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Review Article

Endovascular Treatment for Intracranial Artery Dissections in Posterior Circulation

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Intracranial artery dissections (IADs), although uncommon, are an important cause of cerebral infarction and subarachnoid hemorrhage (SAH). Some IADs can heal spontaneously after reconstitution of the vessel lumen with excellent prognosis. Meanwhile, others can progress to stroke that requires treatment. The incidence of IAD in the posterior circulation is higher than that in the anterior circulation. Anterior circulation dissections are more likely to develop into ischemia and posterior circulation lesions into hemorrhage. The mortality rate after IAD among patients with SAH is 19%–83%. Further, the mortality rate of IAD without SAH is 0%–3%.

Patients with SAH commonly undergo surgery or receive neuroendovascular treatment (EVT) to prevent rebleeding. However, the treatment of IADs is empirical in the absence of data from randomized controlled trials. Recently, EVT has emerged and is considered for IADs because of its less invasiveness and perceived low rates of procedure-related morbidity with good efficacy. EVT strategies can be classified into deconstructive (involving sacrifice of the parent artery) and reconstructive (preserving blood flow via the parent vessel) techniques. In particular, the number of reports on reconstructive techniques is increasing. However, a reconstructive technique for ruptured IADs has not yet been established. This review aimed to provide an overview of IADs in the posterior circulation managed with EVT by performing a literature search.

Keywords endovascular treatment, intracranial artery dissections, intracranial artery dissection aneurysm deconstructive techniques, reconstructive techniques

Introduction

Intracranial artery dissections (IADs) are uncommon. They are an important cause of cerebral infarction and subarachnoid hemorrhage (SAH) in children, young- and middle-aged adults.[1] In 1977, Yonas et al. first reported the pathologic and radiologic features of IADs.[2] Thereafter, IADs have been diagnosed more frequently due to familiarity with its clinical presentation and advancements in imaging techniques.[3] Although dissection of the extracranial cervical arteries has been extensively evaluated and described,[4] there is less information on pure IADs (i.e., excluding the cervical portion of the artery).[5] The optimum treatment for patients with intracranial artery dissection aneurysm (IADA) is unknown. There are no randomized trials, and only observational studies with small sample sizes are available, thereby providing a very low level of evidence. The current review aimed to provide a comprehensive overview of reported studies on the epidemiology and pathophysiology of IADs and a consensus statement regarding neuroendovascular treatment (EVT) for IADA.

Methods

A literature search was conducted on PubMed using a combination of the following terms: “intracranial,” “artery,” “vessel,” “dissection,” and “aneurysm.” Both titles and

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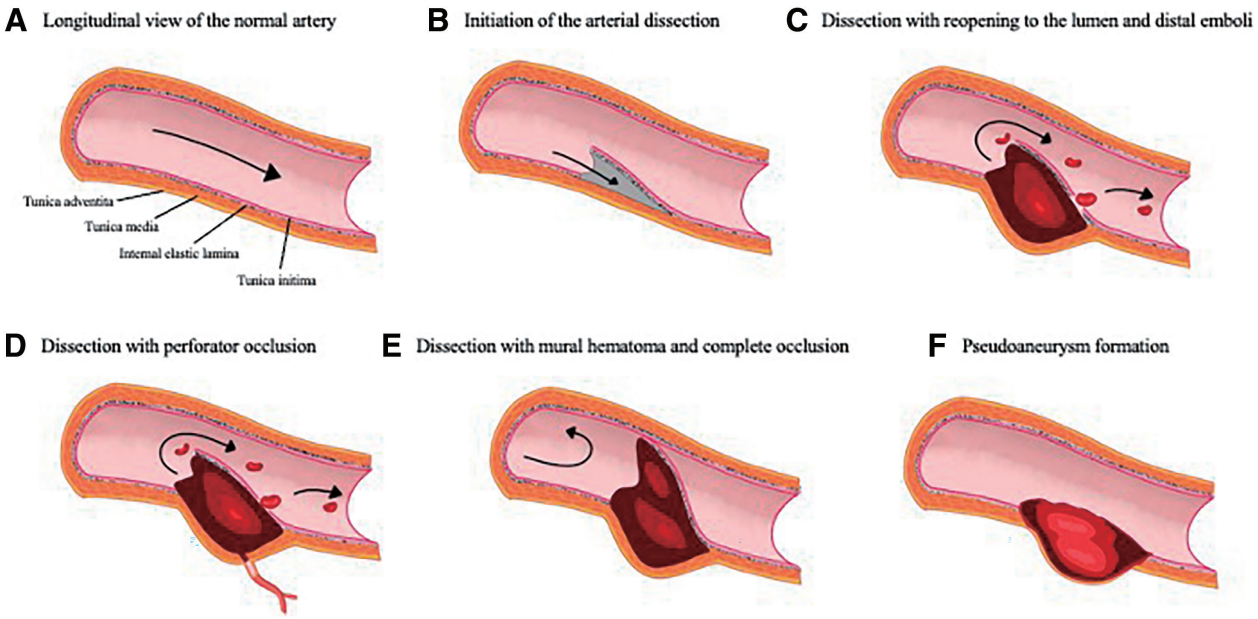

