracranial embolectomy when intra-arterial fibrinolysis cannot be performed, and ICA intimectomy and thrombectomy with a balloon catheter, when stenting open the dissection is not feasible.

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