Chapter 15
Dissecting Pseudoaneurysms and Blister Aneurysms

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Introduction and Pathophysiology of Dissecting Pseudoaneurysm

In normal cerebral arteries, the strongest layer of the arterial wall is the internal elastic lamina (IEL), which consists mainly of elastic fibers [1]. However, once the IEL is disrupted, these fibers may never reconnect [2, 3]. Therefore, the injured arterial wall is repaired with other matrix components in a biological reaction. Disruptions of the IEL do not always develop into detectable arterial dissections and many disruptions may be repaired without clinical manifestation [4]. The underlying etiology of intracranial artery dissection remains uncertain, but two mechanisms have been proposed: (1) an actual tear in the intima, which allows blood from the lumen into the vessel wall, or (2) rupture of the vasa vasorum in the arterial wall itself, which leads to an intramural hematoma, leading to an accumulation of blood and a separation of arterial layers within the vessel. Both mechanisms can occur simultaneously.

The vessel injury may result in (1) stenosis (where thrombus in the false lumen partially compresses flow within the true lumen), (2) complete occlusion (if thrombus in the false lumen completely obstructs flow within the true lumen), (3) false aneurysm formation (where accumulation of blood is subadventitial) with subsequent hemodynamic and embolic infarctions, or lastly (4) rupture with subsequent subarachnoid hemorrhage (SAH) [5–7]. Of the above, spontaneous dissecting pseudoaneurysms, or pseudoaneurysms occurring after trivial trauma, are by far the most commonly encountered scenario. In a recently published systematic review [8] of medically managed pseudoaneurysms in patients with ICA dissection, there were 40 pseudoaneurysms (24%) following a traumatic ICA dissection, while 126 (76%) were classified as having occurred following "spontaneous" ICA dissection. Connective tissue diseases such as Ehlers Danlos or fibromuscular dysplasia "FMD" may be other predisposing factors.

Radiological Findings and Histopathological Correlation

In contrast to aortic dissecting aneurysms extending toward the carotid arteries, opacification of both the false and true lumen occurs very rarely [9]. Mitzutani et al. [10], proposed a classification system for non-atherosclerotic intracranial aneurysms (fusiform and dissecting aneurysms), based on data of treated 85 aneurysms as well as on the pathological patterns of IEL and the state of the intima (Table 15.1):

1. *Type 1, classic dissecting aneurysms*: The most typical angiographic feature ($n = 65$) was a fusiform aneurysm with an irregular wall. Many of the aneurysms had an irregular stenotic portion near the proximal or distal end. The commonly observed pathological features were widespread disruption of the IEL without intimal thickening and the presence of a pseudolumen.

Table 15.1 Angiographic types of dissecting aneurysms and clinical/pathologic features

Type	Name	Clinical symptoms	Pathological features
Type 1	Classical dissecting aneurysm	Rupture Ischemia	Widespread disruption of IEL without intimal thickening
Type 2	Segment ectasia	Asymptomatic	Stretched/fragmented IEL with intimal thickening No luminal thrombus
Type 3	Dolichoectatic dissecting aneurysm	Symptomatic; progressive course	Fragmented IEL and multiple dissections of thickened intima Luminal thrombus
Type 4	Saccular aneurysm, arising from arterial trunk	Rupture	Minimal disruption of IEL without intimal thickening

IEL internal elastic lamina

2. *Type 2, segmental ectasia:* Angiographic findings (n = 8) showed a fusiform aneurysm with a smooth contour, usually larger in size than the Type 1 aneurysms. There was no evidence of luminal thrombus. Clinical and radiological follow-up showed no significant progression. Postmortem pathological examination revealed a stretched or fragmented IEL and a moderately thickened intima.
3. *Type 3, dolichoectatic dissecting aneurysms:* All of the aneurysms were located on the basilar artery and were conservatively followed up for 1–5 years. The most typical angiographic feature (n = 8) was torturous fusiform appearance with irregular contrast caused by organized laminar thrombus. Fragmentation of the IEL combined with multiple dissection of thickened intima, suggesting a chronic response to hemodynamic stress.
4. *Type 4, saccular aneurysms:* All cases (n = 4) manifested with SAH. On postmortem examination, one aneurysm lacked IEL in its dome.

Clinical Impact of Dissecting Pseudoaneurysm

The natural history of dissecting pseudoaneurysms is different from the more common "saccular" or "berry" aneurysms. The most important secondary feature of a dissecting pseudoaneurysm is its rupture status. While dissecting pseudoaneurysms without SAH tend to follow a benign course, cases presenting with SAH carry a high risk of rebleed. Some authors report a high rebleeding risk (between 24% and 57.1%) in the acute phase within 1 week after the initial SAH associated with a high mortality rate [11, 12]. Clinical data suggest that the rate of rebleeding of ruptured dissecting aneurysms decreases 1 week after SAH [2, 13]. Mitzutani et al., reported though nearly a 10% rebleeding rate after 1 month [12]. While the rupture of a dissecting aneurysm is occasionally the presenting event, formation of "pseudoaneurysm" or "dissecting aneurysm" is more commonly a delayed manifestation. Distal ischemic

strokes may also occur with pseudoaneurysms, and imaging studies suggest that more than 90% of infarcts due to dissection are thromboembolic rather than hemodynamic in origin [14, 15]. Other symptoms include headache, neck pain, or loss of consciousness, especially in the posterior circulation (17, 34). In a recent systematic review on the natural history of (mostly distal) ICA dissecting pseudoaneurysms [8], only 3% (5/166) of the pseudoaneurysms increased in size during follow-up, 52% (86/166) remained unchanged in size, 21% (35/166) decreased in size, while 19% (32/166) resolved completely. Only 2% of cases (4 patients) with a conservatively managed pseudoaneurysm after ICA dissection developed new neurological symptoms during follow-up. All four followed traumatic ICA dissection. No pseudoaneurysms that arose after spontaneous ICA dissection developed symptoms.

General Treatment Paradigms for Dissecting Pseudoaneurysms

Conservative and medical treatment is the most common initial management for asymptomatic and unruptured dissecting pseudoaneurysms. In a recent large series of 120 cases of ICA/vertebral artery (VA) dissecting pseudoaneurysms [16], all patients were placed on a regimen of antithrombotic treatment. Antiplatelet treatment was used in 59% of patients (Aspirin and/or Plavix), heparin and warfarin in 26.8%, and combined antiplatelet and anticoagulation agents in 14.3% of patients. Antithrombotic treatment strategies were distributed similarly among patients with or without history of trauma, extracranial or intracranial pseudoaneurysms, and carotid or vertebral artery (VA) involvement. Yet, antithrombotic therapy to prevent further ischemic events is contraindicated in some instances, and there is considerable uncertainty regarding the optimal management of asymptomatic pseudoaneurysms, particularly if they do not increase in size. Indications for endovascular or surgical management include ruptured, symptomatic, or large-size (>10 mm) pseudoaneurysm [17]. Approaches to endovascular treatment of dissecting aneurysms of the intracranial vessels can be divided into deconstructive (involving occlusion or sacrifice of the parent artery) and reconstructive (preserving blood flow through the parent vessel). Deconstructive endovascular techniques include proximal occlusion of the parent artery with detachable coils and/or balloons and occlusion of the dissected segment of the vessel with coils and/or balloons. Such procedures alone can be sufficient if important branch vessels are not originating from the vessel to be occluded, and collateral blood flow to the remainder of the peripheral circulation is adequate.

