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Diagnosing and Treating Atypical Arterial Pathologies of Aortic Arch Vessels: Dissection and Fibromuscular Dysplasia

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Although rare, pathologies of the aortic arch vessels can result in devastating sequelae. This article will address two of these pathologies, fibromuscular dysplasia and arterial dissection, along with diagnosis and treatment options.
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SINCE ITS FIRST description in 1938, the understanding, diagnosis, and treatment of fibromuscular dysplasia (FMD) have evolved through different stages.

Growing use of various noninvasive diagnostic imaging modalities such as computed tomographic angiography (CTA) has increased the incidental diagnosis of asymptomatic FMD. Advance endovascular techniques have made endovascular approaches the first line of treatment for this clinical problem. As such, traditional surgery is now reserved for complicated cases involving carotid and/or an aneurysmal segment as a last-resort option.

The major focus of current research in FMD aims at the identification of genes related to the disease, as well as a better understanding of the risks of disease progression and the targeting of prevention mechanisms.

Epidemiology and Pathophysiology

FMD or fibrodysplasia is a noninflammatory, nonatherosclerotic, systemic degenerative angiopathy of the medium-sized arteries. FMD shares both occlusive and aneurysmal morphology.

It is an idiopathic disease process and multiple factors seem to be involved in the pathogenesis, including vessel wall ischemia, mechanical stress on the arterial wall, smoking, and hormonal and genetic criteria.[1,2] Familial predisposition seems to be important and disease prevalence in first-degree relatives has been reported in at least 11% of documented studies.[3] However, most of these studies are limited in size and no definite etiology has been reported.

Several diseases, including pheochromocytoma, Ehlers-Danlos syndrome, Marfan syndrome, and Alport syndrome, have been associated with FMD. Whether they are phenotypically interrelated to FMD remains to be determined.[4]

FMD is classified into three histological types, depending on the preponderant layer of arterial wall involvement, ie, intima, media, or adventitia. Medial dysplasia represents 80% to 90% of all cases.

The disease can affect virtually every arterial bed, including coronary, mesenteric, hepatic, iliac, or vertebral arteries. The renal arteries are the most frequently affected arteries and represent 75% of the cases of FMD.

Second to renal arteries, internal carotid involvement represent 25% to 30% of the cases. A great proportion of patients are young, white females (female to male ratio = 6-9:1). Patients commonly present between the ages of 20 and 50 years old. The true incidence of carotid FMD in the population is unknown, but based on the largest series of 20,244 postmortem examinations, it seemed to occur at a rate of 0.2%.[5] A much higher incidence (close to 3.5%) has been suggested, but no clear data supports this assumption.

Histologically, most of the carotid FMD is of the medial fibroplasia type, a subtype of the medial dysplasia. These lesions are characterized by areas of thin media alternating with areas of thick fibromuscular ridges, resulting in an alternating sequence of stenosis and poststenotic dilation. This arrangement gives the artery the typical "string of beads" arteriographic appearance (Fig 1A, B). The artery can also become elongated or kinked and a small number of the dilated segments can become aneurysmal, with a risk of subse-

