Basilar Artery Enlargement in an Elderly Female Cadaver

Abstract

An asymptomatic enlarged basilar artery was identified during an educational dissection of a 76-year-old female cadaver. The basilar artery had a uniform arterial wall without evidence of atherosclerotic plaque, intraluminal thrombus, or focal aneurysmal outpouching. The pons and cerebellum showed no signs of infarction or ischemia. The cause of death, malignant neoplasm of the lung, was unrelated to the vascular finding. The current case highlights that vascular pathology can occur without subsequent symptoms. Early identification, coupled with management of modifiable risk factors such as hypertension, hyperlipidemia, and smoking, may decrease the risks of the pathology progressing into strokes.

Introduction

The ventral surface of the pons is covered by the basilar artery, which results from the convergence of two vertebral arteries and provides blood to the brainstem and cerebellum [1]. Dilatation of the basilar artery can be caused by vertebrobasilar dolichoectasia (VBD) or compensatory changes in atherosclerosis. VBD is associated with degeneration of the internal elastic lamina and thinning of the arterial media [2], which may pose a significant clinical risk in the form of ischemic strokes, aneurysm formation, or fatal hemorrhage [3]. The natural history of VBD is progressive, with worsening arterial dilation and increased risk of adverse events over time [4]. A 5-year prognosis is favorable in patients who are asymptomatic at time of diagnosis.

The 2021 American Heart Association/ American Stroke Association practice guidelines suggest patients be treated medically, with strict blood pressure control and use of antithrombotic agents [5]. Surgical and endovascular interventions are rarely performed and lack clinical efficacy [6]. We report a case of an enlargement of the basilar artery found during anatomic dissection.

Case Report

A 76-year-old female cadaver with an enlarged basilar artery was discovered during a routine educational dissection in the gross anatomy laboratory at Philadelphia College of Osteopathic Medicine. The cause of death, malignant neoplasm of the lung, was not related to the vascular finding. The family of the female as well as the body donation program granted permission to allow the case to be published.

The basilar artery measured 33.1 mm in length over the ventral aspect of the pons. The central segment showed a maximum diameter of 8.5 mm, the rostral segment measured 3.9 mm, and the caudal segment measured 4.4 mm (Figure *1A*). Dissection revealed no evidence of intraluminal plaque (Figure *1B*). The arterial wall appeared uniform in thickness across rostral, central, and caudal segments, with no signs of dissection or wall separation. Branches of the basilar artery, (i.e., pontine, anterior inferior cerebellar artery, superior cerebellar artery) were normal in caliber and morphology. No evidence of infarction or ischemia were observed in the adjacent pons or cerebellum.

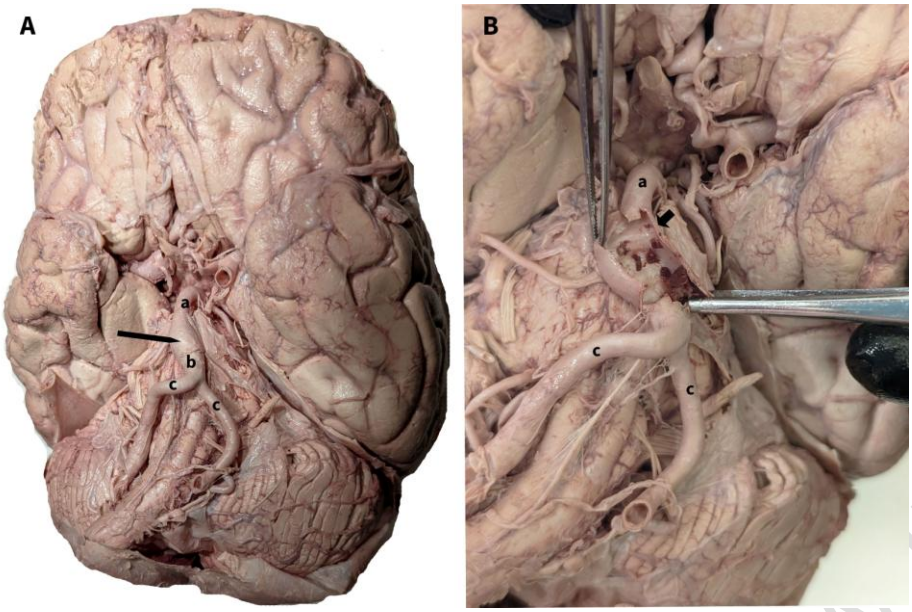

Figure 1. *1A shows an enlarged basilar artery (line) with normal rostral (a) and caudal (b) diameters. Figure 1B highlights that the wall of the enlargement is uniform in thickness (arrow). Labels: a. rostral basilar artery, b. caudal basilar artery, c. vertebral arteries.*

