Blood blisterlike aneurysms of the internal carotid artery

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Object. An aneurysm arising from the anterior wall of the internal carotid artery (ICA) is a poorly understood entity. A small hemispherical bulge from the anterior wall of the ICA, which is called a “blood blisterlike aneurysm” (BBA), may be confused with a tiny berry aneurysm although the clinical features are distinctly different. This paper summarizes the clinical course of patients with this lesion to clarify the nature of the BBA.

Methods. Six patients with BBAs who presented with subarachnoid hemorrhage (SAH) are described. In all patients, the initial angiogram obtained soon after SAH showed only a small bulge from the anterior wall of the ICA. In three of the six patients this bulge had progressed to a saccular appearance within a few weeks. The wall of the lesion was so thin and fragile that the aneurysm ruptured at the base during clipping or within a few hours after clipping in two patients.

Conclusions. From the authors’ experience, as well as a review of the literature, which includes an autopsy study of similar cases, it is inferred that these lesions are focal wall defects covered only with thin fibrous tissue and that they are therefore not true aneurysms. Direct clipping often causes laceration of the lesion, whereas complete wrapping or clipping after wrapping is effective, but may fail to prevent growth of the aneurysm. Endovascular occlusion of the cervical ICA with or without bypass surgery, which is less risky than direct surgery, is another option.

Key Words • aneurysm • internal carotid artery • false aneurysm • atherosclerosis • subarachnoid hemorrhage

Patients with spontaneous subarachnoid hemorrhage (SAH) usually have berry aneurysms or dissecting aneurysms, especially if the lesions are located in the posterior fossa. In this report, we demonstrate that another process involving the anterior wall of the internal carotid artery (ICA) also produces SAH. Our use of the term “blood blisterlike aneurysm” (BBA) refers to a small hemispherical bulge from the arterial wall. Although such an aneurysm is not novel, there are only a few reports of BBAs in the English literature. The lesion resembles a tiny berry aneurysm, which is more familiar to many neurosurgeons, but it has different clinical features and its recognition has surgical importance. In this report, we summarize the clinical course of six patients harboring these aneurysms who presented at our clinics with SAH and we discuss the nature of the lesion.

Clinical Material and Methods

All records of patients who underwent surgery for intracranial aneurysms at our institutions were reviewed for the presence of anterior wall aneurysms of the ICA. A total of 488 patients with aneurysms were treated between 1981 and 1997 in the Department of Neurosurgery at the Saga Medical School, and, of these, four patients (0.8%) were found to have anterior wall aneurysms. Three of these four patients had BBAs (Cases 4–6; Table 1).

Results

Clinical Findings

Four patients were women and two were men; their average age was 56 years (range 43–74 years). Three suffered from hypertension, and all presented with SAH. Preoperative classification according to the Hunt and Kosnik system3 was Grade III in three patients, Grade II in two patients, and Grade IV in one patient.

Angiographic Findings

A small hemispherical bulging was demonstrated on all angiographic studies at the anteromedial wall of the supraclinoid portion of the ICA (C1c segment). In three patients, repeated angiography, including postoperative angiograms, showed growth of aneurysms at intervals of 14 days in two patients and 17 days in one. A false lumen or retention of contrast medium in the lesion was not demonstrated.
Surgical Findings and Procedures

All patients except one (Case 3) underwent direct surgery of the aneurysm in the acute stage; three of them were found to have atherosclerotic ICAs. All aneurysms were located at the anteromedial wall of the supraclinoid portion of the ICA; they had no relation to the arterial division. Direct clipping was performed in three patients (Cases 4–6); in one of these (Case 6), rupture at the aneurysm base occurred during clipping and within a few hours after clipping in another (Case 4). Subsequent procedures included angioplasty of the ICA by suturing with two clips (Case 6). Clipping after wrapping with a strip of gauze was performed in two patients (Cases 1 and 2), whose repeated angiograms showed growth of the aneurysm. These patients underwent subsequent procedures consisting of clipping in one (Case 2) and endovascular occlusion of the cervical ICA in the other (Case 1). One patient (Case 3) was treated conservatively and underwent superficial temporal artery–middle cerebral artery (STA–MCA) anastomosis and endovascular occlusion of the cervical ICA in the chronic stage.