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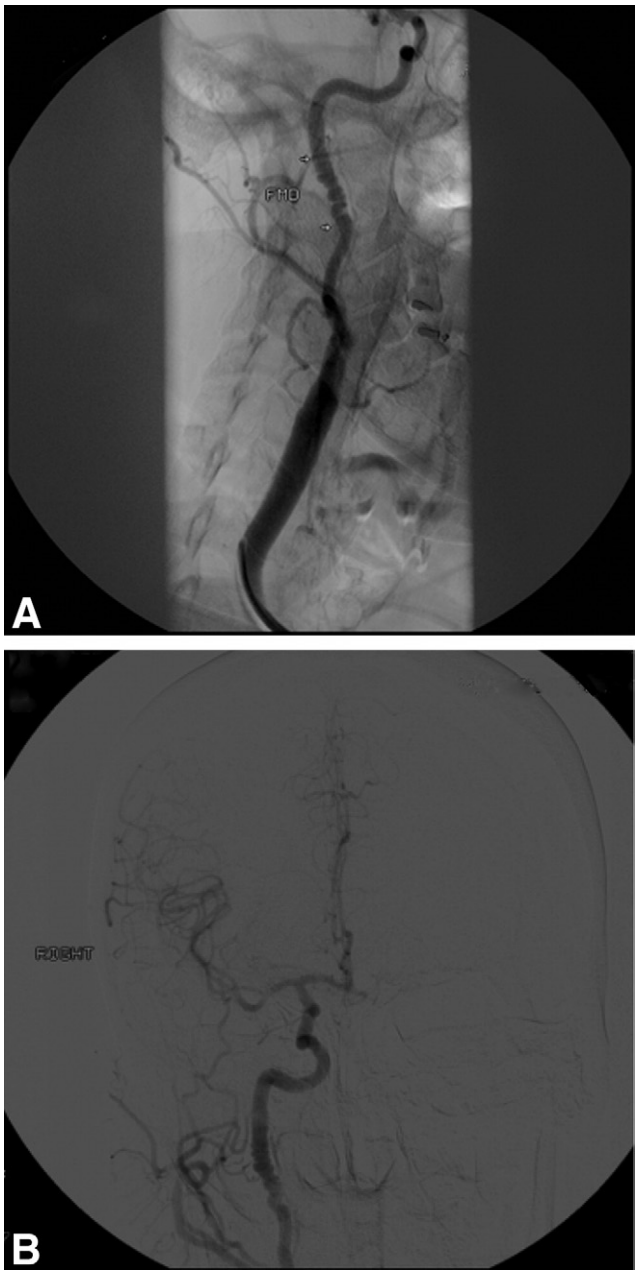

Figure 1 (A) Carotid and (B) cerebral angiography of internal carotid artery fibromuscular dysplasia (FMD) (courtesy of Pegge Halandras, MD and Karthikeshwar Kasirajan, MD).

quent rupture.[6] It has also been demonstrated that 2.3% of extracranial aneurysms are associated with FMD.[7]

FMD involves mostly the middle and/or distal internal carotid artery (ICA), adjacent to C1-2, as opposed to the proximal location of atherosclerotic carotid disease. Bilateral carotid involvement has been reported at different rates, but the rate is likely around 50% to 65%, as documented in the older literature.[8,9]

The prevalence of intracranial aneurysms seems to be lower than the 21% to 51% commonly cited in the literature. A meta-analysis from 18 reports concluded that there was a 7.3% prevalence of incidental, asymptomatic cerebral aneurysms in patients who had ICA and/or verteberal artery FMD.[10] This prevalence is still higher compared to the presence of intracranial aneurysms in the general population, which is estimated at 1% to 5%. It follows that patients with carotid FMD should undergo a head CT for intracranial aneurysm screening.

FMD can affect multiple arterial beds simultaneously. Vertebral FMD, seen in <10% of cases, is virtually only present in association with carotid FMD. Clinically, particular attention should be paid in screening for renal FMD in patients with carotid FMD and hypertension. The screening and treatment of the renal hypertension is even more important when an intracranial or extracranial aneurysm is present because the risk of aneurysm rupture can be related to hypertension that is not controlled.

FMD of the aortic arch and other arch branches, subclavian artery or innominate artery is rare and limited to case reports. They can result in arm weakness, claudication, or even stroke.[11-13] The differential diagnosis of this problem includes vasculitis, such as Takayasu arteritis.

Clinical Presentation

The majority of diagnosed patients with carotid FMD are asymptomatic and the disease is found incidentally during various imaging workups. When symptoms are present, they range from nonspecific to severe neurological impairment.

