

State-of-art in surgical treatment of dissecting posterior circulation intracranial aneurysms

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Abstract Vertebrobasilar (VB) intracranial dissecting aneurysms (IDAs) pose difficult therapeutic issues and are especially among the most difficult to manage surgically. There are, however, some cases where selective aneurysm obliteration by endovascular approach is impossible or is associated with an unacceptable risk of morbidity. This is particularly true when the aneurysm is dissecting, giant, or has a large neck. In such cases, surgical treatment may be the only alternative. Optimal management of these lesions is therefore challenging and treatment decisions have to be made on a case-by-case basis. Ideal treatment should be a complete surgical excision of the lesion; however, this procedure might only be possible after distal and proximal vessel wall occlusion which might not be tolerated by the patient depending on the location of the aneurysm. Therefore, formulation of recommendations

concerning the surgical strategy remains still difficult due to inconsistency of surgical outcomes. The literature describing surgical strategy of VB IDAs is varying in quality and content, and many studies deal with only a few patients. In the presented review, the authors summarize the current knowledge on the incidence, pathogenesis, clinical presentation, and diagnostic procedures with special emphasis on surgical treatment of IDAs in posterior circulation.

Keywords Intracranial dissecting aneurysm · Posterior circulation · Surgical treatment

Introduction

Even still considered relatively rare, intracranial dissecting aneurysms (IDAs) are important causes of subarachnoid hemorrhage (SAH), stroke, or compression of intracranial structures [5, 139] affecting healthy young adults [46] with a high associated morbidity and mortality [67]. Nevertheless, their pathogenesis and management [76] remains unclear and debated, so far. Even though their conservative, endovascular, and surgical treatment have proved successful, with the availability of endovascular procedures, the method has become preferred in treating of IDAs in vertebrobasilar (VB) territory [141]. However, the purely endovascular treatment is unlikely to stop the disease process [156]. Ideal treatment should include a complete surgical excision of the lesion; however, this procedure might only be possible after a complete distal and proximal vessel occlusion which might not be tolerated by the patient [76]. Therefore, formulation of recommendations concerning the surgical strategy remains still difficult due to inconsistency of surgical outcomes [8, 25]. The aim of this paper was to conduct a systemic review of the literature and summarize the current knowledge on the incidence,

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pathogenesis, clinical presentation, diagnostic procedures, and surgical treatment of the IDAs in posterior circulation.

Search strategy

The strategy was designed by the authors to search data base PubMed for English- and Japanese-language articles using text words or terms including “intracranial,” “dissecting,” “dissection,” “aneurysms,” “posterior,” “circulation,” “vertebral,” “basilar,” “vertebrobasilar,” “superior cerebellar artery,” “anterior inferior cerebellar artery,” “posterior inferior cerebellar artery,” “posterior cerebral artery,” “surgical,” “treatment,” “therapy,” “methods,” “risk factors,” “pathophysiology,” “diagnosis,” and “prognosis.” The words/terms were used in both “AND” and “OR” combinations. The authors included studies with radiological, intraoperative, or pathological confirmation of dissection meanwhile studies that did not provide sufficient detail regarding diagnostic criteria for dissection were excluded. The search was not (a) designed to identify studies reporting on conservative or endovascular treatment and (b) restricted by date of publication, patient age, or number of subjects. The authors 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

The IDAs, most frequently detected in Eastern Asian population [5], constitute 2–3 % of all cerebral aneurysms [96, 114]. The IDAs of VB territory occur at a rate of 1–1.5 per 100,000 individuals per year [136] and account for about 28 % of posterior circulation aneurysms [171]. The vertebral artery (VA) dissecting aneurysms often impinging the adjacent basilar artery (BA) [171], or posterior inferior cerebellar artery (PICA) [8, 25] represent the most frequent location of intracranial [8, 11], multiple [172], and posterior circulation dissections [46]. Bilateral VA dissecting aneurysms are reported to account for 7.4 to 16 % of all VA dissecting aneurysms [112, 171]. Whereas the intracranial VA is involved in 81.6 % of posterior circulation arterial dissections [46], the BA IDA represents a rarer entity [45] accounting for about 10.5 % of posterior circulation vessel dissections [122]. Meanwhile, the VA dissecting aneurysms occur in patients with mean age 49.7 ± 8.6 years [171] with a higher prevalence in males [68, 70, 90, 171], and the BA dissections tend to affect even younger people with mean age 31.7 years [90]. Since male predominance observed in children could not be explained by trauma, it suggests a sex influence on their pathogenesis [26]. Finally, whereas the incidence of SAH in VB IDAs ranges

from 61 to 86 % [3, 58, 68, 70, 125, 171] and 24 % of them rebleed [171], ischemic events associated with the dissections were observed only in 26–62 % of patients [70, 150]. In addition, Ono et al. [111] reported that 18 % of initially unruptured IDAs showed clinical symptomatic recurrence and 90 % of them had major or minor strokes caused by occlusion of perforating arteries with a mean interval of 8.6 months. In children, the rate of recurrent ischemic events was observed in 15 % of cases of posterior circulation dissections with rare hemorrhagic complications [26].

