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# Long-term follow-up analysis of microsurgical clip ligation and endovascular coil embolization for dorsal wall blister aneurysms of the internal carotid artery



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# ABSTRACT

Blister aneurysms at non-branching sites of the dorsal internal carotid artery (dICA) are fragile, rare, and often difficult to treat. The purpose of this study is to address the demographics, treatment modalities, and long-term outcome of patients treated for dICA blister aneurysms. A retrospective review of medical records identified all consecutive patients who presented with a blister aneurysm from 2002 to 2011 at our institution. Eighteen patients (M = 7, F = 11; mean age: 48.4 ± 15.1 years; range: 15–65 years) harbored a total of 43 aneurysms, 25 of which were dorsal wall blister aneurysms of the ICA. Eleven (61.1%) patients presented with aneurysmal subarachnoid hemorrhage (aSAH), and 10 (55.6%) patients had multiple aneurysms at admission. Twelve patients had 18 aneurysms that were treated microsurgically. Five (41.7%) of these patients had a single recurrence that was retreated with subsequent repeat clip ligation. Six patients had 7 blister aneurysms that were treated with endovascularly. One (16.7%) of these patients had a single recurrence that was retreated with subsequent coil embolization. Postoperative vasospasm occurred in 8 (44.4%) patients, one of whom suffered from a stroke. This is one of the largest single-institution dICA blister aneurysm studies to date. There was no detected significant difference between microsurgical clip ligation and endovascular coil embolization in terms of surgical outcome. These blister aneurysms demonstrate a propensity to be associated with multiple cerebral aneurysms. Strict clinical and angiographic long-term follow-up may be warranted.

Statement of Significance: Blister aneurysms are focal wall defects covered by a thin layer of fibrous tissue and adventitia, lacking the usual collagenous layer. Due to their pathologically thin vessel wall, blister aneurysms are prone to rupture. The management of these rare and fragile aneurysms presents a number of challenges. Here, we address the long-term outcome of patients treated for blister aneurysms at nonbranching sites of the dICA. The presented data and analysis is imperative to determine the necessary strict long-term clinical and angiographic follow-up.

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# 1. Introduction

Blister aneurysms are focal wall defects covered by a thin layer of fibrous tissue and adventitia, lacking the usual collagenous layer. These aneurysms have a very high risk of intraoperative rupture and are known for causing large lacerations in the parent vessel wall during clip deployment [6]. Their small size, fragility, and ill-defined necks make treatment a unique challenge in terms of approach, exposure, and exclusion modality [11,18,1,7].

Blister aneurysms of the dorsal internal carotid artery (dICA) typically rupture with resultant acute neurological deficits [19]. Although originally described at the non-branching segment of the dICA, blister aneurysms have been observed in the atypical locations of the anterior communicating, middle cerebral, basilar, posterior cerebral, anterior cerebral, and posterior inferior cerebellar arteries [15].

In most surgical analyses, the intraoperative and postoperative rebleeding rates range from 33–80% [14]. Approximately 0.3–1.0% of all intracranial and 0.9–6.5% of all ruptured ICA aneurysms are

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blister aneurysms, while the estimated annual risk of rupture is 0.7% for intracranial saccular aneurysms [16,12,17]. No retrospective study to date has evaluated the annual risk of rupture for blister aneurysms, but it is suggested that rates exceed those of other aneurysm morphologies [12,3,5].

Various treatment options exist for blister aneurysms including primary surgical clip ligation, wrapping, clipping with cotton reinforcement, parent vessel sacrifice with or without bypass, endovascular coiling, and conservative management [8,9]. Each of these interventions carries reasonable risks of complications or failure, yet little is known about the comparative long-term success rate and efficacy among microsurgical and endovascular options [11,18,10].

Both microsurgical and endovascular treatment modalities for blister aneurysms carry a high risk of morbidity and mortality [12,2]. Although there have been reports suggesting positive outcomes in patients treated via microsurgical clip ligation, longterm outcome studies are relatively limited due to the rarity of blister aneurysms [3,9]. This study examines the long-term follow-up results of the patients treated for blister aneurysms of the dICA. We primarily analyzed patient characteristics with respect to microsurgical and endovascular treatment modalities and explored other associated correlations.

# 2. Material and methods

We performed a retrospective review of patients who were diagnosed with dorsal wall-projecting blister aneurysms at nonbranching sites of the dICA. All patients were initially diagnosed with a blister aneurysm via CT-angiography and subsequently treated by means of either microsurgical clip ligation or endovascular intervention. Goodman Campbell Brain and Spine at Indiana University Department of Neurological Surgery prospectively maintains a cerebral aneurysm database since 1976. We identified 22 consecutive patients with at least one dICA blister aneurysm between 2002 and 2011.