Nonspecific symptoms include headaches, neck pain, a subjective swishing sound in the ear, tinnitus, or vertigo. Particular attention should be paid to young patients with a carotid bruit, and in such cases FMD screening should be initiated.[14]

More specific and dramatic neurological symptoms include amorosis fugax, Horner syndrome, or cranial nerve palsies. Transient ischemic attacks, reversible ischemic events, or a stroke can result from cerebral hypoperfusion as a result of carotid stenosis, thrombus formation within the dilated arterial segments, distal embolization, or dissection of the carotid and subsequent arterial thrombosis. Patients can also present with subarachnoid hemorrhage (SAH) secondary to tandem intracranial aneurysmal rupture.

A clinician facing an acute focal neurological deficit in a young patient with no cardiovascular risk factors should suspect the presence of cerebrovascular FMD.

The simultaneous presence in a patient of an ischemic stroke due to carotid stenosis and subarachnoid hemorrhage is characteristic of carotid FMD. An intact tandem aneurysm can also provoke compression of surrounding intracranial structures with resulting neurological deficit.

Diagnosis

Duplex ultrasound (DUS) is commonly the first diagnostic modality in carotid pathologies. Despite the fact that it is operator-dependent, DUS is noninvasive, does not require intravenous contrast, allows direct visualization of the arterial wall (potentially revealing the "string of beads" pattern), and gives an important hemodynamic assessment of the carotid lesions. However, DUS sensitivity to determine carotid

FMD is lower than when used to determine atherosclerotic disease. The disease mostly affects the middle and distal ICA and it is difficult to correctly visualize these distal lesions.

Patients presenting with focal neurological symptoms will likely undergo a head and neck CTA as part of their neurological workup. Although the diagnostic accuracy of CTA in asymptomatic patients and patients with nonspecific symptoms remains to be determined, CTA is a valuable tool to assess intracranial pathologies, such as aneurysms or intracranial bleed. It also allows anatomical assessment of the distal carotid arteries and the extent of the FMD or dissection.

Angiography is considered the gold standard for carotid FMD diagnosis. It can be used as a confirmatory test and allows simultaneous percutaneous treatment when necessary.

In one of the first large studies involving FMD patients with cerebrovascular symptoms, Osborn et al reported 3 characteristic and distinct patterns of carotid FMD, all sparing the carotid bifurcation[8]:

Type 1 has the typical "string of beads" appearance, it is present in 89% of cases and is virtually considered pathognomonic of carotid FMD. The presence of irregular constrictions and dilation differentiates it from arterial waves or spastic constrictions.

Type 2 has the "unifocal or multifocal tubular necrosis" pattern. It was present in 7% of patients. Less specific of FMD, the arteriogram reveals a single or multiple smooth long and concentric tubular lesions sparing the origin of the ICA.

Type 3, named *atypical fibromuscular dysplasia* was present in 4% of patients. Only one wall of the artery is involved, sparing the origin of the ICA, and shows a diverticulum-like outpouching that can subsequently become aneurysmal.

In all cases, the distal ICA can also be elongated and/or kinked.

Carotid FMD is noninflammatory, differing from vasculitis involving arch branches, such as Takayasu arteritis. In the absence of acute infarction, patients with carotid FMD would present with normal C-reactive protein levels or erythrocyte sedimentation rates. They would not have chronic anemia or thrombocytopenia. In equivocal cases, a magnetic resonance angiography can be useful for revealing arterial wall thickening, present typically in patients with Takayasu or giant cell arteritis.

Treatment

Different recent review articles and older data suggest that carotid FMD in asymptomatic patients has a benign course.

Although no randomized study is available, aspirin (81 mg daily) is commonly recommended as a stroke-prevention measure in asymptomatic patients. Patients should also be counseled on the potential neurological complications related to FMD and seek immediate medical attention if symptoms develop. In order to prevent arterial dissection, they should avoid activities involving extreme stretching of the neck, such as bungee jumping or chiropractic manipulation. No specific imaging modality and/or frequency of imaging is recommended as part of follow-up for these patients. Also, there are no defined intervention criteria with respect to asymptomatic stenotic lesions. Most clinicians would, however, follow the progression of the disease clinically and with serial DUS every 6 to 12 months.