Pathology and pathophysiology

A diversion of circulating blood into the weakened arterial wall may come from (a) a tear in the media due to rupture of the vasa vasorum [51] or new vessels formed in response to pathological process, such as atherosclerosis [124]; (b) the intimal surface causing mainly compression of the lumen by blood accumulation between the internal elastic lamina and media [12, 150]; or (c) the dissection plane lies between the media and the adventitia, causing outpouching of arterial wall [12]. Intracranial arteries are prone to subadventitial dissection [164] and subsequently to SAH [8] due to lack of an external elastic lamina and thin muscular/adventitial layer [164]. A recent observation that medial and subadventitial hemorrhage may also underlie ischemic symptoms indicates that some hemorrhagic transformations of unruptured IDAs may be caused by this mechanism [111]. Internal elastic lamina is the most important layer for determining the strength of the intracranial arteries; therefore, the vessel is more prone to damage if the elastic tissue is defective [28]. The intimal cushion formed at branch points in the cerebral circulation by splitting of the internal elastic lamina may serve as a starting point for dissection, especially if an underlying medial defect is present [39]. Dissection may lead to the formation of (a) intraluminal thrombus [12] with hypoperfusion and infarction [13] or (b) a false lumen that may be a source of emboluses [12, 76] or may occlude small perforators [76, 150]. Entry-exit type of dissections is less prone to rupture than entry-only type [151], since the former mentioned lesions contain a constant flow of blood through the pseudolumen and are clinically more stable [98].

Molecular and genetic factors

Congenital and acquired abnormalities of the arterial media and elastic tissue such as Ehlers-Danlos syndrome or fibromuscular dysplasia are seldom found in patients with dissecting aneurysms [138]. Occasionally, ultrastructural abnormalities resembling the aberrations found in Ehlers-Danlos syndrome type II or III have been found in nontraumatic cervical arterial dissections [10]. One study has also reported the

association between alpha 1-antitrypsin deficiency and ruptured intracranial aneurysm or spontaneous dissection of the cervical internal carotid artery (ICA) [137]. Since a fewer than 8 % of patients have connective tissue disorders, it does emphasize the need to shift clinical focus away from seeking VA dissection only in patients with underlying conditions of this type [32].

Risk factors

Although in majority of cases the cause of IDA is unknown, several risk factors (e.g., hypertension, oral contraceptives, cigaret smoking, diabetes mellitus, head or neck trauma, syphilis, polycystic kidney disease, polyarteritis nodosa, systemic lupus erythematosus, or Moyamoya disease) are suspected to be associated with dissection [25, 52, 110, 114, 146, 150, 151, 166]. It has also been suggested that arterial wall edema during a migraine may contribute to the development of IAD as well [49].

Clinical presentation

Patients usually present with either SAH, signs coming from mass effect [12], or ischemic symptoms that have more benign course than those with SAH [37]. Fullerton et al. [26] reported on observation of a 100 % rate of cerebral ischemic presentation in children. A severe, recurrent, steady, or pulsating headache [37, 171] in the occiput with or without its irradiation to the ipsilateral neck preceding neurological disturbances by days to months [86] typically heralds dissections in posterior circulation. However, many patients do not reveal symptoms typically linked to VA IDAs [26] since a pooled prevalence of neck pain in VA dissections was only 46 % [32]. Minor neurological symptoms include diplopia, nystagmus, slight unilateral weakness, vertigo, nausea, vomiting, dysphagia, dysphonia, hemianopsia, dysarthria, Wallenberg's or Terson's syndrome, and Horner's sign [12, 31, 91, 130, 135, 171]. Patients with BA dissection usually present with sudden coma [12] and tetraparesis and extensor plantar reflexes [11]. Cerebral peduncle in the midbrain or pontine tegmental area infarction occasionally causes hemiplegia or coma without pyramidal signs, respectively [11]. Fulminant extension of dissection in case of SAH is one of the important causes of sudden death of the patients [37].

Preoperative diagnosis

Dissections can be imaged either by digital subtraction angiography (DSA) or less invasive modalities, such as

computed tomography (CT)/CT angiography (CTA) or magnetic resonance imaging (MRI)/MRI angiography (MRA). DSA is a gold standard for luminal imaging but, because of its invasive nature and advances in neuroimaging, it is mainly used if CTA or MRI is inconclusive, patients present with SAH, or if surgical or endovascular strategy is being considered [16].

Digital subtraction angiography

The common finding in subintimal dissections is a string sign characterized by a long, narrow column of contrast material; a flame-shaped tapering of the lumen; and occlusion of the artery. A subadventitial dissection is imaged as aneurysmal dilatation preceding or following a focal narrowing of the vessel lumen ("pearl and string sign") [12]. Retention of contrast medium seen in the late angiographic phase due to its influx into the intramural lumina (intramural pooling sign) was defined as a diagnostic indication of arterial dissection [171] and double lumen sign was considered the most indicative of IAD and seems to be necessary for a decisive diagnosis [70, 135, 150, 171]. The string sign and the pearl and string sign show dynamic changes on follow-up angiography, while tapered occlusions or dissections evident as dilatation without luminal stenosis remain unchanged, irrespective of whether the saccular, fingerlike extensions parallel to the artery or fusiform luminal configuration are present [173].

Computed tomography angiography

Thin-section multisection CTA better demonstrate extraluminal abnormalities [87] and frequently depict more findings indicative of dissection, such as more frequently found intimal flaps and pseudoaneurysms than MRI and MRA [158]. The lumen of VA dissection may be of normal caliber and the lesion is easily overlooked if only lumen-opacifying studies such as contrast MRA or DSA are performed and the only visible abnormality on CTA is a dorsal thickening of the arterial wall against the adjacent fat, the "suboccipital rind" sign [87]. The limitations of MRI/MRA, such as obscuration of mural hematoma by the hyperintense signal of thrombus within an occluded artery and lack of hyperintense signal within mural hematoma on T1WI in the first few days after onset of dissection may be overcome by CTA [20]. Chen et al. [50] found that CTA was especially sensitive and accurate for diagnosis of VA dissection with stenotic or expanded lumen when compared with conventional angiography. Comparing sensitivity and specificity, MR techniques and CTA for diagnosis of craniocervical arterial dissection are quite similar [117].