Discussion

The average basilar artery width is 3.6-3.9 mm [7]. In this case, the basilar artery measured 8.5 mm along the central aspect of the pons, indicating pathologic distension. There are numerous modifiable risk factors that contribute to the development of vascular pathology: diabetes, smoking, hypertension, hyperlipidemia, and obesity [8]. Many patients have many of these risk factors. Nicotine use, for instance, is closely linked to hypertension and is frequently accompanied by concurrent alcohol consumption. Similarly, obesity and hypercholesterolemia are commonly observed together, further compromising vascular integrity [8]. Both hypertension and smoking significantly alter intracranial blood flow patterns and are strongly associated with aneurysm development [9]. Damage to the arterial wall is a key contributor to arterial enlargement, which can ultimately lead to aneurysm formation.

Hemodynamic stress is a crucial factor in the development of an aneurysm and its subsequent dilation. According to Laplace's law, wall tension is directly proportional to vessel radius and blood pressure. Risk factors such as hypertension promote turbulent flow, characterized by irregular motion that impose abnormal shear stress on the vascular endothelium. Such damage can initiate a cascade of plaque deposition and narrowing of the lumen [9]. Thinning of the wall and/or increased intracranial pressure would weaken the blood vessel wall, lead to further expansion, and increase the likelihood of rupture (aneurysm) [10].

A basilar artery with a diameter greater than 4.5 mm or a length exceeding 29.5 mm is classified as VBD [11]. This condition involves elongation and tortuosity of basilar or vertebral arteries due to degeneration of the tunica media [12]. VBD affects roughly 1.3% of the population [13] and often results from smooth muscle atrophy, or the dysregulation of antiproteases and matrix metalloproteinases, which disrupt the normal remodeling of arterial collagen and elastin [14]. This disease typically manifests as an enlarged, tortuous artery. Either the enlargement or tortuous nature of VBD can put pressure on cranial nerves and thus leads to a wide array of symptoms (e.g., dizziness, unilateral central facial palsy, and sensorineural symptoms like vertigo and hearing loss) [14, 15].

Another pathology that can cause increased external diameter of the basilar artery is stenosis. In this arterial pathology, the narrowing of the lumen secondary to plaque deposition causes an alteration of blood flow which puts stress on the vessel wall. This stress leads to compensatory outward remodeling of the tunica media [16]. To preserve the inner diameter of the vessel, the wall bulges outward. This happens through the proliferation of vascular smooth muscle, causing the deposition of collagen and degradation of elastin, leading to calcification [17].

The basilar artery, a central component of the posterior circulation within the brain, has been shown to have a greater likelihood of outward remodeling. The basilar artery may be more susceptible to remodeling due to an increased responsiveness to sympathetic activity within the basilar artery versus other cerebral vessels. [18]. There are a few cases of basilar artery stenosis that have been documented. It is difficult to quantify numbers within the population because asymptomatic cases are rarely documented.

Timely diagnosis of arterial enlargements is important clinically, because they pose a significantly greater risk for rupture and stroke. If an enlargement swells to greater than 1.5x its normal diameter, it is diagnosed as an aneurysm. Unruptured cerebral aneurysms can lead to subarachnoid hemorrhages, accounting for approximately 85% of such events, while the remainder typically result from trauma [19]. Aneurysms in the basilar artery pose a unique risk, as they may compress vital brainstem structures, particularly the pontine and cardiac centers, which regulate respiration and cardiac output [20].

Fusiform aneurysms, defined by diffuse circumferential dilation of a vessel segment, are rare findings most frequently in the vertebrobasilar system. They carry a worse prognosis than saccular aneurysms due to their association with thrombosis, infarction, or mass effect associated with brainstem compression [20]. Saccular aneurysms are characteristic of a unilateral focal outpouching with a narrow base. In the current case, the diffuse dilation without focal outpouching suggests a fusiform morphology. The absence of occlusion indicates that blood flow was maintained despite the abnormal vessel shape.

Conclusion

This case describes an asymptomatic enlargement of the basilar artery. Although not the cause of death in this individual, such vascular changes are clinically significant due to their association with ischemic events, aneurysm formation, and brainstem compression. Early identification, coupled with management of modifiable risk factors such as hypertension and smoking, may reduce morbidity and mortality associated with posterior circulation vascular disease.

