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Chapter

Blister Aneurysms

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Abstract

Blister aneurysms are rare carotid lesions characterized by a thornlike appearance in combination with prominent fragility. Comprising less than 2% of all intracranial aneurysms, they are considered to be either dissecting or false aneurysms. Etiogenesis remains poorly understood, though atherosclerosis seems to be playing a prominent role. Although many approaches have been tried throughout the years, treatment of blister lesions remains debatable. Both surgical and endovascular modalities can be used, with every technique having though its own limitations and pitfalls. In this context, when confronted with such a lesion, physicians should consider all available alternatives in order to maximize the chances of a good outcome.

Keywords: blister aneurysms, blister-like aneurysms, carotid aneurysms

1. Introduction

Blister aneurysms are a rare but well-recognized form of cerebral vascular lesions. Comprising less than 2% of all intracranial aneurysms, they are typically found on the dorsal or dorsomedial wall of the internal carotid artery (ICA). With a characteristic thornlike appearance on angiography, blister aneurysms' most prominent feature is the fragility of their wall. This explains for their aggressive clinical course and grave prognosis.

Although most authors agree that blister aneurysms are either dissecting or false lesions, their exact nature as well as their optimal management remains unknown. Traditionally, surgery has been advocated as the first-line treatment. Primary clipping, wrapping, wrap-clipping or even carotid artery sacrifice (with or without a bypass) have all been tried. However, results have always been far from satisfying, often making neurosurgeons reluctant to operate on such cases.

During the past few years, clinicians' interest in blister aneurysm has been renewed with the introduction of endovascular modalities in everyday practice. Among all the different available approaches, flow diversion seems lately to be gaining ground, showing promising results. Of course, until consensus has been reached, blister aneurysms are still to be treated on a case-by-case basis.

2. General features and clinical presentation

Blister aneurysms are a distinct form of cerebral vascular lesions with often mixed characteristics and uncertain pathogenesis. They comprise less than 2% of all intracranial aneurysms [1]. Originally described by Sundt and Murphey more than

40 years ago [2], the typical configuration of blister aneurysms consists of a shallow and broad-based or even semifusiform focal protrusion located on the internal carotid artery [3]. Arising from the anteromedial carotid wall in up to 65% of cases [4], these lesions seem to be unrelated to major arterial junctions, although very fine branches such as those supplying the optic nerve may sometimes be involved [5]. Rarely, blister aneurysms can be found on the anterior communicating artery or even the basilar trunk [6, 7], while in a recent case report, the posterior inferior cerebellar artery has also been implicated as another potential site of origin for such a lesion [8].

A prominent characteristic of blister aneurysms is the marked weakness of their wall, a feature that not only reflects the unique pathology of these lesions but also predetermines their high rupture risk, aggressive clinical course, and tendency for rapid growth and progression. In the most common scenario, a blister aneurysm will be diagnosed after an episode of subarachnoid hemorrhage (WFNS grade > 3 in 68% of cases) [9]. Being initially small and sessile, it will substantially enlarge within days of presentation, reaching finally a shape much similar to that of its saccular counterparts [4]. Commonly, the end result is a rerupture with potentially catastrophic consequences for the patient.

On a side note, and regarding terminology, blister aneurysms were originally known as dorsal, anterior or superior carotid wall lesions, a nomenclature though that soon became obsolete failing to recognize the presence of such anomalies on the medial, posteromedial or lateral surface of the internal carotid artery [10]. Additionally, intracranial vessels have never—by tradition—been designated as dorsal or ventral, adding another reason to abandon at least the first of these denominations [11]. An alternative, broader term used mainly by Japanese authors was carotid trunk aneurysms [4]. Still, confusion remained since bleb-like but essentially stable carotid lesions have been encountered during surgery, an observation that clearly indicates that not all such morphological entities fulfill the requirements to be considered as true blister aneurysms, and as a matter of fact, some of them may even be the precursors of typical berry lesions [12, 13]. Whatever the case, the term blister (or blister-like) aneurysms seems to be by now the dominant one within the relevant literature and as such will be used throughout the present manuscript.

3. Epidemiology and demographics

Blister aneurysms are rare lesions comprising less than 2% of all intracranial aneurysms [1] and 0.9–6.6% of internal carotid artery lesions [14]. In the three largest series published to date, Yaşargil et al. reported 3 blister aneurysms in a total of 319 carotid lesions [15], Nakagawa et al. reported 8 cases in a series of 460 surgical patients [3] and Meling et al. reported 14 lesions in a total of 912 aneurysms [16].

Having a slight female preponderance and occurring more frequently on the right side, blister aneurysms tend to affect patients at a rather younger age than their saccular counterparts [17, 18]. In a series of six patients, Abe et al. found a mean presentation age of 56 years [11], while Park et al. calculated this in their own cases at 35.4 years [19]. Risk factors include arterial hypertension [6] and atherosclerosis [16].