Symptomatic patients with nonspecific symptoms such as headache or patients with debilitating subjective swishing sound in the ear can potentially benefit from percutaneous transluminal angioplasty (PTA).[15] These patients need to be thoroughly evaluated and other etiology, with respect to their symptoms, must be ruled out before any intervention.

Symptomatic patients presenting with transient ischemic attack or stroke directly related to the pathologic carotid, and not resulting from dissection should be treated by angiography and PTA. In these cases, symptoms are either due to stenotic cerebral hypoperfusion or cerebral embolization from thrombus formed within the dilated arterial segments. There are also no large comparative studies between medical therapy alone and PTA. Most studies recommend PTA alone as first-line treatment and translational experience from the treatment of renal FMD suggests it is an efficient and durable treatment.[16] Avoiding overdilation and use of a single long balloon dilation over the entire diseased segment rather than multiple balloon inflations over the entire segment will theoretically avoid an arterial dissection. Despite the theoretical risk of distal embolization from thrombotic debris during PTA, no data or study suggest any benefit using embolic protection devices or a covered stent to treat FMD. Bare metal stent placement is recommended if dissection develops during PTA. No recommendation exists as part of pre and post-PTA antiplatelet and/or anticoagulation treatment.

Although the controversy on antiplatelet versus anticoagulation therapy for carotid dissection is not yet resolved, symptomatic patients presenting with carotid dissection are commonly anticoagulated to prevent clot formation and extension. Angiographic pattern of FMD is found in 17% of patients with spontaneous carotid dissection.[17] Patients with FMD have also higher rates of recurrent carotid dissection. Systemic heparinization should be followed by oral anticoagulation for 3 to 6 months, allowing the dissected carotid to heal. If the patient develops worsening or recurrent symptoms during treatment, the dissected carotid should be stented.[18]

In the presence of a carotid pseudoaneurysm or aneurysm formation, the use of covered stents can be considered as an alternative to the surgical treatment involving resection, with or without venous interposition. Although long-term results are not available, covered stents seem to offer a promising endovascular treatment option. Another endovascular alternative involves placement of a bare metal stent and transstent aneurysm coiling.

One major argument against the use of stents in carotid FMD is the lack of long-term data with respect to this group of typically young patients. Moreover, some of the lesions are located in the distal ICA and the artery is either elongated or tortuous, making stent placement challenging. There is potentially a risk of mechanically "bending" the distal ICA by

the distal end of the stent, resulting in occlusion and thrombosis.

Coexisting intracranial aneurysms should be treated in a similar way to non-FMD patients based on their symptoms and size.

Mid- and distal ICA FMD occasionally coexist with carotid atherosclerotic disease, which commonly affects the bifurcation of the carotid and origin of the ICA.[19] No clear treatment strategy for asymptomatic patients exists in the literature. In symptomatic patients, because the source of the symptoms is ambiguous, both pathologies should be treated simultaneously. Once again, there is no recommendation that exists for treatment options, including percutaneous, hybrid procedures or surgical resection and interposition for this problem.

Dissection

Dissection of the aortic arch branches can be spontaneous, traumatic, or iatrogenic. Current debates surround the role of anticoagulation, antiplatelet therapy, and thrombolysis in the treatment of dissections, as well as indications for endovascular intervention and stent placement.

Epidemiology and Pathophysiology

Arterial dissection is an intimal tear of the vessel wall that results in a breach of pressured blood flow through the arterial wall layers. Dissection can then become subintimal between the intima and media resulting in lumen stenosis or occlusion, subadventitial between the media and adventitia resulting in a weak arterial wall (the artery can develop an acute or chronic base "dissecting aneurysm," which can commonly be saccular), or a combination of both.