In contrast, reconstructive techniques (including stenting, coiling, flow diversion, or a combination) preserve the parent vessel. Surgical options include vessel ligation, clipping (Sundt clip and wrapping), reconstruction with interposition graft, and distal bypass. It should be noted that bland coiling of pseudoaneurysms may have a much higher post coiling growth and recurrence rate, as the aneurysmal sac may consist of adventitia only.

Specific Disease States

Intracranial Dissecting Pseudoaneurysms

Posterior Circulation Dissecting Aneurysms

Dissecting aneurysms have been reported in up to 28% of aneurysms of the intracranial VA, including the PICA [11, 18]. Most of the incidentally detected lesions occurred silently or with minor headache. Flemming et al. [19] reported that the annual prospective risk of hemorrhage from a vertebrobasilar artery non-saccular intracranial aneurysm is 0.9% and that an aneurysm diameter of at least 10 mm is strongly indicative of future rupture. In another series [20], aneurysms of <10 mm had a favorable clinical outcome, but aneurysms of >10 mm with symptoms due to mass effect had a risk of clinical deterioration and enlargement. Therefore, observation with serial radiologic examinations may be the management strategy of choice on patients with asymptomatic unruptured dissecting vertebrobasilar aneurysms (Fig. 15.1).

Medical management of patients with dissecting vertebrobasilar aneurysms who are asymptomatic or present only with pain remains controversial and is not well established. In one series [20], none of the patients (0/56) who presented with pain only at diagnosis had a hemorrhagic or ischemic stroke during observation (no antiplatelet or anticoagulation therapy were used), raising doubt about the necessity for anticoagulation or antiplatelet therapy for patients presenting with pain only. Kim et al. [21] reported a series of unruptured intracranial vertebrobasilar artery dissections (191 patients), including ischemic (n = 110) and nonischemic symptoms (n = 81), where 24.1% received endovascular treatment and 75.9% received medical therapy, with anticoagulation (n = 49), antiplatelet therapy (n = 48), or analgesics (n = 48). Of the medically treated group, 83 patients had aneurysmal dilatation that spontaneously resolved with normal luminal caliber in 9.6% (8/83), stable shape and size of aneurysm in 84.3% (70/83), and progressive enlargement of the dissecting pseudoaneurysm was noted in 6.1% (5 cases). Of these five patients, four were asymptomatic and one patient had brainstem compression symptoms from enlarging basilar dissecting aneurysm.

Proximal artery occlusion entails coil embolization of the non-diseased VA segment proximal to the dissection, which induces distal flow reversal and potentially promotes thrombosis of the aneurysm (Fig. 15.2). However, this technique does not immediately secure the aneurysm. Reports on 196 dissecting VA aneurysms treated with endovascular trapping or sacrifice have shown a re-hemorrhage rate of 3.1% [21–23]. The primary disadvantage of endovascular trapping though is the risk of ischemic stroke when the aneurysm involves a dominant VA [24]. In one report, incidence of ischemic stroke after endovascular trapping of dissecting VA aneurysms was reported at 8% overall and 38% when the aneurysm involved the PICA origin [22].

Recently, flow diversion devices have emerged as an alternative endovascular treatment of ruptured VA dissecting aneurysms [25–28]. Flow diverters preserve flow through the parent VA and branches, but evidence at this point for the efficacy of this

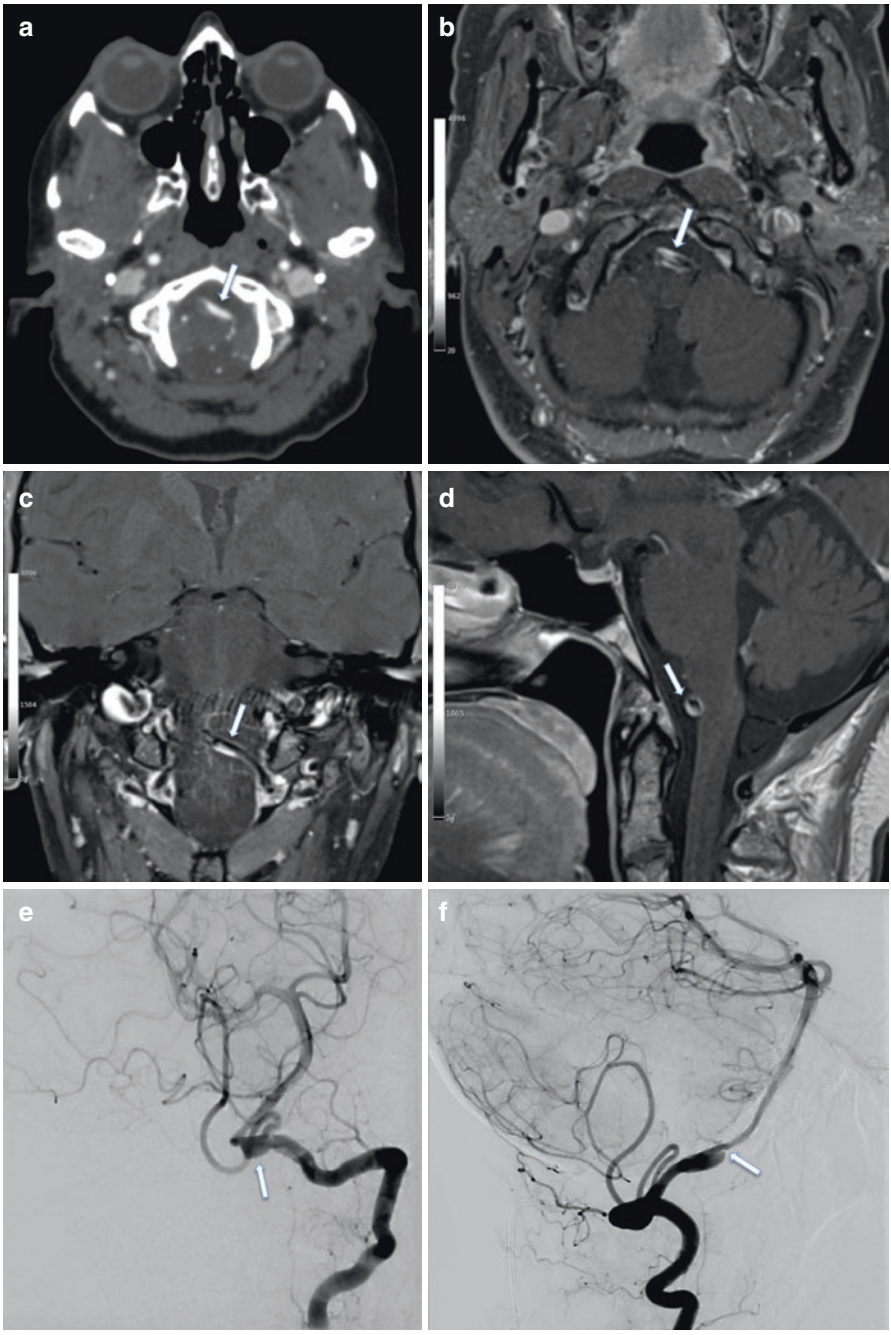

Fig. 15.1 A patient with an incidental left V4 segment fusiform dissecting aneurysm (arrow in **a**). On post-contrast T1-weighted sequence, the mural hematoma (arrow in **b**–**d**) can be appreciated. The digital subtraction angiography (DSA) with a left vertebral artery injection shows clearly the vessel dilatation (arrow in **e**) and the dissection flap (arrow in **f**). A decision was made treat conservatively

