State-of-Art Surgical Treatment of Dissecting Anterior Circulation Intracranial Aneurysms

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Abstract

Intracranial dissecting aneurysms (IDAs) are an important cause of subarachnoid hemorrhage, stroke, or compression of intracranial structures. Since the availability of endovascular treatment and the advantage of intraprocedural anticoagulation, an endovascular strategy has become the mainstay of their therapy. But in some cases selective aneurysm obliteration by the endovascular approach is impossible or associated with an unacceptable risk of morbidity. This is particularly true when the IDA is a blood blister-like aneurysm or when dissection affects peripheral branches of the internal carotid artery. The literature dealing with surgical treatment of IDAs in the anterior circulation is heterogeneous, and formulation of general recommendations concerning the surgical strategy remains difficult. The aim of this study was to conduct a systematic review of the current knowledge on incidence, pathogenesis, clinical presentation, and diagnostic procedures with a special emphasis on the surgical treatment of intracranial dissections of anterior circulation.

Keywords
- intracranial dissecting aneurysm
- anterior circulation
- surgical treatment

Introduction

Intracranial dissecting aneurysms (IDAs) represent a well-defined clinical entity with an increasing occurrence in recent years.[1–13] Even though they continue to be challenging with a high morbidity and mortality,[4] their pathogenesis and appropriate management[14] remain unclear and controversial. Earlier studies on this topic reported on poor outcomes.[4,15] However, recent studies have reported better results.[4,16] Due to the availability of endovascular treatment, this therapy has become the method of choice for most patients.[17] But the literature regarding surgical treatment of these lesions is heterogeneous, and general recommendations are difficult to formulate. This review summarizes the current knowledge on the pathology with a special emphasis on the surgical treatment of anterior circulation IDAs.

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Search Strategy

We searched the database in PubMed for English- and Japanese-language articles using text words or terms including "intracranial," "dissecting," "dissection," "aneurysms," "anterior," "circulation," "internal carotid artery," "middle cerebral artery," "anterior cerebral artery," "anterior communicating artery," "anterior temporal artery," "surgical," "treatment," "therapy," "methods," "risk factors," "pathophysiology," "diagnosis," and "prognosis." The words/terms were used in both "AND" and "OR" combinations. We included studies with radiologic, intraoperative, or pathologic confirmation of dissection. Studies that did not provide sufficient detail regarding diagnostic criteria for dissection were excluded. The search was not designed to identify studies reporting on conservative or endovascular treatment, and it was restricted by date of publication, patient age, or number of subjects. We also performed a manual search of reference lists from eligible articles but did not seek to identify research abstracts from meeting proceedings or unpublished studies. The search was updated through November 2015.

Incidence

Even though IDAs are still considered relatively rare, they represent an important cause of subarachnoid hemorrhage (SAH), brain infarction, or compression of intracranial structures.[17,18] IDAs constitute $<$ 10% of all spontaneous cervicocephalic dissections in adults[19] and $\sim$ 2 to 3% of all cerebral aneurysms.[20–22] They are typically detected in young people (mean age range: 20–50 years)[18,23–25] and most frequently occur in East Asian populations.[26] In the pediatric population, cervicocephalic dissections account for 10% of strokes and are associated with a poor outcome and more commonly with embolic rather than hemodynamic strokes.[1,27] The most frequent localization of the IDAs is the vertebral artery[28,29] followed by the internal carotid artery (ICA) and middle cerebral artery (MCA).[30–32] The reported incidence of spontaneous multivessel dissections ranges from 10 to 15%.[33,34] Blood blister-like aneurysms (BBAs), also known as ICA trunk aneurysms, which are suspected to originate from a dissection, represent $\sim$ 0.9 to 6.5% of all ICA aneurysms,[35–37] 1% of all intracranial aneurysms,[38] and 0.5 to 2% of all ruptured aneurysms.[35,39–42] MCA dissections, usually located in the M1 segment,[43] are relatively rare; only 74 cases of MCA IDAs have been reported since 1990.[1–3,5–10,12,13,16,44–61] Patients presenting with ischemia have dissection more often in M1, whereas pure bleeding is associated with the dissection usually located more distally, in either the M2 or M3 segment.[51,62] Whereas cerebral ischemia as a presenting feature was reported in 73% of patients with anterior circulation dissections,[18] the incidence of SAH due to dissection of ICA and MCA varies between 20% and 65% and 12% and 75%, respectively.[19,25,62–65] However, in cases of BBA, the SAH is observed in up to 99% of cases.[66] In patients with dissection of the anterior cerebral artery (ACA), SAH is observed in 50% of cases,[67] and only 30 such cases were published from 1967 to 2006.[68] Finally, the true anterior temporal artery (ATA) IDA was reported only twice.[69,70]

