

Large Basilar Artery Aneurysm and Recurrent Stroke: A Case Study and Brief Review

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Abstract

We report a patient with a large basilar artery aneurysm with recurrent strokes who presented to the emergency department with stuttering neurologic symptoms. This presentation is uncommon and associated with numerous considerations with respect to management. Our patient illustrates the need to carefully consider the manifestations of the disease, from the aneurysm, to the potential clot within the aneurysm, to the ischemic disease resultant from embolic/thrombotic episodes prior to recommending treatment.

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Introduction

The clinical scenario of a large (generally considered ≥ 10 mm in diameter) basilar artery aneurysm with stroke is uncommon in patients presenting with ischemic disease [1]. Such aneurysms are associated with a high risk of rupture, and have been related to various risk factors, including hemodynamic factors such as hypertension, congenital weakness of the vessel wall, as well as inherited predisposition (such as in connective tissue diseases) [2]. While various considerations of treatment (e.g. coiling, clipping, embolization) have been utilized in order to treat such aneurysms, consensus regarding the most effective approach has not yet been achieved [3]. We describe a case of a patient with a large basilar aneurysm with embolic strokes and discuss the management of this rare clinical scenario.

Case Presentation

The patient was a 70 year old right handed woman with a previous history of hypertension, 15 pack-year history of cigarette smoking (quit 10 years prior), vulvar cancer (resected 15 years prior without recurrence) and ocular migraine who

presented to the emergency department after experiencing an ocular migraine in the morning of admission. This was associated with vertigo and left sided weakness. Because the symptoms did not remit after about 4 hours, the patient called 911 was brought in by ambulance for further assessment. When the patient arrived to the emergency room, a stroke alert was activated, and the patient underwent a CT scan and CT angiogram (CTA). The patient was not taking anticoagulants. Blood glucose was 112. NIH stroke score was 9, with partial gaze palsy, partial hemianopia, facial droop, left leg weakness, dysarthria and extinction visually. CT scan showed a high density nodule arising from the basilar tip as a saccular aneurysm, measuring 19x15x14mm, which was partially thrombosed; CTA revealed the thrombosed aneurysm, with occlusion of the right P1 segment of the posterior cerebellar artery (Figure 1). The patient was not in the 4.5-hour window for thrombolysis, and the presence of the aneurysm was considered a contraindication. The neuro-interventional and neurosurgical services were consulted, who did not indicate any endovascular or surgical approaches were appropriate at the time.



Figure 1: CT scans. A. CT demonstrates high density nodule arising from the basilar tip as a saccular aneurysm, measuring 19x15x14mm, which was partially thrombosed (arrow). B. CTA revealed the thrombosed aneurysm, with occlusion of the right P1 segment of the posterior cerebellar artery (arrow).

The patient was begun on aspirin 325 mg as per stroke protocol, but no permissive blood pressure was allowed, with target systolic blood pressure <150mmHg. An MRI was performed, which demonstrated the thrombosed aneurysm at the basilar tip, and multiple foci of acute ischemia, including the right midbrain, right medial thalamus, right occipital lobe, right

temporal lobe, and right cerebellar hemisphere (Figure 2). The patient was continued on aspirin with stabilization of her headache, with improvement of the gaze palsy and dysarthria, but with persistence of weakness and visual symptoms. Follow up with the neuro-interventional service as an outpatient was scheduled after rehabilitation.