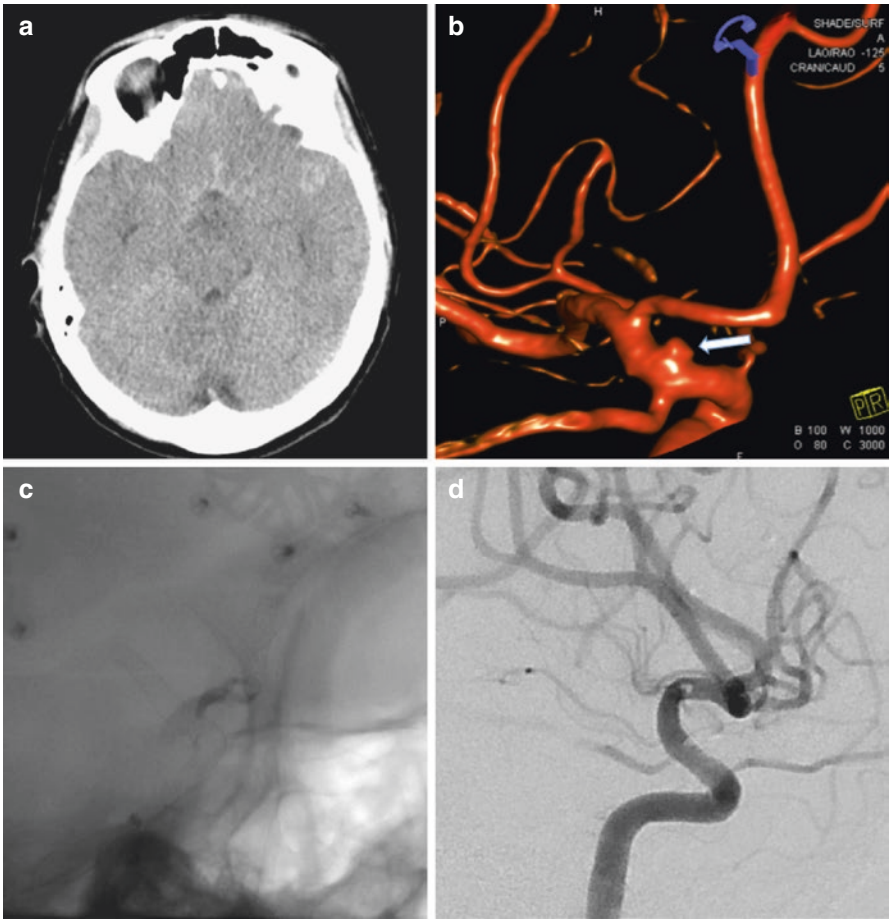

Fig. 15.2 A 56-year-old female with subarachnoid hemorrhage on non-contrast CT (**a**) due to ruptured blister-like aneurysm of the left ICA as seen on the 3D reconstruction of the rotational angiography (arrow in **b**). Clipping was attempted but was unsuccessful in occluding the aneurysm. Pipeline embolization device (PED) was placed in left ICA (**c**, notice the surgical clip). Eight-week follow-up demonstrates complete aneurysm occlusion (**d**)

device in preventing re-hemorrhage is limited to case reports [25, 26]. Flow diversion in basilar dissection is recommended in fetal PCA over sacrifice of parent vessel due to lack of filling through posterior communicating artery (PCOM) to pons. The optimal number of flow diverters necessary to treat such a dissection is unknown. Although overlapping flow diverters have incremental effect on flow diversion, there is an increased risk of perforator occlusion, especially in the perforator rich basilar artery. A relatively new MRI technique, vessel wall imaging [29], might play a role in assessing the vessel wall, especially in giant aneurysms before and after flow-diverter treatment.

Possible complications of flow diversion include device migration, immediate posttreatment aneurysm rupture due to mechanical stretch [30], and hemorrhagic

conversion of ischemic stroke. In patients presenting with SAH, hemorrhagic complications are exacerbated by post-procedural dual antiplatelet therapy.

Surgical treatment options include proximal clipping, trapping, wrapping, or resection with end-to-end anastomosis or bypass (e.g., PICA-to-occipital artery anastomosis or PICA reimplantation to the VA). A recent meta-analysis comparing the clinical outcome of patients with VA dissecting aneurysms treated with proximal occlusion and endovascular trapping found that proximal occlusion was associated with a larger proportion of poor outcomes and mortality ($p = 0.0403$) [31]. There are few case reports in literature about the use of Sundt clip and encircling aneurysm clips in dissecting aneurysm, but these methods are viewed as a last resort in exceptional cases where endovascular treatment is not available [32, 33].

Anterior Circulation Dissecting Pseudoaneurysms

Dissecting aneurysms involving the anterior cerebral artery (ACA) may be classified into three types [34–36]: type I, an extension of the ICA dissection to ACA (occur mostly in young adults and usually present with cerebral infarction); type II, dissection at the A1 segment (usually occurs in young women and often causes SAH); and type III, dissection from the A2 to A4 segment (predominantly in middle-age patients and mostly causes infarctions). Dissecting pseudoaneurysms of the middle cerebral artery (MCA) can also be classified into three types [36]: *type A*, originates from the M1 segment, which poses a treatment challenge in order to preserve the lenticulostriate perforators; *type B*, originates from the M2 segment or MCA bifurcation; and *type C*, originates from the M3 or distal segments. Ischemia has been proposed as the most common symptom in anterior circulation dissections [37], though some studies have observed a comparable occurrence of ischemia and SAH in the case of ACA dissecting pseudoaneurysms [35]. Some authors have suggested that the closer the occlusion is to the end organ, the more likely that ischemia will develop [38].

Dissecting aneurysms revealed by subarachnoid hemorrhage have been effectively treated conservatively with a good outcome and a low rate of rebleeding [39]. Earlier series [40, 41] have reported treatment with stents (single or multiple), presupposing that stents effectively tack down the torn vessel, resulting in aneurysm occlusion and preventing regrowth. This method, which can preserve the parent artery, may be an alternative to parent artery occlusion, especially for patients with high risks of complications after parent artery occlusion. However, there are some limitations, notably the high porosity of the standard stent, which may result in incomplete obliteration of the pseudoaneurysm. Treatment of anterior circulation dissecting pseudoaneurysms with a combination of stents and coils has recently been described in a small number of patients [42–44]. Byoun HS et al. [45] reported a small series of intracranial ICA dissecting pseudoaneurysms ($n = 6$) treated with stent-assisted coiling. There was only one intraoperative aneurysm rupture. Five patients had complete occlusion on follow-up, and good clinical outcome (mRS score 0–1) was achieved in all patients. While preservation of the cervical arteries to

the brain is the ideal goal to reach, there will still be a significant number of cases in which endovascular trapping of the carotid is considered the safest treatment option.