Pathology and Pathophysiology

A dissecting aneurysm is produced by blood entering into the arterial wall due to its splitting or dissecting with subsequent extension of the effused blood for a varying distance between the wall layers.[71] The dissection may come from the tear in three scenarios: (1) in the media due to rupture of the vasa vasorum[3,72] or new vessels formed in response to medial necrosis,[72] (2) at the intimal surface, or (3) the main dissection plane may lie between the media and the adventitia, causing an outpouching of the arterial wall.[30] Owing to the lack of an external elastic lamina and thin muscular and adventitial layer,[73] intracranial arteries are prone to subadventitial dissection. Compression of the lumen followed by encroachment on the blood flow stream and the release of endothelins[30,74] may lead to formation of an intraluminal thrombus[30] with subsequent hypoperfusion and infarction.[75,76] The intramural hematoma creating a false lumen may become a source of embolus as a consequence of intramural clot formation[14,30] and can occlude small perforators.[14,72,74] Aneurysms without aperture at the distal end of the false lumen are more unstable and more prone to rupture.[68] The dynamic nature of dissection is confirmed by observations of increased narrowing due to intramural hematoma, the formation of an aneurysmal bulge in the acute to subacute stage, and the healing process in the chronic stage.[77] Compared with ordinary berry aneurysms that are formed by thickened intima and/or adventitia, with rich collagen and inflammatory cells,[35] the wall of BBAs in which the dissection of the ICA has been proposed as a causative factor[36,38,78] is composed of only normal adventitia and lacks the usual collagenous layer.[79]

These differences are of great importance for surgical treatment.[80] In terms of arteriosclerosis, there is no consensus about its role in the development of IDAs. A few observations suggest two different features of dissection. The first one is the widespread disruption of the entire vessel wall with thin nonatheromatous intima. This indicates that the nonatheromatous thick intima might function as a protective factor in arterial wall disruption. In the second type, the atherosclerosis may predispose to an intra-atheromatous plaque hemorrhage originating from newly formed vessels.[81] According to other studies, a focal wall defect associated with arteriosclerosis-induced ulceration and penetration of the internal elastic lamina may play an important role in the development of BBAs.[35,39,79] Finally the vessel wall edema that develops during a migraine attack is suspected of playing a role in the increased vulnerability of the involved artery to tearing forces.[30,31,82]

Molecular and Genetic Factors

Congenital and acquired abnormalities of the arterial media and elastic tissue such as Ehlers-Danlos syndrome (EDS) or fibromuscular dysplasia are seldom found in patients with dissecting aneurysms.[83] However, ultrastructural abnormalities resembling the aberrations found in EDS type II or III in dermal connective tissue have been found in patients with

proven nontraumatic cervical arterial dissections.[84] A COL3A1 missense mutation leading to a G157S substitution in type III procollagen was detected in one family. The G157S substitution in exon 14 was found in both patients as well as in their mothers who were sisters. The finding suggests that the carriers had genuine vascular EDS. However, neither clinical signs of connective tissue weakness of the patients nor the histology and electron microscopy of their skin biopsies revealed typical signs of vascular EDS.[85] In addition, it was reported that a deficiency of α-1 antitrypsin or one of the other protease inhibitors could result in degradation of the arterial wall through an imbalance between proteolytic enzymes and their inhibitors, thereby predisposing the arterial wall to dissection or aneurysm formation.[86]