Fig. 1 (**A**) Normal layers of the intracranial. There was an internal elastic lamina; however, an external elastic lamina was not observed. (**B**) Arterial dissection occurred and blood dissociated into the subintimal space, thereby forming a false lumen and an intramural hematoma. (**C**) An embolus from an intramural hematoma in the false lumen can cause an embolic stroke. (**D**) If an intramural hematoma is involved at the origin of a perforating branch vessel, perforating branch occlusion may occur. (**E**) Intramural hematomas can accumulate and eventually cause parent artery occlusion. (**F**) Hematomas can accumulate around the perineum and cause pseudoaneurysm formation.

abstracts of the retrieved articles were meticulously screened for relevance. We did not exclude articles according to study type such as case reports and meta-analyses. Given the rarity of IADs, instances of more extensive extracranial vessel dissections have occasionally been reported.

A thorough examination of the references cited in each relevant article was undertaken to ensure a comprehensive review of the available literature and to avoid missing any pertinent information.

Epidemiology

To date, no population-based studies have assessed the incidence of IADs. It is probably lower than that of dissection within the cervical portion, which is approximately 2.6–3 per 100000 people per year.[4] The mean age upon presentation is 50 years.[5] The proportion of IADs in all cervicocephalic dissections substantially varies according to ethnic origin and age groups, and it is based on study recruitment strategies and ascertainment methods used. Previous studies on different geographic regions with substantially different ethnic populations have reported different intracranial and extracranial dissection ratios. In two studies that exclusively included East Asian patients, 67% and 78% of cervicocerebral dissection cases were intracranial.[6] In three series from Japan, IADs affect the posterior circulation more frequently than the anterior circulation (78%–98%). The most common site for intracranial dissection is the V4 segment of the vertebral artery. According to these reports, the proportion of people with pure IADs (i.e., without dissection of the cervical portion of the artery) leading to SAH was 52%–58%.[7–9] However, the causality between these events and IADs remains unclear.

Pathogenesis

The intradural segments of the arteries have distinct differences in their wall structure compared with cervical arteries. The arterial walls comprise three layers: (1) the tunica intima, which is the inner lining of the endothelium; (2) the tunica media, which is the middle muscular layer; and (3) the outermost tunica adventitia, which comprises longitudinally arranged collagen fibers. However, an external elastic lamina is absent in intracranial vessels (**Fig. 1**). Furthermore, the media lacks elastic fibers, and the adventitia is notably thin. Traditionally, arterial dissection is attributed to an intimal and internal elastic lamina (IEL) tear, leading to blood extravasation along the subintimal or subadventitial planes within the media layer.[10,11]

The pathophysiological manifestations of intracranial dissections are based on multiple factors, including the dissection location and vessel wall layers.[12] Cerebral hypoperfusion caused by severe stenosis or occlusion of the dissected artery occurs when the intramural hematoma builds up in the subintimal layer, and there is no outflow of blood from the distal portion of the intima. The characteristic double-lumen sign is observed if the dissection extends longitudinally along the subintimal layer and reopens into the vessel lumen. The false lumen generally exhibits slower blood flow than the true lumen, posing a risk of clot formation and distal embolization. If a perforating artery arises from the parent vessel and its ostium is involved in the intramural hematoma (e.g., intradural vertebral artery, basilar artery, posterior cerebral artery, and middle cerebral artery), there is a risk of perforator infarction. Approximately half of intracranial dissections are observed in aneurysmal diseases, including SAH and fusiform/thrombosed aneurysms, as no adventitial tissue contains the intramural hematoma in the vessel wall. After SAH for a dissecting aneurysm, the aneurysm is protected from further extravasation by a layer of thrombus only, which accounts for the fragility of these lesions and high rerupture rates.

