Updates in the Management of Cerebral Infarctions and Subarachnoid Hemorrhage Secondary to Intracranial Arterial Dissection: A Systematic Review

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Key words
- Aneurysm
- Dissection
- Hemorrhage
- Intracranial
- Ischemia

Abbreviations and Acronyms
CT: Computed tomography
CTA: Computed tomography angiography
IAD: Intracranial arterial dissection
MCA: Middle cerebral artery
MRA: Magnetic resonance angiography
MRI: Magnetic resonance imaging
SAH: Subarachnoid hemorrhage

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OBJECTIVE: **Intracranial arterial dissection (IAD) is a rare cerebrovascular disease that is likely underdiagnosed because of the inherent difficulty of visualizing the subtle radiographic signs of the pathologic small intracranial arteries. No widespread consensus exists on the treatment of IAD, and thus it is often managed empirically because of the absence of major randomized controlled trials. In this study, we conducted a systematic review to evaluate the management and treatment options for IAD.**

METHODS: **We performed a systematic review in accordance with the PRISMA guidelines using the following databases: MEDLINE (PubMed) and Cochrane Library. Included studies were limited to human patients with dissections in intracranial vessels only.**

RESULTS: **A total of 82 studies were included in this systematic review. The most common complications of IAD were cerebral infarction and subarachnoid hemorrhage, and thus, patients with IAD can be subdivided into those presenting with either ischemia or hemorrhage, respectively. Those with ischemia were predominantly managed with antiplatelet therapy, whereas patients presenting with hemorrhage often were amenable to treatment with endovascular techniques.**

CONCLUSIONS: **Given these findings, clinicians should prescribe antiplatelet therapy for patients with IAD presenting with ischemia and consider endovascular treatment for those presenting with hemorrhage. However, further investigation is required given the heterogeneity of methods and reporting outcomes in the investigated studies.**

INTRODUCTION

Intracranial arterial dissection (IAD) has become recognized as a more frequent cause of cerebral infarction and hemorrhage as a result of improvements in diagnostic imaging and an increasing awareness of the disease state.[1-3] IAD has a significant clinical impact, with nearly 20% of strokes in young patients being attributed to it.[4-7] Subarachnoid hemorrhage (SAH) is a feared complication of IAD and is often associated with a poor mortality. As well as the initial hemorrhagic insult, mass effect can compress the cranial nerves and brainstem, thus contributing to decreased long-term morbidity.[8] Symptomatic recurrence can extend to months after the ictus, with 1 study[9] finding that it affected 33% of patients.

There exists a scarcity of literature regarding IAD, with most consisting primarily of case reports and retrospective studies; there are no large randomized clinical trials investigating optimal management strategies. The number of cases that involve patients presenting with ischemia outnumbers those with hemorrhage. This dichotomization reaches clinical practice logistics in which patients with neck pain, symptoms of mass effect, or ischemia are treated by neurologists, whereas those presenting with SAH are often treated by neurosurgeons and endovascular neuroradiologists. Along with the skewed epidemiology, this tendency can lead to a publication bias and make drawing substantial conclusions on treatment decisions more difficult. Because of differences in the presentation and clinical course of IAD, this review aims to evaluate and present the current knowledge on the management of these lesions.

METHODS

A comprehensive systematic literature review using the MEDLINE (PubMed) and Cochrane Library databases was conducted

as per the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines. The search was focused on only clinical studies (randomized controlled trials, prospective and retrospective cohort analyses, case series, and case-control studies). One reviewer conducted the initial search, and the search was then verified by 2 reviewers.

The following keywords were used in our inquiry: intracranial dissection, intracranial carotid artery, intracranial vertebral artery, basilar artery, vertebrobasilar artery, anterior circulation, posterior circulation, ischemia, SAH, dissecting aneurysm. Furthermore, each individual topic pertaining to the objective of this review was subjected to its own search. The timeframe of the literature search was primarily restricted to articles published within the past 2 decades (1997–2017), but cornerstone articles within the field were included to provide historical perspective. We included all studies on human patients who had presented with intracranial arterial dissections. Studies in which the dissections were extracranial with some extension into the intracranial vessels were excluded.