4. Pathological considerations

Due to the rarity of blister aneurysms, our understanding of the relevant pathology is only limited. For most authors these lesions are of a dissecting nature [20], and, as such, they are considered to be the consequence of a tear in the affected artery's inner wall followed by intramural hemorrhage [21, 22]. The resultant protrusion is

covered by only the adventitial layer. At this point, it should be noted that the adventitia of intracranial arteries is well known to be thicker over bifurcation sites, partially compensating for the underlying medial defect. Arising exclusively at nonbranching arterial segments, blister aneurysms lack this relatively rigid coverage and are thus much more fragile than their saccular counterparts [23]. This difference between the two accounts for the prominently aggressive clinical course of blister lesions (i.e. increased risk of bleeding and tendency for rapid subsequent growth and rerupture). In their series of 40 blister aneurysms, Ogawa et al. found signs of dissection (double lumen, arterial narrowing or dilation, etc.) in 10 cases [4], while Satoh et al. reported a similar association in as many as 16 out of their 18 in total patients [13].

An alternative hypothesis on the pathology of blister aneurysms is that they are in fact false lesions, essentially representing a focal arterial wall defect [8]. In this direction, Abe et al. suggested a few years back that the characteristic bleb-like protrusion seen by surgeons in such cases is nothing more than an organized blood clot covering the diseased arterial site [11]. Removal of this clot during surgical preparation of the aneurysmal dome is highly likely to cause a rerupture [4]. The pseudoaneurysm theory was initially based on macroscopic intraoperative findings but was later confirmed in at least two cases reported by Charbel et al. [24]. According to these authors, pathological examination of the aneurysmal sacs, which were resected in both cases, in their entirety revealed only blood clot and no fibrous, elastic or smooth muscle tissue (Verhoeff-vanGieson staining plus immunostaining) [24].

The first-ever complete microscopic description of a blister aneurysm has been delivered by Ishikawa et al. in their, classic by now, publication in neurosurgery in 1997. To do this, the authors of the article applied an Elastica Masson stain on cross section specimens of the internal carotid artery of a patient who sustained a fatal rupture of such a lesion [25]. According to their report, the examined blood vessel was heavily atherosclerotic with prominent underlying thickening of the intima. Near the edge of the atheromatous plaque, both the internal elastic lamina and the tunica media were abruptly terminated, and the resultant gap (i.e. the aneurysm sac) was covered only with fibrinous tissue and adventitia. The latter was neither thickened nor rich in collagen as is usually seen in a saccular aneurysm. At the rupture point, the adventitia was lacerated and fragmented. Notably, and contrary to what it would be expected in a dissecting lesion, no inflammatory infiltration or dissection of the artery outside the actual aneurysm was observed.

In a more recent publication, Kim et al. reported a blister aneurysm with a typical clinical course whose dome was resected during surgery for histologic examination. Their conclusions, although interesting, seem to have only added to the confusion: immunohistochemical staining for smooth muscle actin proved to be positive, a finding compatible with an intact tunica media and thus a true lesion. Medial fibroblast proliferation and laminar thrombosis were also noted [26].

It is obvious that the literature on pathology of blister aneurysms has been, up until now, far from conclusive. Speculations stray widely with data supporting each theory being only limited. A possible explanation is that we are dealing with different stages of a rapidly evolving disease or, alternatively, that there is no single entity but a whole range of these. Our only certainty seems to be that morphological changes taking place in blister aneurysms are much more destructive than those seen in berry lesions.

5. Etiogenesis

Similar to their pathology, etiogenesis of blister aneurysms remains, despite our best efforts, unclear. Both Stehbens and Ohara have traditionally associated such

lesions with atherosclerosis, a concept that has by now gained wide acceptance [12, 27]. Atheromatous changes affecting the carotid artery lead to degeneration of the internal elastic lamina [28]. The latter is reported to be the major anatomic structure resisting the pressure of blood flow within a given vessel [29]. The end result is an overall weakening and subsequent laceration of the arterial wall (penetrating ulceration) [30]. Focal subadventitial dissection leads to the formulation of a blister aneurysm [14]. For reasons not yet fully understood, this whole process usually takes place at the periphery of an atherosclerotic plaque where its stiff degenerated wall borders that of a normal elastic vessel segment [25]. Further research is needed to explain carotid vulnerability at these locations.

Apart from atherosclerosis, another factor that seems to play an important role in the formation of blister aneurysms is hemodynamics. The anteromedial surface of the supraclinoid carotid segment, where most of blister lesions arise, is curved in such a way that blood flow directly impinges on the arterial wall [4, 11]. In the most probable scenario, increased hemodynamic stress acts on an already diseased, sclerotic arterial segment, and it is the combination of these two that finally results in an aneurysm formation.

6. Diagnosis

Given that the vast majority of blister aneurysms presents with subarachnoid hemorrhage, the diagnostic evaluation of such a lesion usually starts with a brain CT scan. Hemorrhage is typically lateralized and mainly involves the carotid and suprasellar cisterns as well as the Sylvian fissure. Of note is that, when performed within 24 hours after ictus, CT scans detect subarachnoid blood with a sensitivity of up to 95%, a figure though that quickly drops to less than 50% a week later [31]. This is attributed to the rapid decrease of the hemorrhage density due to dilution by cerebrospinal fluid [32].