Risk Factors

Risk factors for IDAs include hypertension, oral contraceptives, cigarette smoking, diabetes mellitus, head or neck trauma, syphilis, polycystic kidney disease, polyarteritis nodosa, systemic lupus erythematosus, or Moyamoya disease.[18,20,22,62,65,67,68,72,74,87–91]

Classifications of Dissecting Aneurysms

Based on the condition of the intimal and internal elastic lamina, dissecting lesions may be arranged in four types.[92] Type 1 poses acute widespread disruption of the internal elastic lamina without intimal thickening. Type 2 presents with intimal thickening and stretched and/or fragmented internal elastic lamina. Type 3 is characterized by fragmented internal elastic lamina, multiple dissections of the thickened intima, and an organized thrombus in the lumen. Finally, type 4 shows minimal disruption of the internal elastic lamina without intimal thickening.[92] Even though BBAs may belong to type 4 dissections,[93] the pathophysiologic overlap of IDAs with BBAs is still controversial, and some authors state that the BBAs are caused by a degeneration of the internal elastic lamina and media without associated arterial dissection (no mural hematoma or double lumen on pathologic examination).[79] Mizutani et al[94] suggest that the risk of bleeding is related to the extent of disruption of the media and to an entry-only type of dissection.

Anterior Cerebral Artery

Dissecting aneurysms involving the ACA may be classified into three types.[95] Extension of the dissection to the ACA from the ICA represents type I. Type II includes dissection at the A1 segment, and type III involves the dissection from the A2 to A4 region.[95] Most type I dissections seem to occur in young adults (mean age: 24 years), and in 90% the presenting factor is cerebral infarction with the same mortality rate.[95] Type II most frequently occurs in young women and often causes SAH.[95] Type III predominates in middle age and mostly causes infarctions.[67,95]

Middle Cerebral Artery

The MCA IDAs originating from the M1 segment represent type A, and the main difficulty in their treatment is preserving the lenticulostriate perforators that are often incorporated into the aneurysm base or dome. In type B the aneurysm originates from the M2 segment or MCA bifurcation. The dissection originating from the M3 or distal segments belongs to type C.[12]

Clinical Presentation

Based on the pathophysiology, site of the aneurysm, and the plane of the cleavage, the patients with intracranial dissections present with either SAH or brain infarction. Ischemia has been proposed as the predominant symptom in anterior circulation, even though some studies have observed a comparable occurrence of ischemia and SAH in the case of ACA IDAs.[67] Infarcts are usually found within the MCA or ACA territory.[63,65,96,97] The closer the occlusion to the brain, the more likely the infarction will develop.[30] A severe dull headache[95] localized in the forehead and temple or pain in the ipsilateral eye are common findings followed by contralateral hemiparesis,[31] ipsilateral paresis, aphasia, or choreoathetotic movements.[95]

Diagnostic Procedures

The common digital subtraction angiography (DSA) findings in patients with subintimal dissection are a string sign characterized by a long narrow column of contrast material, a flame-shaped tapering of the lumen, and occlusion of the artery. In patients with subadventitial dissection, aneurysmal dilatation is preceded or followed by a focal narrowing of the vessel lumen ("pearl and string sign").[30,98] The double-lumen sign is considered the most indicative of IDA.[11,74,98,99] Although the conventional catheter angiography cannot visualize in situ pathology of the arterial wall such as intramural hematoma,[98,100] magnetic resonance imaging (MRI)/MRI angiography (MRA) or computed tomography (CT)/CT angiography (CTA) enables visualization of both intramural and luminal pathology.[72] MRI includes routine T1-weighted imaging (T1WI), T2-weighted imaging (T2WI) usually with spatial presaturation, either with or without fat suppression, and MRA with or without gadolinium enhancement. In particular, time-of-flight (TOF) MRA and T1WI can provide direct visualization of subacute intramural hematoma as a high-signal structure due to its short T1WI feature.[101–108] Because intraluminal signal depends on flow velocity and the presence of laminar flow and/or turbulence, a differentiation between intraluminal flow and intramural hematoma is often difficult solely on the basis of TOF MRA.[106,109,110]

