Turk Neurosurg 32(4):571-577, 2022

Original Investigation

DOI: 10.5137/1019-5149.JTN.32794-20.6

Received: 15.10.2020
Accepted: 05.04.2021

Published Online: 18.10.2021

Can Intracranial Vertebral Artery Hypoplasia be an Etiopathogenetic Factor for Barré–Lièou Syndrome Other than Arcuate Foramen? A Retrospective Clinical Study and Review of Literature

Esin Derin CİCEK[1]*, Nese KESER[2]*, Merih IS[3], Ozkan ATES[4]

[1]The University of Health Sciences, Istanbul Fatih Sultan Mehmet Training and Research Hospital, Department of Radiology, Istanbul, Turkey
[2]The University of Health Sciences, Istanbul Fatih Sultan Mehmet Training and Research Hospital, Department of Neurosurgery, Istanbul, Turkey
[3]Retired Neurosurgeon, Istanbul, Turkey
[4]Koc University, Department of Neurosurgery, Istanbul, Turkey

*Equal contributors

Corresponding author: Nese KESER ✉ nskeser@gmail.com

ABSTRACT

AIM: To investigate the co-occurrence of the arcuate foramen (AF) variation of atlas and intracranial vertebral artery (V4) hypoplasia and, therefore, to understand the pathophysiology of Barré–Lièou syndrome (BLS). The AF localizes on the vertebral artery (VA) sulcus posterior to the atlas and has incomplete and complete types. Complete-type AF can exert pressure on the VA that passes through it, resulting in vertebrobasilar insufficiency finding, a BLS component. By the surgical decompression of VA at the AF level, complaints could be decreased in some cases. However, a reliable theory regarding BLS has not yet been established; therefore, the cases that do not respond to AF decompression have not been fully elucidated. We assumed that V4 hypoplasia that accompanies AF might be the main factor in the pathophysiology of BLS.

MATERIAL and METHODS: Cervical computed tomography and magnetic resonance angiography images of 139 patients aged 14–88 years with head and neck pain and dizziness were retrospectively evaluated.

RESULTS: Of the patients, 19.4% exhibited complete AF and 32.4% exhibited VA hypoplasia (VAH); 10% of the patients with VAH had accompanying contralateral complete AF variation. There was no significant relationship between complete AF and contralateral and ipsilateral VAHs (right side: p=0.527 and p=0.433, respectively; left side: p=1.000 and p=0.740, respectively).

CONCLUSION: Our findings indicate that V4 hypoplasia is not the main factor of BLS pathophysiology. Furthermore, the rarity of the relationship suggests why some cases do not respond to decompressive surgery.

KEYWORDS: Arcuate foramen, Barré Lièou syndrome, Decompression of vertebral artery, Intracranial vertebral artery hypoplasia, Vertebrobasilar insufficiency

ABBREVIATIONS: AF: Arcuate foramen, **BLS:** Barré-Louie syndrome, **C1:** First cervical vertebra, Atlas, **CT:** Computed tomography, **MRA:** Magnetic resonance angiography, **VA:** Vertebral artery, **VAH:** VA hypoplasia, **V3:** The third segment of VA, **V4:** Intracranial VA, **VBI:** Vertebrobasilar insufficiency

Esin Derin CICEK : 0000-0002-0391-3003
Nese KESER : 0000-0002-6024-8855
Merih IS : 0000-0002-5035-2229
Ozkan ATES : 0000-0002-3132-4839

INTRODUCTION

The arcuate foramen (AF) variation localized on the vertebral artery (VA) sulcus posterior to the first cervical vertebra (C1) is referred to by more than one name in the literature (Table I). Its morphology is highly variable and may be unilateral or bilateral and complete or incomplete (Figure 1), and the complete form fully encircles the transversing vessels (14,15,25,35). AF has been hypothesized to be caused by ossification of the connective tissue around VA or late ossification of the lower edge of the atlantooccipital membrane. However, the fact that the frequency of its occurrence among the elderly does not increase has excluded this conclusion (1,7). Moreover, it is considered a regressive primitive structure because of its higher prevalence in lower primates (14,36). The incidence varies regionally and ethnically, and the only clinically positive aspect is the low fracture risk. However, it has been reported to cause Barré-Liéou syndrome (BLS) by compressing the third segment of VA (V3), C1 spinal nerve, and periarterial sympathetic plexus while passing through the complete form (Figure 2). Furthermore, it can cause VA dissection through stretching, VB ischemia during strong interventions in the cervical spine, and complications in cases where C1–C2 stabilization is required (3,4,7-9,17,18,22, 25,27-29,31,35).