The second step in the diagnostic triage of subarachnoid hemorrhage is, for most centers nowadays, a CT angiogram. Blister aneurysms appear, initially at least, as shallow, broad-based lesions, usually less than 2 mm and with a characteristic triangular or thorn-like shape (**Figure 1**) [33, 34]. Unfortunately, their small size in combination with their unusual location (i.e. nonbranching arterial sites) and close proximity to the skull base often makes detection of these lesions obscure [35]. Adding to the difficulty, the aneurysmal dome presumably collapses right after rupture, while its parent artery contracts as a reaction to the presence of subarachnoid blood. The end result can be a significant delay in diagnosis and initiation of treatment. Sensitivity of single slice CT angiography in the investigation of intracranial aneurysms smaller than 3 mm has been reported to be 25–64% [36]. Better results with improved image quality and spatial resolution have been achieved with the introduction of multidetector row technology [37]. Blister lesion diagnosis can be also greatly facilitated through the application of a meticulous technique (decreased section thickness, increased pitch, proper bolus timing and elimination of venous contamination) along with appropriate postprocessing of CTA scans (maximum intensity projections, multiplanar reconstructions and volume-rendered 3D images) [33]. Notably, most false-negative CT angiograms, when evaluated in retrospect, do reveal suspicious anomalies that could be well associated with a blister aneurysm. This observation underlines the significance in such cases of a high index of suspicion.

Despite advances in the field of CT angiography, conventional DSA with its excellent spatial resolution remains the gold standard for the detection of cerebral aneurysms, and, as such, it should be performed whenever initial investigations prove to be negative. The appearance of blister lesions on a DSA closely resembles that of their CTA counterparts, but luminar irregularities related to atherosclerosis of adjacent arterial segments may obscure the diagnosis (**Figure 2**) [35]. Multiple

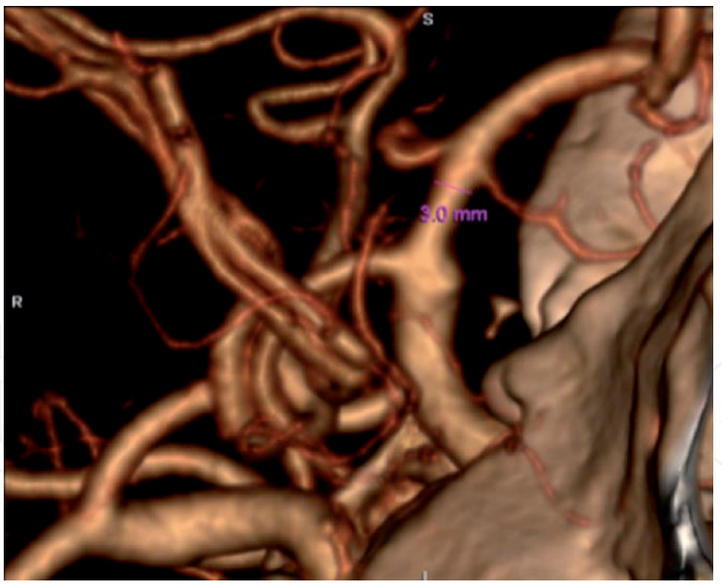

Figure 1.
Reconstructed CTA showing a blister-like aneurysm on the dorsal wall of the internal carotid artery (ICA).

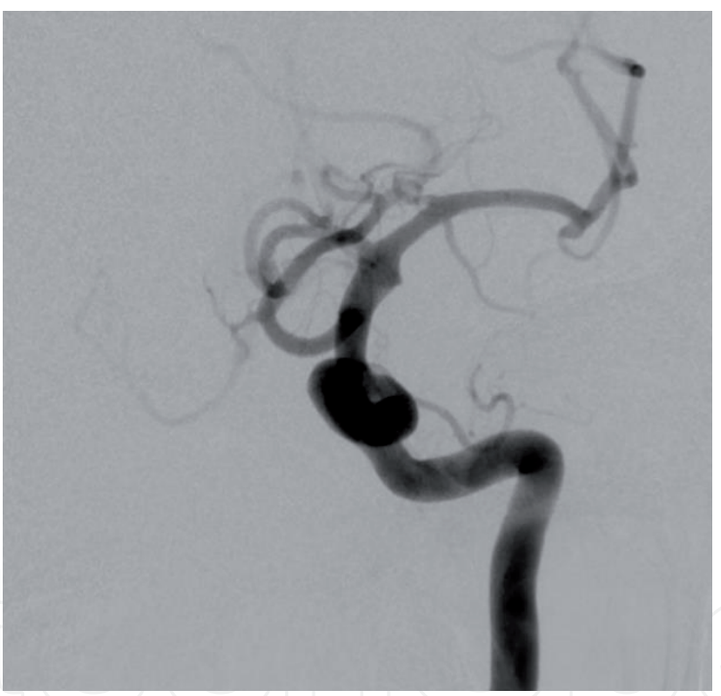

Figure 2.
Internal carotid injection, AP view. A characteristic thornlike blister aneurysm is noted opposite to the anterior choroidal artery origin. Large posterior communicating artery supplying the posterior circulation noted.

oblique views or even rotational 3D scans significantly increase the sensitivity of the method [38]. Signs of dissection have been reported in up to 89% of blister aneurysm cases and include a false lumen, an intimal flap, a filling defect or contrast pooling [39]. Should the presence of a blister aneurysm be suspected on the basis of DSA findings, the evaluation of collateral flow through the circle of Willis is always advisable in case an occlusion procedure is to be carried out [18, 33].
Cross-compression carotid injections may help demonstrate the anterior communicating artery, while patency, size and collateral potential of its posterior counterpart can be assessed through an Alcock test (vertebral injections with carotid artery compression). For a more detailed study, temporary balloon occlusion will be required [31].