Magnetic resonance imaging/angiography

MRI of IDA include routine T1-weighted images (T1WI), T2-weighted images (T2WI) usually with spatial presaturation, either with or without fat suppression, and MRA with or without gadolinium enhancement [74, 81, 118, 121]. The intensity of an intramural hematoma on T1WI and proton images varies according to its age [74]. It appears isointense or slightly hyperintense during the first few days after onset, then becomes hyperintense in the subacute stage. The abnormal intensity of the hematoma resolves after several months [29]. Conventional MRI is able to detect the intramural hematomas in less than 35 % of VB IDAs because T1WI isointense hematoma in the acute stage is often obscured by surrounding tissue [71]. Contrary, Yoshimoto and Wakai [173] demonstrated that T1WI between 3 days and 2 months after the ischemic episodes shown intramural hematoma in 70 % of examinations. The T2WI seem to have less diagnostic value for imaging intramural hematoma in the posterior circulation because they are prone to image distortion due to hyperintense cerebrospinal fluid [74], bony structures, and air in mastoid bones [71]. An intimal flap is better visualized on MRI than angiography [56] and MRA seems to be useful in demonstrating pseudolumen, aneurysmal dilatation, and occlusion of the affected vessels [173]. Time-of-flight MRA can provide direct visualization of subacute intramural hematoma as a high-signal structure due to its short T1WI feature [74, 81, 117, 121]; however, differentiation between intraluminal flow, intramural hematoma, and surrounding tissue may be difficult due to (a) the intraluminal signal changes depending on flow velocity, presence of laminar flow and/or turbulence [20, 118] and (b) confusing high-signal intensity caused by the venous plexus and fat surrounding the VAs [74]. For the last decade, a variation of the black blood three-dimensional turbo spin-echo technique has been introduced with visualization grading scores of the intramural hematomas significantly higher than those of MRA [148]. Advanced high-resolution MRI provides detailed information of hidden structures such as intimal flap, inlet of the false lumen, size of the intramural hematoma, and ostium of small branching vessel [162]. It helps to differentiate between intramural hematoma and intraluminal thrombus [162]. Susceptibility-weighted technique provides images with higher resolution, flow compensation, less image distortion in the detection of micro bleeds and can differentiate hemorrhage from calcification [85].

Intraoperative diagnosis

The clinical diagnosis of VB IDAs is based on the characteristic angiographic features but is sometimes difficult to make with certainty [37]. Characteristic sign confirming preoperative diagnosis of dissection is a reddish discoloration of affected artery around aneurysmal dilatation owing to hematoma in the vessel

wall observed at surgery within acute stage [25, 37, 109, 135, 171]. Approximately 1 month after ictus, the aneurysm becomes whitish gray in color with shiny, smooth surface, firm, and not compressible due to organized intramural clot. Occasionally the vascularization of the outer wall is visible, too [171].

Differential diagnosis

Characteristic angiographic findings of aneurysmal dilatation were observed in 89 % of patients with SAH, but only in 14 % of cases with ischemic symptoms [37]. Limitation of CTA, MRA, and DSA is that they cannot differentiate between different vascular pathologies that exhibit similar luminal changes [87]. Occasionally, the lumen of affected artery appears normal in caliber on CTA, despite the vessel wall hematoma [87]. The string sign may be misdiagnosed as sign of spasm in patients with SAH [131]; however, the vasospasm would not be expected early (until fourth to tenth day) after bleeding and is often multifocal [79]. Moreover, the string sign may be distinguished from ordinary vasospasm by the presence of the mural irregularity [135]. Similarly, a localized abnormality in a single vessel would make an atherosclerotic lesion less likely. Vasculitis also tends to involve vessels in a diffuse manner and would be characterized by areas of arterial wall dilatation in addition to stenosis [79]. Since the term fusiform aneurysms may include both acute dissecting and chronic fusiform lesions [102] including giant so-called dolichoectatic aneurysms [19], a definitive diagnosis should be made on the basis of pathological dissection or angiographic and intraoperative findings. A certain subset of fusiform aneurysms may be caused by dissection of the aneurysmal wall [18, 100], but progressive dilatation of vessels due to defect of the internal elastic lamina may be an important factor in pathogenesis of these lesions as well [174]. Therefore, giant fusiform aneurysms should probably be regarded as distinct entities [30]. A fusiform or irregular aneurysmal dilatation located at a non-branching site of an artery is very suggestive of IDAs if associated with a segmental stenosis [103]. However, additional radiological elements are needed to confirm the diagnosis of intracranial artery dissection, including rapid change in morphology [16]. Interestingly, Yamaura reported on that while 81 % VA dissecting aneurysms bleed and 24 % of these rebleed, no such feature was observed in atherosclerotic fusiform aneurysms [168]. Strictly, definite diagnosis of IDAs can be done only by pathological diagnosis [37].