In patients with BLS, which was defined in 1926, complaints could be decreased by surgical decompression of V3 and the periarterial sympathetic plexus at the AF level (1). However, a reliable theory regarding BLS has not been established yet; in addition, the reason why some of the cases do not respond to AF decompression has not been fully elucidated (19,20). We assumed that the presence of intracranial VA (V4) hypoplasia might be a main contributor to BLS. Furthermore, because intracranial VA hypoplasia (VAH) contralateral to AF variation could lead to a "double crush" effect in the posterior fossa, it might explain some of the cases' unresponsiveness to the surgical decompression. Although it has been shown that C1 variation is accompanied by V3 variations (15,24,37), so far, to the best of our knowledge, no studies published in English have investigated the relationship between AF variation and intracranial VAH. We aimed to investigate the possibility of the co-occurrence of these variations.

Table I: Nomenclatures Used For Describing Arcuate Foramen Variation In Humans

Arcuate foramen
Atlas bridging
Canalis arteria vertebralis
Foramen atlantoideum posterius/vertebrale
Foramen retroarticular superior
Foramen sagittale
Kimmerle's anomaly/variant/deformity
Pons posticus
Ponticulus posticus
Posterior atlantoid foramen
Posterior glenoid process
Posterior glenoid spiculum
Posterior ponticle of the atlas
Retroarticular canal
Retroarticular VA ring
Retroarticular vertebral ring
Retrocondylar bony foramen
Retrocondylar VA ring

VA: *Vertebral artery.*

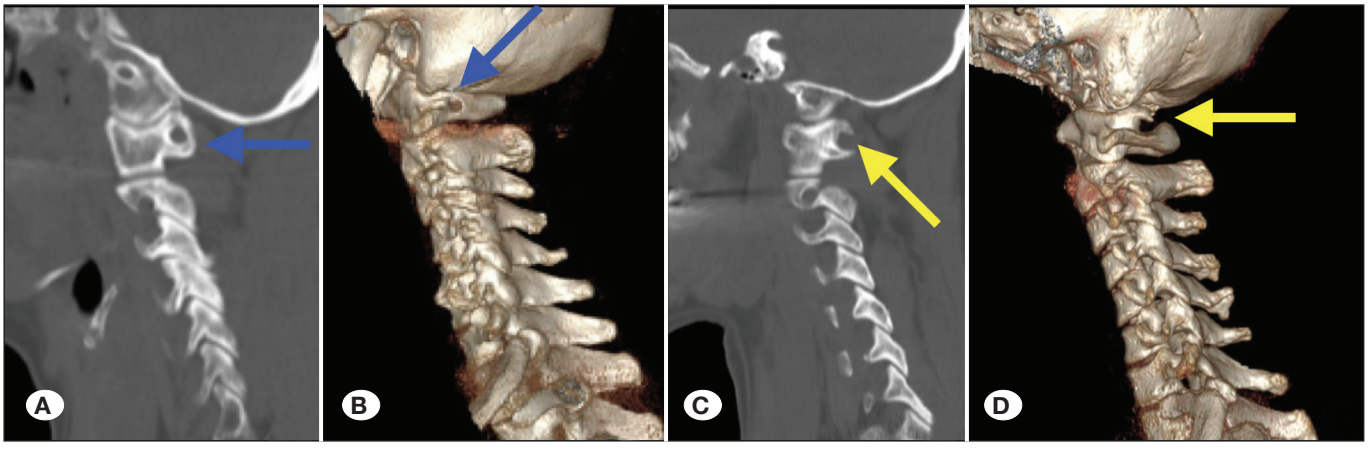

Figure 1: Photographs showing computerized tomographic (CT) imaging of the arcuate foramen (AF) variations. **A)** Sagittal cervical computed tomography (CT) scan. A left-sided complete AF variation of the atlas (blue arrow). **B)** 3D reconstruction of the cervical CT, the left-sided complete AF variation (blue arrow). **C)** A left-sided incomplete AF variation (yellow arrow). **D)** 3D reconstruction of the cervical CT, the left-sided incomplete AF variation (yellow arrow).

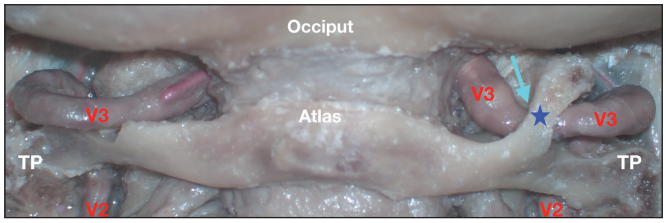

Figure 2: The cadaveric dissection showing a right-sided complete arcuate foramen (AF). **Blue star:** complete AF; **cyan arrow:** compression sign of the AF on the third segment (V3) of the vertebral artery (VA); **V2:** the second segment of the VA; **TP:** Transverse process of the first cervical vertebra (Atlas).