The mechanism of acute IADs involves invasion of the circulating blood into the arterial wall through an entry formed by widespread disruption of the IEL.[13,14] Type 1 aneurysms comprise classical dissecting aneurysms with widespread disruption of the IEL, and they do not present with thrombus. Type 2 aneurysms comprise segmental ectasia dissecting aneurysms with stretched or fragmented IELs, moderately thickened intima composed mainly of collagen fibers, and a smooth luminal surface of the intima. Moreover, they do not present with thrombi. Type 3 aneurysms comprise dolichoectasia dissecting aneurysms. This histological feature represents laminar thrombi in the lumina of the aneurysms and thrombus formation at the sites of intimal dissection. Types 1–3 often appear as fusiform aneurysms on imaging. However, type 4 aneurysms take on a saccular appearance and can originate from intact sections of the IEL within pre-existing fusiform aneurysms or directly from the arterial trunk. These aneurysms are characterized by a fragile dome with sparse connective tissues, and they present with adventitial components, but not thrombi.

Diagnosis

IAD and IADA diagnoses are commonly based on clinical features and radiological findings. There are no unequivocal radiological criteria for IAD diagnosis. These diagnoses are often confirmed by performing various imaging modalities, including magnetic resonance imaging (MRI), magnetic resonance angiography, computed tomography scan, computed tomography angiography, and digital subtraction angiography. The characteristic radiological features indicative of IAD and IADA include the following[5]:

1. *Double-Lumen Sign*: This involves the presence of a false lumen, an intramural hematoma, or an intimal flap within the affected artery.
2. *Pearl-and-String Sign*: This indicates alternating widening and narrowing of the arterial lumen.
3. *Morphological Changes*: Serial imaging studies often reveal morphological alterations in the affected arterial segment. These can include a regular crescent-shaped thickening of the arterial wall, a dissected artery with increased external diameter, and, frequently, a reduced arterial lumen size.

Improved detection of mural hematomas can be achieved using high-resolution MRI imaging and three-dimensional acquisition of fat-suppressed sequences with a black-blood effect or susceptibility-weighted imaging (SWI). These techniques can enhance the sensitivity and specificity of imaging, making the identification of hematoma more reliable. The detection of a mural hematoma can be improved by using high-resolution MRI imaging and three-dimensional acquisition of fat-suppressed sequences with black-blood effect or SWI, which increase the sensitivity and specificity of images. Although these are characteristic imaging findings, to obtain a definite diagnosis of IAD and IADA, the baseline and follow-up imaging results are often compared.[12]

Treatment

The management of IAD is controversial due to the lack of randomized controlled trials and internationally accepted guidelines.[15] The incidence of IAD in the posterior circulation is higher than that in the anterior circulation. Anterior circulation dissections are more likely to develop into ischemia and posterior circulation lesions into a hemorrhage.[16] Previous reports have shown that the mortality rate after IAD among patients with SAH is 19%–83%.[5,16] In contrast, the mortality rate of IAD without SAH ranges from 0% to 3%.[5] The management of intracranial dissections excludes dissection from the circulation to prevent dissection propagation or rupture/rerupture. To treat intracranial dissection

caused by ischemia, medical treatment is often prioritized over surgical treatment. The benefit of medical treatment such as antiplatelet or anticoagulant therapy for cervical artery dissection is inconclusive in several randomized controlled trials and retrospective studies. The appropriate medical therapy for IADs has not been identified.[17] Surgical treatment, including EVT, is more frequently reported in IADs in the posterior circulation than in IADs in the anterior circulation. Hence, this study focused on EVT for IADs in the posterior circulation with respect to the treatment section. Dissecting basilar artery aneurysms was not included in this review because no case series exist, and there are several mixed reports on dolichoectasia.

Vertebral Artery Dissecting Aneurysm

Intracranial vertebral basilar artery dissecting aneurysms (VBADs) are the most common arterial dissection aneurysm among intracranial arteries. Early treatment is recommended for VBAD presenting with SAH because up to 40% of patients can rebleed within the first few days after onset.[9,18] Recently, EVT has emerged and is recommended for VBADs due to the perceived low rates of procedure-related morbidity with good efficacy and less invasiveness. The nationwide, retrospective, multicenter registries in Japan (JR-NET3) study has reported that the number of EVTs for ruptured VBADs has increased in the past decade.[18] Although there are several reports on its efficacy against ruptured VBADs, a consensus on the appropriate management strategy for unruptured VBADs has not been achieved. EVT is generally performed for unruptured VBADs with the following indications: (a) dissecting aneurysm with a size of >10 mm or with a mass effect, (b) early unfavorable change in VBADs in terms of shape and size during the follow-up period, and (c) recurrent or progressive ischemia. Kobayashi et al. have found that unruptured VBADs are typically associated with sluggish growth patterns.[19] Therefore, aneurysms measuring >10 mm or symptomatic aneurysms associated with a mass effect were more likely to rupture and should be treated. The reviews and case series on EVT for VBADs are summarized in **Table 1**.