Treatment strategy

As widely accepted guidelines are still missing, an interdisciplinary team of neurosurgeons and endovascular interventionalists should bear in mind patient's medical and

neurological condition, age, and personal preference, location and morphology of IDA, its relation to afferent and efferent vessels, critical structures, presence of neck calcification, dome-to-neck ratio [40], whether the PICA is reduplicated or its territory is supplied by an alternative vessel; whether the contralateral artery is present; whether there persists fetal posterior cerebral circulation [48]. In patients with ischemic symptoms, the treatment strategy should be focused on reduction of a risk of thromboembolic recurrent ischemic stroke by administration of anticoagulants or antiplatelet agents [46, 68] while stenting [12] and surgical treatment [37] is rarely indicated, because in patients without occlusions the arterial lumens usually open of their own accord over time, and anticoagulants are effective at preventing further thromboembolism. As ruptured IDAs are unstable and their tendency to rebleed is associated with high rate mortality, the endovascular approach is the first choice in their treatment [139]. However, a subset of IDAs not amenable to endovascular treatment still requires operative intervention [68]. Beside PICA-origin aneurysms, this subset includes giant, partially thrombosed, fusiform-like aneurysms, those with unfavorable neck-to-dome ratios, and those that have failed attempted endovascular treatment [175]. The purpose of surgery of IDAs is complete isolation of the dissection from the circulation in order to prevent rebleeding and extension of the dissection [37], avoid or reduce ischemic damage, and achieve neural decompression in case of giant aneurysm [97]. Management strategies that combine surgical and endovascular techniques in a complementary way can be designed accordingly [35]. Ventricular drainage can be a necessary emergent procedure, because many patients with rupture of VB IDAs have SAH complicated by hydrocephalus [37, 171].

VA dissecting aneurysm

Approximately 24 % of ruptured VA dissecting aneurysms may rebleed and 80 % of the patients die or become comatose, rebleeding should be prevented [168] and the lesion secured as soon as possible, since rebleeding occurs in early period [37, 155]. The factors that determine the appropriate treatment modalities for ruptured IDAs of the VA are guided by tolerance of the affected VA to a 20-min balloon test occlusion (BTO) and the involvement of the PICA [84].

PICA non-involvement and tolerance of BTO

Patients with IDA without PICA involvement who tolerate the BTO without compromising cerebral perfusion were treated using the Guglielmi Detachable Coils by a double-microcatheter technique with favorable outcomes [36, 40]. The occlusion of critical perforating vessels arising from the distal VA, especially the non-dominant VA, and from

proximal and distal part of VB junction [88], may cause underlying treatment-related complications [36]. Affected VA can also be effectively proximally clipped [25, 135, 171], but only when a patient's contralateral VA and posterior communicating arteries (PComA) are well developed and blood supply to the brain stem and cerebellum is sufficient [135, 161]. However, the blood flow from the contralateral VA may persist; this may retard thrombosis and organization of the dissection site, resulting in postoperative rebleeding [4, 25, 72]. Friedman and Drake [25] reported three VA occlusions by proximally placed clip to the PICA without deficit. The PICA filled retrogradely through (a) the partially thrombosed aneurysm, (b) its high origin just beyond the distal segment of the thrombosed aneurysm, or (c) a collateral vessel from the anterior inferior cerebellar artery (AICA). Interestingly, even trapping cannot prevent the postoperative rebleeding [55, 60]. Mechanism of development of such an aneurysms is not clearly understood, but three anticipated mechanisms have been mentioned: (a) a normal VA and BA developed a dissecting aneurysm due to hemodynamic stress after complete trapping; (b) a preexisting VA or BA dissecting aneurysms angiographically occult enlarged due to hemodynamic changes after trapping; and (c) incomplete trapping [60]. Finally, if the patient remains asymptomatic during the BTO but has evidence of cerebral perfusion compromise, a low- or high-flow bypass should precede parent vessel occlusion [40].

PICA involvement and tolerance of BTO

Even though some authors wrapped [171] or directly clipped neck of PICA-involved VA IDA with favorable outcome [37, 144], in patients with IDAs with PICA involvement who tolerate the BTO, aneurysm trapping and PICA transposition into the VA by interposing a superficial temporal artery (STA) [36] or radial artery (RA) graft [15] is performed either by end-to-end (STA) or end-to-side (STA or RA) anastomosis to PICA and end-to-side anastomosis (STA or RA) to VA facilitating anterograde flow to the PICA territory without requiring the extensive mobilization of brainstem perforating vessels and the proximal PICA [15, 36].

Intolerance of BTO

In the patient who are unable to tolerate the BTO, the trapping of the aneurysm via the posterior transpetrosal approach along with VA–posterior cerebral artery (PCA) bypass procedure may be performed by means of the superior petrosal sinus sacrifice, the tentorium cerebelli cutting and an end-to-side anastomoses between the P2 segment and the extradural VA using RA [36, 40] or vein graft [40]. The wider, proximal end of the RA should be attached to the VA and the smaller one, distal end to the PCA. Finally, trapping of the dissected portion of the VA is performed [36]. Provided that PICA is

involved into dissection, trapping of the VA should be preceded by a VA–PCA anastomosis and revascularization of the PICA [36]. When clinically significant mass effect exists and the affected VA is dominant, revascularization along with decompression by aneurysmectomy or aneurysmotomy is demanded [7, 41, 43, 130, 161].