Visualization grading scores of the intramural hematomas on three-dimensional (3D) black blood T1WI (3D-BB-T1WI) were highest among the different sequences, and they were significantly higher than those of MRA.[111] Unlike spin-echo T1WI, 3D-BB-T1WI has a higher craniocaudal spatial resolution.[111] A limitation of CTA, MRA, and DSA is that they cannot differentiate between vascular pathologies that exhibit similar luminal defects. The lumen of the affected artery occasionally appears normal in caliber on CTA, despite the vessel wall hematoma.[112] In addition, intramural hematoma in the

acute stage is rarely detectable on T1WI because isointense hematomas are often obscured by surrounding tissue.[113] Conventional MRI was able to detect the intramural hematomas in only 32 to 34% of intracranial vertebrobasilar dissections.[98,114] Recently advanced high-resolution MRI provided detailed information on hidden structures such as intimal flap, inlet of the false lumen, size of the intramural hematoma, ostia of small branching vessels, and helped clearly differentiate between intramural hematoma and intraluminal thrombus.[115–119]

Susceptibility-weighted imaging (SWI) and a phase map is highly sensitive to both paramagnetic compounds such as deoxyhemoglobin, ferritin, and hemosiderin and to diamagnetic compounds (bone minerals and dystrophic calcifications).[120,121] It is able to detect intramural hematoma in 90% of patients.[113] The detection rate is comparable with the studies that used 3D spoiled gradient-recalled acquisition images with contrast medium or 3D-BB-T1WI.[98,111,114] Dissecting aneurysms in the posterior circulation are prone to image distortion on T2-weighted gradient-echo images due to bony structures and air in the mastoid bones.[113] Compared with a T2-weighted gradient-echo image, SWI provides images with higher resolution, flow compensation, less image distortion in the detection of microbleeds, and can differentiate hemorrhage from calcification.[122–124]

BBAs usually present various typical angiographic findings (e.g., tiny hemispheric bleb, broad-based bulge, irregular protrusion of the anterior wall of the ICA, and rapid preoperative growth of the dome or regrowth).[93] Angiographic evidence of a saccular shape does not always correlate with the nature of the aneurysm wall; therefore, intraoperative observation is required for the final diagnosis of BBA.[35] Interestingly, CTA seems to be a more sensitive diagnostic method for BBAs of the anterior communicating artery (AComA) than DSA.[80]

A transcranial Doppler ultrasound shows diminished intracranial velocities in the ICA siphon and the MCA in case of dissecting aneurysms. Duplex ultrasound findings include narrowed or dilated vascular segments.[125–127]

Intraoperative Diagnosis

The characteristic sign confirming preoperative diagnosis of dissection is a reddish discoloration of the affected artery around aneurysmal dilation owning to hematoma in the vessel wall observed at surgery within the acute stage.[11,63,64,87,128] Approximately 1 month after ictus, the aneurysm becomes whitish gray with a shiny, smooth surface, firm, and not compressible due to an organized clot. Occasionally the vascularization of the outer wall is visible.[11] Because the term *fusiform aneurysms* may include both types of lesions, that is, acute dissecting[6] and chronic fusiform aneurysms[129] including giant so-called dolichoectatic aneurysms,[130] a definitive diagnosis should be made on the basis of pathologic dissection or angiographic and intraoperative findings. A certain subset of fusiform aneurysms may be caused by dissection of the aneurysmal wall,[131,132] but progressive dilatation of vessels due to a defect of the internal elastic lamina may be an important factor in the pathogenesis of these lesions as well.[133] Therefore giant fusiform aneurysms should probably be regarded as distinct entities.[66] A fusiform or irregular aneurysmal dilation located at a nonbranching site of an artery is very suggestive of IDAs if associated with a segmental stenosis.[134] However, additional radiologic features are needed to confirm the diagnosis of IDA including a rapid change in morphology.[135] Yamaura reported that while 81% vertebral artery dissecting aneurysms bled and 24% of these rebleed, no such observation was made in terms of atherosclerotic fusiform aneurysms.[136] In conclusion, the final diagnosis of IDAs can only be made by pathologic diagnosis.[128]