In some circumstances, a spontaneous primary intramural hematoma, occurring within the media, can initiate the dissection. This latter sequence is more commonly observed in the aorta. Acutely, an intramural hematoma can develop that can narrow the arterial lumen and reduce distal blood flow. The lumen can become occluded either through the intimal flap or as a result of the expanding hematoma. Subsequent thrombus can form within the vessel wall or at the intimal tear, which can then cause distal embolization or in situ arterial thrombosis. Additionally, a dissection plane can extend distally, for example up to the intracranial circulation, with a risk of arterial wall rupture and subarachnoid hemorrhage.

Spontaneous, traumatic, or iatrogenic dissections of the aortic arch branches have different epidemiology and pathophysiology. The incidence of spontaneous ICA dissection is between 1 and 3/100,000 per year.[20] Spontaneous dissection of the carotid or vertebral arteries represent 2% of all ischemic strokes. Ten to twenty-five percent of these strokes occur in the young adult population, with a peak incidence in the fifth decade of life. Atherosclerosis is uncommon in these patients.[21] Five percent of patients will report a familial history of spontaneous arterial dissection of the aorta or its major branches.[22]

Underlying arteriopathy and extracellular matrix abnormalities have been incriminated in the pathogenesis. Skin biopsy and electron microscopy analysis of dermal structures reveal that half to two thirds of patients with spontaneous carotid dissections have some connective tissue abnormalities.[23,24] However, <5% of these patients will reveal clinical manifestations of a known connective tissue disorder,[24,25] such as Ehlers-Danlos type 4, Marfan's disease, cystic medial necrosis disease, polycystic kidney disease, osteogenesis imperfect type 1, FMD, and α1 antitrypsin deficit.[26] Theoretically, the structure of the arterial wall is weaker and therefore susceptible to dissection under the influence of hemodynamic shear forces, within the arterial lumen, and/or mechanical external forces such as minor or major trauma.

In 12% to 34% of spontaneous cases, a causal minor trauma such as rapid stretching or rotation of the neck (eg, sneezing, vomiting, chiropractic manipulation) is reported.[27]

Traumatic cerebrovascular dissections result from high-energy injuries. Blunt carotid and vertebral injuries, respectively, present in 0.1% to 1.55% and 0.2% to 0.77% of all trauma patients.[28] Forty to seventy percent of these cases are secondary to motor vehicle collisions. During the last decade, aggressive screening protocols have been adopted by trauma centers and reported mortality rates have decreased to <10%.[29,30] Dissection occurs from rapid deceleration and stretching of the neck, or a crush injury to the artery, either directly, or by stretch against surrounding structures, such as the vertebral bodies.

Innominate artery and left subclavian artery dissections are rare. There are only a few case reports that describe this disease.[31] It can be spontaneous, traumatic, or iatrogenic. Infectious or atherosclerotic degeneration can play a role in some cases. Similar to carotid artery dissection, connective tissue diseases such as Ehlers-Danlos syndrome are likely involved in the spontaneous dissection of the innominate artery.[32]

Isolated common carotid dissection is also a very rare condition and shares most ICA dissection presentations although, for anatomical reasons, most cranial nerve palsies are usually spared. A recent literature review reported 46 cases, including 20 spontaneous, 11 traumatic, 4 iatrogenic and 12 that were associated with aortic arch dissection, with positive outcomes overall.[33] Furthermore, innominate artery, common carotid artery, and subclavian artery dissection can occur as an extension of aortic arch dissection in antegrade or retrograde fashion.

Iatrogenic dissections of the aortic arch branches result from direct intimal injury during endovascular device manipulation (eg, wire, catheter, embolic protection device), and can be caused by excessive pressure within the vessel lumen (eg, balloon inflation of stenotic vessel), or even retrograde dissection during endovascular treatment of the aortic arch, or descending thoracic aorta (eg, endograft placement). There are no clear data reporting the frequency of this type of injury, but with advances in endovascular treatment options, one can only expect an increase in the incidences of iatrogenic dissections. In contrast to other types of dissections, most of the iatrogenic types occur during treatment of

atherosclerotic, calcified lesions. The hard, intimal plaque ruptures creating a dissection plane that can extend. The carotid bifurcation, the origin of the common carotid, innominate, and subclavian arteries, are all important locations of atherosclerotic plaques, which are prone to injury.