RESULTS

A total of 82 studies were included in this review (**Figure 1**). When organized by location of the dissection, 25 studies involved the anterior circulation, 53 were on the posterior circulation, and 4 included dissections in both the anterior and posterior circulations. In total, 669 patients had anterior circulation dissections, with 492 (74%) presenting with ischemic symptoms. For the posterior circulation, 2948 patients were included, with 960 (33%) presenting with ischemic symptoms (**Table 1**). Concerning treatment modality, 13 studies discussed medical management, 52 pertained to endovascular management, 6 discussed surgical management, and 11 examined a combination of treatment modalities.

DISCUSSION

Epidemiology

As alluded to earlier, the incidence of IAD is difficult to determine because of the heterogeneous reporting standards across different populations; recruitment bias is a substantial factor in the reported literature. Asian countries seem to recruit patients through their neurosurgery and endovascular neuroradiology services, which skews the etiology of those with IAD favoring SAH. In contrast, European countries tend to recruit patients with IAD predominately through their neurology departments as a result of ischemia. Nevertheless, studies have shown that IADs represent <10% of all cervicocephalic dissections in adults and that approximately 3%–5% of SAH cases are caused by intracranial dissections.[10,11] In addition, approximately 2%–3% of aneurysms feature some form of dissection within the intracranial circulation.[12,13] There is a predisposition for IAD in the East Asian population, particularly in males.[4,14-16] However, this predisposition could be confounded by the fact that physical activity and trauma predispose one to the development of an IAD, which may explain the difference in prevalence between males and females.

Pathophysiology

The distinct histologic features of cerebral blood vessels contribute to the pathophysiology of IAD. Intracranial arteries, in contrast to extracranial arteries, have a thick internal elastic lamina, no external elastic lamina, a thinner media with fewer elastic fibers, and a thinner tunica adventitia.[17,18] Prolonged stress on the arterial wall, especially at points of turbulent blood flow because of luminal disease or mechanical factors, can lead to a weakening and deterioration of the arterial wall, thus predisposing to a dissection.[12]

Dissections can be categorized into 2 groups based on the degree of separation between the intima and media. A subintimal dissection occurs when blood accumulates between the tunica intima and the tunica media after the initial tear of the intima.[19] A subadventitial dissection occurs when there is a tear across the media and blood collects between the media and adventitia.[20] Some studies[21-23] have suggested that the intracranial arteries are predisposed to subadventitial dissection because of the lack of an external elastic lamina, a thin adventitia, and lack of a muscular layer. The rate of subadventitial dissection conversion from a subintimal dissection is not known.

After the initial tear in the intima, an intramural hematoma forms in subintimal dissections, which can lead to subsequent stenosis or occlusion of the vessel caused by the extravasated blood.[20] This situation can lead to symptoms of ischemia in the vascular territory of the affected artery. The stagnated flow of blood in the false lumen can result in a thrombus that can reconnect to the true lumen, leading to embolization of the intramural thrombus and subsequent infarction.[24] The reduced perfusion of the vessel also leads to a

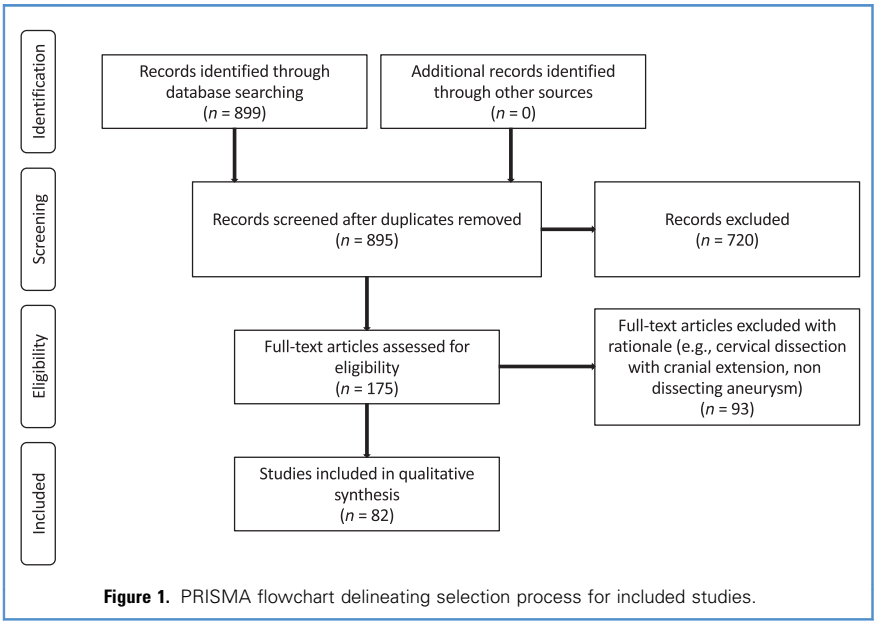

Figure 1. PRISMA flowchart delineating selection process for included studies.