Another important aspect of imaging in cases of blister aneurysms is that, on short-term follow-up angiography after initial presentation, these lesions usually show rapid growth to a saccular configuration [19]. Being at least partially related to lysis of an intra-aneurysmal clot [26], this progression is considered to be a good indicator of a blister lesion even though the only real way to authenticate such a diagnosis is through direct intraoperative inspection [1, 40].

7. Treatment

Management of blister aneurysms is associated with a high overall rate of mortality and morbidity [35]. The main causes for this include the small size and broad neck morphology along with the prominent fragility of such lesions, features that often lead to intraprocedural rupture when traditional surgical or endovascular techniques such as clipping or primary coiling are to be applied [6, 19]. Additionally, and even if an initial intervention proves successful, subsequent regrowth requiring further treatment has been commonly reported [9]. Other factors contributing to the grim prognosis of blister aneurysms include a commonly grave clinical presentation as well as delays in an appropriate diagnosis.

Given the lack of universal consensus in blister aneurysm treatment, a wide variety of approaches and methods has been employed up to date and will be discussed below. These have included both reconstructive and deconstructive surgical and endovascular techniques, with different authors competing for best results in an ever-changing field. In any case, attention should be given to specific measures to prevent rerupture while awaiting final treatment (tight arterial blood pressure control, cautious cerebrospinal fluid drainage in patients with a ventricular drainage in place and selective use of aminocaproic acid) [41]. The latter must be instituted as soon as possible to secure the aneurysm and to allow aggressive management of subarachnoid hemorrhage-related complications such as vasospasm and hydrocephalus.

The alternative treatment modalities for blister aneurysms are:

a. Reconstructive techniques

- Surgery: primary clipping (including encircling clips), wrapping, clip-wrapping, wrap-clipping and direct suturing
- Endovascular therapy: primary coiling, stent-assisted coiling, telescopic stenting (stent-in-stent technique) and flow diverters

b. Deconstructive techniques

- Parent artery occlusion with surgical or endovascular means with or without bypass surgery

7.1 Surgical treatment

7.1.1 Clipping procedures (primary clipping and wrap-clipping)

7.1.1.1 Technique

Traditionally, surgical clipping has been the preferred mode of treatment for all forms of cerebral aneurysms, including blister lesions. In a typical case, the procedure starts with exposure of the cervical internal carotid artery to ensure

proximal control in case of an intraprocedural rupture. This is usually achieved with an incision along the medial border of the ipsilateral sternocleidomastoid muscle [42]. Subsequently, a standard pterional craniotomy with generous sphenoid ridge drilling is carried out, and through this, the Sylvian fissure is opened widely. Gentle retraction of the frontal lobe provides access to the supraclinoid internal carotid artery which, in most cases, is found to be prominently sclerotic [43]. The aneurysm itself is usually seen protruding from the dorsomedial carotid wall. Careful preparation of the aneurysmal dome is crucial. As a matter of fact, should the frontal lobe be attached to it, most authors propose a subpial dissection in order to minimize direct manipulation of the lesion [44]. Special care is needed to avoid removing the platelet plug that typically covers the aneurysm as this may result in a large wall defect and uncontrollable bleeding [4].

Direct clipping of a blister aneurysm is performed under temporary trapping and in such a way that the blades of a usually angled or curved Sugita clip are parallel to the longitudinal axis of the carotid artery [19, 45]. This has been shown to lower the risk of intraprocedural rupture [17]. Given that the underlying pathological process seems to extend well beyond the limits of the aneurysm itself, the surgeon should try, when closing the clip, to include part of the "normal" arterial wall outside the lesion in order to avoid breakage of the transitional zone found in between them [4]. A valid alternative is to envelope the entire diseased arterial segment with a wrapping material such as gauze, cotton or Gore-Tex on top of which the clip can be applied to obliterate the lesion [46]. Wrap-clipping not only reinforces the carotid wall as a whole but also helps avoid slippage of the clip, a complication not uncommonly seen in blister aneurysms surgery [47]. One yet alternative is to wrap the lesion after the clip has been applied (clip-wrapping). Whatever the exact technique, induced hypotension [11], burst suppression with desflurane or thiopental, cooling of the patient or even transient flow arrest with adenosine can all prove useful adjuncts [11, 24, 41, 48].

7.1.1.2 Results of clipping procedures, associated complications and rescue measures

Primary clipping of blister lesions is known to carry an up to 30% risk of complications, both intraoperative and postoperative [9, 49]. In the former group, aneurysm avulsion and internal carotid artery laceration seem to be our major concerns, while progression of the lesion often accompanied by rerupture seems to be the main danger during the postoperative period [50].

When confronted with a laceration of the carotid artery during surgery, the most commonly used rescue technique has traditionally been sacrifice of the vessel to control bleeding. As proven by numerous studies, this is associated with a high risk of cerebral ischemia, not only because collaterals may be inadequate in the first place but also due to hemorrhage-related vasospasm that often further aggravates the whole situation [16]. Other alternatives include direct suturing, placement of an encircling clip graft and reapplication of the originally placed clip to intentionally narrow the carotid artery and thus achieve hemostasis [4, 43]. In every case, surgeons must be always prepared for such an event, and large aspirators need to be available throughout the procedure. Initial hemorrhage control is usually achieved by applying direct pressure on the artery with oxidized cellulose and a small cottonoid. Brief periods of cardiac arrest with the aid of adenosine are also useful to improve visibility and help gain control of the situation [41].

