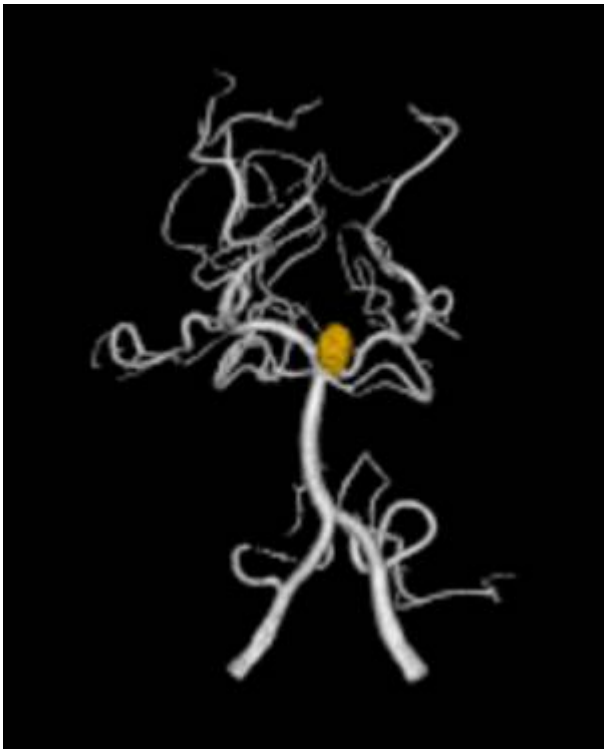
Basilar bifurcation aneurysm



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AKA Basilar apex aneurysms, including basilar tip aneurysm and basilar artery-superior cerebellar artery (BA-SCA) aneurysms.

Epidemiology

Basilar bifurcation aneurysm epidemiology.

Pathogenesis

The pathogenesis of basilar apex aneurysm (BAA) are still poorly understood. Embryologically, basilar apex anatomical disposition is formed by the fusion of both caudal internal carotid divisions on the midline. To compare basilar apex morphology by embryological classification among patients with BAAs, anterior circulation aneurysms (ACAs), and controls. Prospectively collected data of 47 consecutive patients with unruptured BAAs (42 females and five males), age- and gender-matched 47 patients with unruptured ACAs, and 47 controls without any aneurysms were analyzed. Based on embryology, basilar apex morphology was classified into symmetric cranial fusion (SCrF), symmetric caudal fusion, and asymmetric fusion type. Posterior communicating artery (Pcom) was classified into hypoplastic, adult, or fetal type. The asymmetrical Pcom was defined as bilaterally different type Pcom. The ACAs located at the anterior communicating artery (n = 18), paraclinoid portion (n = 12), middle cerebral artery (n = 8), anterior cerebral artery (n = 5), the top of internal carotid artery (n = 2), and anterior choroidal artery (n = 2). Compared with the ACA group and controls, smoking, asymmetrical Pcom (fetal and adult type), and SCrF type were more prevalent in patients with BAAs by residual analysis. The multinomial logistic regression comparative analysis demonstrated that SCrF type was associated with BAAs (vs. ACA group; odds ratio, 13; 95% confidence interval, 3.8-41 and vs. controls; odds ratio, 25; 95% confidence interval, 5.4-121). The assessment of basilar apex morphology may aid in the understanding of the pathogenesis of BAA and the prediction of BAA formation [1].

BA bifurcation aneurysms are significantly associated with patients' age, female sex, wider bifurcation angles, and smaller vascular diameter at the BA bifurcation [2].

Wide-Necked Basilar tip Aneurysm

Wide-Necked Basilar tip Aneurysm

Rupture risk

Bifurcation angle and aspect ratio are independent predictors for aneurysm rupture. Bifurcation angle, which does not change after rupture, is correlated with hemodynamic factors including inflow coefficient and WSS, as well as rupture status. Aneurysms with the hands-up bifurcation configuration are more prone to rupture than aneurysms with other bifurcation configurations [3].