Surgical management is recommended if there is a high risk of rebleeding (rebleeding under conservative treatment, growing aneurysm, giant aneurysm, or uncontrolled hypertension). In a recent meta-analysis [8] of distal ICA dissecting aneurysm, only 9 out of 166 cases (5%) required surgical treatment; 5 underwent resection and interposition bypass, 3 were treated by carotid ligation, and 1 patient underwent extracranial to intracranial bypass.

Extracranial Dissections and Pseudoaneurysms

The estimated annual incidence of cervical artery dissection is 2.6 to 5 per 100,000 [46]; however this is probably an underestimation, as dissections may be asymptomatic. Approximately 1–2% of all ischemic strokes are attributed to cervical artery dissections but are responsible of up to 25% of strokes in young patients [46, 47]. The reported rate of pseudoaneurysm development following carotid and VA dissection varies widely between 5% and 40% [48, 49]. A large number of these pseudoaneurysms are detected at a later time point after the dissection, emphasizing the importance of follow-up imaging in dissection. After imaging documentation of extracranial dissecting pseudoaneurysm, the majority of these cases are reported to either remain stable or decrease in size [48–50], while others have reported an increase in pseudoaneurysm size on follow-up [16]. Rupture of these pseudoaneurysms occurs rarely, and the rate of ischemic complications of dissecting ICA pseudoaneurysms is low [16, 50]. In a large series of extra- and intracranial artery dissection (370 patients), 30.3% developed a dissecting pseudoaneurysm, 81.7% of which were extracranial (53.4%were located at C1; cervical division, 20.8% C2; petrous division, 10.8% at V2 segment and 6.7% at V3 segment and 5.8% at V4 segment) [16].

The most commonly utilized treatment in these cases is medical therapy. The 2014 American Heart Association/American Stroke Association Guidelines recommend an initially conservative treatment strategy in patients with ICA dissection [51], but there is no specific recommendation for ICA dissection pseudoaneurysms. Indication for either endovascular or surgical treatment would include enlargement on follow-up, thromboembolic ischemia and compressive symptoms from large aneurysms. In a recent report [52] on endovascular management of cervical dissections in 116 patients (93 had ICA and 23 had VA dissection), stent placement was used in 90% (n = 104) and coil occlusion (Fig. 15.3) of parent artery in 9.2% (n = 11). Stroke rate was 0.9% and mortality 3.4%, indicating that endovascular treatment is an effective treatment in specific indications. Surgical treatment is reserved for the management of symptomatic patients with lesions in accessible locations and often traumatic dissections (either blunt or penetrating); however, surgical artery preserving techniques are time-consuming compared to endovascular treatment [53].

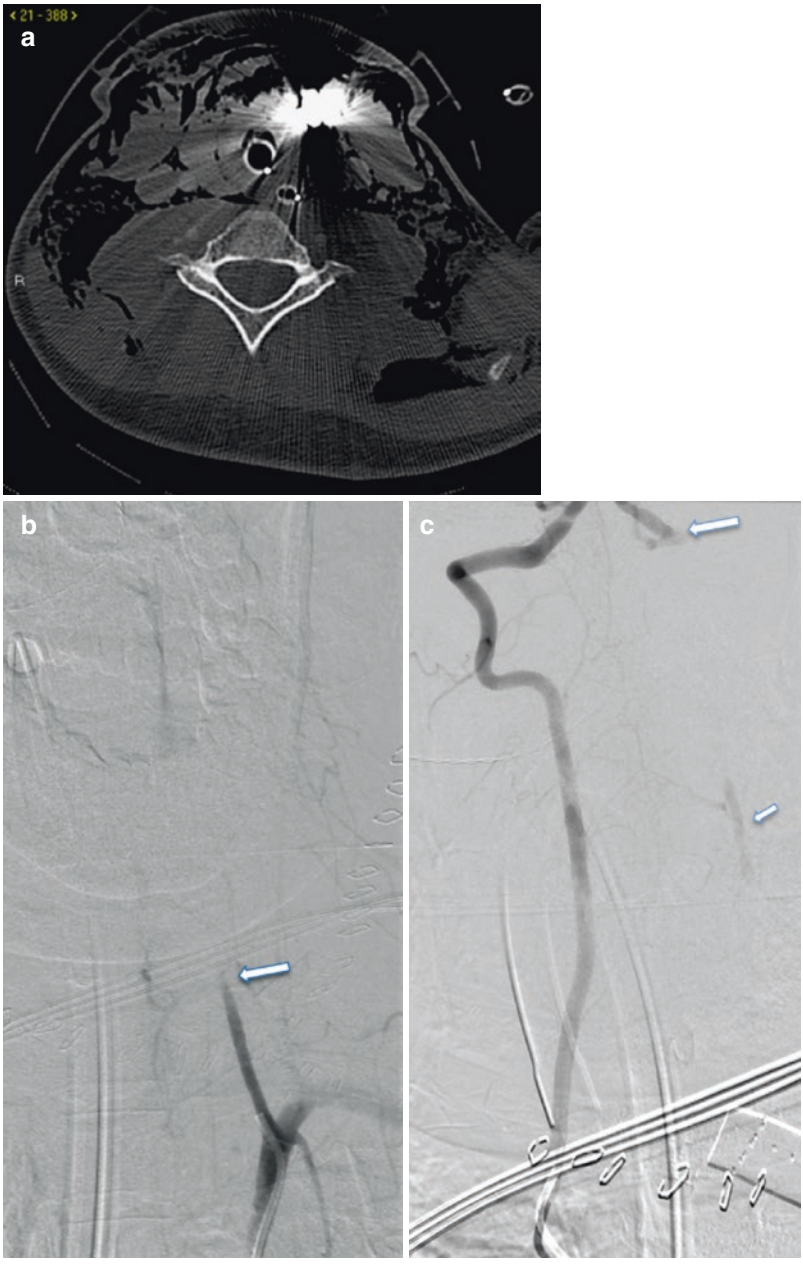

Fig. 15.3 A 23-year-old female with status post gunshot wound to the neck (**a**) presents with surgically uncontrollable bleeding from a left vertebral artery (VA) injury. On digital subtraction angiography (DSA), a selective left (**b**) and right (**c**) VA injection shows a tapered occlusion of left V1–V3 segments (long arrow in **b**–**c**) with partial reconstitution of V2 segment (short arrow in **c**) through the right VA. First coil trapping of the left V1 segment was performed (**d**), followed by coiling of the left V4 segment through catheterization of the right VA and basilar arteries (**e**–**f**). Successful control of bleeding was reached