MATERIAL and METHODS

The hospital ethics committee approved this retrospective study (approval number: 17073117-050.06). Initially, we included 481 patients who presented with head and neck pain and dizziness to the neurosurgery outpatient clinic between October 2019 and March 2020. Patients with a history of cervical surgery, cervical trauma, cervical spine malignancy, inner-middle ear pathology, congenital diseases related to the craniovertebral junction (Arnold–Chiari malformation, down syndrome, Klippel–Feil syndrome, atlantooccipital fusion), rheumatoid arthritis, cardiovascular disease, patients with posterior circulatory infarction or aneurysm, and children under 13 years of age were excluded from the study. Of the remaining 387 patients, 143 had undergone cranial magnetic resonance angiography (MRA) and cervical computed tomography (CT). After excluding 4 patients because of artifacts on CT, the remaining 139 patients were included.

The first author evaluated all radiological examinations. AF was classified as either complete or incomplete. Superiorly, the vertebral groove is arched by the posterior atlantooccipital membrane. When this membrane is totally ossified, it forms a whole bony bridge over the vertebral groove called complete AF (4) (Figures 1A, B). Moreover, partial ossification of the atlantooccipital membrane is called incomplete AF (Figures 1C, D). Incomplete AF could be identified in three types as follows (15): type I, a bony spicule extending only from the superior articular facet; type II, a bony spicule projects from the posterior arch of the atlas toward the superior articular facet; and type III, a bony spicule originates both from the superior articular facet and posterior arch.

As the VAH criterion, a diameter of <2 mm in the V4 segment of VA was used as per Chuang et al.'s report (6).

Cervical CT examinations were performed using a 64-detector 128-slice CT scanner (Optima CT 660, GE Healthcare, Tokyo, Japan) (kVp=120, mAs=120–300, slice thickness=0.625 mm). 3D images were obtained using a soft tissue algorithm. Cranial MRA was performed on a 1.5 Tesla imaging system (Optima MR 450w, GE Healthcare, Milwaukee, WI, USA) without contrast media. Three-dimensional time-of-flight images (Short TE (2.5 ms) and TR (<30 ms) values, flip angle: 20°, slice thickness: 1.4 mm, FOV: 22 cm, matrix: 256 × 192) were acquired in the transaxial plane. A presaturation band was applied above each slice to suppress the venous signal. A maximum intensity projection algorithm was used to minimize the vessel overlap. CT and MRA images were evaluated using a GE Advantage Workstation (GE Healthcare, Buc, France) and picture archiving communication systems.

Statistical Analysis

Number Cruncher Statistical System statistical software (Utah, USA) program was used for statistical analysis. In addition to descriptive statistical methods (mean, standard deviation, median, frequency, and ratio), Shapiro–Wilk test and box plot graphics were used to test for the variables' normal distribution. Kruskal–Wallis test was used to perform intergroup comparisons of the variables that did not show normal distribution. Pearson's Chi-Square test, Fisher's exact test, and Fisher–Freeman–Halton test were used for comparing qualitative data. Significance was evaluated at $p<0.05$.

RESULTS

Among the 139 patients, 30% were men, and 70% were women; the mean patient age was 47.65 ± 15.60. Complete AF variation was observed in 16.5% of the patients on the right and 15.1% of the patients on the left side. Incomplete AF variation was observed in 33.8% of the patients on the right and 41% of the patients on the left side (Table II). Intracranial VAH was found in 23.7% of the patients on the right and 15.1% of patients on the left side (Figure 3). Among every 10 VAH cases, one presented with the contralateral complete AF variation (Figure 4). According to the complete and incomplete AF groups, no significant difference was found between the patients' age and gender distributions ($p>0.05$). Similarly, no significant difference was found between the age and gender distributions of the patients according to the presence of VAH ($p>0.05$) (Table III); furthermore, there was no significant relationship between the AF variations and ipsilateral and contralateral VAHs ($p>0.05$) (Table IV).