Neurophysiologic intraoperative monitoring

Since BTO cannot be applied to patients with severe SAH due to their deteriorated neurological condition [36], neurophysiologic intraoperative monitoring (NIOM) including electroencephalography (EEG) and evoked potentials are likely best used in combination and can be continuously monitored during the surgical procedure [84]. The final decision which type of NIOM will be used is determined by vascular territory at risk and the patient's baseline neurologic status [84, 113]. Brainstem auditory evoked potential monitoring detects functional changes along the auditory brain stem pathway, however, ischemia in the cerebellum or PCA territories could be missed [84]. On the other hand, EEG provides a more global assessment of cerebral ischemia but cannot be used to monitor the posterior fossa region. It may be used to detect ischemia in the PCA territory, but its sensitivity is limited [84]. Alternative procedures to BTO include also the use of xenon CT [92] or single-photon emission computed tomography, but they are not very practical [134]. However, when supplemented with cerebral blood flow studies, modern series of carotid sacrifice after normal BTO show false-negative rates of up to 25 %. Moreover, in case of SAH, the cerebrovascular insufficiency may develop in consequence to vasospasm which could impair otherwise sufficient collateralization [113].

Surgical reconstruction of VA

Sano et al. [126] separated VA dissecting aneurysms into unilateral and circumferential group depending on whether the dissection involves only one side of the arterial wall or its entire circumference. The patients with unilateral dissecting aneurysms underwent surgical reconstruction of the VA, whereas patients with circumferential lesions were treated with either proximal clipping or trapping. Of the six patients in whom direct surgical clipping was performed, four had excellent outcomes, one fair outcome, and one died from cardiac failure 12 days after the surgery.

Unilateral/bilateral VA occlusion in bilateral dissecting aneurysms

Since an unilateral VA occlusion of bilateral VB dissections or bilateral VA dissecting aneurysms may be followed by development of stenotic or occlusive dissection or enlargement of a contralateral aneurysm occurs, follow-up angiography is

necessary to assess the extent of aneurysm thrombosis, resolution of occlusive dissections, enlargement of contralateral dissecting aneurysms, and direction of flow in the BA. If recurrent or progressive aneurysm enlargement appears, prompt consideration of staged bilateral VA occlusion as definitive therapy is necessary [119, 172].

Wrapping

Even though results of wrapping techniques for saccular aneurysms are generally unsatisfactory [128, 142], Uhl et al. [155] reported on stabilization and elimination of further expansion and rebleeding of dissecting aneurysms following a wrapping of the entire lesion with a cuff resisting tensile stress during the 7 years follow-up period. The potential disadvantage of this method is that the clip might obliterate arterial branches [155] and damage the lower cranial nerves [73]. Well fitting cuffs formed from Dacron or PTFE (Gore-Tex™) strips are pulled through underneath the aneurysm. The ends are then approximated and secured with angled titanium aneurysm clips or malleable clips. Pulling the strip of material around the aneurysm is risky as it requires thorough dissection and removal of clot which may lead to rupture. To avoid this, a strip of PTFE may be first attached to one blade of a fenestrated angled aneurysm clip and then passed around the dissected artery [155]. Sliding a Sundt type encircling clip is certainly the wrapping method that imposes the smallest amount of mechanical stress onto the lower cranial nerves, but these clips are only useful if its size corresponds to the outer diameter of the dissected segment [155].

Surgical approaches

VA can be approached through suboccipital [135] or far lateral inferior suboccipital craniectomy [37] whereas the VB junction may be exposed using far lateral [38], transpetrosal [64], or combined far lateral-transpetrosal approach [38].

BA dissecting aneurysm

The BA IDAs pose difficult therapeutic problems and have even less favorable prognosis than IDAs arising from VA [24]. Furthermore, their rarity limits the acquisition of experience with management of these lesions. Surgical clipping of BA aneurysms has always been a complex procedure with high potential morbidity related to the deep location of the aneurysm and the proximity of critical perforators [65]. If an aneurysm poses a clippable neck, the most effective treatment is its direct obliteration [2, 130]. Unfortunately, in many cases, the aneurysm neck is extremely large and often filled with mural thrombus; therefore, an effective method of aneurysm exclusion from circulation is induction of its thrombosis [43].

Although the natural history of dissecting aneurysms is unique and differs from other vascular lesions, the ischemic effects of proximal ligation should be similar [2]. A standard treatment option for the unclippable giant VB aneurysms generally include endovascular [101] or surgical unilateral/bilateral VA occlusion either distally or proximally to the PICA, BA occlusion, or trapping [17, 38, 40, 43, 130, 161].

Trapping, wrapping, Hunterian ligating, and revascularization

Even though perforators arising from the wall of giant IDAs are thrombosed or nonfunctional [130] and of minimal importance [161], trapping should be avoided until the segment which should be isolated is very short and devoid of perforators [17]. Visualization of perforators behind the neck of a BA aneurysm is vital to safe clip application. However, often the size and projection of a BA aneurysm coupled with the operative approach through a deep surgical corridor preclude safe clipping [65]. While wrapping is associated with a persistent risk of aneurysmal rupture, proximal ligation/occlusion carries a risk of incomplete aneurysmal thrombosis and thromboembolic strokes [17, 18, 145]. Contrary to VA, in case of BA it may be dangerous, unless a sufficient collateral supply exist at least through 1 robust PComA [65].

Unilateral vs. bilateral VA occlusion

Unilateral ligation of a VA seems to be reasonable providing that the aneurysm fills principally or entirely through this VA and the other VA exists, unites with its fellow, and seems large enough to supply the BA. In case of VB aneurysms, a unilateral intracranial occlusion of a VA was performed in eight cases by Drake [17], with no morbidity and complete or nearly complete thrombosis in all but one aneurysm. Seven patients had excellent or fair results while one showed little recovery from an existing medullary syndrome. The best results were achieved when the artery was ligated intracranially just proximal to the aneurysm since an extracranial ligation of VA may be followed by development of an extensive collateral flow from deep cervical arteries with subsequent aneurysm enlargement [17]. If an aneurysm is filled from both VAs, their bilateral occlusion in the neck seems to be much less suitable with reported fair outcome only in about 30 % of patients [17]. Even bilateral PComA collateral flow from anterior to posterior circulation did not prevent development of an acute brain stem edema after bilateral VA occlusion [53].