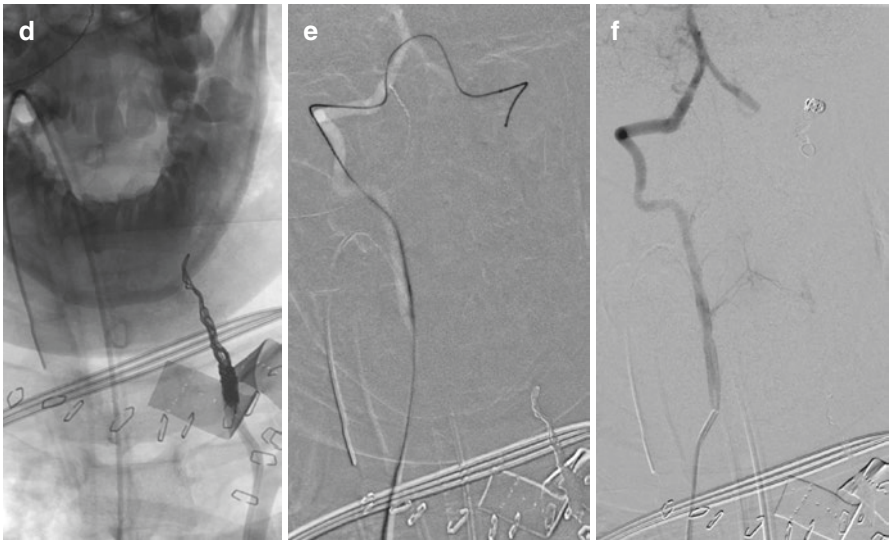

Fig. 15.3 (continued)

Blister Aneurysms

Blister "aneurysms" are rare, fragile, thin-walled, and often broad necked, arising at non-branching sites, typically discovered after rupture, and it is believed they represent a subadventitial dissection with a focal vessel wall defect due to disrupted internal elastic lamina and tunica media, with a thin residual wall consisting only of adventitia and fibrous tissue, and thus not a "true" aneurysm.

Blister "aneurysms" are usually found in the anterior circulation, along the dorsal aspect of the ICA in either the paraophthalmic or paraclinoid [54, 55] regions. Although blister-like aneurysms represents only 0.3–1% of all intracranial aneurysms, they account for about 0.9–6.5% of aneurysmal subarachnoid hemorrhage. Ruptured blister pseudoaneurysms are associated with a high mortality rate and spontaneous or treatment-related rebleed regardless of the treatment type [56–59].

Endovascular treatment of blister-like aneurysms involves stenting, coiling, trapping, and more recently flow diversion (Figs. 15.4 and 15.5). A recent meta-analysis [59] of endovascular treatment of ruptured blister-like aneurysms (265 procedures and mean dome size 2.4 mm) showed that the most common treatment option reported was stent-assisted coiling (44.2%), followed by flow diversion (25.8%) and stenting alone (18.8%). Other less frequently employed endovascular treatment options were deconstructive treatment (9.4%), coiling, with or without balloon assistance (6.3%), combined treatment (3.8%), and Onyx plus stenting (1.3%). As expected, endovascular deconstructive treatment carries a higher chance of immediate complete occlusion of the aneurysm than reconstructive treatment (77.3% vs.

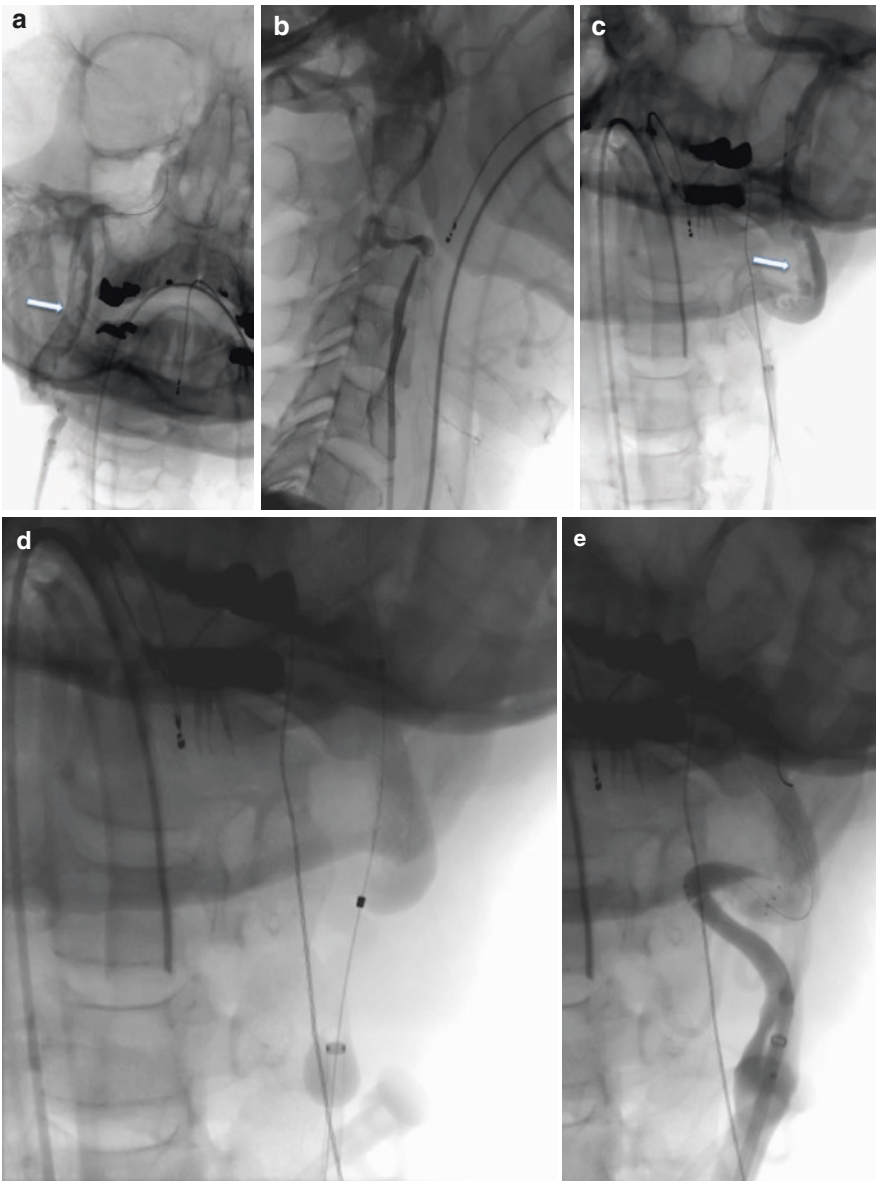

Fig. 15.4 A patient with symptomatic cervical internal carotid artery (ICA) fusiform dissecting aneurysms (right anteroposterior; AP in **a** and left lateral; **b** and AP; **c**) with irregular filling defect on digital subtraction angiography (DSA) representing clot formation (arrow in **a**, **c**). A double stent construct was used to oppose the clot and restore the lumen diameter of the left cervical ICA (**d**–**e**). The right cervical ICA dissecting aneurysm was managed conservatively