Direct suturing of an arterial tear during surgery for a blister aneurysm is made possible because the edge of the lesion is relatively well outlined [51]. It entails removal of the original clip, trimming of the aneurysmal sac with microscissors (to prevent tissue buckling into the lumen) and repair of the arterial wall defect with

8/0 nylon stitches [43]. Suturing in the acute stage is usually extremely difficult due to the deep and narrow surgical field. As a result, initial attempts at this required prolonged trapping of the diseased segment invariably led to cerebral ischemia [52, 53]. To avoid this, recent studies propose reformation of the arterial wall with only a few stitches rather than its complete restoration [43]. Subsequent circumferential wrapping of the artery or, alternatively, placement of an encircling clip graft should be enough to stop oozing and stabilize the end result [54].

Encircling clip grafts have been developed by Sundt almost 40 years ago and specifically to address the problem of repairing the wall defect associated with a blister aneurysm [2, 55]. They can be used either as a primary modality (instead of the classic clip) or in cases of intraoperative rupture. Applied typically with the aid of a straight or right angle clip holder, Sundt clips provide a rigid sleeve and a soft woven fabric lining, allowing surgeons to essentially reconstruct the lacerated arterial wall [56]. Produced in various sizes, the appropriate clip for each case can be estimated on the basis of preoperative angiographic studies with a 3–4 mms diameter and a 3–5 mms length being the most widely used options [42]. Major disadvantages associated with these devices are that they cannot be used in cases of lesions close or opposite to carotid branches (i.e. posterior communicating and anterior choroidal arteries) and also that they come in certain fixed diameters that may or may not fit the vessel involved [48]. The latter limitation occasionally leads to postprocedural stenosis, a risk that surgeons must always be aware of and act accordingly [6]. Of note is a recent publication by Cho et al. who used a Sundt clip in combination with an endovascularly placed carotid stent to treat a ruptured blister aneurysm with good results [42]. Being a valuable adjunct in blister lesion surgery, encircling clip grafts should be readily available in all such procedures. Sadly, these adjuncts are rarely part of the modern day aneurysm clip tray, and young neurosurgeons are often not even aware of their existence.

Despite all efforts, intraoperative rupture of a blister aneurysm carries a mortality rate of up to 25% [57]. On this basis, and with surgeons trying for improved results, novel techniques continue to emerge. An interesting addition to our armatorium has been recently proposed by Kazumata et al. in the form of a protective bypass concept. According to these authors, when dealing with a blister aneurysm, and knowing the fragility of such a lesion, surgeons should at least contemplate an STA-MCA bypass prior to any attempted clipping. Ensuring adequate cerebral perfusion, this technique allows, in case of an intraoperative laceration, time for corrective measures to be applied accordingly. The duration of temporary occlusion for direct suturing or clip reapplication seizes to be a limiting factor, while, should the need for a carotid occlusion arise, this can be done safely, combined with a proper high-flow, radial artery graft bypass [58].

7.1.2 Wrapping procedures

In an effort to limit intraoperative manipulation of the lesion, neurosurgeons, especially in the past, have widely used wrapping of blister aneurysms as an alternative to clipping mode of treatment. In this context, wrapping is undertaken when a traditional clip cannot be properly applied due to either the morphology of the aneurysm itself or the underlying sclerotic changes of the parent vessel (i.e. the carotid artery) [46]. The technique entails encasing of the entire diseased segment of the carotid artery with an appropriately sized thin sheet of—usually—cotton. On occasions, cuts along its longitudinal axis are made to accommodate for branch vessels and perforators. Creation of ample room to allow free movement of surgical instruments and circumferential dissection of the aneurysm dome can prove dangerous but are, at the same time, essential. The wrapping material is applied with

the aid of microforceps, and an angled clip is used to fit it snugly around the artery. Once the wrapping is secured, any excess length is cut and removed [39, 41].

The concept behind wrapping is that mechanical reinforcement of the diseased carotid artery provides protection from a potential rerupture during the immediate postoperative period while, in the long term, induced inflammatory changes eventually lead to connective tissue formation and remodeling of the weak arterial wall into a histologically competent structure [47, 59, 60]. Wrapping materials that have been tried out include muscle, muslin gauze, Teflon, silicon, collagen-impregnated Dacron fabric and cotton [46]. The latter seems to dominate today the relevant literature with most authors proposing it as the most effective option. Notably, Biobond, a cyanoacrylate glue widely used in the past, is by now obsolete due to its toxicity [60–62].

Complications associated with wrapping are rare. Perforator injury or parent vessel narrowing following placement of whichever material has been chosen make postoperative angiography an absolute necessity [63]. Delayed development of a granuloma or arachnoiditis has also been reported resulting in cranial neuropathies [64, 65], an observation that has led many authors to suggest that cotton must be used with the utmost care and never be placed in contact with adjacent cranial nerves, especially the chiasm [46, 63].

In the premicrosurgery era, numerous studies have been published on the efficacy and safety of wrapping. Among these, the largest series has been the one presented by Todd et al. in 1989. According to it, the risk of early (within 6 months) rehemorrhage after wrapping of an aneurysm has been calculated to be 8.6%, while late incidents were at 1.5% per year [66]. Results seem to have been significantly better after the introduction of the surgical microscope although only few relevant articles exist. Characteristically, Cudlip et al. reported in 1998 a series of 15 wrapped aneurysms with no rehemorrhages within 1 year after surgery [67].