BA occlusion

BA occlusion may be indicated as a fallback treatment if direct surgical or endovascular intervention is not considered safe due to a number of factors, including failure of previous

treatments, large size of the aneurysm, posterior projection of the dome, neck-to-dome ratio > 1, incorporation of major vessels in the neck, and position of the thalamoperforators. Patients with recurrences after neck clipping or coil embolization are also potential candidates for this therapeutic option. Moreover, patient must either demonstrate spontaneous filling of at least one PCA via carotid arterial beds and the preoperative angiogram or trial BTO of the BA should document filling of the BA apex via at least one PComA. A surgical clip can be precisely placed at a single point on the BA between perforators under direct observation and the occlusion is thus stable and nonthrombogenic [65]. Since BA occlusion followed by positive outcome ranges from 40 to 80 % [17, 18, 65], one should keep in mind that the major risk from BA ligation is brain stem ischemia, which may cause disaster in approximately one third of patients [18, 43]. Steinberg et al. [143] reported that only 11 % of the patients treated with lower BA occlusion for predominantly giant intracranial aneurysms developed irreversible ischemic neurological deficits, whereas upper BA occlusions carried a 14 % risk of irreversible ischemic complications, with an additional 8 % of patients unable to tolerate complete BA occlusion but showing reversible ischemia with release of the tourniquet. The presence of only small PComAs correlated significantly with an increased risk of developing brain stem ischemia after upper (P 0.004) or lower (P 0.036) BA occlusion; two small PComA represented the highest risk group. Kellner et al. [65] reported that 80 % patients after proximal clip occlusion of the BA experienced no new postoperative neurological deficits whereas the rest made excellent recoveries at long-term follow-up. No patient experienced postoperative hemorrhage, rebleeding, or delayed neurological deterioration [65].

Revascularization

In circumstances where occlusion is not tolerated, extracranial-intracranial unilateral or bilateral anastomosis may provide the collateral flow that will allow safe occlusion of the BA [17]. High flow ICA to BA bypass with a saphenous vein may provide the collateral flow that will allow safe occlusion of the BA; occasionally complemented by aneurysmectomy [130]. However, a low flow bypass, such as STA anastomosed to either the SCA or the PCA may bear a risk of a high incidence of early graft occlusion and inadequate flow provided to support the upper basilar circulation [145].

BTO and BA aneurysm

If BA fills spontaneously and well from carotid injection then BA occlusion will be probably tolerated. However, even the existence of well-developed PComA either at angiography or operation does not ensure sufficient blood supply to brain

stem after BA occlusion [17]. The existence of collateral circulation can be evaluated either by carotid artery compression called as Allcock's maneuver [18], by BTO as described above for VA during angiography, or intraoperatively when the PComAs could be visualized [65]. While BTO predicts outcome following ICA occlusion with accuracy [157] and neurological deterioration during BTO of the ICA would usually contraindicate parent vessel occlusion, this test has less value in the BA due to the possible perforators obstruction by endovascular balloon. In these situations, patients have to be examined for visual problems indicating ischemia in the PCA distribution and the BTO failure. Symptoms consistent with brainstem ischemia (such as arm or leg weakness, cranial nerve lesions, or loss of consciousness) would not contraindicate surgical BA occlusion, as long as anatomical evidence of filling of the top of the BA via the carotid circulation is angiographically documented. In the case of complex BA tip aneurysms, therefore, the BTO retains its negative predictive value while the positive predictive value of the test is decreased [65]. The use of a temporary BTO with monitoring of auditory and somatosensory evoked potentials may be useful to identify appropriate candidates for this approach [2]. The monitoring of somatosensory and motor evoked potentials is important during the surgical BA occlusion [65].

Surgical approach

There are many surgical approaches that enable access to VB system with their inherited disadvantages. A suboccipital and subtemporal transtentorial approach need significant brain retraction [17] whereas a transoral, transmaxillary, and transclival exposure suited for some midbasilar aneurysms offer a narrow surgical field with a higher risk of cerebrospinal fluid leakage and infection [6, 44, 140]. A combined subtemporal-suboccipital route is suited for visualization of the rostral-to-caudal extent of the brain stem requires a transection of the tentorium and occasionally the transverse and sigmoid sinuses as well. Even though partial or total petrosectomy enables reconstruction of difficult giant VB and midbasilar aneurysms with no brain retraction, a permanent hearing loss and potential damage of facial nerve are its inherited disadvantages [14, 129, 130]. The facial nerve can be mobilized with recovery of normal or nearly normal function provided that at least two of its feeding arteries were preserved [130]. The orbitozygomatic approach extended by removal of the anterior and posterior clinoid process and dorsum sellae, exposing the upper two fifths of the BA and far-lateral approach, exposing the lower two fifths of the BA [38, 78] can replace transpetrosal approaches [130]. They have lower morbidity rates than transpetrosal approaches [78]. If the exposure from these two approaches is insufficient because of the size or complexity of a particular aneurysm, then one of the transpetrosal approaches may be selected [78].