Two patients (Cases 4 and 5) died of massive infarction, and one (Case 2) became disabled due to delayed spasm after SAH.

Illustrative Cases

Case 1

History and Examination. This 57-year-old woman experienced a sudden-onset headache followed by loss of consciousness when going to bed late at night. A computed tomography (CT) scan showed a thick and diffuse subarachnoid clot. Her neurological status was unremarkable except for drowsiness and signs of meningeal irritation. An angiographic study performed on the day of onset revealed a large aneurysm at the cavernous portion and a small bulge on the anteromedial wall at the supraclinoid portion of the left ICA (Fig. 1 upper left). Because an aneurysm at the cavernous portion does not usually cause SAH, the small lesion at the supraclinoid portion of the artery was thought to be the source of hemorrhage.

Operation. On the day of onset, a left frontotemporal craniotomy was performed after exposure of the left cervical ICA, and the left frontal lobe was carefully elevated. The walls of the ICA as well as the anterior cerebral artery (ACA) and the MCA were not sclerotic but seemed thinner than usual. A blood blisterlike bulge was located at the anteromedial wall of the C1 portion of the artery (Fig. 1 lower left). During temporary occlusion of the cervical ICA, the lesion was clipped with two Sugita clips so that the blades of the second one could catch the arterial wall beyond the lesion after the lesion and the ICA had been wrapped with a strip of gauze (Fig. 1 lower center and right).

Postoperative Course. The patient tolerated the procedure well, but on the 17th day postsurgery repeated angiographic studies revealed growth of the aneurysm (Fig. 1 upper right). Because the patient had an aneurysm at the cavernous portion of the artery in addition to the growing lesion, we decided to occlude the cervical ICA rather than reoperate on the aneurysm. A balloon occlusion test of the left cervical ICA was performed on the 23rd day postsurgery with monitoring of the electroencephalographic and stump pressure readings and neurological examination. The patient tolerated the test for 20 minutes with no detectable changes in the results of the examinations. The cervical ICA was then occluded with a detachable balloon (Nycomed Ingenor, Paris, France) and an interlocking detachable coil (Target Therapeutics, San Francisco, CA) to prevent migration of the balloon. The patient tolerated the procedure well and was discharged with no neurological deficits. On repeated angiographic studies obtained at 3 and 6 months after occlusion, the two aneurysms could not be visualized, although the supraclinoid portion of the ICA was reconstituted via the ophthalmic artery.

Case 2

History and Examination. This 56-year-old man experienced a sudden-onset headache while working in an ironworks. A CT scan was obtained and showed a thin but diffuse subarachnoid clot predominantly on the left side. The patient’s neurological status was unremarkable except for moderate signs of meningeal irritation. Angiographic studies performed on the day of onset revealed no remarkable abnormalities except for a faint bulge on the anteromedial wall of the right ICA (Fig. 2 left).

First Operation. On the day of onset, a right frontotemporal craniotomy was performed. The cervical ICA was not exposed but was prepared for manual compression. The sylvian fissure was opened widely and the right frontal lobe was carefully elevated to expose the right ICA.

TABLE 1
Summary of characteristics of six patients with BBAs on the anterior wall of the ICA