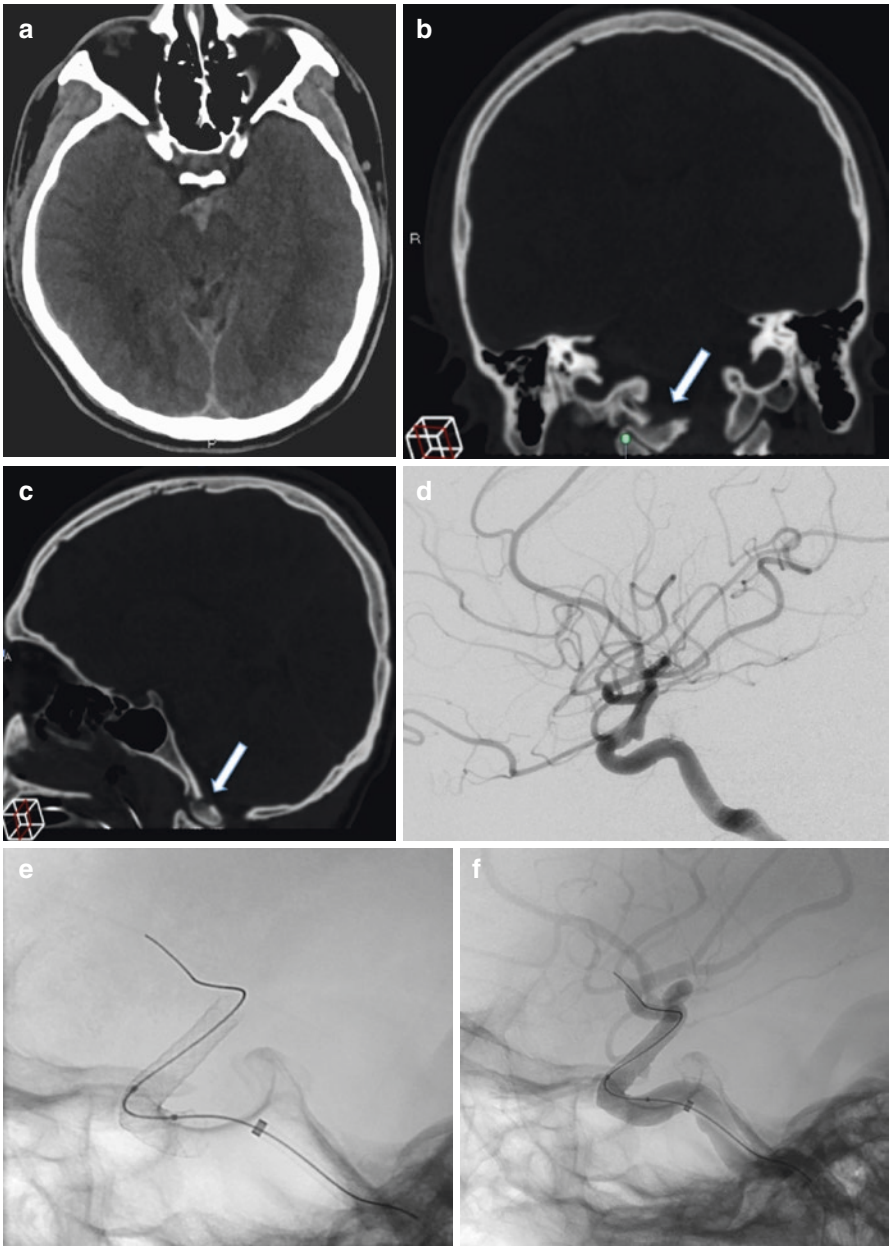

Fig. 15.5 A 21-year-old patient with motor vehicle accident and severe head trauma. (**a**) Non-contrast head CT showing subarachnoid hemorrhage, especially perimesencephalic and along the tentorium. (**b–c**) Head CT in bone window, reveling a dislocated right occipital condyle fracture extending into the clivus (arrow). (**d**) Diagnostic cerebral angiography shows a traumatic blister aneurysm of the communicating segment of the right ICA. (**e–f**) A week later, second angiography verified the persistence of the aneurysm with no dissection flap visible. After deployment of two flow diversion devices across the blister aneurysm (**e**), flow reduction into the aneurysm can be observed (**f**)

33%, $p = 0.0003$), yet it was associated with higher perioperative stroke (29.1% vs. 5%, $p = 0.04$). At follow-up there was no statistical difference in mid- to long-term aneurysm occlusion and retreatment or good neurological outcome. Stenting with or without coiling has a reportedly good outcome (usually mRS score 0–2) between 66% and 100% with mortality rate of 0–30%, while the rate of rebleed and retreatment ranged between 8% and 50% [59].

Recently, flow diversion has been described as a treatment option for blister "aneurysms," as it decreases the manipulation of the fragile wall of the blister-like aneurysm and preserves the parent vessel, reducing the rate of iatrogenic aneurysm rupture and strokes, respectively. In their meta-analysis, Rouchaud et al. [59] reported that treatment with flow diversion (mostly pipeline embolization device; Covidien) was associated a higher rate of mid- to long-term occlusion comparing to other endovascular reconstructive methods (90.8% vs. 69.7%, $p = 0.005$) and consequently lower retreatment rate. There was no statistically significant difference in regard to perioperative strokes, ICH, initial occlusion, or good clinical outcome. Disadvantages of flow diversion include obligatory dual antiplatelet therapy, possible perforator compromise, delayed aneurysm rupture (due to persistent flow into the aneurysm), in-stent thrombosis, and delayed parenchymal hemorrhage [54, 55, 59]. In regard to endovascular deconstructive treatment, it offers a higher initial occlusion rates in comparison with endovascular reconstructive methods, yet with associated higher rate of perioperative stroke [59]. Surgical treatment options for blister pseudoaneurysms include direct clipping, clip reinforced wrapping, and trapping, with or without bypass. It should be noted that because the blister is essentially adventitia only, clipping typically requires approximation of vessel that still contain media and intima and may result in some degree of luminal compromise. Recent reports on surgical treatment options had a range of good clinical outcome (mRS score 0–2) between 59% and 100% and an intraoperative rupture of 0–41% [60–62].

Conclusion

- Pseudoaneurysms differ from "true" aneurysms in that they do not contain the full complement of mural layers.
- Management of intracranial hemorrhagic pseudoaneurysms requires more aggressive management due to high re-rupture and aneurysm growth risk.
- Extracranial, asymptomatic pseudoaneurysms may be managed conservatively with medical therapy consisting of antiplatelet or anticoagulative therapies.
- Unruptured intracranial dissections, such as in the V4 segment, are often managed conservatively with antiplatelet agents.
- Long-term follow-up of dissections is needed to recognize the usually delayed development of dissecting pseudoaneurysms.
- Endovascular or open surgical treatment options include reconstructive and deconstructive methods and should be selected with attention to preserving adequate perfusion to the end organ while reducing risk of rupture, by reducing or

eliminating flow stress on the lesion. Treatment approach must then be carefully tailored based on individual patient vascular anatomy.
– Blister "pseudoaneurysms" are rare, fragile high-risk lesions. Endovascular options, especially flow diversion therapy, may offer higher occlusion and lower retreatment rate.