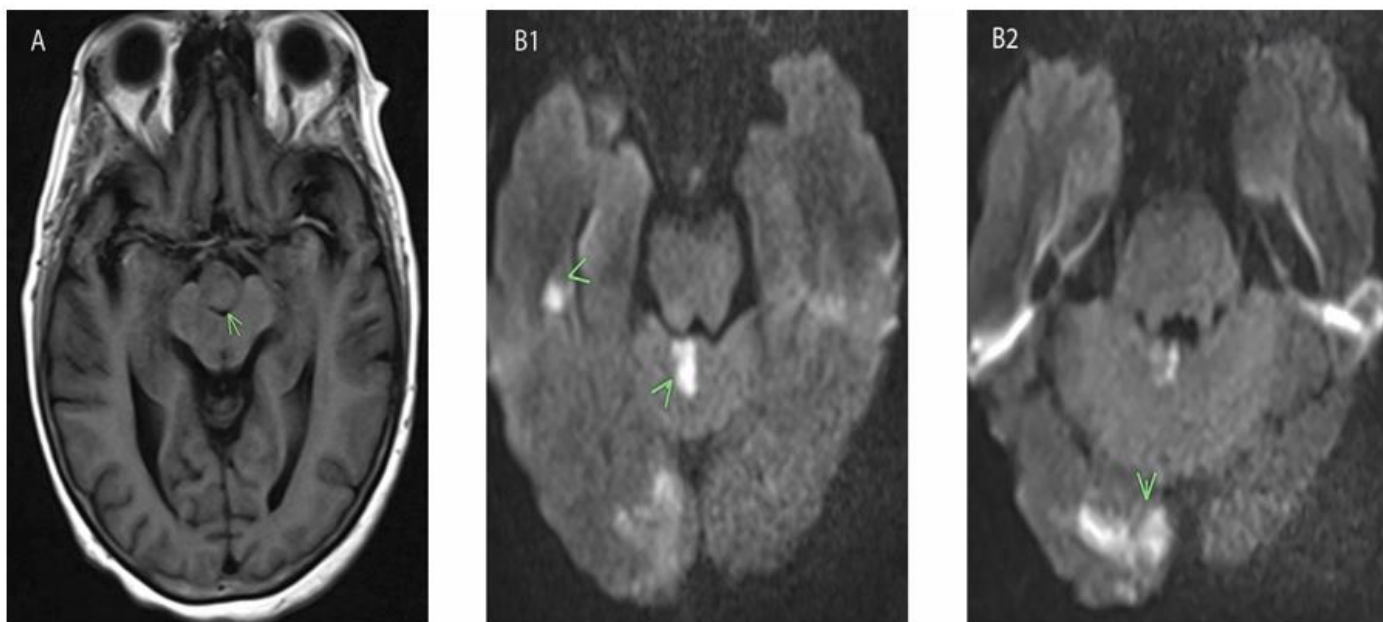


Figure 2: MRI scan. A. T1 sequence. Thrombosed aneurysm (arrow). B. Trace sequence. Evidence of acute ischemia in the right temporal lobe and cerebellum (B1) and occipital lobe (B2) (arrows).

Upon return home, the patient discontinued all medications, including aspirin against medical advice. Subsequently, the patient re-entered the emergency department two weeks later with new stroke-like symptoms, including worsening speech, hemiparesis, and confusion. An MRI of the brain was obtained, which demonstrated new right dorsal midbrain, superior cerebellar, and right dentate nucleus acute infarcts, with multiple

evolving subacute infarcts and increased size of the thrombus within the basilar tip aneurysm (Figure 3). Despite more aggressive anti-platelet therapy, the patient worsened with progressive encephalopathy and decreased awareness; the patient's family opted for comfort measures only, and the patient expired shortly thereafter.