Nonsurgical Treatment

Anticoagulants/Antithrombotics

A presumed mechanism of cerebral ischemia is thromboembolic in origin. Thus the treatment strategy should focus on the reduction of the risk of ischemic recurrence by the administration of anticoagulants or antiplatelet agents.[23,25,137] A risk reduction of recurrent ischemic stroke in patients receiving anticoagulants is comparable with those treated with antiplatelet agents.[137] Even though the use of anticoagulants in the acute stage may bear some risk of progression of the dissection, expansion of the intramural hematoma with subsequent progression of stenosis or intracranial hemorrhagic transformation,[18,138] no such complications or progression of the dissection have been observed so far.[18,23,137,139] Thrombolytic agents might be effective in patients with intracranial red clot embolization appearing soon after the onset of neurologic symptoms who do not already have large brain infarcts.[30,140] However, their effects are limited due to the narrow time window and inadequate recanalization rate for large arterial occlusions.[1] In addition, their application remains controversial due to the potential expansion of intramural hematoma or hemorrhagic complications.[141,142]

Endovascular Treatment

In case of ischemic presentation, stenting is rarely indicated because without occlusions the arterial lumen usually opens of its own accord over time, and anticoagulants effectively prevent further formation of thrombi and subsequent embolization. Stenting might be useful in the very rare situations when a patient has tight luminal narrowing and continual brain ischemia despite anticoagulant treatment.[30,143] The choice of therapeutic strategy in pediatric patients may also consist of selective thrombus aspiration.[1] In unstable ruptured IDAs with a tendency to rebleed, the endovascular approach is the therapeutic measure of first choice.[17] The deconstructive technique represents proximal occlusion of the parent artery and internal coil trapping of the dissected segment of the vessel. Reconstructive techniques preserve the parent vessel and consist of stent placement including double-stent placement, flow-diverting stents, and stent-assisted coil embolization.[10,17,66]

BBAs are difficult to treat with coil embolization, owing to their small size, wide necks, and location, making the coil

placement into the cavity technically difficult.[40] A balloon-assisted technique and stent-assisted coil embolization may help overcome the problem.[41,93,144–146] Endovascular ICA trapping is an optimal treatment for patients tolerating the ICA obstruction; however, this method is unsuitable when the posterior communicating artery (PComA) or anterior choroidal artery (AChoA) originate in close vicinity to the lesion.[41] Finally, an overlap stenting or flow-diverter stenting can preserve the flow of a parent vessel and may overcome the drawbacks of saccular embolization and trapping.[144,146,147] A recent study demonstrated that a recurrent risk in patients treated by preserving blood flow through the parent vessel was comparable with those treated with a deconstructive technique.[24]

Surgical Treatment

Surgical treatment includes clipping, trapping, proximal ligation, reinforcement, aneurysmectomy/aneurysmotomy or revascularization depending on localization and clinical representation of dissecting aneurysms.

Internal Carotid Artery

The treatment of patients with ICA IDAs is controversial.[31] Anterior circulation dissections most often involve the ICA in its supraclinoid portion, and the tear often extends to the carotid bifurcation, sometimes even to the ACA and MCA.[30] Due to the architecture of dissecting aneurysms, direct microsurgical clipping is usually not feasible.[73] With the availability of endovascular methods and the advantage of intraprocedural anticoagulation, endovascular strategy has become the primary treatment for most patients. The endovascular treatment is performed under general anesthesia, and balloon occlusion tests with angiographic analyses are performed when necessary. The evaluation of collateral circulation is usually based on meticulous arterial anatomical analyses, reconstitution of the distal arterial territory, and venous delay analysis ($<$ 2-second delay) conducted under general anesthesia. A patient is considered for trapping of the dissecting aneurysm if the lesion does not incorporate an important branch. If the dissecting segment incorporates an important branch or represents the sole supply to the territory, stent alone or stent-assisted embolization should be considered.[73]