References

1. E. GL. Medial defects in the circle of willis and their relation to aneurysm formation. J Pathol Bacteriol. 1940;51(2):169–316.
2. Mitchell GM, McCann JJ, Rogers IW, Hickey MJ, Morrison WA, O'Brien BM. A morphological study of the long-term repair process in experimentally stretched but unruptured arteries and veins. Br J Plast Surg. 1996;49(1):34–40.
3. W.E. S. Pathology of the Cerebral Blood Vessels. St. Louis: C.V. Mosby; 1972.
4. Mizutani T, Kojima H, Asamoto S. Healing process for cerebral dissecting aneurysms presenting with subarachnoid hemorrhage. Neurosurgery. 2004;54(2):342–7. discussion 347–348.
5. Schievink WI. Spontaneous dissection of the carotid and vertebral arteries. N Engl J Med. 2001;344(12):898–906.
6. Fusco MR, Harrigan MR. Cerebrovascular dissections – a review part I: spontaneous dissections. Neurosurgery. 2011;68(1):242–57. discussion 257.
7. Fusco MR, Harrigan MR. Cerebrovascular dissections: a review. Part II: blunt cerebrovascular injury. Neurosurgery. 2011;68(2):517–30. discussion 530.
8. Paraskevas KI, Batchelder AJ, Naylor AR. Fate of distal false aneurysms complicating internal carotid artery dissection: a systematic review. Eur J Vasc Endovasc Surg. 2016;52(3):281–6.
9. Lasjaunias P, Ter Brugge KG, Berenstein A. Surgical neuroangiography, vol. 2.1/2.2. Switzerland: Springer; 2004.
10. Mizutani T, Miki Y, Kojima H, Suzuki H. Proposed classification of nonatherosclerotic cerebral fusiform and dissecting aneurysms. Neurosurgery. 1999;45(2):253–9. discussion 259–260.
11. Yamaura I, Tani E, Yokota M, Nakano A, Fukami M, Kaba K, Matsumoto T. Endovascular treatment of ruptured dissecting aneurysms aimed at occlusion of the dissected site by using Guglielmi detachable coils. J Neurosurg. 1999;90(5):853–6.
12. Mizutani T, Aruga T, Kirino T, Miki Y, Saito I, Tsuchida T. Recurrent subarachnoid hemorrhage from untreated ruptured vertebrobasilar dissecting aneurysms. Neurosurgery. 1995;36(5):905–11. discussion 912-903.
13. Poole JC, Cromwell SB, Benditt EP. Behavior of smooth muscle cells and formation of extracellular structures in the reaction of arterial walls to injury. Am J Pathol. 1971;62(3):391–414.
14. Droste DW, Junker K, Stögbauer F, Lowens S, Besselmann M, Braun B, Ringelstein EB. Clinically silent circulating microemboli in 20 patients with carotid or vertebral artery dissection. Cerebrovasc Dis. 2001;12(3):181–5.
15. Srinivasan J, Newell DW, Sturzenegger M, Mayberg MR, Winn HR. Transcranial Doppler in the evaluation of internal carotid artery dissection. Stroke. 1996;27(7):1226–30.
16. Daou B, Hammer C, Chalouhi N, Starke RM, Jabbour P, Rosenwasser RH, Tjoumakaris S. Dissecting pseudoaneurysms: predictors of symptom occurrence, enlargement, clinical outcome, and treatment. J Neurosurg. 2016;125(4):936–42.
17. Mokri B, Piepgras DG, Houser OW. Traumatic dissections of the extracranial internal carotid artery. J Neurosurg. 1988;68(2):189–97.
18. Yamaura A. Diagnosis and treatment of vertebral aneurysms. J Neurosurg. 1988;69(3):345–9.
19. Flemming KD, Wiebers DO, Brown RD, Link MJ, Nakatomi H, Huston J, McClelland R, Christianson TJ. Prospective risk of hemorrhage in patients with vertebrobasilar nonsaccular intracranial aneurysm. J Neurosurg. 2004;101(1):82–7.

20. Kobayashi N, Murayama Y, Yuki I, Ishibashi T, Ebara M, Arakawa H, Irie K, Takao H, Kajiwara I, Nishimura K, et al. Natural course of dissecting vertebrobasilar artery aneurysms without stroke. AJNR Am J Neuroradiol. 2014;35(7):1371–5.
21. Kim BM, Kim SH, Kim DI, Shin YS, Suh SH, Kim DJ, Park SI, Park KY, Ahn SS. Outcomes and prognostic factors of intracranial unruptured vertebrobasilar artery dissection. Neurology. 2011;76(20):1735–41.
22. Madaelil TP, Wallace AN, Chatterjee AN, Zipfel GJ, Dacey RG, Cross DT, Moran CJ, Derdeyn CP. Endovascular parent vessel sacrifice in ruptured dissecting vertebral and posterior inferior cerebellar artery aneurysms: clinical outcomes and review of the literature. J Neurointerv Surg. 2016;8(8):796–801.
23. Lv X, Jiang C, Li Y, Wu Z. Clinical outcomes of ruptured and unruptured vertebral artery-posterior inferior cerebellar artery complex dissecting aneurysms after endovascular embolization. AJNR Am J Neuroradiol. 2010;31(7):1232–5.
24. Sugiu K, Tokunaga K, Watanabe K, Sasahara W, Ono S, Tamiya T, Date I. Emergent endovascular treatment of ruptured vertebral artery dissecting aneurysms. Neuroradiology. 2005;47(2):158–64.
25. Ducruet AF, Crowley RW, Albuquerque FC, McDougall CG. Reconstructive endovascular treatment of a ruptured vertebral artery dissecting aneurysm using the Pipeline embolization device. J Neurointerv Surg. 2013;5(4):e20.
26. Narata AP, Yilmaz H, Schaller K, Lovblad KO, Pereira VM. Flow-diverting stent for ruptured intracranial dissecting aneurysm of vertebral artery. Neurosurgery. 2012;70(4):982–8. discussion 988–989.
27. McTaggart RA, Santarelli JG, Marcellus ML, Steinberg GK, Dodd RL, Do HM, Marks MP. Delayed retraction of the pipeline embolization device and corking failure: pitfalls of pipeline embolization device placement in the setting of a ruptured aneurysm. *Neurosurgery*. 2013;**72**(2 Suppl Operative):onsE245–50. discussion onsE250-241.
28. Munich SA, Tan LA, Keigher KM, Chen M, Moftakhar R, Lopes DK. The pipeline embolization device for the treatment of posterior circulation fusiform aneurysms: lessons learned at a single institution. J Neurosurg. 2014;121(5):1077–84.
29. Mandell DM, Mossa-Basha M, Qiao Y, Hess CP, Hui F, Matouk C, Johnson MH, Daemen MJ, Vossough A, Edjlali M, et al. Intracranial vessel wall MRI: Principles and expert consensus recommendations of the American Society of Neuroradiology. AJNR Am J Neuroradiol. 2017;38(2):218–29. doi:10.3174/ajnr.A4893. Epub 2016 Jul 28.
30. Fox B, Humphries WE, Doss VT, Hoit D, Elijovich L, Arthur AS. Rupture of giant vertebrobasilar aneurysm following flow diversion: mechanical stretch as a potential mechanism for early aneurysm rupture. BMJ Case Rep. 2014;2014
31. Hernández-Durán S, Ogilvy CS. Clinical outcomes of patients with vertebral artery dissection treated endovascularly: a meta-analysis. Neurosurg Rev. 2014;37(4):569–77.
32. Park PJ, Meyer FB. The Sundt clip graft. Neurosurgery. 2010;66(6 Suppl Operative):300–5. discussion 305.
33. Jafar JJ, Kamiryo T, Chiles BW, Nelson PK. A dissecting aneurysm of the posteroinferior cerebellar artery: case report. Neurosurgery. 1998;43(2):353–6.
34. Hirao J, Okamoto H, Watanabe T, Asano S, Teraoka A. Dissecting aneurysms at the A1 segment of the anterior cerebral artery – two case reports. Neurol Med Chir (Tokyo). 2001;41(5):271–8.
35. Ohkuma H, Suzuki S, Kikkawa T, Shimamura N. Neuroradiologic and clinical features of arterial dissection of the anterior cerebral artery. AJNR Am J Neuroradiol. 2003;24(4):691–9.
36. Zhu W, Liu P, Tian Y, Gu Y, Xu B, Chen L, Zhou L, Mao Y. Complex middle cerebral artery aneurysms: a new classification based on the angioarchitecture and surgical strategies. Acta Neurochir. 2013;155(8):1481–91.
37. Hensler J, Jensen-Kondering U, Ulmer S, Jansen O. Spontaneous dissections of the anterior cerebral artery: a meta-analysis of the literature and three recent cases. Neuroradiology. 2016;58:997.
38. Chaves C, Estol C, Esnaola MM, Gorson K, O'Donoghue M, De Witt LD, Caplan LR. Spontaneous intracranial internal carotid artery dissection: report of 10 patients. Arch Neurol. 2002;59(6):977–81.