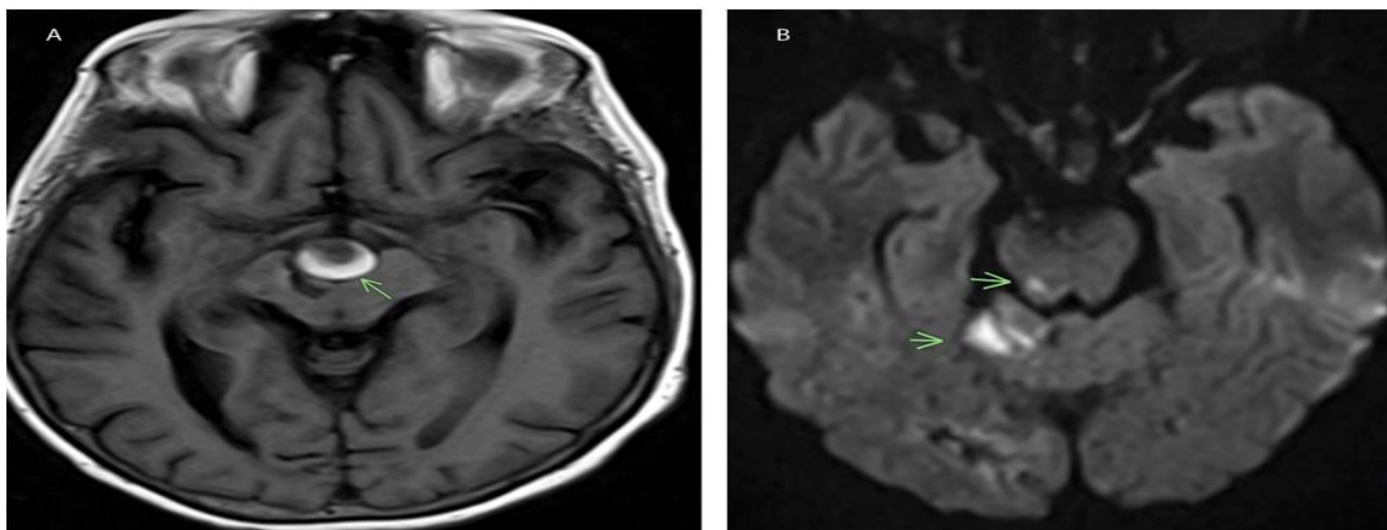


Figure 3: Repeat MRI. A. T1 sequence, showing altered signal characteristics and volume indicating increased size of thrombus (arrow). B. Trace sequence. New right dorsal midbrain and superior cerebellar acute infarcts demonstrated (arrows).

Discussion

Basilar artery aneurysms associated with stroke are a rare presentation of ischemic disease, but one which are complicated with regard to management. The usual aspects considered for management of thrombosed unruptured aneurysms (e.g. blood pressure control, single anti-platelet therapy, limited consideration of anti-coagulants) are in contrast to that for ischemic disease alone of the nervous system (e.g. permissive blood pressure, dual anti-platelet therapy, anti-coagulation with atrial fibrillation). The clinician must be cognizant of both avoidance of extension of ischemia (and recurrence) balanced with the avoidance of increasing the risk of aneurysm rupture, both with catastrophic consequences. While treatment options include medical, endovascular and neurosurgical modalities, there is little consensus on what approaches are optimal. A multidisciplinary approach, considering not only the clinical scenario and risks of rupture, as well as need for other mitigation of ischemic stroke (such as presence of atrial fibrillation), and expertise (such as for neurosurgical and endovascular approaches) is paramount in management of these patients.

Risk factors for intracranial aneurysm rupture

Many reviews have identified the key risk factors of rupture. Some challenges exist due to differing definitions of various aspects of scoring (e.g. size of aneurysm). In general, the key components as noted are size (“large” e.g. ≥ 10 mm diameter versus “small” < 7 mm diameter) and location (posterior circulation being the highest, and cavernous carotid and middle cerebral artery being the lowest). Additional risk factors include smoking, hypertension (either un- or poorly controlled), history of growth over time or previous subarachnoid hemorrhage, and structure as daughter sac or bleb [4]. Unfortunately, while scales have been developed for prediction of risk of aneurysm rupture, none has been validated with high sensitivity and specificity to be useful routinely in the clinic [5].

Medical therapy

As noted, detection of aneurysms presents with potential management dilemmas given the risk of rupture. With greater sizes, such as those large aneurysms as in this case, the risk is greater [6], with posterior fossa location having an even higher risk. The treatment of large aneurysms has not been specifically

addressed, and treatment is extrapolated from the overall general data from intracranial aneurysms.

Anti-thrombotics

Medical management typically includes anti-thrombotic therapy, with agents decreasing the (hypothetical) inflammatory mediators and has been shown to potentially decrease the risk of rupture [7]. While some have argued reducing thrombus formation can be associated with an *increase* in rates of rupture and complicate hemorrhagic complications [8], use of aspirin at least thrice weekly was found to have a lower risk of aneurysm rupture [7], and further, amongst patients with ischemic stroke, having an intracranial aneurysm did not result in increased risk of complications with anti-platelet therapy [9]. Moreover, use of anti-platelet treatment in those patients undergoing invasive treatments for aneurysms, including large aneurysms, were not associated with increased rupture [10,11] with lower complication rates [12]. Use of aspirin seems reasonably safe in this population.

Thrombolysis

The use of thrombolytic agents in aneurysm treatment in general is less well studied compared to that of anti-thrombotics. The presence of an intracranial aneurysm is a contraindication for both tissue plasminogen activator (tPA) and tenecteplase [13,14]. No prospective treatments nor large retrospective studies have evaluated this question, but two small studies [15,16] did not find use of tPA associated with increased risk of hemorrhage. Hence, while no definitive conclusions can be drawn, such small studies (n=22 and n=11, respectively) suggest further evaluation is warranted for use of thrombolytics in these patients when presenting with ischemia.

Anti-coagulants

The data with respect to anti-coagulants in the treatment of aneurysms, large or otherwise, is also limited and conflicting. While one study in patients with ischemic stroke treated with anti-coagulants in the presence of an intracranial aneurysm (including large posterior fossa aneurysms) was not associated with increased adverse events [9], a case controlled population based study in contrast did show increased rupture of aneurysms in patients receiving short term (less than one month) of vitamin K antagonists, which diminished with longer term use [17]. Moreover, patients who do have subarachnoid hemorrhage

while being treated with anti-coagulants perform significantly worse clinically [18], although other studies have not confirmed this [19]. Treatment with anti-coagulants must thus be clearly individualized, *vis-a-vis* other clinical factors requiring such therapy, and the risk of rupture of a present aneurysm.