Table 1. Study Characteristics Based on Dissection Location and Clinical Presentation

		Clinical Presentation	
Circulation	Number of Patients	Ischemic, n (%)	Hemorrhagic, n (%)
Anterior	669	492 (74)	177 (26)
Posterior	2948	960 (33)	1988 (67)

decreased washout of the embolus and contributes to progression of the ischemic stroke.[25,26] In addition, the intramural hematoma may be formed by direct bleeding of the vasa vasorum, which supplies the arterial walls. However, because vasa vasorum is absent in the intracranial arteries, this situation is usually only seen in the proximal arteries (i.e., the internal carotid, vertebral, and basilar arteries), which can result in a significant intracranial extension.[27]

Subadventitial dissections are initiated similarly to subintimal dissections, but the dissection pierces the tunica media and allows for the hematoma to accumulate between the media and adventitia. Because of the more fragile outer layers of the vessel bearing the stress of the expanding hematoma, a pseudoaneurysm can form, leading to compression of cranial nerves and the brainstem. Sustained stress on and expansion of this aneurysm can result in SAH.[8]

Diagnosis

Patients with IAD typically present with either infarction or SAH.[28] A few patients present with headaches or signs of mass effect impinging on susceptible areas of the brain parenchyma or cranial nerves. Still, others may be asymptomatic with extensive dissection aspects. Radiologic studies provide crucial information that can help arrive at a conclusive diagnosis. Magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) alongside computed tomography (CT) and CT angiography (CTA) are most often used because they allow for the best visualization of the internal and external portions of the vessel. However, digital subtraction angiography remains the gold standard for diagnosis, although it is more invasive than MRI, MRA, CT, or CTA.[29] More recently, susceptibility-weighted imaging has emerged as a potential option, with 1 study finding a 90% sensitivity and a 96.6% specificity for IAD.[30]

The following are key radiographic features of IAD: string sign (intraluminal stenosis of the pathologic portion of the vessel), string-and-pearl sign (proximal narrowing of the vessel with distal dilatation often seen in patients with fibromuscular dysplasia), double-lumen sign (the visualization of both a true and false lumen), intimal flap, stasis of contrast, fusiform dilatation, or pseudoaneurysm formation.[12,14,15,31] Of these features, the double-lumen sign and intimal flap have been regarded as pathognomonic for dissections, but some investigators have questioned the sensitivities.[2,15,19,29]

Because of the distinct presentations of IAD (i.e., ischemia or hemorrhage), these radiographic features can vary based on cause. Patients presenting with SAH are more often seen with associated aneurysms or fusiform dilatations. Conversely, in patients presenting with cerebral ischemia caused by subintimal dissection, the string sign is predominantly noted.[31,32] When MRI, MRA, CT, or CTA fail to provide a clear diagnosis, angiography is performed.[33] Here, the characteristic findings are the string sign or aneurysmal sacs and intimal flaps.

CEREBROVASCULAR DISSECTIONS IN THE ANTERIOR CIRCULATION

Background

Anterior circulation dissections present more often with ischemia than with SAH; 1 study found that 72.7% of patients with dissections in the anterior circulation presented with ischemia.[5] Among middle cerebral artery (MCA) dissections, ischemia was found to be the presenting symptom in 88% of cases.[34] SAH, conversely, occurs in about 20%–65% of anterior circulation dissections and, specifically, in about 12%–75% of MCA dissections.[10,31,34-36] However, larger studies evaluating the incidence of ischemia and hemorrhage in anterior circulation dissections are lacking, making it difficult to extrapolate conclusions from these smaller case series and to determine an accurate representation of the proportion of patients presenting with ischemia or hemorrhage.