DISCUSSION

Our study revealed that 19.4% of the patients had complete AF formation (Figure 4), and intracranial VAH does not usually accompany the AF variation of the atlas (Table IV). It has been reported in the literature that the prevalence of AF variation varies from 1%–68%, and it is more common in North American populations but less common in Indians and South Koreans (12,25,26). Besides, it has been reported that AF can be overlooked on lateral radiographs. CT used for the diagnosis is the gold standard and provides high-quality images similar to those obtained from cadaveric studies (Figure 1, 2) (24,25). We found that the AF prevalence in our study participants was almost similar to the that (22.5%) reported by Saleh et al. (26) in their study that evaluated the CT of 2917 cases; however, contrary to their study, we found that AF was more common in women and on the right side. V4 (Figure 5A) variations reportedly include VAH or aplasia. Although aplasia is very rare (1%), the VAH prevalence ranges from 2% to 42%, and similar to AF variations, VAH is more common in African-Americans than in Caucasians and less common in Indians (30,32). Although no consensus has been reached

Table II: Distributions of the Descriptive Properties

Characteristic		Value
	Min-Max (Median)	14-88 (48)
	Mean ± SD	47.65 ± 15.60
		n (%)
Age (year)	<30	23 (16.5)
	31-45	38 (27.3)
	46-60	46 (33.1)
	≥61	32 (23.0)
Gender	Male	42 (30.2)
	Female	97 (69.8)
Complete AF	R	23 (16.5)
	L	21 (15.1)
	None	112 (80.6)
	R only	6 (4.3)
	L only	4 (2.9)
	Bilateral	17 (12.2)
Incomplete AF	R	47 (33.8)
	L	57 (41.0)
	None	77 (55.4)
	R only	5 (3.6)
	L only	15 (10.8)
	Bilateral	42 (30.2)
VAH	R	33 (23.7
	L	21 (15.1)
	None	94 (67.6)
	R only	26 (18.7)
	L only	12 (8.6)
	Bilateral	7 (5.0)

AF: *Arcuate foramen,* ***R:*** *Right,* ***L:*** *Left,* ***VAH:*** *Vertebral artery hypoplasia*

on the definition of VAH in the literature, a vessel diameter of ≤2 mm and a threshold for asymmetry ratio of >1:1.7 were frequently used (2,13,21,34). Similar to our study, that of Chuang et al., who defined the VAH criterion as a V4 diameter of <2.0 mm and used a 1.5-tesla MRI scanner, reported that the VAH prevalence was 42% in Chinese patients (6). In our study, the patients' MRA examination for V4 variation revealed only hypoplasia at a rate of 32.4%. Moreover, similar to AF variations, VAH at a higher rate was observed on the right side and in female patients (Figure 5B), which agrees with the findings in literature (5). Although an association was shown

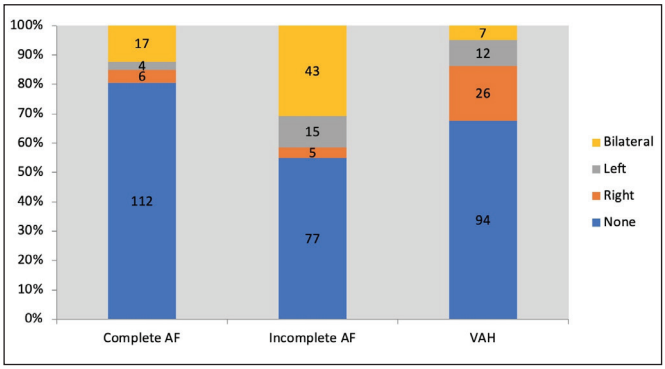

Figure 3: Graphics showing the cases' distribution with the complete and incomplete arcuate foramen (AF) variation and vertebral artery hypoplasia (VAH).

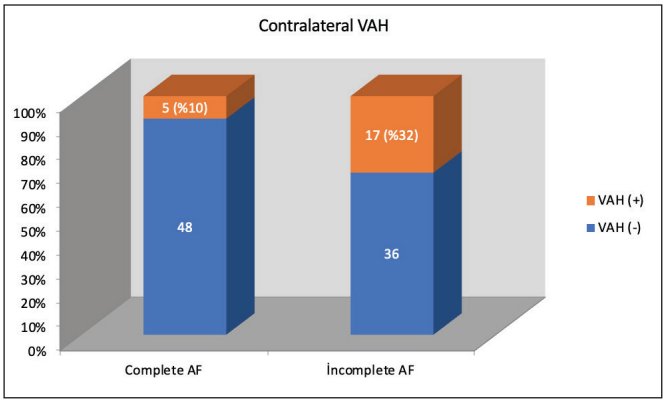

Figure 4: Graphics showing the co-occurrence of the complete and incomplete arcuate foramen (AF) variations and contralateral vertebral artery hypoplasia (VAH).

between AF and V3 variations (15,24,37), we did not find this relationship with V4 hypoplasia (Table IV). This difference can be explained by the embryological development of VA (10,23). Furthermore, this finding suggests that V4 hypoplasia may not be the main factor in BLS pathophysiology. Therefore, this can explain why most BLS cases respond to V3 decompression.