Deconstructive or reconstructive treatment

EVT strategies can be divided into deconstructive (involving sacrifice of the parent artery) and reconstructive (preserving blood flow via the parent vessel) techniques. Specific reconstructive treatments include stents alone, stent-assisted coils (SACs), or flow diversion devices (FDDs).[20] A previous meta-analysis revealed that patients treated with deconstructive techniques (parent artery occlusion or aneurysm trapping) had higher rates of immediate complete occlusion than those treated with reconstructive techniques. This is an advantage of deconstructive treatments.[20,21] However, recent advances in devices have also reported the usefulness of reconstructive treatments.

Deconstructive treatment for VBADs

Parent arterial occlusion (PAO) is a representative deconstructive treatment. PAO, including the dissected segment of the vertebral artery (VA), remains the most reliable treatment option for ruptured VBAD. However, in some VBADs involving a dominant VA without sufficient collateral flow or incorporating the posterior inferior cerebellar artery (PICA) into the dissected segment, PAO may not be suitable due to a greater risk of medullary or cerebellar infarction. To perform trapping for ruptured supra-PICA VBADs and to sacrifice the PICA in cases with other poor collateral circulation covering the PICA territory, bypass techniques such as occipital artery-PICA anastomosis should be considered to reduce the complication of symptomatic PICA territory infarction.[22] If the origin of PICA involves the distal segment of VBAD, some ingenuity to preserve the PICA is required.[23] In addition to the ischemic complications of PAO, the possibility of the anterior or posterior spinal artery arising from the dissected segment should be considered. Guan et al. reported that severe ischemic complications such as quadriparesis or respiratory disturbance, might occur if the anterior spinal artery originates only from the side of the dissected VA in patients with VA aneurysms located distal to the PICA.[24] Furthermore, PICA, anterior spinal artery (ASA), and perforator infarctions to the brainstem are also a concern with PAO. Some studies have reported that postoperative medullary infarction is a risk factor for poor clinical outcomes. Endo has shown that occlusion or the blind alley of the terminal-type perforator caused by the PAO was associated with postoperative medullary infarction.[25] Therefore, preventing occlusion beyond the necessary range is considered better. In addition, there is a risk of developing contralateral vertebral artery dissecting aneurysms (VADA) as a long-term complication following PAO. Occlusion of the affected VA may cause increased blood flow in the contralateral VA, resulting in increased hemodynamic stress, as shown in several studies.[26] Excessive hemodynamic stress

Table 1 Summary of case series and reviews on EVT for VBADs

Studies	Types	Surgical approach	Results and conclusion
Lee et al.[28]	Ruptured (36), Unruptured (80)	Coil without a stent (9), Single stent with or without a coil (43), Multiple stents or stents without a coil (16), FDDs (13), and PAO (46)	The multiple-stent group had a higher complete occlusion rate and a lower recurrence rate than the other reconstructive treatment groups. Coil embolization without a stent was significantly associated with recurrence.
Oh et al.[30]	Unruptured (26)	FDDs (26)	FDDs are safe and effective against unruptured VADA. However, the complete occlusion rate in patients with VADA who presented with stenosis or those with a previous history of stent placement had a relatively lower FDD utilization rate than those with dilation-only lesions.
Catapano et al.[31]	Ruptured (54), Unruptured (37)	SACs (15), FDDs (29), PAO (47)	Endovascular FDD treatment of VADAs can be associated with lower retreatment and complication rates than stenting/coiling.
Dmytriw et al.[32]	Ruptured (21), Unruptured (39)	FDDs alone (50), FDDs with a coil (10)	FDD treatment in VA lesion with a coverage of ASA or PSA/LSA is not associated with higher occlusion rates in these branches or any cases of cord infarction.
Maybaum et al.[33]	Ruptured (31)	FDDs (31)	ADA reconstruction is a promising approach for acute SAH with FDDs. However, the severity of the condition is reflected by high overall morbidity and mortality rates, despite a technically successful endovascular treatment.

ASA: anterior spinal artery; EVT: endovascular treatment; FDDs: flow diversion devices; LSA: lateral spinal artery; PAO: parent arterial occlusion; PSA: posterior spinal artery; SACs: stent-assisted coils; SAH: subarachnoid hemorrhage; VA: vertebral artery; VADA: vertebral artery dissecting aneurysm; VBADs: vertebral basilar artery dissecting aneurysms

can disrupt the IEL, resulting in another artery dissection on the contralateral VA. As mentioned above, deconstructive therapy for ruptured VADA is the first choice of treatment. However, ischemic complications have been a cause of concern. By contrast, PAO for unruptured VADA is controversial. Li et al. and Lee et al. compared PAOs and SACs for unruptured VADA and reported fewer ischemic complications with SACs.[27,28] Thus, PAO is currently not actively recommended for unruptured VADA.