Clinical Presentation

Most patients with spontaneous ICA dissections present with at least two symptoms.[20] The typical patient will present with neck pain on one side and oculosympathetic palsy (miosis and ptosis with no facial anhidrosis) due to interruption of the sympathetic plexus around the ICA. These symptoms can be followed by cerebral or retinal ischemia hours or days later.

Unilateral facial or orbital pain is present in half of the patients and a gradual unilateral headache, usually frontotemporal, is present in two thirds of patients.[34] This pain usually constitutes an initial symptom. Cerebral and retinal ischemias develop in 50% to 95% of patients,[35,36] with TIA episodes commonly preceding stroke. Retinal infarction and permanent blindness are rare.[37] Subsequent cranial nerve palsies can develop in 12% of patients, affecting in particular the hypoglossal nerve that overlays the ICA. Other symptoms and signs such as pulsatile tinnitus or a carotid bruit may also be present.

Spontaneous vertebral artery dissection can be present, with pain manifesting in the back of the neck. Ischemic symptoms involving the posterior circulation occur in at least 90% of diagnosed patients.

Common carotid dissection can present, with symptoms similar to ICA dissections including head and neck pain, hemispheric ischemic and monocular visual deficit.

Patients with innominate artery dissection may report a syncopal episode due to cerebrovascular compromise or simple shoulder pain. Other symptoms, such as hoarseness or cough due to recurrent laryngeal nerve injury or compression and tracheal compression may be present.

Patients with traumatic or iatrogenic dissections can be completely asymptomatic or present with the same clinical symptoms as those with spontaneous dissections.

Diagnosis

There seems to be no single optimal diagnostic modality for aortic arch branch dissection. DUS is noninvasive and safe, but it is operator-dependent. The combination of hemodynamic abnormalities and direct B-mode anatomical inspection of the arteries has >90% sensitivity with respect to detection of carotid dissections.[38] Because of the difficulty visualizing the vessel, DUS is not as reliable when dissection involves the very distal portion of the ICA, the vertebral arteries, or innominate and subclavian arteries.

CTA has 92% to 100% sensitivity with regard to diagnosis of dissections. It has supplanted the more invasive diagnostic angiography. Furthermore, it offers the advantage of being a commonly available, fast imaging modality and provides an accurate anatomical image of the arterial wall and lumen. It will reveal pseudoaneurysms or aneurysms, the presence of flap or thrombus in the vessel lumen, as well as the extension of the dissection. It can also evaluate deeper structures up to the brain, including the aortic arch and branches, as well as challenging structures such as the vertebral arteries and very distal ICA. The most reliable finding in dissection diagnosis is the "target lesion," with an annular contrast filling pattern. Most trauma patients will undergo CTA as part of their trauma workup. The screening of CTA protocols has increased the diagnostic rate of traumatic cerebrovascular dissections.

Magnetic resonance imaging/magnetic resonance angiography has the advantage of revealing ischemic complications of dissections occurring in the brain, as well as exposing subtle mural hematomas. However, it overestimates arterial lumen narrowing and cannot differentiate intramural hematoma from intraluminal thrombus.

Angiography is a valuable tool that allows assessment of the arterial lumen as well as exposure of the overall aspect of the involved artery and associated lesions. On the other hand, it has the potential risk of complications ranging from access hematoma to stroke. As such, angiography can be used as a complementary diagnostic modality in equivocal cases, or when endovascular treatment is planned.