Blood pressure management

The balance between maintaining cerebral perfusion pressure whilst minimizing risk of increasing risk of a compromised vessel wall in the context of an aneurysm is a key understanding for those managing these patients. Rigid control of blood pressure has been shown in a variety of studies to protect against aneurysm rupture [20,21]. Indeed, both as a treatment and a modifiable risk factor, hypertension has been shown to be present in over half those with unruptured intracranial aneurysms [20]. Recent data has shown significant declines in rupture risk, including those with the highest risk (large, posterior fossa location) with aggressive treatment of blood pressure [22].

Recently, in a 3044 patient retrospective study, intriguing data suggests the use of renin-angiotensin-aldosterone system (RAAS) inhibitors have a particularly effective preventative effect in aneurysm rupture [23]. Compared to non-RAAS inhibitor antihypertensive agents, the use of angiotensin-converting enzyme inhibitors/receptor blockers was associated with higher reduced rupture risk, including in those patients with large (≥ 5 mm diameter) aneurysms. Further prospective studies evaluating the reduction of blood pressure with RAAS inhibitors *versus* other antihypertensive agents will be interesting to evaluate the generalizability of this result. Regardless, the prompt normalization of blood pressure in patients with intracranial aneurysms is a key *de rigueur* aspect of management of these patients.

Invasive approaches

Both surgical and endovascular treatments of aneurysms, both large and small, have been used with good results. Generalities have been made about both approaches, but these have been mostly from observational studies rather than randomized evaluations. However, a recent report [24] evaluated surgical clipping and endovascular approaches in a randomized non-blinded multicenter trial. This study used a composite primary adverse outcome of failed aneurysm occlusion, intracranial hemorrhage during follow up, and residual aneurysms at one year. The study revealed surgical clipping was associated with less such adverse outcome (13/142, 9%) than endovascular treatment (28/148, 19%) [relative risk (RR): 2.07; 95% confidence interval (CI) 1.12-3.83; $p=0.021$]. While there was no difference in mortality/morbidity at 1 year (2% per group), post-procedure neurologic deficits (22% *versus* 12%, RR: 1.74; 95% CI 1.04-2.92, $p=0.04$) and hospitalizations >5 days (48% *versus* 8%, RR 0.18; 95% CI 0.11-0.31; $p<0.001$) was higher for the surgical compared to the endovascular group, respectively.

A recent meta-analysis evaluated 114 studies which described either endovascular treatments and surgical therapy for unruptured intracranial aneurysms [25]. The types of studies were mostly observational, and revealed a higher complication rate for surgical approaches *versus* endovascular treatments (8.3% *versus* 5%, respectively). For surgical cases, complications were associated with high age, vascular risk factors (diabetes, smoking, hypertension, congestive heart failure), female sex, posterior circulation location, calcification in the aneurysm, and use of anti-coagulants/coagulopathy. For

endovascular cases, similarly complications were associated with female sex and diabetes; in addition, any cardiac morbidity and wide aneurysm neck were also associated with complications. Mortality was 0.1% in surgical patients, and 0.3% in endovascular cases. Hence, both surgical clipping and endovascular treatments are both safe and effective in patients with aneurysms, both small and large, and can be used in appropriate patients in combination of maximal medical therapy and risk reduction strategies.

Conclusion

Basilar artery aneurysms, large and small, represent especially in the context of stroke, a challenging clinical scenario, involving medical, endovascular and neurosurgical approaches. Current recommendations suggest balancing the clinical characteristics of the patient with respect to risk factors for aneurysm hemorrhage *versus* stroke recurrence. Aspirin seems to have a relative safe profile, with data suggesting it may even have protection against rupture. Anticoagulation has a small but definite risk of worsening aneurysmal rupture and hemorrhage, but may be appropriate in certain scenarios. Clipping, coiling, embolization are all important components of the armamentarium of the treating clinician, but careful selection of patients is required to avoid worsening prognosis. The need for considered multidisciplinary approach and expertise is paramount in the treatment of these patients.

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Author contributions

SL conceived, collected data and wrote/reviewed the manuscript; BL conceived, collected data, wrote/reviewed and supervised the creation of the manuscript.

Statements and Declarations

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