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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.*

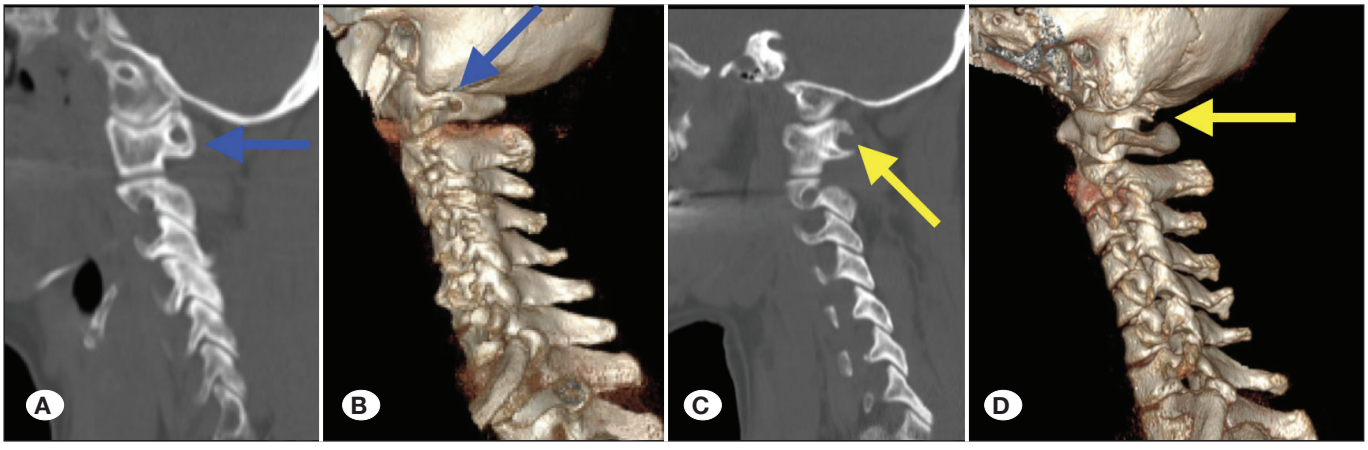

Figure 1: Photographs showing computerized tomographic (CT) imaging of the arcuate foramen (AF) variations. **A)** A left-sided complete AF variation of the atlas (blue arrow). **B)** 3D reconstruction of the cervical CT, the left-sided complete AF variation (blue arrow). **C)** A left-sided incomplete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

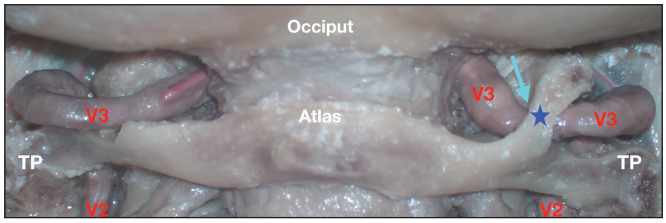

Figure 2: The cadaveric dissection showing a right-sided complete arcuate foramen (AF). **Blue star:** complete AF; **cyan arrow:** compression sign of the AF on the third segment (V3) of the vertebral artery (VA); **V2:** the second segment of the VA; **TP:** Transverse process of the first cervical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

		n (%)
Age (year)	Min-Max (Median)	14-88 (48)
	Mean ± SD	47.65 ± 15.60
	<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 between AF and V3 variations (15,24,37), we did not find this

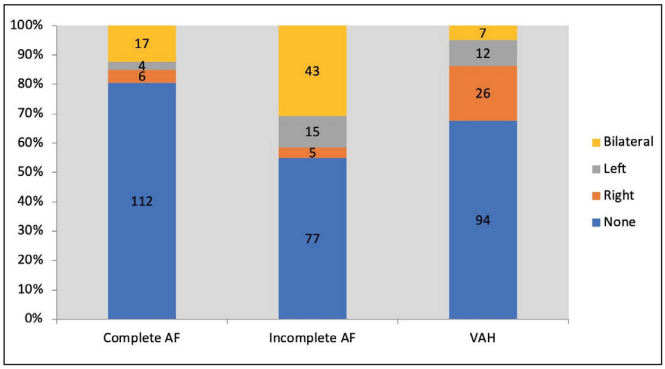

Figure 3: Graphics showing the cases' distribution with the complete and incomplete arcuate foramen (AF) variation and vertebral artery hypoplasia (VAH).