However, the BBAs are small hemispheric-shaped lesions, bulging from unbranched site of the ICA and are suspected to originate from a dissection.[35–37,40,42,78,148–151] Patients typically present with a severe SAH.[35] The critical points are (1) diagnostic problems due to their small size, (2) the extremely fragile wall of BBAs with a high probability of intraoperative rupture due to abrupt histologic transition from the sclerotic ICA wall to the fragile aneurysm neck,[35,42,152,153] (3) the presence of important branches (ophthalmic artery, AChoA and PComA),[66] and (4) a high probability of losing parent vessel patency, either intentionally or as a consequence of the treatment.[35,40,152]

Several surgical approaches have been proposed to treat this condition, such as clipping[40,42] on wrapping material (cellulose fabric)[35,42] enveloping the entire circumference of the ICA or parallel clipping of the ICA (including the normal arterial wall beyond the lesion under decreased pressure produced by temporary trapping of the ICA).[35,42] The latter choice may occasionally create a residual arterial stenosis resulting in unfavorable outcomes.[35] Wrapping as another technique does not prevent rebleeding and is associated with a high incidence of postoperative SAH and death.[35] However, clipping on wrapping material was recently observed to be an effective treatment strategy with good outcomes without surgical complications.[35,42] Even though surgical clipping is the most common technique, 30 to 50% of cases involve perioperative complications including intraoperative rupture of the aneurysm.[40,42,66] A second treatment modality is necessary to secure or exclude the BBA in 21% of patients.[66] The frequent intraoperative ruptures are thought to directly reflect fragility of the aneurysm walls, which are covered with adventitia or clot alone.[79] Clot removal from the aneurysm dome during dissection of its adhesions to surrounding tissues may further increase the risk of intraoperative bleeding.[35]

To diminish the risk, careful subpial dissection of the BBA is recommended.[42,154] The profuse intraoperative bleeding from a large visible defect/tear in the vessel wall invariably led to ICA trap ligation if applying an encircling Sundt clip (Codman, Raynham, Maryland, United States),[155] wrapping[42] or suturing[40] of the thin vessel walls surrounding the tears is unsuccessful.[40] The ICA trapping requires sufficient cross-flow through the AComA,[35,42] preservation of PComAs and the AChoAs.[42,93] However, the sacrifice of the ICA within 48 hours following an SAH may lead to a poor outcome even in patients with adequate collateral capacity revealed by preoperative DSA (good cross-filling of the MCA and ACA, good-sized AComA, and good-sized PComAs), probably because of vasospasm-induced compromise of the cerebral collaterals.[40,93] Meling et al[40] pointed out that because the incidence of cerebral vasospasm after ICA closure is far greater in an acute than in an elective treatment situation, the results of tests to evaluate tolerance to elective ICA closure preoperatively are invalid in situations of acute SAH. The patients in whom the ICA is preserved usually have a benign clinical course.[40]

These data suggest that if acute ICA sacrifice is performed prior to or within the very first days of the vasospasm phase following a major SAH, it should be accompanied by concomitant construction of a high-flow bypass.[40,150,156–162] Satoh et al[150] warn that any attempt for simple clipping at the acute stage should be avoided without sufficient preparation including securing the cervical ICA before opening the dura, if the facing ICA dorsal aneurysm shows any clinical or angiographic signs different from ordinary aneurysms. Postoperative outcome of early surgery within 96 hours after the onset of SAH is significantly less favorable than that of the delayed procedure.[163] At the chronic stage, healing processes make the fragile tissue much firmer, thus making clipping possible in most cases.[150] The BBAs often regrow and rerupture even after seemingly successful clip application.[36,39,42,63,64,164–168] The postoperative rupture of the aneurysm may occur as a result of clip torsion or slippage and elevated blood pressure. Confirming the stability of clips with induced blood pressure elevation

and repeated irrigation may prevent the complication.[42] The most used rescue surgical techniques are trapping without bypass and arterial suturing. The rest constitutes a second clipping attempt, coiling, coiling with stenting, endovascular or surgical trapping with or without bypass, or just bypass.[66]