Wrapping is unquestionably inferior to clip ligation for the treatment of ruptured aneurysms. However, it does offer a degree of protection during the immediate postoperative period, and, by doing so, it remains today a valid—even though last resort—therapeutic option when confronted with a blister aneurysm [47].

7.2 Endovascular treatment

Initial attempts at endovascular treatment with primary coiling of blister aneurysms (**Figure 3**) have returned, in most cases, disappointing results [68]. A high risk of intraprocedural rupture and coil protrusion or migration were problems commonly encountered due to the small size and shallow morphology of such lesions, their fragile nature and their proximity to vascular curves on the carotid wall (the latter, in combination with the typical orientation of blister aneurysms, has been known to necessitate a difficult and extremely gentle catheterization maneuver in order to gain access to the sac while avoiding a potential perforation) [19, 35]. Additionally, the lack, in many cases, of a true wall often allowed for post-treatment progression and—possibly—rebleeding [40]. As a consequence, most authors advocated that blister aneurysms are unsuitable for endovascular treatment and should therefore be left to surgery [69].

With the introduction of intracranial stents in clinical practice, our conception of blister lesion management has gradually changed. Surgical techniques are increasingly looked down upon as outdated and old-fashioned while stent-assisted coiling (**Figure 4**) became, initially at least, the new trend in the field. The procedure is carried out either by first placing the stent (e.g. Neuroform or Enterprise stents) and then introducing coils through its struts (trans-stent coiling) or by catheterizing the aneurysm sac and deploying the stent over the microcatheter

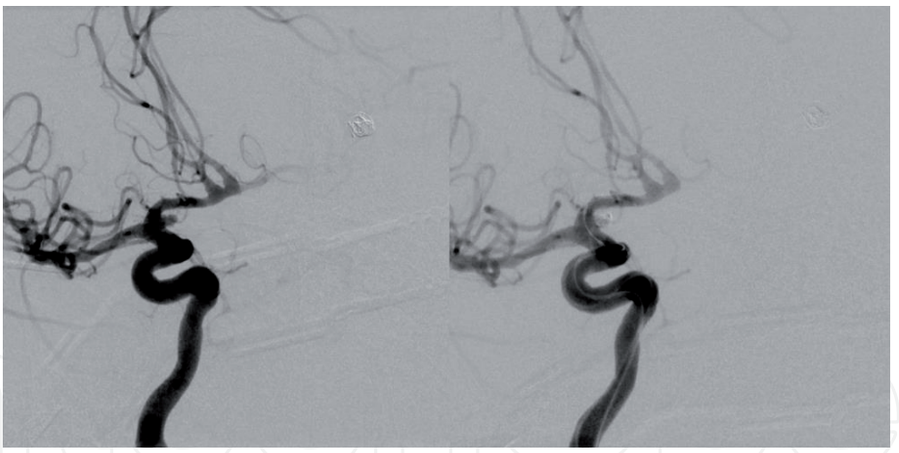

Figure 3.
Simple coiling of a right ICA blister aneurysm. Intraprocedural images.

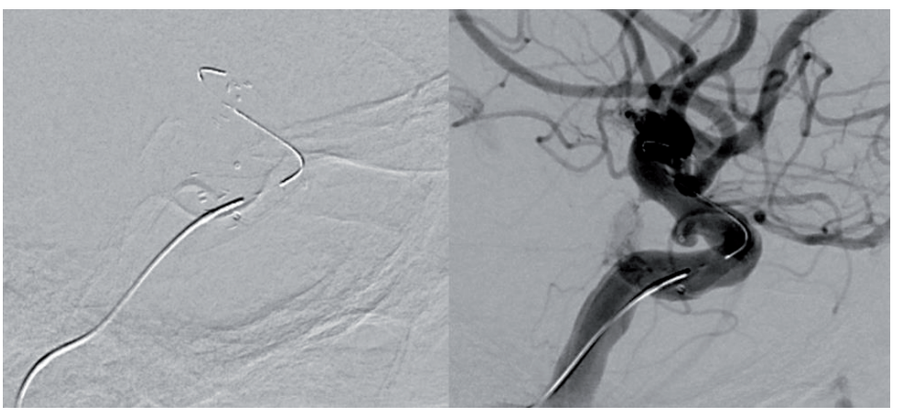

Figure 4.
Intraprocedural images showing stent-assisted coiling of a left ICA blister lesion.

prior to coiling (jailing technique). Facilitating stable intrasaccular coil deployment while at the same time reinforcing the underlying diseased arterial wall, stent-assisted coiling promised to provide a safe and reliable therapeutic alternative [20, 70]. However, it was soon realized that results, even though better than those of surgery, were far from optimal. Intraoperative complications, mainly bleeding, were encountered in up to 17% of cases, while the risk for recurrence of the lesion, need for further treatment and postoperative repeat hemorrhage were reported at 65, 50 and 13%, respectively [9, 35, 71]. Notably, Meckel et al. found that the latter, a potentially catastrophic and fatal event, is seen only in cases that show incomplete occlusion (i.e. neck remnant or residual sac) by the end of the initial already procedure and especially if the patient receives full double antiplatelet treatment postoperatively or if the aneurysm in question is atypically large or partially thrombosed [35]. In this context, early and tight angiographic follow-up of partially obliterated lesions is essential, while any signs of regrowth should prompt complementary treatment. In the face of all these, most authors are by now moving away from stent-assisted coiling being the preferred primary and sole mode of treatment. Instead, they are using it, if possible, as a preliminary means to achieve a certain degree of protection until definite treatments, in the form of some other techniques, can be instituted.