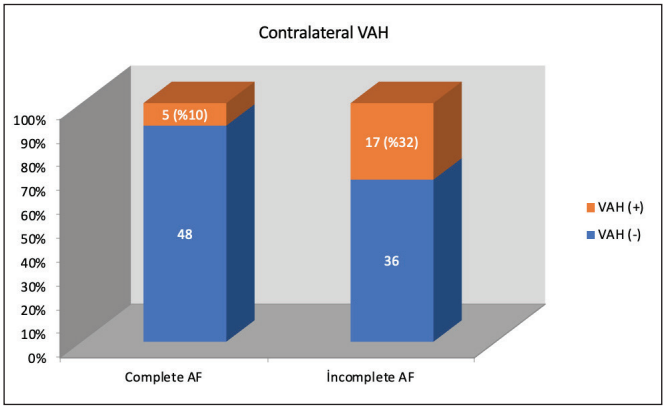

Figure 4: Graphics showing the co-occurrence of the complete and incomplete arcuate foramen (AF) variations and contralateral vertebral artery hypoplasia (VAH).

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 Ki kare 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

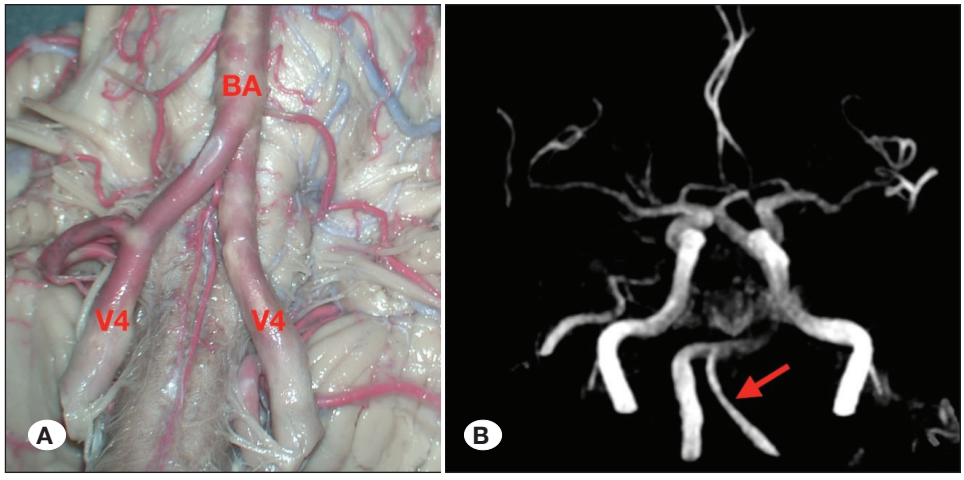

Figure 5: A) A cadaveric specimen showing the average-sized intracranial vertebral arteries (V4) and basilar artery (BA). **B)** The magnetic resonance angiography of a case with left-sided vertebral artery hypoplasia (VAH) (red arrow). Note that since the case does not have a contralateral arcuate foramen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

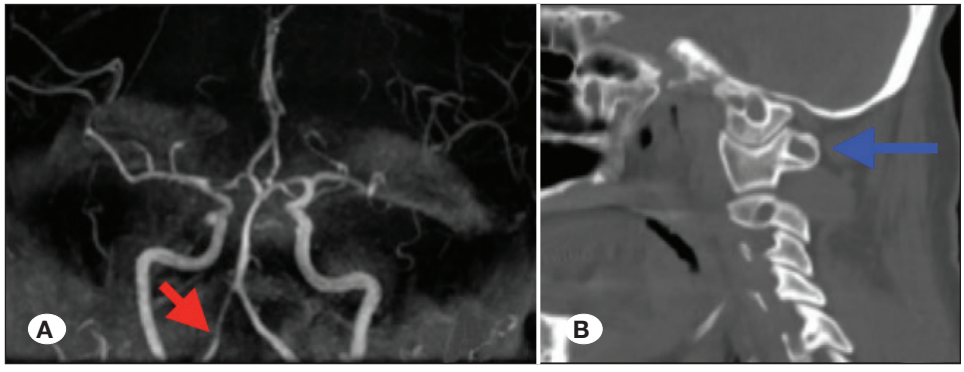

Figure 6: A case with both a right-sided vertebral artery (VA) hypoplasia (red arrow) **(A)** and also a left-sided arcuate foramen (blue arrow) **(B)**. Note that left-sided VA has not a compensatory dilatation.

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.

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