Middle Cerebral Artery

Patients with MCA IDAs presenting with SAH have better outcomes if they are treated surgically.[62] Even though wrapping the M2 IDA presenting with SAH with the help of Teflon mesh may decrease the bleeding risk,[62] it is not considered an effective treatment method for dissecting aneurysms.[12] Furthermore, it is still unclear if revascularization surgery can be indicated for patients with IDA without any evidence of ischemia. Revascularization may minimize the risk of cerebral infarction after trapping, eliminate the risk of rebleeding by exclusion of damaged vessel from circulation, and reduce potential progression of the dissection.[62] Because the IDAs cannot be treated with direct aneurysm neck clipping or dome coiling, tailored surgical treatment is required for each individual case. A preoperative intracarotid amobarbital test (Wada test),[7] direct intraoperative flow measurement with an ultrasonic flow probe,[169] and electrophysiologic assessment plays an important role in predicting the possibility of surgery-related cerebral ischemia.[12]

The dissecting M1 aneurysm with severe calcification of the aneurysmal wall (which prevents direct aneurysm clipping) without the lenticulostriate perforators originating from the part of the artery with the aneurysm may be trapped after a superficial temporal artery (STA)-M3 bypass was successfully performed.[12] Provided that the trapping of the aneurysm originating from the M1 segment would be unavoidable to sacrifice the lenticulostriate perforators, STA-radial artery (RA)-M3 bypass followed by a staged cervical ICA occlusion (placing a balloon or staged clipping of the cervical ICA endovascularly with a Silverstone clamp) should be done.[12] Even though an alternative technique such as aneurysm trapping or resection combined with an M1-M3 excimer laser-assisted nonocclusive anastomosis (ELANA) technique flow replacement bypass have been reported too, its surgical-related ischemic complication rate was higher.[170] Although thrombosis initiated by a STA-RA-M3 bypass and staged ICA occlusion can also potentially occlude the lenticulostriate perforators, their delayed occlusion allows the involved neurons to tolerate the ischemia or promotes the construction of potential collateral circulation.[12] The utilization of a STA-RA-M3 bypass has some advantages such as a shorter interpositional graft, avoiding an additional cervical skin incision, and high-flow carrying capacity.[12]

The treatment strategy for a type B dissecting MCA aneurysm includes performance of a STA-M3 bypass,[3,12] STA-RA-M3, intracranial M3-M3 side-to-side or a M2-M2 in situ anastomosis bypass, confirming the graft patency, temporary occlusion of the proximal and distal M2 segment with intraoperative motor evoked potentials (MEPs) and somatosensory evoked potentials monitoring, followed by aneurysmal trapping and resection.[12] In the long term, leptomeningeal collateral vessels of the anterior and posterior cerebral arteries may take over the function of the bypass.[3] Interestingly, a spontaneously occluded parent artery with obliteration of the aneurysmal lesion on M2 with subsequent recurrence as an aneurysmal dilatation in the same segment, may be successfully resected without revascularization, especially if an intracarotid amobarbital test demonstrates ischemic tolerance to occlusion of the parent vessel.[7]

The MCA dissecting aneurysm originating from the M3 segment may be treated by proximal parent artery temporary clipping under MEP monitoring. If no MEP changes are detected during the 30-minute monitoring period, simple clipping of the proximal M3 segment is a definitive solution. Provided that the aneurysm originates from the central arteries or angular arteries, low-flow bypasses, such as an STA-M3 bypass or M3-M3 in situ anastomosis, are required before aneurysm trapping or resection.[12] Avoiding occlusion of the bypass graft, perforators, and reduction of anastomosis-related ischemia requires meticulous intraoperative aneurysm dissection, perioperative administration of antiplatelet therapy and calcium channel blocker agents, mild hypothermia or the use of mannitol, dexamethasone, barbiturate, or propofol during the temporary occlusion of the parent artery.[12]