Either barbiturate-induced electroencephalic burst suppression and mild hypothermia [40] or deep hypothermic circulatory arrest [17, 78, 130] is critical to clipping large and giant BA aneurysms directly. If crucial dissection or debulking of thrombosed or atherosclerotic aneurysms is needed, adenosine (an endogenous purine nucleoside) may be administered intravenously with the aim to produce cardiac arrest for a few tens of seconds and subsequent profound hypotension for another tens of seconds. Administration of these therapeutic measures may be repeated if previous dose was without complication [33].

Peripheral cerebellar artery dissecting aneurysm

Isolated IDA confined to more peripheral cerebellar arteries have been observed only cursorily in the neurosurgical practice [31]. Majority of peripheral cerebellar artery dissecting aneurysms (PCADAs) arise from their proximal segment (88 %) and present with SAH (58 %) [31]. PICA is the most frequently affected vessel (65 %) followed by SCA (23 %) and AICA (12 %) [31]. Extremely quaint case in this region is represented by ruptured dissecting labyrinthine artery aneurysm successfully surgically treated by trapping without hearing loss or facial weakness [34].

Ischemic group

In patients with ischemic symptoms, some authors performed proximal occlusion of the parent artery [75, 95], trapping with bypass [99], resection and bypass [61], encasement [25], or circumferential wrapping [107] with good postoperative recovery. Other surgeons are inclined to treat the patients conservatively [159, 167].

Subarachnoid hemorrhage group

In patients with SAH, surgical occlusion of the parent artery by trapping [23, 120, 147] or entrapment [170] of the site of dissection, without [149, 163] or with [1, 77, 98, 107] bypass surgery, clipping [104, 163, 170], wrapping [57, 154], circumferential wrapping [107], or coil embolization [93, 123, 152] were applied with favorable results except in one patient who underwent coating of the dissected SCA [31, 93]. If circumferential wrapping with clipping reconstruction or trapping with distal revascularization are technically difficult, Nussbaum et al. [106] suggested a novel approach called remote distal outflow occlusion. The authors reported favorable outcomes and dramatic angiographic resolution of the aneurysmal dilation without ischemic injury or cerebellar edema following application of this method.

AICA

AICA dissecting aneurysms are divided into P group (pons) and C group (cerebellum). The pons group consists of pA (AICA anterior pontine segment), pL (lateral branch on the pons to the meatal loop), and pM (medial branch on the pons). Cerebellum group consists of m-loop (meatal loop), cL (lateral branch post meatal loop), and cM (medial branch on the cerebellum) [27]. Occlusion of the parent artery of AICA dissecting aneurysms pons group without revascularization may entail the risk of death. On the other hand, the parent artery of AICA IDAs cerebellum group may be occluded without severe consequences, but hearing loss and/or cerebellar infarction may be expected [27].

PICA

PICA occlusion distal to the telovelotonsillar segment generally does not result in brainstem injury and is well tolerated in majority of cases [1, 47, 59, 61, 82, 83, 106, 163]. Some patients have a very large PICA that also supplies the AICA territory [106]. When the perforators already are involved in the dissection, proximal occlusion will not cause brainstem infarction. However, if these perforators are still intact great care must be taken to preserve them during occlusion of the parent artery [31, 132].

SCA

The evaluation of cerebellar collateral blood flow is sometimes rather difficult. Using the BTO to assess occlusion of the small arteries of the posterior circulation has not been validated [77]. The more proximal the SCA segment, the more unpredictable the clinical repercussions can be after occlusion. More distal occlusions are more frequently associated with ischemic lesions, but this does not tend to cause clinical manifestations. Larger arteries with small adjacent ones, as well as the presence of a vasospasm, could indicate insufficient collateral blood circulation [77]. The safety of occlusion without revascularization in treatment of the SCA lesions remains unclear. Tohgi et al. [153] reported that after SCA infarcts, 61 % of cases resulted in independent outcomes, 26 %, in dependent outcomes, and 6 % of patients died. Consequences of ischemia in SCA territories were much less favorable than in PICA or AICA regions [153].

Surgical approach

PICA IDAs can be approached by a far-lateral suboccipital craniotomy with C1 laminectomy and partial condylar resection [107], AICA IDAs by lateral suboccipital retrosigmoid approach [27] or transpetrosal approach [64] and SCA IDAs by subtemporal [77] or combined subtemporal presigmoidal

approach [105]. Of the revascularization methods, the PICA-PICA, PICA-occipital artery [27, 107] or STA-SCA bypasses [77] were applied.

PCA dissecting aneurysm

Isolated dissection of the PCA is rare [9, 26, 63, 79, 80, 89, 133]. They are predominant in females, with a female–male ratio of 3.1:1 [26, 63]. Isolated PCA dissections tend to present with ischemic symptoms and have a more benign clinical course and prognosis [26, 63, 80, 89, 108]. They are most commonly located near the P1–P2 junction, adjacent to the free border of the tentorium cerebelli [79] suggesting shearing injury of the artery. However, dissections of P3–P4 [54, 79] or P2 segment [79, 133] have been observed as well. Majority of cases are treated conservatively [54, 63, 89, 133]. Anticoagulation, specifically heparin [79, 133] or antiplatelet therapy [133] is recommend for patients with IDAs presenting with stable ischemic symptoms. Patients with ruptured IDAs, repeated intramural hemorrhage, enlargement of an associated aneurysm, or progressive neurologic deficits typically require treatment by surgical or endovascular means. Following placement of the Guglielmi detachable coils (GDCs) proximal to the aneurysm had favorable outcome 67 % of patients [79, 127]. In the surgical group, the P2–3 [8] and P3 [127] segments were involved. One patient underwent a left frontotemporal craniotomy and the abnormal segment was clipped on both sides and resected. In this patient, a hemiparesis developed postoperatively, which remained stable [8]. In the second case, a proximal ligation via temporal craniotomy was followed by thrombosis of the aneurysm without resulting in infarction in the region of the PCA. The occlusion of distal PCA where perforators branch off can be indicated with less risk of infarction especially in cases in which collateral supply of the distal posterior cerebral artery is rich [127]. Of the three patients with SAH managed conservatively, two remained asymptomatic at 6 and 12 months, respectively [116], while the third had rebleeding at 8 months [108]. This suggests that despite demonstration of angiographic improvement in short-term follow-up, the inherent vessel wall abnormality persists, predisposing to recurrent hemorrhage [108].