It has been reported that VAH-induced regional hypoperfusion causes posterior circulation strokes, vestibular neuropathy, migraine, and lateral medullary ischemia; this variation is also associated with the pathologies of the other arterial structures of the posterior circulation (13,16,38). Moreover, hypoplastic VA may be more sensitive to prothrombotic or atherosclerotic processes than normal VA due to a reduced flow volume and flow velocities; therefore, when other risk factors are present, it can cause stroke in the posterior fossa even in young patients (30,32,33). Furthermore, it was reported that ipsilateral flow volume insufficiency (<100 mL/min net flow volume) was significantly higher in individuals with VAH than those without VAH, whereas contralateral VA showed a compensatory increase in the flow volume (11). Because many cadaveric studies have shown that AF compresses V3 (Figure 2) (17,35), the association of AF with the contralateral side of V4 hypoplasia was assumed to increase posterior fossa ischemia. In our study, 10% of the patients with complete AF

Table III: Evaluation of the Defining Characteristics of Vertebral Artery Hypoplasia (VAH) with Complete and Incomplete Arcuate Foramen (AF) Variations

		Normal	Right	Left	Bilateral	p
Complete AF	n	112	6	4	17	
Year	Min-Max(median)	14-78 (48)	25-70 (38)	23-52 (43,5)	23-88 (54)	[a]0.648
	Mean ± SD	47.61 ± 15.53	44.0 ± 18.79	40.5 ± 13.12	50.94 ± 15.95	
Gender	Male	38 (33.9)	0	1 (25.0)	3 (17.6)	[b]0.211
	Female	74 (66.1)	6 (100)	3 (75.0)	14 (82.4)	
	None	86 (76.8)	4 (66.7)	2 (50.0)	11 (64.7)	
Incomplete AF	n	77	5	15	43	
Year	Min-Max(median)	14-88 (52)	22-58 (49)	25-74 (44)	18-75 (46)	[a]0.734
	Mean ± SD	48.83 ± 15.90	43.8 ± 14.18	47.33 ± 15.85	46.07 ± 15.44	
Gender	Male	22 (28.6)	0	4 (26.7)	14 (38.1)	[b]0.335
	Female	55 (71.4)	5 (100)	11 (73.3)	26 (61.9)	
	None	56 (72.7)	3 (60.0)	11 (73.3)	33 (78.6)	
VAH	n	94	26	12	7	
Year	Min-Max(median)	14-78 (47)	22-88 (50)	28-66 (54)	27-62 (54)	[a]0.695
	Mean ± SD	46.52 ± 16.35	50.46 ± 14.80	50.50 ± 12.11	47.57 ± 14.35	
Gender	Male	28 (29.8)	11 (42.3)	2 (16.7)	1 (14.3)	[b]0.360
	Female	66 (70.2)	15 (57.7)	10 (83.3)	6 (85.7)	
	None	69 (73.4)	21 (80.8)	9 (75.0)	4 (57.1)	

*[a]Kruskal Wallıs test, [b]Fisher Freeman Halton test, **AF:** Arcuate foramen, **VAH:** Vertebral artery hypoplasia.*

Table IV: Relationship Between Vertebral Artery Hypoplasia (VAH) and Complete and Incomplete Arcuate Foramen (AF) Variations

		VAH/ R			VAH/ L		
		N	VAH	p	N	VAH	p
Complete AF/ R	Absent	87 (75.0)	29 (25.0)	[c]0,433	97 (83.6)	19 (16.4)	[d]0.527
	Present	19 (82.6)	4 (17.4)		21 (91.3)	2 (8.7)	
Incomplete AF/ R	Absent	69 (75.0)	23 (25.0)	[c]0,626	77 (83.7)	15 (16.3)	[c]0.582
	Present	37 (78.7)	10 (21.3)		41 (87.2)	6 (12.8)	
Complete AF/ L	Absent	90 (76.3)	28 (23.7)	[d]1,000	99 (83.9)	19 (16.1)	[d]0.740
	Present	16 (76.2)	5 (23.8)		19 (90.5)	2 (9.5)	
Incomplete AF/ L	Absent	60 (73.2)	22 (26.8)	[c]0,305	69 (84.1)	13 (15.9)	[c]0.768
	Present	46 (80.7)	11 (19.3)		49 (86.0)	8 (14.0)	

*[c]Pearson Chi-Square test, [d]Fisher Exact test, **AF:** Arcuate foramen, **R:** Right, **L:** Left, **VAH:** Vertebral artery hypoplasia.*

had an accompanying contralateral VAH (Figures 4 and 6). The finding may indicate why some cases do not respond to surgical decompression, and we suggest MRA evaluation of V4 before AF decompression surgery. Therefore, if contralateral intracranial VAH is found, surgical decompression might not be considered for these cases.