High flow conditions, characterized by large and concentrated inflow jets, complex and oscillatory flow patterns, and wall shear stress distributions with focalized regions of high shear and large regions of low shear, are associated with aneurysm rupture, especially for basilar tip aneurysms. The higher flow conditions in basilar tip aneurysms could explain their increased rupture risk compared with internal carotid bifurcation aneurysms [4].

Clinical features

Most present with SAH indistinguishable from SAH due to anterior circulation aneurysmal rupture. Enlargement of the aneurysm prior to rupture may rarely compress the optic chiasm →bitemporal field cut (mimicking pituitary tumor), or occasionally may compress the oculomotor nerve as it exits from the interpeduncular fossa →oculomotor nerve palsy.

These aneurysms can cause a variety of clinical features, depending on their size and location, and may present with symptoms such as:

Headache: A sudden, severe headache is a common symptom of a basilar bifurcation aneurysm.

Vision problems: The aneurysm may press on the nerves that control vision, causing blurred vision, double vision, or loss of vision in one eye.

Cranial nerve dysfunction: The aneurysm may compress or damage cranial nerves, leading to facial weakness, hearing loss, difficulty swallowing, or difficulty speaking.

Nausea and vomiting: An aneurysm can cause pressure on the brainstem, leading to nausea and vomiting.

Loss of consciousness: In severe cases, a basilar bifurcation aneurysm may cause loss of consciousness, seizures, or coma.

Sensory and motor deficits: The aneurysm may press on the brainstem or other parts of the brain, leading to sensory and motor deficits such as numbness, tingling, weakness, or paralysis on one side of the body.

It is important to note that some people with a basilar bifurcation aneurysm may not experience any symptoms at all, and the aneurysm may only be detected incidentally on brain imaging studies.

Diagnosis

CT/MRIscan

May occasionally be seen on CT or MRI as round mass in region of suprasellar cistern. With SAH, tend to see blood in interpeduncular cistern with some reflux into 4th (and to a lesser extent, third and lateral) ventricle. Occasionally may mimic pretruncal nonaneurysmal SAH.

Angiography

Dome usually points superiorly. Should evaluate flow through posterior communicating arteries (may require Allcock test) in case trapping is required. Need to assess the height of the basilar bifurcation in relation to the dorsum sella.

Critical angiographic features to assess: On angiogram or CTA:

1. general features

2. orientation: determines whether surgery is an option. Posteriorly pointing aneurysms obscure perforators which may be adherent to the aneurysm, making surgery more difficult.

3. patency of PCAs & SCAs

4. patency and size of p-comms

a) diameter of p-comm >1 mm is needed to support collateral flow (expert opinion)

b) to determine if the P1's can be sacrificed

c) P-comm patency and size is important for endovascular treatment as a potential route for deployment of horizontally oriented stent extending from P1 to contralateral

d) which can facilitate temporary clipping, or sacrifice, or placement of stents.

5. height of the aneurysm relative to the posterior clinoid process which will affect the selection of surgical approach (the range of height of the posterior clinoid is 4–14 mm)

a) supraclinoidal: aneurysm neck >5 mm superior to posterior clinoid process

b) clinoidal: aneurysm neck within 5 mm of posterior clinoid process

c) infraclinoidal: aneurysm neck >5 mm inferior to posterior clinoid process

Treatment

see Basilar bifurcation aneurysm treatment.

Outcome

If the aneurysm cannot be treated with endovascular technique, then the surgical option can be considered. Overall mortality is 5%, and morbidity is 12% (mostly due to injury to perforating vessels) [5)].

Case reports

A study aimed to report the retrograde technique of horizontal stenting through the PCoA using a Solitaire AB stent.

A self-expandable stent was deployed from one posterior cerebral artery to the opposite, across the neck of a ruptured wide-neck basilar apex aneurysm.

The technique allowed successful aneurysm embolization with coils in a 53-year-old woman.

Different from clipping, where the fetal-type PCoA may pose an obstacle, in endovascular treatment, the Solitaire AB stent provides an alternative route to the aneurysm. It is easy to navigate, being fully retrievable and repositionable, which enables accurate deployment [6].