Complications and their avoidance

Since VB IDAs pose specific anatomical proximities which increase the chance of branching vessels injury, perforators or cranial nerves during surgical procedure and indocyanine green video-angiography visualization is limited to the vessels directly observed under operating microscope [165], the application of the endoscope in deep

seated aneurysm may further increase the feasibility of the procedure to minimize the retraction of brainstem, vascular, and neural structures resulting in reduced morbidity [22, 115]. In patients with VA IDAs, the IX. and X. cranial nerves deficits were observed in 16 %, meanwhile XI., XII. cranial nerve paresis and sensory long tract lesions occurred in 5, 5, and 16 % of cases, respectively [171]. Yamaura et al. [169] found that such neurological deficits were commonly seen when the aneurysm was located within 10 mm of the midline or more than 13 mm posterior to the clivus. Thrombosis of the VA and its perforators to the medulla is known to play a more important role in the development of lateral medullary infarction than thrombosis of the PICA [21]. When the PICA is a major supplier of blood to the medulla, the PICA should remain patent proximal to the clip. When the VA is the main supplier to the lateral medullary region, clip occlusion should be performed proximal to the PICA origin so that the PICA will provide collateral perfusion to the distal segment of the VA. This procedure will prevent thrombosis of the distal VA and its perforators [168]. Therefore, the location at which a clip is applied should be carefully decided and its relation to the origin of PICA conditioning the blood supply to the medulla carefully considered.

Prognosis

The most predictive factor for surgical outcome in patients with aneurysms of the VB system is the size of the aneurysm. Sixteen percent morbidity and mortality rates have been reported for small aneurysms (<12 mm), 25 % for large aneurysms (12 to 25 mm), and 50 % for giant aneurysms (>25 mm) [18, 42, 66, 94, 145]. Probability of rebleeding and morbidity/mortality rates of IDAs of the VB circulation were reported as high as 71 and 50 %, respectively [4, 17, 18, 73, 94, 97, 145, 160]. Kawaguchi and associates [62] reported in their series of VA dissecting aneurysms 21 % rebleeding rate during the first 72 h with 100 % mortality. Yamaura et al. [171] reported 24 % of rebleeding in time period from third to 17th postictus day with 80 % mortality rate. Aoki and Sakai [4] reported 30 % rebleeding with 80 % occurring in the first few hours and Mizutani et al. [97] reported 71 % rebleeding, with the mortality 46.7 %, compared to a mortality of 8.3 % of patients, who bled only one time. Acute and chronic dissecting aneurysms of the basilar trunk are known to have a higher mortality rate if aneurysm ruptured (30 vs. 0 %, respectively) [69]. Overall outcome in the patients with BA IDAs was poor, with a mortality rate of 53 % within days to months; an additional 12 % experiences moderate to severe disability, and only 26 % made a fair recovery [3]. Kim et al. [69] reported that 100 % of conservatively managed patients with acute BA IDAs experienced rebleeding with the mortality 67 %, while the rest 33 %

were only moderately disabled. Interestingly, the chronic BA dissecting aneurysms, also known as chronic mural bleeding ectasias, are diagnosed incidentally in 64 %, as a cause of compressive syndrome or stroke in the remaining 36 % of cases [125]. Even though, these aneurysms have traditionally been considered to follow a benign course without clinical or imaging progression, they may increase in size over time, rupture, or cause a stroke in 25, 4, and 14 % of cases, respectively [125].

Conclusions

Surgical management of IDAs in posterior circulation is challenging because of difficult surgical access, frequent occurrence of a broad-neck, and the proximity of the perforating branches. Treatment decisions are often complicated by an unpredictable clinical course and controversies in treatment strategy. Nevertheless, the surgical treatment still plays a significant role in their treatment. A better understanding of their incidence, pathology, and natural history may help to improve their management strategy. Although in patients with ischemic symptoms, conservative therapy may be sufficient and effective, if recurrent or progressive ischemic neurological deficit or enlargement of the dissection occur despite appropriate medical therapy, the surgical treatment is justified. Since ruptured IDAs are unstable and their tendency to rebleed is associated with high mortality, the endovascular treatment is the first choice, however, a certain subset of IDAs are not amenable to endovascular approach and still require surgical strategy. The rebleeding occurs in an early period, particularly in the first 24 h, so the treatment should be performed as soon as possible. If the aneurysms pose a clippable neck, the most effective is direct clipping; in case it is unclippable, standard surgical treatment options include Hunterian ligation of VA or BA distally or proximally to the PICA, aneurysmal trapping or circumferential wrapping. Each procedure may be combined with revascularization. If circumferential wrapping with clipping reconstruction or trapping with distal revascularization is technically difficult and the neurosurgeon is confronted with peripheral PICA AIDAs, a remote distal outflow occlusion may be considered.

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