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Interval Preop to Op (days)</th>
<th>Treatment &amp; Course</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>57</td>
<td>F</td>
<td>III</td>
<td>1st: clipping after wrapping, regrowth; 2nd: endovascular occlusion of the ICA</td>
<td>excellent</td>
</tr>
<tr>
<td>2</td>
<td>56</td>
<td>M</td>
<td>II</td>
<td>1st: clipping after wrapping, rebleeding; 2nd: clipping of regrown aneurysm</td>
<td>poor (spasm)</td>
</tr>
<tr>
<td>3</td>
<td>43</td>
<td>M</td>
<td>III</td>
<td>STA–MCA anastomosis &amp; endovascular occlusion of ICA</td>
<td>excellent</td>
</tr>
<tr>
<td>4</td>
<td>51</td>
<td>F</td>
<td>III</td>
<td>1st: clipping, rebleeding on the day of op; 2nd: angioplasty of the ICA by suturing</td>
<td>dead (spasm)</td>
</tr>
<tr>
<td>5</td>
<td>57</td>
<td>F</td>
<td>IV</td>
<td>clipping</td>
<td>dead (spasm)</td>
</tr>
<tr>
<td>6</td>
<td>74</td>
<td>F</td>
<td>II</td>
<td>clipping after rupture on initial clipping</td>
<td>excellent</td>
</tr>
</tbody>
</table>

* Graded according to the Hunt and Kosnik system.
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The supraclinoid portion of the ICA was seen to bend laterally and was sclerotic with atheromatous plaques. A BBA was seen on the anteromedial wall of the C1 segment. Under manual compression of the cervical ICA and after wrapping the lesion and the ICA with a strip of gauze, a right-angle Sugita clip was applied parallel to the ICA so that the clip blades could catch the arterial wall beyond the lesion.

The patient tolerated the procedure well, but on Day 14 postsurgery he experienced sudden recurrence of a severe headache. A CT scan was obtained and showed a thick and diffuse subarachnoid clot. Repeated angiography at that time demonstrated growth of the aneurysm extending posteriorly at the medial side of the clip (Fig. 2 right).

Second Operation. On postoperative Day 15, the frontotemporal wound was reopened after exposure of the right cervical ICA. The right pericarotid cistern was found to be packed with a fresh clot. There was a newly devel-

![Figure 1](image1.png)

**Fig. 1.** Case 1. *Upper Left:* Left carotid angiogram showing a large aneurysm at the cavernous portion and a small bulge (arrow) on the anteromedial wall at the supraclinoid portion of the ICA. *Upper Right:* Postoperative carotid angiogram obtained on Day 17 after the first angiogram, showing growth of the small aneurysm. *Lower:* Drawing of surgical findings showing a BBA on the anteromedial wall of the left ICA (left) and drawings of the operative procedure showing wrapping with a strip of gauze (center) and clipping with two clips (right). AChA = anterior choroidal artery; An = aneurysm; II = optic nerve; PCoA = posterior communicating artery.

![Figure 2](image2.png)

**Fig. 2.** Case 2. *Left:* Right carotid angiogram showing a faint bulge (arrow) on the anteromedial wall of the ICA. *Right:* Postoperative angiogram obtained on the Day 14 after the first angiogram, showing growth of the aneurysm (arrow) extending posteriorly in the lateral view.
oped aneurysm with a tough organized clot behind the ICA bifurcation. The aneurysm was clipped with a fenestrated Sugita clip over the right MCA after dissecting the anterior choroidal artery and the right ACA from the aneurysm.

**Postoperative Course.** The patient did well initially, but his condition deteriorated a few days later. Repeated angiography on the 6th day after the second operation revealed no recurrence of aneurysm; however, the patient experienced a spasm of the right ACA and entered a vegetative state.

**Case 3**

**History and Examination.** This 43-year-old man had a sudden-onset headache, slipped down a flight of stairs, and hit his back. A CT scan was obtained and showed a thick and diffuse subarachnoid clot. Physical examination revealed no scalp wound but a bruise on the patient’s back. He was drowsy and mildly hemiparetic on the left side. Angiographic studies obtained on the following day revealed a questionable bulge in the anteromedial wall of the right ICA (Fig. 3 left), and the patient was treated conservatively. Repeated angiography on Day 14 after the first angiogram showed an aneurysm that had grown superiorly from the bulging of the ICA (Fig. 3 right). The patient underwent a balloon occlusion test for 20 minutes. Neurological examination and electroencephalographic studies showed no changes; however, the stump pressure was as low as 45 mm Hg, whereas the systemic blood pressure was 120 mm Hg.