Prognosis

Patients who present with an infarct caused by an IAD in the anterior circulation generally have good clinical outcomes at follow-up when managed with antiplatelet and anticoagulation therapies. Endovascular and surgical treatments have also proved to be effective when medical therapy is unsuccessful. The mortality among patients with IADs who present with ischemia ranges from 0% to 17%.[5,28] Those with an IAD who present with SAH generally have poorer outcomes because of a worse initial presentation and an increased risk for subsequent rebleeding.[5] The mortality among patients presenting with SAH caused by anterior circulation IAD ranges from 31% to 50%, with a greater risk of death if rebleeding occurs.[31,35]

CEREBROVASCULAR DISSECTIONS IN THE POSTERIOR CIRCULATION

Background

The annual incidence of vertebral artery dissection ranges between 1 and 1.5 of 100,000 people.[37] Although this review focuses on intracranial dissections, the most commonly involved regions of the vertebral artery are the extradural V1 and V3 segments because they are not bounded securely by dura and are thus more mobile and susceptible to injury. Extracranial dissections can extend intracranially and may consequently involve the posterior inferior cerebellar artery and other distal posterior circulation arteries.[38,39] Dissecting aneurysms of the posterior cerebral artery represent approximately 28% of posterior circulation aneurysms.[40-43] In contrast to anterior circulation IADs, posterior circulation IADs tend to present more frequently with SAH. In vertebrobasilar dissections, the incidence of SAH reaches almost 70%.[15,44,45] Some investigators have hypothesized that the long course of the vertebrobasilar artery within the

subarachnoid space contributes to the increased rate of pseudoaneurysm formation and SAH.[1] The rebleeding rate is particularly high in these dissections, ranging from 30% to 58%.[16,46-48] Approximately 26%–62% of IADs in the posterior circulation present with either infarction or ischemia.[15,19] Expanding dissecting hematomas and aneurysms can also present with symptoms of mass effect because of the proximity to the brainstem and cranial nerves.[13,20,49] The incidence of brainstem compression for patients with basilar artery dissections is 24%.[50] This situation is especially worrisome for patients because they may experience occipital headaches, vertigo, vomiting, and cranial nerve deficits.[20]

Prognosis

Patients presenting with posterior circulation IADs tend to have worse clinical outcomes because of the higher likelihood of SAH. Posterior circulation dissections with SAH have a mortality of up to 63%, which can increase with rebleeding.[15,16,44,51] The incidence of recurrent hemorrhage ranges between 22% and 58%, with most occurring within the first 24 hours after the initial hemorrhage. Patients with ischemia who were treated with medical management had low rates of subsequent ischemia.[4,14] However, whether this was because of a more benign disease course or the targeted management strategies is uncertain.

MANAGEMENT OF INTRACRANAL DISSECTIONS

Ischemic

Patients with anterior circulation IADs who present with ischemia are generally managed conservatively.[5] Treatment is initially targeted toward the dissolution of an intramural thrombus, and anticoagulants and antiplatelets are used to prevent recurrent strokes. The superiority of either therapy in the management of these patients has not been established. However, the use of anticoagulants in acute IAD poses a risk of increased bleeding into the dissection flap or hemorrhagic transformation of the original infarct.[4,5] Ohkuma et al.,[35] for example, cite poor outcomes in patients with MCA dissecting aneurysms treated with anticoagulants. Other studies contend that complications are unlikely, and that antiplatelet and anticoagulant therapies are safe and efficacious at preventing the formation of additional thrombi, which pose a greater risk to the patient's overall outcome.[1,31,34,52,53] Antiplatelet and anticoagulation therapy is generally recommended until the affected vessel has recanalized.[4,28] Intravenous thrombolytic therapy can also be used to treat IAD.[33,54,55] However, a narrow window of time and poor recanalization rates in vessels with larger occlusions limit its use.[56]

A stepwise approach to the treatment of anterior circulation IAD presenting with ischemia entailing escalating levels of antiplatelet and anticoagulant intervention has been propositioned. The first line of treatment is a single antiplatelet agent, such as aspirin. If the patient shows angiographic progression of the dissection or worsening of symptoms, a second antiplatelet agent, such as clopidogrel, may be considered on a case-by-case basis. An important consideration before escalating therapy is the patient's response to antiplatelet agents, which can now be monitored with various assays to optimize treatment.[57,58]