Reconstructive treatment for VBADs with SACs

There are several reports on reconstructive techniques used for dominant vertebrobasilar artery dissecting aneurysms that cannot be managed with deconstructive treatment. Guan et al. reported no significant differences between PAO and stent-assisted treatments (single stent with or without a coil) in clinical outcomes or ischemic complications. There were no significant differences in favorable clinical outcome rates, recurrence rates, and perioperative mortality rates between the two groups, particularly among patients with SAH.[24] Antithrombotic therapy is an issue in SAH cases, but the usefulness of SACs has also been reported. Therefore, stent-assisted treatments might be effective and safe for ruptured VA dissecting aneurysms. Among stent-assisted treatments, a comparison of the recurrence and retreatment rates between single stent alone or SACs were 22.5% and 7%, respectively. In addition, the metal coverage of stent(s) at the aneurysmal neck is an independent factor associated with the recurrence of vertebrobasilar dissecting aneurysms (VBDAs) after SAC.[29] Hence, SACs are better than stents alone. Further, the use of a braided stent with a higher metal coverage is preferred in SACs. However, in Japan, stent-assisted treatment in the acute phase of VBAD rupture has an off-label use.

Reconstructive treatment for VBADs with FDDs

Several mechanisms work for FDDs to achieve complete occlusion of VBADs. These include decreased aneurysmal inflow, antegrade flow diversion at the lesion artery, thrombus formation, and endothelial healing along the stent via epithelialization. Based on these effects, several FDD treatments for VBADs have been reported.[30] Several studies have reported good outcomes on FDD treatment for unruptured VADA with favorable occlusion rates (56%–91%) and safety outcomes (0%–17%).[29–31] This may be attributed to the fact that the VA where the lesion is located has a relatively simple vascular path. Hence, the FDDs attached extremely well to the arterial wall with lesions.

FDDs remain a concern in perforating branch infarction. Dmytriw et al. reported that in the ASA or lateral spinal artery, occlusion was not achieved with FDD placement for a VA aneurysm.[32] Under adequate antiplatelet therapy, FDDs for unruptured VBDAs can be effective. Although there are some reports on the efficacy of FDDs for unruptured VBADs, large-scale multicenter studies on ruptured VBADs have not been performed. To the best of our knowledge, most reports included small case series. According to the study of Maybaum et al., which has the largest number of cases, 31 patients with VADA underwent FDDs. In total, 18 (58%) patients had good outcomes. Moreover, six (19%) patients died, and two (6%) of them died due to procedure-related causes. Of these two patients, one had intraoperative rebleeding, and the other presented with delayed parenchymal hemorrhage.[33] By contrast, Catapano et al. compared FDDs and SACs for ruptured VBDAs. The rate of poor neurological outcome did not significantly differ, and the complication rate of FDDs placement was lower than that of SAC treatment (7% vs. 27%). Moreover, the FDDs group had a higher complication rate due to acute rebleeding compared with the SAC group.[31] Rebleeding after FDDs placement may be associated with the use of antiplatelet agents preoperatively and postoperatively to reduce the risk of thrombotic ischemic events. FDDs treatment for ruptured VBADs may be challenging unless the problem of antiplatelet therapy is resolved. A recently published report has shown that a new-generation flow diverter (FD), which has a modified surface, potentially results in reduced thrombogenicity. This novel device might be a viable alternative. However, further research on the use of FDDs for ruptured VADAs, including shielding technology, is required. The use of FDDs in the acute phase of rupture of VBADs is also an off-label use in Japan.

Posterior Cerebral Artery Dissecting Aneurysm

Posterior cerebral artery dissecting aneurysms (PCADAs) are rare. Posterior cerebral artery (PCA) aneurysms account for only 0.8%–1.7 % of all intracranial aneurysms, and the frequency of PCADA has not yet been reported.[34] PCA aneurysms are difficult to diagnose as fusiform or dissecting aneurysms accurately. Therefore, nonsaccular PCA aneurysms are treated as PCADA here, including past reports. Previous studies have reported that the most common locations of PCADA are the proximal segments (i.e., P1 segment, P1-2 junction, and P2 segment).

Some reports have suggested that anatomical factors are involved in the pathogenesis of PCADA. The proximal PCA and the tentorial incisura free edge have an intimate anatomic relationship. This may be an important factor in the genesis of PCA dissection and secondary aneurysm formation.[35]

Essibayi et al. performed a systematic review of 331 PCADA cases and observed that 82.8% and 92% of patients with ruptured and unruptured aneurysms, respectively, presented with favorable clinical outcomes. Approximately 50% of patients who received conservative treatment had favorable clinical outcomes, and 78.6% received retreatment.[34] Regarding treatment indications, other reports have shown that ruptured PCADA is associated with a poor prognosis, and it typically requires some treatments to achieve a better outcome. However, the treatment benefits of unruptured PCADA are significantly unclear. In addition, they reported a high complication rate for surgical treatment in both unruptured and ruptured cases. Hence, EVT is considered the recommended treatment for PCADA. There are several reports on the use of EVT as a treatment method in PCADA, and EVT strategies in PCADA can also be divided into deconstructive and reconstructive techniques. The reviews and case series on EVT for PCADA are summarized in **Table 2**.