Treatment

The goal is to find the best approach to limit ischemic damage and its progression, as well as prevent early and late recurrence. There is no single best treatment approach because there is no published randomized data comparing early anticoagulation to single or dual antiplatelet therapy. The role of thrombolysis and early endovascular intervention is even less clear. The treatment approach for spontaneous and traumatic dissections should be tailored to individual cases, but at the same time it should follow similar principles.

In iatrogenic dissections, lesions can be immediately treated by endovascular means, either by prolonged balloon inflation or stent placement. It is important to preserve wire access through the true lumen of the artery until a satisfactory result is achieved.

The following discussion will focus on cerebrovascular arterial dissections:

Several large, nonrandomized reports and meta-analyses have failed to demonstrate any difference in clinical outcomes, intracranial bleeds, or mortality rates between early medical treatments: anticoagulation versus antiplatelet therapy.[39-41] Consequently, the approach should be tailored and anticoagulation should be considered in at least some cases, including those involving symptomatic patients who are already on aspirin.[42] Additionally, patients with recurrent ischemic events, multiple embolic infarction, free-floating thrombus, or occluded dissection, can probably benefit from anticoagulation in order to prevent thrombotic extension.

Anticoagulation can sometimes be contraindicated in traumatic dissections, due to concomitant injuries or the presence of SAH. However, it is apparently safe in intracranial

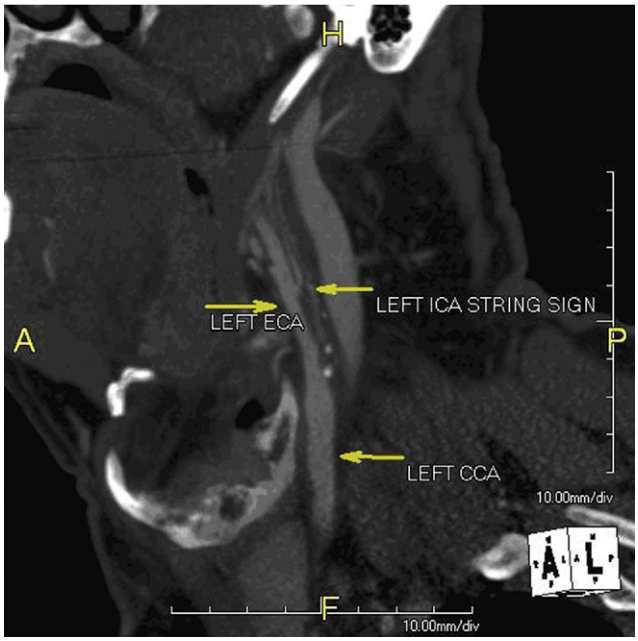

Figure 2 Computed tomographic angiogram of partially recanalized ICA after spontaneous fibromuscular dysplasia dissection (courtesy of Pegge Halandras, MD and Karthikeshwar Kasirajan, MD). CCA, common carotid artery; ECA, external carotid artery; ICA, internal carotid artery.

arterial dissections if there is no associated intracranial aneurysm or SAH.[43]

The natural course of cerebrovascular arterial dissection favors recanalization. Most recanalizations commonly occur within the first few months, but can appear after a year (Fig 2). Several studies report ICA recanalization in the range of 60% to 70% within 6 months, with corresponding antiplatelet or anticoagulation therapy. Baracchini et al[44] used DUS to closely monitor patients after spontaneous dissection of their ICA and vertebral arteries. DUS was obtained daily while the patient was in hospital, then monthly for 6 months and after this time, every 6 months. They reported 55% ICA and 46% vertebral artery recanalization—all but one within 9 months. There was an equal rate of recanalization for stenotic or occlusive lesions. They suggested medical treatment (antiplatelet or anticoagulation) until complete recanalization or, alternatively, a change to antiplatelet therapy after 6 months in cases where there is residual stenosis or occlusion.

One can draw a treatment strategy from these results. If anticoagulation is started initially, a DUS at 3 months and 6 months should be obtained. Anticoagulation then can be stopped if there is no recanalization at the 6-month mark. If the artery is recanalized, a switch to antiplatelet therapy until there is complete recanalization. Due to the lack of data, it is unclear whether interventions play a role in cases involving asymptomatic patients with persistent severe (>80%) stenosis at the dissection site.