The strength of this study is that it is the first study in the literature that investigates the relationship between the AF variation of the atlas and intracranial VAH.

The limitation of our study is the broad age range of the study patients. Reportedly, the frequency of secondary or acquired

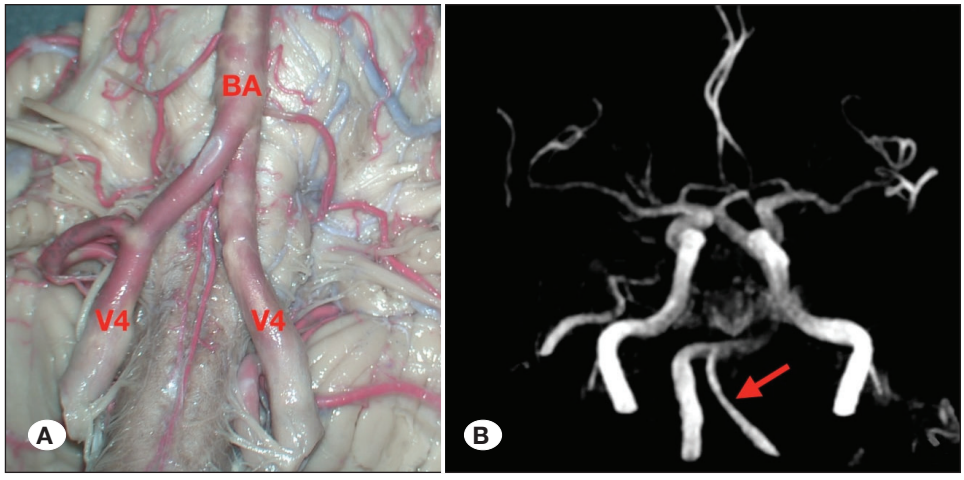

Figure 5: A) A cadaveric specimen showing the average-sized intracranial vertebral arteries (V4) and basilar artery (BA). **B)** The magnetic resonance angiography of a case with left-sided vertebral artery hypoplasia (VAH) (red arrow). Note that since the case does not have a contralateral arcuate foramen variation, the right-sided VA has a compensatory dilatation.

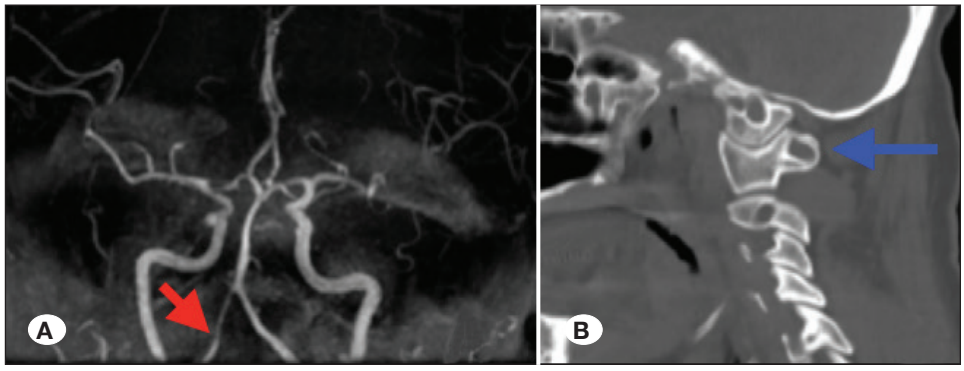

Figure 6: A case with both a right-sided vertebral artery (VA) hypoplasia (red arrow) **(A)** and also a left-sided arcuate foramen (blue arrow) **(B)**. Note that left-sided VA has not a compensatory dilatation.

VAH increases with age, but that of AF variation remains unchanged (35,37). Further studies should be conducted on a large number of young cases.

CONCLUSION

In conclusion, our findings indicate that V4 hypoplasia cannot be the main factor in the pathophysiology of BLS because it does not usually accompany the AF variation of the atlas. Furthermore, the other finding that presents concomitant contralateral VAH in only 10% of patients with complete AF may suggest why a small proportion of cases do not respond to the surgical decompression of VA. Therefore, this finding may help determine the treatment strategy. If V4 hypoplasia is detected contralateral to the AF side, decompression of extracranial VA may not be considered.

ACKNOWLEDGMENT

Authors thank Ozden Gemalmaz and Hatice Sari for their kind assistance in providing patients' 3D reconstructions of the CT scans.