In this study, we assessed patient demographics, Fisher grade, Hunt and Hess scores at admission, preoperative presentation, treatment modalities, postoperative complications, and outcomes. Glas-

#### Table 1

### Preoperative characteristics.

cow Outcome Scale (GOS) scores at discharge, 6 months, and at 1 year were also recorded. Long-term follow-up was defined as availability of imaging >1 year from the time of the initial surgery. We defined blister aneurysms as shallow, broad-based aneurysms that projected from the dorsal wall at non-branching sites of the ICA.

Data analysis was performed using SPSS (version 19.0, SPSS Inc.) and Microsoft Excel (2010). Statistical significance was set at a probability value  $\leq 0.05$ . Data is presented as means ± standard deviations. We performed a Mann-Whitney *U* test on nonparametric ordinal data including GOS at discharge, 6-month, 1-year follow-up, and last recorded GOS scores.

# 3. Results

We identified 22 consecutive patients (M = 8, F = 14; mean age: 48.4 ± 14.5 years; range: 15–65 years) who harbored 50 aneurysms, 30 of which were dICA blister aneurysms. Four patients died before treatment could be rendered and were not included in the analysis. Our final cohort therefore contained 18 patients. This group harbored a total of 43 aneurysms, 25 of which were dICA blister aneurysms. The mean size of these blister aneurysms was 4.4 mm (median: 3 mm; range: 1.0–14.5 mm), and all were dorsal wall-projecting at non-branching segments of the ICA. Preoperative characteristics for individual patients are displayed in Table 1.

Relevant clinical data and surgical outcomes are summarized in Table 2. Eight patients had blister aneurysms identified incidentally while being treated for other pathologies, and 2 patients had blister aneurysms identified after unrelated trauma. There were 11 (61.1%) patients who presented with aneurysmal subarachnoid hemorrhage (aSAH). The mean Hunt and Hess grade at presentation was 1.7 (median: 2; range: 0–4), and the mean Fisher grade on admission was 3.2 (median: 3; range: 3–4). Notably, 11 (50.0%) patients presented with multiple aneurysms on admission.

Treatment modality analysis is presented in Table 3. Microsurgical clip ligation was used for 12 patients who harbored a total of 18 blister aneurysms (Fig. 1). There was one case of intraoperative rupture during microsurgery and no cases of postoperative

Patient number	Age (y)	Sex	Presentation	HH grade	Fisher grade	Total aneurysms	Bilster aneurysms	Blister aneurysm size (mm)	Side	Location	Other aneurysms
1	47	F	sSAH	3	3	2	1	1	L	ICA	Not specified
2	64	F	sSAH	1	3	2	1	N/A	R	ICA	R. SCA
3	48	F	Incidental	-	1	8	3	L: 4, 1; R: 2	В	L: PC, ICA; R: PC	L Anterior Choroidal, L MCA, L S. Hypophyseal; R MCA, R Pcomm
4	49	F	Incidental	-	1	5	2	L: N/A; R: 3	В	ICA	L SCA, Bilateral Vertebral
5	15	Μ	Trauma	-	1	1	1	14.5	L	ICA	-
6	63	F	sSAH	2	3	2	1	3	R	PC	L Acomm
7	36	Μ	Incidental	3	3	2	2	L: 1; R: 5.4	В	PC	-
8	61	F	sSAH	4	4	1	1	8.7	L	SC	-
9	59	F	Incidental, sSAH	2	3	6	3	L: 7; R: 8, 3	В	L: ICA; R: PC, ICA	L S. Hypophyseal; 2 R Cavernous
10	30	Μ	sSAH	2	3	1	1	N/A	R	ICA	-
11	23	F	sSAH	3	3	1	1	N/A	R	ICA	-
12	65	F	Incidental	-	1	2	1	6	L	PC	R S. Hypophyseal
13	44	Μ	tSAH	1	3	1	1	1.5	R	SC	-
14	44	Μ	sSAH	1	3	1	1	9	R	PC	-
15	40	Μ	Incidental	-	1	1	1	3	L	PC	-
16	64	F	Incidental	-	1	4	2	6, 6	L	ICA	L Opthalmic, L S. Hypophyseal
17	62	F	Incidental	-	1	2	1	1.5	R	PC	Cavernous
18	57	Μ	sSAH	3	3	1	1	N/A	R	ICA	-

tSAH, traumatic subarachnoid hemorrhage; sSAH, spontaneous subarachnoid hemorrhage; HH, Hunt and Hess; L, left; R, right; B, bilateral; ICA, internal carotid artery; PC, paraclinoid region; SC, supraclinoid region; N/A, non-applicable; S, superior; SCA, superior cerebellar artery; MCA, middle cerebral artery; Pcomm, posterior communicating artery; Acomm, anterior communicating artery.

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