39. Thines L, Zairi F, Taschner C, Leclerc X, Lucas C, Bourgeois P, Lejeune JP. Subarachnoid hemorrhage from spontaneous dissection of the anterior cerebral artery. Cerebrovasc Dis. 2006;22(5–6):452–6.
40. Mehta B, Burke T, Kole M, Bydon A, Seyfried D, Malik G. Stent-within-a-stent technique for the treatment of dissecting vertebral artery aneurysms. AJNR Am J Neuroradiol. 2003;24(9):1814–8.
41. Ahn JY, Han IB, Kim TG, Yoon PH, Lee YJ, Lee BH, Seo SH, Kim DI, Hong CK, Joo JY. Endovascular treatment of intracranial vertebral artery dissections with stent placement or stent-assisted coiling. AJNR Am J Neuroradiol. 2006;27(7):1514–20.
42. Ahn JY, Chung SS, Lee BH, Kim SH, Yoon PH, Joo JY, Kim JK. Treatment of spontaneous arterial dissections with stent placement for preservation of the parent artery. Acta Neurochir. 2005;147(3):265–73. discussion 273.
43. Lylyk P, Cohen JE, Ceratto R, Ferrario A, Miranda C. Combined endovascular treatment of dissecting vertebral artery aneurysms by using stents and coils. J Neurosurg. 2001;94(3):427–32.
44. Lanzino G, Wakhloo AK, Fessler RD, Hartney ML, Guterman LR, Hopkins LN. Efficacy and current limitations of intravascular stents for intracranial internal carotid, vertebral, and basilar artery aneurysms. J Neurosurg. 1999;91(4):538–46.
45. Byoun HS, Yi HJ, Choi KS, Chun HJ, Ko Y, Bak KH. Comparison of endovascular treatments of ruptured dissecting aneurysms of the intracranial internal carotid artery and vertebral artery with a review of the literature. J Korean Neurosurg Soc. 2016;59(5):449–57.
46. Lee VH, Brown RD, Mandrekar JN, Mokri B. Incidence and outcome of cervical artery dissection: a population-based study. Neurology. 2006;67(10):1809–12.
47. Béjot Y, Daubail B, Debette S, Durier J, Giroud M. Incidence and outcome of cerebrovascular events related to cervical artery dissection: the Dijon Stroke Registry. Int J Stroke. 2014;9(7):879–82.
48. Guillon B, Brunereau L, Biousse V, Djouhri H, Lévy C, Bousser MG. Long-term follow-up of aneurysms developed during extracranial internal carotid artery dissection. Neurology. 1999;53(1):117–22.
49. Touzé E, Randoux B, Méary E, Arquizan C, Meder JF, Mas JL. Aneurysmal forms of cervical artery dissection: associated factors and outcome. Stroke. 2001;32(2):418–23.
50. Djouhri H, Guillon B, Brunereau L, Lévy C, Bousson V, Biousse V, Arrivé L, Tubiana JM. MR angiography for the long-term follow-up of dissecting aneurysms of the extracranial internal carotid artery. AJR Am J Roentgenol. 2000;174(4):1137–40.
51. Biller J, Sacco RL, Albuquerque FC, Demaerschalk BM, Fayad P, Long PH, Noorollah LD, Panagos PD, Schievink WI, Schwartz NE, et al. Cervical arterial dissections and association with cervical manipulative therapy: a statement for healthcare professionals from the american heart association/american stroke association. Stroke. 2014;45(10):3155–74.
52. Moon K, Albuquerque F, Cole TS, Gross BA, McDougall CG. 355 endovascular management of cervical carotid and vertebral artery dissection: indications, techniques, and outcomes from a 20-year experience. Neurosurgery. 2016;63(Suppl 1):205.
53. Horowitz MB, Purdy PD. The use of stents in the management of neurovascular disease: a review of historical and present status. Neurosurgery. 2000;46(6):1335–42. discussion 1342–1333.
54. Linfante I, Mayich M, Sonig A, Fujimoto J, Siddiqui A, Dabus G. Flow diversion with pipeline embolic device as treatment of subarachnoid hemorrhage secondary to blister aneurysms: dual-center experience and review of the literature. J Neurointerv Surg. 2017;9(1):29–33.
55. Griessenauer CJ, Ogilvy CS, Foreman PM, Chua MH, Harrigan MR, He L, Fusco MR, Mocco JD, Stapleton CJ, Patel AB, et al. Pipeline embolization device for small intracranial aneurysms: evaluation of safety and efficacy in a multicenter cohort. Neurosurgery. 2017;105:232–7.
56. Ogawa A, Suzuki M, Ogasawara K. Aneurysms at nonbranching sites in the surpaclinoid portion of the internal carotid artery: internal carotid artery trunk aneurysms. Neurosurgery. 2000;47(3):578–83. discussion 583-576.
57. Ahn JY, Cho JH, Jung JY, Lee BH, Yoon PH. Blister-like aneurysms of the supraclinoid internal carotid artery: challenging endovascular treatment with stent-assisted coiling. J Clin Neurosci. 2008;15(9):1058–61.

58. McLaughlin N, Laroche M, Bojanowski MW. Blister-like aneurysms of the internal carotid artery – management considerations. Neurochirurgie. 2012;58(2–3):170–86.
59. Rouchaud A, Brinjikji W, Cloft HJ, Kallmes DF. Endovascular treatment of ruptured blister-like aneurysms: a systematic review and meta-analysis with focus on deconstructive versus reconstructive and flow-diverter treatments. AJNR Am J Neuroradiol. 2015;36(12):2331–9.
60. Yu J, Xu B, Guo Y, Xu K. Direct clipping of a blister-like aneurysm in the supraclinoid segment of the internal carotid artery: a clinical analysis of nine cases. Int J Clin Exp Med. 2015;8(11):21786–95.
61. Owen CM, Montemurro N, Lawton MT. Blister aneurysms of the internal carotid artery: microsurgical results and management strategy. Neurosurgery. 2017;80(2):235–47.
62. Pahl FH, de Oliveira MF, MeQ TG, Capel Cardoso AC, Rotta JM. Blister-like aneurysms: report of successful surgical treatment of consecutive cases and review of the literature. World Neurosurg. 2016;89:376–81.