Patients with posterior circulation IADs presenting with ischemia can be treated conservatively if the dissections are not aneurysmal.[15,49,59-61] These patients often have good outcomes, with 1 study[59] reporting that only 14% of patients experienced rupture, which were subsequently treated via parent vessel occlusion. However, a 20%–28% risk of further infarction exists when the dissections are treated conservatively.[49,61] Nevertheless, aneurysmal dissections and dissections of the posterior circulation are often treated more aggressively because they can expand or bleed if left untreated.[62,63]

Endovascular therapy has become a mainstay treatment for patients with IADs presenting with ischemia who experience worsening symptoms despite optimal medical management. This finding is especially true if, on follow-up imaging, the dissection propagates further distally or if there is involvement of the posterior circulation, which can lead to severe deficits. Treatment options include intra-arterial thrombolysis, coils, and stents. Endovascular therapies are not only minimally invasive but they offer the ability to both diagnose and treat patients within a single procedure and are thus being adopted into practice more broadly.[16] By allowing for the direct delivery of thrombolytic agents at the site of dissection, intra-arterial thrombolysis is preferred to intravenous thrombolysis because it decreases the resulting dose requirements and reduces the risk of complications. Intra-arterial thrombolysis in conjunction with either a bypass or a stent has also been shown to be effective.[64,65]

Reconstruction can be performed using flow diversion devices or stents to separate the true and false lumens. This strategy leads to a controlled thrombosis of the dissection or the dissecting aneurysm and preserves patency of the parent vessel, thereby preserving physiologic cerebral blood flow.[66] The use of a stent alone or in conjunction with a flow diverter in the treatment of anterior circulation IADs has been performed successfully without any instances of rebleeding or progression of ischemia.[67-69] The occlusion of a vessel is usually preceded by a balloon occlusion test to evaluate the collateral supply to the vascular territory. These methods have been shown to be effective in small retrospective studies that have found good outcomes and no incidences of rupture or additional infarcts.[11,29,44,61,70] Reconstructive techniques minimize the effects of poor collateral supply on tissue that otherwise would have been sacrificed via a deconstructive procedure.

Overlapping of stents has also emerged as a viable treatment for IADs. The use of stents allows for the redirection of blood flow away from the dissection into the parent artery and enables stasis of blood in the aneurysmal sac.[37] These factors promote thrombosis of the aneurysm and lead to the occlusion of the aneurysm and neointimal growth over the stent scaffold. Several studies have been reported[71-77] showing the safety and efficacy of stenting in reconstructing dissected vessels. The procedure also has a lower risk of aneurysm rupture, because no coils are inserted into the thin-walled lesions.

Deconstructive techniques involve vessel takedown using embolization material or detachable coils. The Guglielmi detachable coil has been shown to be an effective method for thrombosis of these aneurysms.[61,78] A limitation of this coil lies in its inability to be used to treat fusiform aneurysms that lack a defined sac. These wide-necked aneurysms have a high risk of coil

herniation, which can lead to occlusion of the parent vessel. In turn, stent-assisted coiling has become an effective option to treat these dissections and to maintain the patency of the parent vessel.[59,60,71,79,80]

Surgical treatment options include trapping and resection or bypass of the occluded segment of the artery.[81] Ono et al.,[82] for example, reported performing a superficial temporal artery–MCA bypass to successfully treat an MCA dissecting aneurysm. Wakabayashi et al.,[83] alternatively, used surgical trapping and resection to treat an anterior cerebral artery dissecting aneurysm. Surgical treatment for IADs is not often performed in the context of posterior circulation dissections in patients presenting with ischemia. Data regarding this subject are limited. In one study, Kim et al.[15] reported that surgical trapping and clipping can be performed for vertebrobasilar dissections successfully without the risk of rupture or worsening of the infarction.

Despite the numerous treatment options available to patients, limited randomized controlled trial data are available to justify the use of certain treatment regimens over others. Treatments are selected for patients on a case-by-case basis.

Hemorrhagic

Because of the risk of rebleeding and higher mortality, anterior circulation dissections in patients presenting with hemorrhage are treated more aggressively than those involving ischemia.[3,16,79] Patients who are treated conservatively for SAH have been found to have poor clinical outcomes, resulting from frequent rebleeds.[35,84] Ruptured dissections are unstable and have a tendency to rebleed.[3,16,79] Studies on the treatment of posterior circulation IADs presenting with SAH[3,16,46,49,85] report high mortality and poor outcomes. However, this situation is often confounded by the poor initial presentation and preexisting contraindications to more aggressive therapies.