Deconstructive treatment for PCADA

Several studies have shown that PAO has a good aneurysm occlusion rate. This is an advantage of deconstructive treatments. This is because the PCA territory has a rich collateral supply.[36] However, the PAO procedure complications have been reported in 33% of ruptured cases and 22.3% of unruptured cases.[34] Further, the indication of PAO for PCADA remains controversial. Therefore, the balloon test occlusion (BTO) before PAO is useful for identifying patients who cannot tolerate PAO following PCA occlusion. Meanwhile, vast clinical data show that BTO is useful for occluding the internal carotid and vertebral arteries. However, there is significantly less information about the usefulness of selective BTO for PCADA. Some reports have reported the usefulness of PAO for PCA aneurysms. Park et al. reported the usefulness of BTO for PCA aneurysms wherein six patients who did not receive BTO had newly developed PCA territory infarctions. In contrast, only one in five patients who received PAO after passing the BTO experienced such an infarction. Nevertheless, no BTO-associated complications were detected.[37] Thus, BTO may

Table 2 Summary of case series and reviews on EVT for PCADA

Studies	Types	Surgical approach	Results and conclusion
Essibayi et al.[34]	Ruptured (152), Unruptured (139)	EVT (53), PAO (187), conservative treatment (22), surgery (27), and combined therapy (2)	Ruptured aneurysms treated with EVT (22.6%) had higher recanalization rates than PAO (9.2%) and microsurgery (3.8%). In unruptured cases, microsurgery (40.4%) and PAO (21.5%) had higher complication rates than EVT (13.2%). Moreover, EVT had higher recanalization rates (15.6%). Hence, it is a promising approach for PCADA, with good clinical and safety profiles. However, it has a higher recurrence rate than PAO and microsurgery.
Park et al.[37]	Ruptured (9), Unruptured (12)	PAO (14), SACs (3), and surgery (4)	Six patients who did not receive BTO had newly developed PCA territory infarctions. One patient who received PAO after passing the BTO experienced such an infarction. BTO before PAO was helpful in identifying candidates who require surgical revascularization.
Tang et al.[38]	Ruptured (9), Unruptured (11)	SACs (20)	The complete occlusion rates were 83% in unruptured cases and 79% in ruptured cases. The complication rate was only 1% in ruptured cases. Braided stent-assisted coiling, which has a high occlusion rate and a relatively low complication rate, can be an alternative strategy against PCA aneurysms.
Wallace et al.[39]	Unruptured (29)	FDDs (29)	The preservation rate of the parent artery and perforating arteries was 92%. PCADA is more frequently associated with complete occlusion compared with saccular aneurysms (100% vs. 70%). However, it had a higher complication rate than (43% vs. 9%). The safety and efficacy profiles of FDDs of PCA aneurysms are acceptable in specific cases.

BTO: balloon test occlusion; EVT: endovascular treatment; FDDs: flow diversion devices; PAO: parent arterial occlusion; PCA: posterior cerebral artery; PCADA: posterior cerebral artery dissecting aneurysm; SACs: stent-assisted coils

be the best option for minimizing ischemic complications after PAO for PCADA.

Reconstructive treatment for PCADA with SACs

In patients who cannot tolerate the BTO outcomes or those with a lesion close to the origin of the perforating artery, the PCA blood flow and the patency of the parent artery should be maintained. To the best of our knowledge, there are no reports on the favorable outcomes of coil embolization for PCADA. Stent-assisted treatment is required to preserve the parent vessel, and the use of stents is reasonable for the reconstruction of dissecting vascular lesions. Several studies have reported the usefulness of SACs for PCADA. Tang et al. reported the treatment outcomes of SACs with a braided stent in 20 PCADA cases (11 unruptured and 9 ruptured PCADAs). The mean follow-up time was 11.2 ± 4.9 (range: 6–26) months. Nine patients with unruptured PCADAs presented with Raymond class I outcome and one with class II. Further, one patient had retreatment. Five patients with ruptured PCADAs presented with Raymond class I outcome and two with class II. Moreover, one patient had retreatment. One patient with a ruptured PCADA had perioperative hemorrhagic events.[38] SAC can be an effective treatment for PCADA. However, the treatment of lesions more distal than P3 is challenging and cannot be performed in all cases. In addition, there are no clear comparisons between the braided stent and other types of stents. Larger-scale or randomized studies may be required to identify the safety and long-term efficacy of SACs for PCADA. SACs in the acute phase of PCADA rupture have an off-label use in Japan.