Prior to moving on, and in order to close the discussion of stent-assisted coiling, it should be noted that one more major argument against it is the need for subsequent antiplatelet therapy [72]. This, combined with a potentially still unsecure aneurysm, requires careful consideration. Additional risks include the frequent

need for a surgical intervention at a later time (e.g. extraventricular drainage or shunting procedures) [73, 74] and delayed spontaneous intraparenchymal bleeding with the rate of the later though not being affected by the initial subarachnoid hemorrhage [75, 76]. The exact therapeutic antiplatelet regimen that should be used in such a setting is still debatable. Some authors suggest that a similar to elective case scheme should be followed: loading with full doses of aspirin and clopidogrel just prior to the procedure, double antiplatelets for 3–6 months (usually 75 mg aspirin and 75 mg clopidogrel daily) and then continuation of only aspirin for another 6 months to life [77]. Going even further, Lee et al. augmented this regimen by a full-dose heparinization for 24–48 hours postoperatively [1]. On the contrary, other authors favor a reduced or even single-drug scheme. The rationale for this is that in the presence of a high-flow state within the stented lumen of large-size vessels (e.g. internal carotid artery), fibrin formation or clotting is relatively unlikely. Moreover, the risk of and the consequences of a potential intraprocedural rerupture are minimized, while at the same time occlusion of the aneurysm through thrombus formation is significantly facilitated. Using such an approach, Meckel et al. reported in 2011 only 1 fatal rehemorrhage in between 11 patients who were primarily treated with stent-assisted coiling of blister aneurysms. In other reports, stent-assisted coiling under systemic heparinization and loading with dual antiplatelets only after the procedure was found to be associated with a cumulative risk of thrombotic and hemorrhagic complications as low as 2% [78], while the intraprocedural use of aspirin and later introduction of clopidogrel carried a 21% overall risk of perioperative complications [79]. In any case, and because of significant heterogeneity of response to antiplatelet drugs, platelet function should be performed prior to any intervention as well as during the postoperative period (a patient's response may be found to change over time) [80, 81].

A recent development in blister aneurysm treatment and a welcome addition to our armatorium has been flow diversion. According to advocates of such a concept, and given that blister lesions are regarded by many as pseudoaneurysms, flow diversion is the only endovascular technique capable of actually reconstructing the vessel wall and sealing off any underlying defect [8, 82]. This can be achieved either through a stent-in-stent technique or with the aid of devices such as the pipeline or the Silk flow diversion systems. The former typically entails telescopic deployment of multiple overlapping stents within the diseased vessel in an effort to increase the total mesh density and thus restore proper parent artery laminar flow [8]. This results in thrombosis of the lesion, an effect augmented by endothelial proliferation along the length of the implanted stents. In the same direction, and specifically designed with this in mind, proper flow diverters like the Silk or the pipeline systems provide significantly better hemodynamic results at the expense however of increased perioperative complications due to their stiffness and thrombogenicity [83–85]. Notably, and regardless of the technique selected, flow diversion allows for preservation of branching vessels, an important feature when it comes to blister aneurysms since such lesions are usually located close to the posterior communicating or the anterior choroidal arteries [86].

Major concerns with the use of flow diversion for the treatment of blister lesions include an even more prominent need for antiplatelets as well as the fact that such an approach does not guarantee protection from postoperative progression and rerupture. Regarding the latter, and despite reports of a marked decrease in intraaneurysmal flow on the intraoperative already angiogram, hemodynamic stress upon the lesion theoretically remains at least for a few days [40]. Rasskazoff et al. recently reported that even with the use of double SILK flow diverters, complete occlusion of a blister aneurysm they treated did not occur till 18 days after the intervention, while Consoli et al. verified obliteration of a similar lesion no less than

6 months postprocedurally [8, 75]. A valid alternative possibly addressing the whole issue is the combination of flow diversion with coiling. On this basis, Kim et al. have reported favorable results with stent-assisted coiling as a primary treatment augmented by deployment of a second flow diverting stent if needed (i.e. postoperative progression of the lesion) [39]. In cases with extremely small lesions where coil deployment is perceived as carrying a significant risk, the reverse route can also be followed: telescopic stenting and subsequent trans-stent coiling should the lesion further grow to allow that [40]. Another attractive option involves covered stents; their use however is still limited due—mainly—to their stiffness, a feature that makes their intracranial delivery not only difficult but also dangerous since they may impinge on the fragile aneurysm neck portion resulting in intraoperative rupture. In addition, they often prove impossible to fully conform to the curved supraclinoid carotid wall, and, in this way, they can potentially leave an underlying aneurysm essentially open [39]. Whatever the case, further experience is needed should such devices gain a significant role in blister aneurysm treatment.

7.3 Parent artery occlusion

Although often considered as inherently inferior, deconstructive techniques allow for definitive occlusion of a blister aneurysm with minimal direct manipulation of its walls. In this context, carotid artery sacrifice and trapping of blister lesions significantly reduce the overall risk of rerupture during the perioperative period [19, 87].