Endovascular therapies include the deconstructive and reconstructive techniques described previously. These options are individually chosen for patients based on vessel morphology and anatomy (i.e., the involvement of critical perforating branches). Studies on deconstructive techniques have shown good clinical outcomes with both rebleeding and infarction rates <33%.[3,13,15,44,46,60,70,80,85-100] Comparisons between these studies are limited by the heterogeneous outcome measures and varied reporting systems for complications. Reconstructive techniques are also efficacious, with reported bleeding rates <50% and infarction rates between 0% and 14%.[49,60,67,73-75,79,80,90,96,100-110] In 1 study consisting of 111 patients,[43] no statistical difference in recurrence rates after reconstructive and deconstructive regimens was observed. In the few cases of ruptured dissections in the anterior circulation treated with these techniques, a few investigators[31,69,111,112] have reported good outcomes with occasional mild neurologic deficits at follow-up. The use of liquid embolic agents is a novel approach to IAD; in 1 notable case,[113] embolization with Onyx was performed without any instances of rebleeding or rupture. Vessel reconstruction via a double stent-assisted coil embolization has also been reported.[114]

Surgical techniques to treat posterior circulation IADs include clipping and bypass with external trapping.[11,15,97,115,116] Morbidity and mortality are higher with this treatment modality than with an endovascular approach primarily because of a higher rate of cranial nerve injury.[3,11,45] In addition, achieving total occlusion of a dissecting aneurysm via clipping is difficult, and there is a risk of intraoperative rupture because of the thin adventitia, which bulges outward to form the wall of the aneurysm. However, bypass surgery can be a useful option in patients who have dissections involving the posterior inferior cerebellar artery, or when the contralateral vertebral artery is hypoplastic and the patient has failed the total balloon occlusion test. In these cases, perfusion can be maintained and the risk of infarction with aneurysm trapping decreases.[117]

Surgical treatment for IADs in the anterior circulation featuring hemorrhage has not been extensively investigated. One study[118] found that surgical treatment of MCA dissecting aneurysms resulted in improved outcomes compared with those treated without surgery. Ohkuma et al.[31] assessed the efficacy of surgical trapping, wrapping, and clipping of anterior circulation dissecting aneurysms and, among patients who had SAH after dissection, poor clinical outcomes were observed in 66% of cases after surgery as a result of rebleeding. This finding was corroborated in another study as well.[35] However, because anterior circulation dissections frequently present with ischemia, data regarding the efficacy of different surgical treatment in patients with SAH are limited. Because of the limited randomized controlled trials pertaining to surgical management, patients must be treated on an individual basis with consideration for dissection morphology, collateral circulation, and risk factors for surgical or endovascular treatment.

The lack of randomized clinical trial data compels clinicians to draw conclusions from retrospective and observational studies concerning the management of patients with these lesions. The retrospective studies, case series, and case reports included in this review did not have standardized reporting criteria or outcome measurements, obscuring the ability to discern the true risks and benefits of different treatment modalities. Reports of IADs are also prone to recruitment bias, which further limits the ability to draw conclusions and treatment recommendations. Because of the difficulty of recruiting large numbers of patients for clinical trials, increased efforts must be placed in assessing nationwide and device registries to gain more meaningful recommendations regarding the management of IAD. Treatment decisions should be made by a multidisciplinary team composed of neurosurgeons, neurologists, and neuroradiologists on an individual patient basis after weighing the risks and benefits of each type of treatment.

CONCLUSIONS

IAD is an uncommon cause of infarct and hemorrhage that has gained further awareness as a result of more refined noninvasive imaging techniques and an increased appreciation of the pathologic implications. IADs presenting with ischemia tend to have a more benign disease course compared with those presenting with SAH. The former is more amenable to treatment with anticoagulants, antiplatelet therapies, thrombolysis, or endovascular options. More aggressive therapy is warranted in patients with SAH because of the high initial mortality and risk of rebleeding. Several factors such as collateral blood supply and involvement of the posterior circulation

must be taken into consideration before pursuing reconstructive, deconstructive, or surgical treatments. Although no consensus exists, more studies endorse reconstructive techniques because they not only can simultaneously occlude the dissection and maintain patency of the parent vessel but they also pose less risk compared with open surgical intervention and deconstructive techniques.

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