Reconstructive treatment for PCADA with FDDs

Recently, some studies have reported the use of FDDs for the treatment of PCADA. These studies have reported favorable clinical and angiographic outcomes and the ability of FDDs to preserve the parent artery and perforating arteries. However, aneurysm recurrence or incomplete aneurysm occlusion is common after reconstructive EVT regardless of the morphology and rupture status; thus, close follow-up is required.[39] Otherwise, there is no FD stent compatible with PCA diameter such as distal PCA. Thrombotic complications are of more concern for PCA because of its smaller diameter than VA. Therefore, there

is currently no consensus regarding FDDs treatment for ruptured and unruptured cases. Further studies with a larger case series should be performed to validate the durability and efficacy of FDDs. Currently, several reports have revealed that constructive treatment for PCADA is more effective with SACs than with FDDs. Further, the use of FDDs for PCA lesions, even unruptured lesions, has an off-label use in Japan.

Reconstructive treatments for PCADA such as SACs and FDDs, have some limitations. PAO is the first treatment choice if these devices cannot be used for lesions located more than distal to P3.

Posterior Inferior Cerebellar Artery Dissecting Aneurysm

There are few large case series on PICA dissecting aneurysm (PICADA), and they all report a small number of cases. PICA aneurysms account for 0.5%–3% of all intracranial aneurysms. Isolated PICADA is extremely rare, with an incidence rate of 0.5%–0.7%. SAH is the most common initial clinical presentation (74%) of PICADA, with ischemia accounting for the remaining cases. Ruptured PICADA has a 24% risk of rerupture; hence, early diagnosis and management can prevent rerupture.[40] Early treatment is essential; however, its management remains controversial. Treatment decisions were based on the individual clinician's experience, without a standardized treatment approach. In most reports, the lesion has been treated via surgery, with direct clipping, wrapping, or trapping with or without distal revascularization.[41] More recently, the efficiency of EVT for PICADA has been reported. EVT is associated with a lower incidence of cranial nerve palsy than surgical treatment for PICA lesions.[42] Deconstructive treatment has been reported mainly in EVT for ruptured PICADA, with only a few case reports of reconstructive treatment performed. There are very few reports of surgical procedures for PCADA in patients with ischemia. The reviews and case series on EVT for PICADA are summarized in **Table 3**.

Deconstructive treatment for PICADA

PAO is considered the best management for ruptured PICADA. Even in PICADA, PAO is highly effective in preventing rebleeding and can be safely performed if a good collateral flow is present. Currently, the most important factor in determining PAO is the location of the lesion. PICA supplies blood to the caudal medulla, cerebellar tonsils, inferior portion of the cerebellar hemisphere, vermis, and choroid plexus of the fourth ventricle. The location of the involved PICA segment was recorded according to the classification scheme described by Lister et al.[43] (I, anterior medullary; II, lateral medullary; III, tonsilomedullary; IV, telovelotonsillar; and V, cortical segment). In particular, the proximal (I and II, anterior and lateral medullary) segments contribute perforating branches to the brainstem. Meanwhile, the distal (IV and V, telovelotonsillar and cortical) segments have no perforators. Therefore, anatomic consideration of whether the vessel does not possess perforating vessels to the brainstem is essential for deciding whether PICA can be sacrificed. If planning PAO for PICA, when the PICA is nondominant, and the dissection has developed on the distal segment, PICA sacrifice can be a suitable option. As the distal PICA lesion is always located in a twisty parent artery, PAO may be a simpler option. Lim et al. reported no additional abnormalities in the five patients who underwent PAO for distal PICADA, and they did not experience procedure-related complications.[44] Conversely, the risks and benefits of PAO should be cautiously discussed if a lesion involves the three proximal PICA segments (I, II, or III), all of which have perforators to the brainstem. Malcolm et al. reported that 12 (57%) of 21 patients had evidence of ischemic stroke on follow-up imaging and that one patient required suboccipital decompression.[45] Therefore, endovascular PAO of the proximal PICA is not a viable option for patients with poor collateral perfusion. Maimon et al. divided PICA cases into two types based on their angiographic anatomy. In the first type, the ipsilateral anterior inferior cerebellar artery is greater than or equal to more than half the size of the PICA. In this type, PICA occlusion could not be associated with significant sequelae. The second type is a small anterior inferior cerebellar artery and a dominant PICA. In this type, occlusion possessed a higher risk of cerebellar damage.[46] In cases where poor collateral perfusion is expected, alternative surgical treatments such as occipital artery-PICA bypass or reconstructive treatment of EVT should be considered.