**Operation.** On the 31st day after the onset of headache, frontal and parietal branches of the right STA were anastomosed to the frontal and temporal arteries, respectively, of the right MCA. The right cervical ICA was occluded using the endovascular technique in the same manner as in Case 1.

**Postoperative Course.** The patient tolerated the procedure well. He had a resolving left-sided hemiparesis when he was discharged 1 month postsurgery. No sign was seen of aneurysm of the ICA on postoperative angiographic studies, although the distal ICA was visualized via the ophthalmic artery.

**Discussion**

**Terminology and Clinical Features**

Aneurysms arising from the anterior wall of the ICA are rare, reportedly comprising 0.3 to 1% of intracranial aneurysms or 0.9 to 6.5% of aneurysms of the ICA.10,19 The patients often suffer from hypertension and have atherosclerotic cerebral arteries.6,7,15 The lesions are located at the curvature of the ICA and not at the arterial division where berry aneurysms generally originate. The term “internal carotid (IC) dorsal wall aneurysm” was once preferred because it implies that the lesion develops at the greater curvature of the bend of the ICA.5,11 However, because the term “dorsal” is traditionally never used in relation to intracranial arteries, the terms “IC anterior wall aneurysm” or “superior wall aneurysm” have begun to be used in recent Japanese literature.6,11 Other terms describing these lesions are “sclerotic cerebral aneurysm”12,15 and “IC distal medial wall aneurysm.”19 These aneurysms have been classified into two groups according to shape: one is a small hemispherical bulge that is called a BBA and the other is a saccular type aneurysm. A BBA is also called a semifusiform aneurysm11 or a blisterlike aneurysm.4,18 Aneurysms at this particular location have been reported to have a fragile wall regardless of their shape.5,11,19

The BBAs of the ICA found in our patients had certain unique characteristics. They were located at the anterior wall of the supraclinoid portion of the ICA. Although these patients presented with massive hemorrhage in most cases, initial angiograms obtained soon after SAH showed only a small bulge, which progressed within a few weeks to a saccular appearance in three cases. The walls of these aneurysms were so thin and fragile that they easily lacerated at the base when they were clipped in the acute stage.

**Pathological Considerations**

It appears from measurements that the wall thickness of berry aneurysms is proportional to the aneurysm radius.14 Early berry aneurysms therefore have a very thin aneurysm wall, although the risk of rupture of these tiny aneurysms is considered to be extremely small. The wall of berry aneurysms consists largely of collagen, and it can
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FIG. 4. Illustrations demonstrating structural differences among three types of aneurysms. The BBA (left) is a focal wall defect covered with thin fibrous tissue located where the vessel displays the most curvature, generally associated with atherosclerosis. The wall of a berry aneurysm (center) is composed of a thickened intima and adventitia. In a dissecting aneurysm (right), the wall is dissected for a certain distance, and both stenotic and dilated portions can be observed.

tolerate very high pressure unless fibrinoid necrosis or hemorrhage takes place within it, because collagen is much stiffer than elastin and has a breaking point that is higher than that of elastin or smooth muscle. 13,14

The demonstration on angiography of a small bulge and its rapid evolution on repeated studies in our patients, as well as in several reported cases of BBAs, indicates extreme weakness of the aneurysm wall.10,16,17 Operative findings that the wall can be lacerated easily during manipulation also support this evidence. This marked weakness of the wall seems to constitute the very nature of BBAs that differentiates these lesions from berry aneurysms. In an autopsy study of similar cases it was demonstrated that these lesions are focal wall defects covered with thin fibrous tissue14 and are therefore not true aneurysms. A true aneurysm is formed by an attenuated arterial wall that grows to become saccular and has one or two layers of normal arterial structures within it. A berry aneurysm has a thickened intima and adventitia in its wall.13,14 Blood blisterlike aneurysms and saccular-type aneurysms at the anterior wall of the ICA were once thought to be dissecting aneurysms,10 however, a dissecting aneurysm typically shows an intimal flap and a false lumen, which were not demonstrated in our patients or in similar cases in the literature.15 The structural differences between BBAs, berry aneurysms, and a dissecting aneurysm are illustrated in Fig. 4.