One fourth of patients with spontaneous dissection will have an early dissection recurrence, which is much more frequent in the first 2 months, with most occurring within 1 week. Interestingly, this can also occur in unaffected arteries, suggesting the presence of acute and transient arterial state of disorder.[44,45] A minority of the patients (2% to 3%) will have a late recurrence at the site of original dissection, likely due to underlying arterial wall weakness. Recurrence is more frequent in patients with known underlying connective tissue disorder, or a familial history of arterial dissection.[21,45]

The role of systemic and focal thrombolysis, or focal percutaneous mechanical thrombectomy is also not clear. There is no large study that addresses these issues. Thrombolytic therapy can potentially worsen the intramural hematoma, which can result in dissection extension, or formation of dissecting aneurysms. Systemic thrombolytic for the treatment of concomitant stroke seems to be safe although the recovery of carotid artery dissection patients is not as good as in other stroke patients.[46] Local thrombolysis or percutaneous mechanical thrombectomy can potentially be performed before endovascular treatment of dissection.

Asymptomatic, extracranial dissecting aneurysms seem to have a benign course and can be left alone.[47,48] Due to the potential devastating sequel of SAH, it is prudent to treat intracranial dissecting aneurysms. Endovascular treatment of intra- and extracranial aneurysms include coil embolization and/or stent placement that initiate spontaneous thrombosis of the aneurysm. Alternatively, covered stents can offer another treatment option. Assadian et al reported six patients treated with covered stents, including two with dissecting aneurysms. Mean follow-up was 38.3 months and no stenosis, occlusion, or persistent neurological deficit was ultimately reported.[49]

The most common current indication of endovascular therapy is the failure of medical therapy. It becomes an option when patients have recurrent or progressive ischemic symptoms, despite antiplatelet and anticoagulation therapy, or have a contraindication to anticoagulation and fail antiplatelet therapy. A recent report from Pham et al encompassed a systemic review of 31 case series including 140 patients with extracranial ICA and/or vertebral artery dissection.[50] The study included 61 patients with their dissecting aneurysms treated by endovascular means. Although it can be challenging to remain intraluminal at the dissection level, the overall reported technical success of stent placement was >99%. Reported asymptomatic periprocedural complication rates were 1.3% and transient symptoms manifested at 1.4%, with no permanent neurological events. Although the study is based on nonrandomized case series, it suggests that stent placement is feasible and safe in cases involving cerebrovascular dissections.

Silvestrini et al recently reported the outcomes of 66 cases involving stroke patients with ICA occlusion secondary to spontaneous dissection.[51] Transcranial Doppler was obtained within 24 hours of the stroke in each of the patients, focusing on the three major intracranial collaterals (ie, ophthalmic artery, anterior, and posterior communicating arteries). Subsequent progressive recanalization of the occluded arteries was also examined. Recovery was not associated with the presence of recanalization, severity of the stroke at presentation, or vascular risk factors. In fact, early activation and adequacy of collateral vessels was the most important prognostic factor for recovery. Significant disability was expected if none or only one collateral

vessel was compensating. Early assessment of these collaterals could help with predicting clinical outcomes, as well as provide guidance for a proactive and more aggressive approach through application of endovascular recanalization. The importance of collateral pathways has been established in cases of atherothrombotic carotid occlusion.[52] However, with respect to dissection occlusion, the collaterals have to adapt acutely to the given hemodynamic change, therefore, an early intervention aiming at recanalization may be crucial. Further studies are necessary to investigate this approach.

There are many other controversial areas to examine, including the role of proximal embolic protection devices during endovascular treatment of dissections, the role of dual antiplatelet therapy, and the best antithrombotic regimen after stent placement. Large randomized trials are necessary to investigate these areas.

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