Reconstructive treatment for PICADA

The first method, which is not a reconstructive treatment, is selective coil embolization of the dissecting aneurysm. This procedure preserves the parent vessel artery. The dissected portion of the PICADA is often small, and selective coil embolization is commonly challenging to perform. Therefore, the use of stents is required to preserve PICA. There have been several reports on the use

Table 3 Summary of case series and reviews on EVT for PICADA

Studies	Types	Surgical approach	Results and conclusion
Petr et al.[42]	Ruptured (660), Unruptured (136)	EVT (134), surgery (328)	The complication rates of cranial nerve palsy were 9.6% in the surgical group and 3.7% in the endovascular treatment group. Endovascular therapy could be an appropriate first-line treatment for proximal PICA aneurysms. However, surgery remains a highly effective first-line treatment for distal PICA aneurysms.
Lim et al.[44]	Ruptured (5)	PAO (5)	In all cases, complete occlusion was achieved There were no immediate procedure-related and perioperative complications. PAO is recommended for distal PICADA.
Malcolm et al.[45]	Ruptured (21)	PAO (21)	Twelve (57%) patients had evidence of stroke (cerebellar [n = 8], medullary [n = 1], and both [n = 3]) on follow-up imaging. One patient required suboccipital decompression for brainstem compression. If surgical revascularization is either high risk or is technically not feasible, proximal PICA sacrifice is considered.
Maimon et al.[46]	Ruptured (5), Unruptured (1)	PAO (6)	PAO was successful in all cases, without aneurysmal bleeding, persistent neurologic deficits, or signs of infarction. There were no procedure-related complications. PAO of isolated dissecting PICA aneurysms can be a safe and effective alternative to surgery if vessel occlusion can be tolerated, as determined by adequate collateral circulation on preprocedural angiography.
Wallace et al.[47]	Ruptured (1), Unruptured (2)	FDDs (3)	None of patients developed complications. One of three patients had complete occlusion, and two had residual aneurysm. The size of both aneurysms decreased, and they were not re-treated. FDDs of PICA aneurysms is a safe and viable treatment option if traditional endovascular options are not likely to preserve parent vessel patency.
Lauzier et al.[48]	Ruptured (2), Unruptured (1)	FDDs (3)	All aneurysms achieved complete occlusion at the 6-month and final follow-up in patients undergoing subsequent angiography. Pipeline embolization of distal PICA aneurysms is feasible in specific cases. However, operator experience is important for these procedures, with consideration that this approach has technical challenges.
Bhogal et al.[49]	Ruptured (2), Unruptured (1)	FDDs (3)	There were no procedure-related complications. Two of three aneurysms had complete occlusion at the 6-month follow-up. The use of FDDs for the treatment of PICA aneurysms can be useful in proximal PICA aneurysms.

EVT: endovascular treatment; FDDs: flow diversion devices; PAO: parent arterial occlusion; PICA: posterior inferior cerebellar artery; PICADA: posterior inferior cerebellar artery dissecting aneurysm

of each. However, all studies involved case reports.[47–49] Based on these reports, the FDDs of PICA aneurysms can be a safe and viable treatment option. The issue with the treatment approach is the placement of a microcatheter distal to the PICA. The more recent introduction of FD, which can be delivered via 0.017- and 0.021-in microcatheters, may also facilitate FD placement in the distal PICA. The use of FDDs requires strict antiplatelet medication. However, at present, FDD treatment for PICA is preferred for unruptured PICA aneurysms, particularly recurrent PICA aneurysms after selective coiling. The use of FDDs in the acute rupture stage is an issue that should be discussed in the future. Further, the use of FDDs for PICA lesions, even unruptured lesions, has an off-label use in Japan.

The application of the FD device requires strict antiplatelet medication; thus, the FD device is preferred in treating unruptured PICA trunk aneurysms, particularly recurrent PICA aneurysms, after selective coiling. Therefore, more case studies on FDDs for PCADA should be performed.

Conclusion

Intracranial dissection is a relatively rare entity that can present in various ways, including subarachnoid

hemorrhage. The natural history of the disease can be good in the case of an ischemic lesion. However, lesions that present with hemorrhage have a high rate of rebleeding if left untreated and generally require prompt surgical treatment, especially endovascular techniques. Despite the availability of various treatment options, the key to a successful outcome is a personalized approach that considers the presentation, natural history, and anatomical location of the dissection. An individualized approach can decrease risks and optimize the possibility of a good outcome.

Disclosure Statement

The authors declare no conflicts of interest.

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