A BBA tends to progress to a saccular appearance.10,16,17 This saccular lesion seems to result from a blood clot covering a focal wall defect; the blood clot subsequently organizes and grows to become a saccular lesion. The lesion has none of the normal arterial structures in its wall and therefore a false aneurysm. There is a possibility that the saccular-type anterior wall aneurysm that is detected on post-SAH angiographic studies is actually a lesion that results from progression of a BBA.

Among various causal factors, hemodynamic stress as well as atherosclerosis seem to be important in the formation of a BBA, because the anteromedial wall of the supraclinoid portion of the ICA is curved where the flux of blood flow impinges on the arterial wall.6,7,15,16 Morphological changes that take place in a BBA are more destructive than those seen in berry aneurysms. Stanson, et al.,12 have described an atherosclerotic lesion with ulceration that penetrated the internal elastic lamina and allowed hematoma formation within the media of the aortic wall, and they designated it as a penetrating atherosclerotic ulcer. This may have been a focal dissection, but Stanson, et al., distinguished it from both classic dissection and aneurysm formation because the clinical features are quite different. We speculate that ulceration and hemorrhage play important roles in the development of focal wall defects in BBAs, in the same way that penetrating atherosclerotic ulcers are produced in the descending thoracic aorta.

Treatment Options

It has been reported repeatedly that direct clipping of a BBA often causes rupture of the lesion, resulting in an apparent wall defect of the feeding artery.6,10,16,17 Various neurosurgical tactics have been proposed to treat a BBA. First, the cervical ICA should be exposed for pressure control of this vessel. The lesion should be carefully dissected; a subpial dissection of the frontal lobe attached to the lesion is advised.5 If clipping is attempted, the clip should be applied under decreased pressure of the ICA with the clip blades parallel to the parent artery and catch-
ing the arterial wall beyond the lesion. A Sundt clip–graft may be useful, provided that the segment of the ICA is free of branches or that the branch can be preserved using the window of the clip graft. Either complete wrapping or clipping of the lesion reinforced by wrapping has been applied most often for this kind of lesion. Previous reports have described successful treatment by this method with materials such as gauze, muscle, muscle fascia, cellulose fabric, and Gore-Tex (W. L. Gore & Associates, Inc., Flagstaff, AZ). It is difficult to select the most appropriate wrapping material to reinforce the wall of this kind of lesion. We failed to prevent growth of aneurysms in two cases. In retrospect, it seemed that the border of the lesion was difficult to identify on the medial side and wrapping with a strip of gauze was not sufficient to reinforce the very fragile aneurysm wall, if clip blades could not catch the wall of the parent artery. Angioplasty by suturing after temporary trapping was reported to be successful after intraoperative rupture because the edge of the lesion was relatively well outlined. However, a disastrous outcome due to ischemia during angioplasty of the ICA has also been reported. Preparation for extracranial–intracranial bypass is recommended before direct manipulation of the lesion.

Surgery in the chronic stage is safer than in the acute stage because a clot covering the lesion organizes. As is shown in two of our cases, endovascular occlusion of the cervical ICA in the chronic stage with or without bypass surgery seems effective and less risky than direct surgery. Reduced blood flow in the supraclinoid ICA may cause thrombosis of these aneurysms and prevent rerupture.

Conclusions

We describe unique characteristics of a BBA of the ICA in six cases. The initial angiograms obtained soon after SAH showed only a small bulge from the anterior wall of the supraclinoid portion of the ICA, although these patients presented with massive SAH in most cases. The lesion progressed within a few weeks to a saccular appearance in three cases. The walls of the BBAs were so thin and fragile that they were easily lacerated at the base during clipping in the acute stage. We propose that BBA is a new entity that produces SAH and needs special neurosurgical tactics for treatment.

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References


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