Case reports from the HGUA

A 69-year-old male admitted for Basilar bifurcation aneurysm. Subarachnoid hemorrhage. [7]

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Title: Acute Subarachnoid Hemorrhage Secondary to a Ruptured Basilar Tip Aneurysm: Endovascular Treatment and Delayed Ischemic Complications

Abstract: We report the case of a 45-year-old male who presented with a thunderclap headache during sexual activity, followed by a rapid decline in consciousness. Imaging revealed an extensive subarachnoid hemorrhage (SAH) secondary to a ruptured basilar tip aneurysm. The patient underwent successful endovascular embolization with stent-assisted coiling. Despite initial stabilization, the patient developed severe vasospasm in both middle cerebral arteries (MCAs) leading to delayed ischemic deficits. This case highlights the challenges in managing SAH-related vasospasm and the role of multimodal interventions.

Introduction: Spontaneous subarachnoid hemorrhage (SAH) is a life-threatening condition, most commonly caused by aneurysmal rupture. SAH associated with basilar tip aneurysms is particularly challenging due to the complex anatomy and high risk of morbidity and mortality. This report presents a case of SAH managed with endovascular treatment, complicated by vasospasm and ischemic stroke.

Case Presentation:

Patient Information: - **Age/Sex:** 45-year-old male. - **Chief Complaint:** Sudden onset severe headache during sexual activity, followed by altered consciousness. - **Initial GCS Score:** 5 (O1, V2, M2). - **Initial Blood Pressure:** 180/103 mmHg. - **Pupillary Response:** Bilateral mydriasis with sluggish response to light. - **Emergency Treatment:** Endotracheal intubation, hemodynamic stabilization, and neuroprotective measures.

Imaging and Diagnosis: - **CT Scan (Day 1):** Extensive SAH in the supratentorial and infratentorial compartments, including Sylvian fissures, interhemispheric fissure, basal cisterns, perimesencephalic region, and fourth ventricle. - **CT Angiography:** Saccular aneurysm at the basilar tip (7.6 mm maximum diameter, 3.5 mm neck). - **Doppler Transcranial Ultrasound (Day 16):** Severe vasospasm in bilateral MCAs.

Treatment and Intervention: 1. **Endovascular Procedure (Day 1):**

1. Stent-assisted coiling via right femoral artery.
2. Neuroform Atlas stents deployed in Y-configuration.
3. Administration of Tirofiban due to intraoperative thrombus formation.

2. **Neurocritical Care:**

1. Invasive intracranial pressure (ICP) monitoring.
2. External ventricular drain (EVD) placement.
3. Blood pressure optimization (target SBP 140–160 mmHg).
4. Nimodipine infusion for vasospasm prophylaxis.

3. **Follow-up CT Scan (Day 5):**

1. Residual SAH with no acute hydrocephalus.
2. Ischemic changes in the right frontoparietal cortex.

4. **Clinical Progression (Day 16):**

1. Persistent somnolence.
2. Hemiplegia on the left side.
3. Complete plegia of the right lower limb.
4. Preserved movement in the right upper limb.
5. Motor aphasia.

Outcome and Follow-Up: Despite aggressive treatment, the patient developed delayed cerebral ischemia (DCI) affecting the right frontoparietal cortex. The external ventricular drain was removed after normalization of ventricular size. Physical rehabilitation was initiated. Further monitoring and consideration of rescue therapies for vasospasm, including intra-arterial vasodilators or balloon angioplasty, were discussed.

Discussion: Basilar tip aneurysms pose unique challenges in management due to their deep-seated location and high risk of complications. This case underscores the need for: - **Early and aggressive management of SAH.** - **Endovascular strategies to achieve aneurysm occlusion.** - **Close monitoring for vasospasm and DCI.** - **Multimodal treatment for vasospasm, including pharmacologic and mechanical interventions.**

Conclusion: This case highlights the complexities of SAH management, particularly in the context of basilar tip aneurysms. While endovascular embolization remains a cornerstone of treatment, delayed ischemic complications can significantly impact neurological outcomes. A multidisciplinary approach is essential for optimizing prognosis.

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