Anterior Cerebral Artery

Trapping of a ruptured A1 (type II) IDA is indicated when collateral flow through the AComA is sufficient.[95] However, due to subsequent cerebral vasospasm, the trapping may be followed by an unfavorable clinical course.[67] In contrast, clipping[67] and wrapping of the aneurysmal bulge in the A1[68] may result in postoperative aneurysmal growth and/or rebleeding with an unfavorable prognosis. In case of the A2 dissected artery, a wrapping procedure may achieve stabilization of the dissection and a favorable outcome.[171] One patient with a type I ruptured dissection survived with mild hemiparesis and aphasia after surgical clamping of the affected ICA.[172] Occlusion at the earlier stage may prevent further extension of the dissection and possibly preserve collateral flow through the AComA. Therefore occlusion of the affected ICA may be the best choice.[95] Trapping and resection of the aneurysm[95] or trapping and side-to-side anastomosis[173] was applied in patients with type III dissection. They achieved a good postoperative recovery.[95] In addition, favorable results without recurrence were also observed in conservatively managed patients with A1,[68] A2, and A3[67] lesions with SAH[67,68] or a fresh cerebral infarction.[174] The patient with cerebral infarction recovered without complications by blood pressure control and administration of brain-function protection therapies without antithrombotic therapy.[174] If there is a high risk of rebleeding under conservative treatment (growing dissecting aneurysm, giant dissecting aneurysm, traumatic dissecting aneurysm, or dissection associated with uncontrolled hypertension), direct surgical treatment of the dissection should be proposed.[3,68,174]

Anterior Communicating Artery

BBAs of AComA are generally thin-walled lesions without a surgical neck as well as involvement of the ACA branches arising from a horizontal portion of the AComA.[80] Contrary to

BBAs on ICA that are associated with difficulties with clipping[40,42,155] and a high rate of intraoperative rupture[40,42,66] owing to abrupt transition from the sclerotic ICA wall to the fragile aneurysm neck[35,42,152,153] besides temporary clipping,[80] no surgical measure such as trapping, wrapping, or suturing had to be done in the surgical management of the BBAs of AComA. The lesions are usually able to be clipped with straight fenestrated clips through the A1-AComA junction. Good or fair outcomes are achieved in 80% of cases, even though 40% of the aneurysms rupture intraoperatively.[80]

Anterior Temporal Artery

True ATA dissecting aneurysms are extremely rare.[69,70] Because distal branches are thinner, the aneurysms carry a higher risk for rupture.[70] They may present as a dilatation and thrombosed mass with infarction in a corresponding region of the brain[70] or with SAH and intracerebral hematoma.[69] Trapping and resection of the aneurysms were not followed by new neurologic deficits.[69,70]

Prognosis

In general, a good prognosis without hemorrhagic complications and zero mortality was reported in conservatively treated patients with ICA IDAs presenting with stroke.[31] However, the overall surgical morbidity and mortality of patients with BBA is estimated at 18% and 14%, respectively, with the highest risk for patients undergoing clipping (75% of the overall morbidity/mortality in the surgical group).[66] Regrowth and rebleeding was observed in 5% and 30% of cases, respectively. An additional intervention due to rebleeding or evidence of BBA growth was required in 21% and 46% of the surgical and endovascular procedures, respectively.[66] Susceptibility to rebleeding within 14 days of MCA IDAs was reported in $\sim$ 50%, and $>$ 50% of them died.[65] Even though rebleeding from a dissecting aneurysm at the peripheral site of the ACA has been seen less frequently,[67] all previously mentioned facts suggest that early surgery is critical for preventing rerupture and SAH in cases of IDAs of the anterior circulation.[62] However, IDAs can resolve spontaneously; therefore the risk of rebleeding decreases considerably over time[91] with the complete neointimal reinforcement of the disrupted internal elastic lamina within 2 months.[21,175]

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