AUTHORSHIP CONTRIBUTION

Study conception and design: NK

Data collection: NK

Analysis and interpretation of results: EDC

Draft manuscript preparation: NK

Critical revision of the article: NK, OA, MI, EDC

Other (study supervision, fundings, materials, etc...): MI, OA

All authors (EDC, NK, MI, OA) reviewed the results and approved the final version of the manuscript.

REFERENCES

1. Ahn J, Duran M, Syldort S, Rizvi A, D'Antoni AV, Johal J, Iwanaga J, Oskouian RJ, Tubbs RS: Arcuate foramen: Anatomy, embryology, nomenclature, pathology, and surgical considerations. World Neurosurg 118:197-202, 2018
2. Akar ZC, Dujovny M, Slavin KV, Gomez-Tortosa E, Ausman JI: Microsurgical anatomy of the intracranial part of the vertebral artery. Neurol Res 16:171-180, 1994
3. Arslan D, Ozer MA, Govsa F, Kıtıs O: The ponticulus posticus as risk factor for screw insertion into the first cervical lateral mass. World Neurosurg 113:e579-e585, 2018
4. Cakmak O, Gurdal E, Ekinci G, Yildiz E, Cavdar S: Arcuate foramen and its clinical significance. Saudi Med J 26:1409-1413, 2005
5. Chen YY, Chao AC, Hsu HY, Chung CP, Hu HH: Vertebral artery hypoplasia is associated with decreased net vertebral flow volume. Ultrasound Med Biol 36:38-43, 2010
6. Chuang YM, Chern CM, Liao WH, Hsu LC, Lien CF, Lirng JF, Shiao AS, Ko JS: Contribution of intracranial vertebral artery asymmetry to vestibular neuropathy. J Neurol Neurosurg Psychiatry 82:823-825, 2011