References

1. Adigun OO, Reddy V, Sevensma KE, et al.: Anatomy, Head and Neck: Basilar Artery. StatPearls [Internet, Treasure Island (FL): StatPearls Publishing; 2025.
2. Samim M, Goldstein A, Schindler J, et al.: Multimodality imaging of vertebrobasilar dolichoectasia: clinical presentations and imaging spectrum. Radiographics. 2016, 36:1129-1146. 10.1148/rg.2016150032
3. Ubogu EE, Zaidat OO, Smith J, et al.: Vertebrobasilar dolichoectasia diagnosed by magnetic resonance angiography and risk of stroke and death: a cohort study. J Neurol Neurosurg Psychiatry. 2004, 75:22-26.
4. Passero SG, Rossi S, Brown M, et al.: Natural history of vertebrobasilar dolichoectasia. Neurology. 2008, 70:66-72. 10.1212/01.wnl.0000286947.89193.f3
5. Kleindorfer DO, Towfighi A, Chaturvedi S, et al.: 2021 guideline for the prevention of stroke in patients with stroke and TIA. Stroke. 2021, 52:364-467. 10.1161/STR.0000000000000375
6. Wolters FJ, Rinkel GJ, Vergouwen MD, et al.: Clinical course and treatment of vertebrobasilar dolichoectasia: a systematic review. Neurol Res. 2013, 35:131-137. 10.1179/1743132812Y.0000000149
7. Vijayakumar AU, Sudhakaran M, Yovel LJ: Human basilar artery: morphology and variations. AIMS Med Sci. 2020, 7:278-292. 10.3934/medsci.2020017
8. Swiatek VM, Fischer I, Khajuria R, et al.: The MARVIN hypothesis: linking unhealthy lifestyles to intracranial aneurysm rupture risk and prognosis. Medicina. 2024, 60:1813. 10.3390/medicina60111813
9. Singh P, Jones L, Carter B, et al.: Role of computational fluid dynamics in the analysis of haemodynamic and morphological characteristics of intracranial aneurysms. 2015.
10. Falkovich G, Turner A, Lee C: Fluid Mechanics: A Short Course for Physicists. Cambridge University Press, 2011.
11. Tanaka M, Sakaguchi M, Miwa K: Basilar artery diameter is an independent predictor of cardiovascular events. Arterioscler Thromb Vasc Biol. 2013, 33:2240-2244. 10.1161/ATVBAHA.113.301467
12. Zang X, Feng Z, Qiao H: Vertebrobasilar dolichoectasia as a rare cause of simultaneous abducens and vestibulocochlear nerve symptoms: case report and review. Int J Gen Med. 2020, 13:523-527. 10.2147/IJGM.S269649
13. Yuan YJ, Xu K, Luo Q: Research progress on vertebrobasilar dolichoectasia. Int J Med Sci. 2014, 11:1039-1048. 10.7150/ijms.8566

14. Yuh SJ, Alkherayf F, Lesiuk H, et al.: Dolichoectasia of the vertebral basilar and internal carotid arteries: a case report and review. Surg Neurol Int. 2013, 4:153. 10.4103/2152-7806.122397
15. Lee YJ, Baek W, Nam GS, et al.: Vertebrobasilar dolichoectasia manifesting as sudden sensorineural hearing loss with vertigo: a case report. Res Vestib Sci. 2024, 23:32-36. 10.21790/rvs.2024.004
16. Golledge J, Shi GP, Norman P: Pathogenesis of aortic aneurysms. In: Fitridge R, Thompson M (eds). Mechanisms of Vascular Disease. University of Adelaide Press; 2011.
17. van Varik BJ, Rennenberg RJ, Reutelingsperger CP, et al.: Mechanisms of arterial remodeling: lessons from genetic diseases. Front Genet. 2012, 3:290. 10.3389/fgene.2012.00290
18. Qiao Y, Anwar Z, Intrapiromkul J, et al.: Patterns and implications of intracranial arterial remodeling in stroke patients. Stroke. 2016, 47:434-440. 10.1161/STROKEAHA.115.009955
19. Ziu E, Khan SMZ, Mesfin FB, et al.: Subarachnoid hemorrhage. StatPearls [Internet, Treasure Island (FL): StatPearls Publishing; 2024.
20. Barletta EA, Ricci RL, Silva RDG, et al.: Fusiform aneurysms: pathogenesis to treatment options. Surg Neurol Int. 2018, 9:189. 10.4103/sni.sni_133_18

Author contributions

All authors contributed to writing the manuscript draft and reviewed and edited final drafts of the manuscript. All authors reviewed and approved the final manuscript.

Funding None.

Data availability No sets of data were created or analyzed.

Declarations

Conflict of interest None.

Consent to publish by the PCOM body donation program.