Should carotid artery occlusion be contemplated as a final treatment, endovascular rather than surgical trapping is recommended due to its convenience, rapidity and safety. In an often cited article, Park et al. have described an elegant technique entailing the use of two microcatheters and a proximal balloon to control blood flow intraoperatively [19]. The first microcatheter is placed distal to the aneurysm, while the second is positioned just at the level of its neck. With the aid of the latter, and after inflation of the balloon, a framing coil is deployed inside the carotid artery. Prior to detachment, the distal microcatheter is retrieved and repositioned within the coil mess. Additional coils are deployed till a stable result is achieved. The use of trapping coils instead of detachable balloons is perceived as having a lesser risk of intraprocedural rupture.

When occlusion of the carotid artery is suggested, postoperative patency of its cardinal branches requires careful consideration. A detailed study of the ophthalmic, the posterior communicating and the anterior choroidal arteries as well as their collaterals is essential:

i. Ophthalmic artery

Blister aneurysms arise on the communicating segment of the internal carotid artery, and therefore the ophthalmic artery does not usually present a problem when dealing with such a lesion. If a choroidal blush via the external carotid circulation is visualized on preoperative angiograms, the trapped segment of the carotid artery is typically extended to include the origin of the ophthalmic artery as well, achieving thus a more robust result. As an additional precaution, proximal occlusion of the cervical internal carotid artery may also be performed. If, on the other hand, no ophthalmic collateral vessels are visualized, preservation of the ophthalmic artery is absolutely essential, and carotid trapping should be kept short. Accordingly, the proximal carotid artery cannot be occluded, and, as a consequence, close follow-up is mandatory to exclude recanalization of the trapped segment.

ii. Posterior communicating artery

Due to its proximity to blister lesions, the posterior communicating artery often needs to be included in the trapped carotid segment. In most cases this is safe with the only exception being a fetal-type vessel. In the latter case, an alternative therapeutic strategy should be considered.

iii. Anterior choroidal artery

Preservation of the anterior choroidal artery is essential in order to avoid postoperative hemiplegia. On this basis, precision of segmental occlusion offers obvious advantages. Should the origin of the anterior choroidal artery prove impossible to save, the procedure should be abandoned.

Apart from carotid side branches, another important concern with all deconstructive procedures is postoperative early or late ischemia. Unselective, abrupt occlusion of the internal carotid artery is known to carry a 26% risk of cerebral infarction and 12% risk of death [88]. A balloon occlusion test should always be performed when such an approach is contemplated. This entails inflation of a nondetachable balloon at the site of the intended occlusion and subsequent clinical and angiographic evaluation of hemispheric collateral circulation [89, 90]. However, one should always keep in mind that—in the setting of a subarachnoid hemorrhage—results of a balloon occlusion test can be misleading. This is due to the fact that a balloon occlusion test does not take into account the hemodynamic effects of a posthemorrhagic vasospasm which may complicate such cases [16].

For patients who fail a balloon test occlusion, surgical bypass should be contemplated. Traditionally, this is in the form of an artificial communication between the superficial temporal and the middle cerebral artery with or without an interposed vascular graft (radial artery or saphenous vein) (STA-MCA bypass) [16, 58]. In the case a graft is to be used, this is termed as a high-flow bypass and ensures significantly better results [91]. As a matter of fact, there are authors recommending high-flow bypasses as a primary mode of treatment for blister aneurysms. However, experts in the field seem skeptical advocating that a high-flow bypass should never be thought of as complete substitute for normal carotid artery supply, quoting an 80% rate of postoperative complications, including graft occlusion and vasospasm-related cerebral infarction [41]. Additionally, surgical bypass in patients with severely atherosclerotic vessels typically requires antiplatelets, a feature that only adds to the overall risk.

8. Conclusion

Our understanding of blister aneurysms is still today incomplete. Having a relatively wide spectrum of pathological differentiations, they can be classified as either true or false lesions. Whether this represents consecutive stages of the same entity or a different, in each case, disease remains to be clarified. Further research on the field is an absolute necessity, and young physicians should thus be encouraged toward this direction by their senior colleagues.

A direct consequence of our limited knowledge on the nature of blister aneurysms is the lack of an established and universally accepted treatment modality. This lack of consensus has led to numerous attempts at novel and often promising therapies. However, and with the sole exception of the recently introduced flow diverters, all previous options have invariably failed to rise to initial expectations.

Flow diversion, despite its limitations, arises today as probably the most attractive future prospect. However, until our technology reaches that point, blister aneurysms are still to be treated on a case-by-case basis. In this context, when confronted with such a lesion, physicians should consider all available alternatives, both surgical and endovascular, in order to maximize the chances of a good outcome.

Whichever end treatment modality is to be used, an important point when dealing with a patient that has suffered rupture of a blister aneurysm is amelioration of perihemorrhage management in its entity. Careful consideration of all measures to prevent and, if needed, manage rebleeding or any other related complications (i.e. vasospasm, hydrocephalus, seizures) is essential. All these issues would preferably be addressed within a multidisciplinary team consisting of neurosurgeons, interventionists and ICU specialists should the best possible result be achieved.

As with every other such case, and despite initial enthusiasm, each new therapeutic approach proposed for blister lesions needs careful consideration and long periods of follow-up to evaluate its efficacy, safety and durability of results. It is obvious that further research in the field is an absolute necessity and young physicians should be encouraged toward this direction.

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