7. Cossu G, Terrier LM, Destrieux C, Velut S, François P, Zemmoura I, Amelot A: Arcuate foramen: "Anatomical variation shape or adaptation legacy?". Surg Radiol Anat 41:583-588, 2019
8. Cushing KE, Ramesh V, Gardner-Medwin D, Todd NV, Gholkar A, Baxter P, Griffiths PD: Tethering of the vertebral artery in the congenital arcuate foramen of the atlas vertebra: A possible cause of vertebral artery dissection in children. Dev Med Child Neurol 43:491-496, 2001
9. Elliott RE, Tanweer O: The prevalence of the ponticulus posticus (arcuate foramen) and its importance in the Goel-Harms procedure: Meta-analysis and review of the literature. World Neurosurg 82:e335-343, 2014
10. George B, Bruneau M: Embryology of the vertebral artery. In: Pathology and Surgery Around the Vertebral Artery. Paris: Springer, 2011: 5-24
11. Giannopoulos S, Kosmidou M, Pelidou SH, Kyritsis AP: Vertebral artery hypoplasia: A predisposing factor for posterior circulation stroke? Neurology 68:1956; author reply 1956-1957, 2007
12. Gibelli D, Cappella A, Cerutti E, Spagnoli L, Dolci C, Sforza C: Prevalence of ponticulus posticus in a Northern Italian orthodontic population: A lateral cephalometric study. Surg Radiol Anat 38:309-312, 2016
13. Harati A, Zeh D, Rohde S, Schultheiß R, Schmieder K, Hernesniemi J: Association between vertebral artery hypoplasia and vertebral artery aneurysms: A case-control study. J Neurol Surg A Cent Eur Neurosurg 80:365-370, 2019
14. Hasan M, Shukla S, Siddiqui MS, Singh D: Posterolateral tunnels and ponticuli in human atlas vertebrae. J Anat 199:339-43, 2001
15. Hong JT, Lee SW, Son BC, Sung JH, Yang SH, Kim IS, Park CK: Analysis of anatomical variations of bone and vascular structures around the posterior atlantal arch using three-dimensional computed tomography angiography. J Neurosurg Spine 8:230-236, 2008
16. Katsanos AH, Kosmidou M, Kyritsis AP, Giannopoulos S: Is vertebral artery hypoplasia a predisposing factor for posterior circulation cerebral ischemic events? A comprehensive review. Eur Neurol 70:78-83, 2013
17. Keser N, Cikla U, Ozaydın B, Baskaya MK: The importance of arcuate foramen, a variation of the atlas: A microsurgical cadaveric study and review of the literature. Istanbul Medical Journal 20:377-381, 2019
18. Lamberty BG, Zivanović S: The retro-articular vertebral artery ring of the atlas and its significance. Acta Anat (Basel) 85:113-122, 1973
19. Li Y, Peng B: Pathogenesis, diagnosis, and treatment of cervical vertigo. Pain Physician 18: E583-595, 2015
20. Limousin C: Foramen arcuale and syndrome of Barre-Lieou. Its surgical treatment. Int Orthop 4:19-23, 1980
21. Mitsumura H, Miyagawa S, Komatsu T, Hirai T, Kono Y, Iguchi Y: Relationship between vertebral artery hypoplasia and posterior circulation ischemia. J Stroke Cerebrovasc Dis 25:266-269, 2016
22. Morinaga Y, Nii K, Sakamoto K, Inoue R, Mitsutake T, Hanada H: Focus on diagnosis, treatment, and problems of Barré-Lièou syndrome: Two case reports. Drug Discov Ther 13:239-243, 2019
23. Motomura M, Watanabe K, Tabira Y, Iwanaga J, Matsuuchi W, Yoshida D, Saga T, Yamaki KI: A case of duplicated right vertebral artery. Kurume Med J 64:69-73, 2018
24. Natsis K, Piperaki ET, Fratzoglou M, Lazaridis N, Tsitsopoulos PP, Samolis A, Kostares M, Piagkou M: Atlas posterior arch and vertebral artery's groove variants: A classification, morphometric study, clinical and surgical implications. Surg Radiol Anat 41:985-1001, 2019
25. Pękala PA, Henry BM, Pękala JR, Hsieh WC, Vikse J, Sanna B, Walocha JA, Tubbs RS, Tomaszewski KA: Prevalence of foramen arcuale and its clinical significance: A meta-analysis of 55,985 subjects. J Neurosurg Spine 27:276-290, 2017
26. Saleh A, Gruber J, Bakhsh W, Rubery PT, Mesfin A: How common is the ponticulus posticus?: A computed tomography based analysis of 2917 patients. Spine (Phila Pa 1976) 43:E436-E441, 2018
27. Sanchis-Gimeno JA, Llido S, Guede D, Nalla S, Martinez-Soriano F, Blanco-Perez E, Caeiro JR: Atlases with arcuate foramen present cortical bone thickening that may contribute to lower fracture risk. World Neurosurg 117:e162-e166, 2018
28. Senoglu M, Gumusalan Y, Yuksel KZ, Uzel M, Celik M, Ozbag D: The effect of posterior bridging of C-1 on craniovertebral junction surgery. J Neurosurg Spine 5:50-52, 2006
29. Simsek S, Yigitkanli K, Comert A, Acar HI, Seckin H, Er U, Belen D, Tekdemir I, Elhan A: Posterior osseous bridging of C1. J Clin Neurosci 15:686-688, 2008
30. Singh R, Kumar R, Kumar A: Vascular anomalies of posterior fossa and their implications. J Craniofac Surg 28:2145-2150, 2017
31. Song MS, Lee HJ, Kim JT, Kim JH, Hong JT: Ponticulus posticus: Morphometric analysis and its anatomical Implications for occipito-cervical fusion. Clin Neurol Neurosurg 157:76-81, 2017
32. Sparaco M, Ciolli L, Zini A: Posterior circulation ischaemic stroke-a review part I: Anatomy, aetiology and clinical presentations. Neurol Sci 40:1995-2006, 2019
33. Szárazová AS, Bartels E, Bartels S, Turčáni P: Possible morphological pathomechanisms of ischemic stroke in the posterior circulation of patients with vertebral artery hypoplasia. J Neuroimaging 25:408-414, 2015
34. Thierfelder KM, Baumann AB, Sommer WH, Armbruster M, Opherk C, Janssen H, Reiser MF, Straube A, von Baumgarten L: Vertebral artery hypoplasia: Frequency and effect on cerebellar blood flow characteristics. Stroke 45:1363-1368, 2014
35. Tubbs RS, Johnson PC, Shoja MM, Loukas M, Oakes WJ: Foramen arcuale: Anatomical study and review of the literature. J Neurosurg Spine 6:31-34, 2007
36. Wakao N, Takeuchi M, Nishimura M, Riew KD, Kamiya M, Hirasawa A, Kawanami K, Imagama S, Sato K, Takayasu M: Vertebral artery variations and osseous anomaly at the C1-2 level diagnosed by 3D CT angiography in normal subjects. Neuroradiology 56:843-849, 2014
37. Wang Y, Cai A, Liu L, Wang Y: Sonographic diagnosis of congenital variations of the extracranial vertebral artery and assessment of its circulation. J Ultrasound Med 28:1481-1486, 2009
38. Zhou M, Zheng H, Gong S, Guo J, Chen N, Zhou D, Yang R, Zhu C, He L: Vertebral artery hypoplasia and vertebral artery dissection: A hospital-based cohort study. Neurology 84:818-824, 2015