Vertebral artery compression of the medulla oblongata: A benign radiological finding?

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ABSTRACT

Background: To the best of our knowledge, no study has documented the natural history of rostral medullary compression of the vertebral artery (RMCVA) as radiological finding. The aim of this study was to explore it.

Methods: A total of 57 patients with RMCVA and not presenting symptoms of medullary compression syndrome were enrolled. These participants underwent cerebral magnetic resonance imaging with contrast, and 19 of them who were followed for 5.7 ± 1.9 years (range: 3.0–10.3 years) were analyzed in detail. For comparison, clinical courses of two other patients with vertebrobasilar dolichoectasia (VBDE) were presented.

Results: RMCVA was well delineated in all 57 patients. In the 19 patients analyzed, RMCVA was found in 17 sides on the right and 15 on the left. Moreover, the ventrolateral medulla was the most frequent compression site, and it was found in 69% of cases, with 84.2% presenting as mild compression and 15.8% as considerable compression. During the follow-up period, no patients showed neurological deterioration or radiological progression. In contrast, the two VBDE patients demonstrated both neurological and radiological progressions during the follow-up period.

Conclusion: Unlike VBDE, RMCVA seems to be a benign condition without progression, even when with a considerable compression. Degree of the compression in RMCVA may not be relevant to the patient's neurological status.

Keywords: Magnetic resonance imaging, Medullary compression, Natural history, Vertebral artery

INTRODUCTION

The medulla oblongata forms the lower part of the brainstem. The upper limit of it is adjacent to the bulbous pons, while the lower limit is estimated to lie approximately 3.4 mm caudal to the olivary inferior margin and 6.4 mm caudal to the obex or 10–12 mm above the foramen magnum plane. At the level of the rostral medulla, the pyramid is located anterior to the inferior olive, with the spinal trigeminal nucleus located in its lateral part. Based on extensive studies of microvascular decompression syndrome studies and surgical treatment outcomes, the rostral ventrolateral medulla has been thought to have a role in arterial blood pressure control. Notably, symptoms caused by rostral medullary compression of the vertebral artery (RMCVA) are characterized as medullary compression syndrome (MCS), including intractable dizziness, vertigo, ataxia, dysarthria, dysphagia, dyspnea, progressive or acute paralysis, hemisensory loss, central sleep apnea, excessive daytime sleepiness, and cervical

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myelopathy.\(^5\)\(^,\)\(^11\)\(^,\)\(^12\)\(^,\)\(^15\)\(^,\)\(^25\)\(^,\)\(^26\)\) In contrast, asymptomatic RMCVA has been documented as a frequent radiological finding.\(^\text{[9,10]}\) For these reasons, mechanisms of neurovascular compression syndrome and trauma- and age-associated vertebral artery (VA) elongation have been extensively investigated using magnetic resonance imaging (MRI).\(^\text{[2,6,10,13,18,20]}\) However, to the best of our knowledge, no studies have documented the natural history of RMCVA.

Dolichoectasia is an arterial disease causing abnormal dilation and tortuosity in affected vessels, which more frequently affects the vertebrobasilar system than the anterior circulation.\(^8\) Vertebrobasilar dolichoectasia (VBDE) commonly results in poor prognosis due to brainstem compression of progressively enlarging vessels that causes diverse neurological dysfunctions and ischemic and/or hemorrhagic stroke.\(^\text{[8,9,21,23,28]}\)

Thus, the aim of the present study was to explore asymptomatic RMCVA in comparison with VBDE.

### Table 1: Demographics of 57 patients with rostral medullary compression of the vertebral artery.

| Compression identification rate | 64.0% (57/89) |
|--------------------------------|---------------|
| Sex composition               | Man 30; woman 27 |
| Age                           | 50.0±17.1 years (range, 18–77) |
| Body mass index               | 22.5±3.8 (range, 14.1–32.7) |
| Hypertension                  | 7.0% (4/57) |
| Diabetes mellitus             | 1.8% (1/57) |
| Dyslipidemia                  | 3.5% (2/57) |
| Smoking history               | 1.8% (1/57) |
| Mild compression              | 84.2% (48/57, right 37; left 39) |
| Considerable compression      | 15.8% (9/57, right 8; left 2) |

### MATERIALS AND METHODS

The present retrospective study initially involved 89 outpatients and two VBDE patients. Patients in the former group, who presented with headaches, focal seizures, transient dizziness, tinnitus, hearing disturbance, and hemisensory disturbances, visited our hospital outpatient department from September 2010 to September 2013. Before MRI examination, detailed interviews and neurological evaluations confirmed that none of the 89 patients had a history of the previous cerebral infarction, intracerebral hemorrhage, meningitis, neurodegenerative disease, traumatic brain injury, hydrocephalus, or brain tumor. They were excluded using conventional MRI examination with axial T1- and T2-weighted imaging, T2 gradient echo, fluid-attenuated inversion recovery, diffusion-weighted sequences. Then, the patients underwent contrast studies through intravenous gadolinium infusion (0.1 mmol/kg) on T1-weighted sequences of the axial, coronal, and sagittal sections. Consecutively, 57 of the 89 patients (64.0%) were determined to have radiological RMCVA. Patients suspected of having MCS were not found. Following exclusion, the patient population finally comprised 30 men and 27 women, with a mean age of 50.0 ± 17.1 years (range: 18–77 years) and a mean body mass index (BMI) of 22.5 ± 3.8 (range: 14.1–32.7). Medically controlled hypertension, diabetes mellitus, and dyslipidemia and smoking history were found in 4 (7.0%), 1 (1.8%), 2 (3.5%), and 1 (1.8%) patient respectively [Table 1]. The medullary VA compression site was localized on postcontrast axial T1-weighted images, where the anterolateral 1/4 area of the medulla was divided into three parts: ventral (V), ventrolateral (VL), and lateral (L) [Figure 1a]. Moreover, the degree of medullary VA

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**Figure 1:** Schematic drawings explaining the methodology for localizing the site of vertebral artery compression on the rostral medulla oblongata, carried out on the anterolateral 1/4 area of the axial plane divided into three parts (a) and that for defining the degree of medullary compression in the longitudinal or transverse dimension (b). D: maximal dimension of the hypothetically unaffected, intact medulla oblongata at the vertebral artery compression site, Lat: Lateral, R: Maximal vertebral artery dimension at the medullary compression site, V: Ventral, VA: Vertebral artery, VL: Ventrolateral; dotted line: hypothetically intact contour of the medulla oblongata.
compression was assessed on postcontrast axial T1-weighted images in the longitudinal or transverse dimensions, based on the maximal dimension of the hypothetically unaffected intact medulla at the compression site (D) and maximal VA dimension at the medullary compression site (R). In this study, when the angle formed by two lines connecting the center of the VA and the anterior and posterior margins (θ) were <120° or D was <R/2, the compression was defined as mild. In contrast, when θ was more than 120° or D was greater than R/2, the compression was considered to be considerable [Figure 1b]. Of the 57 patients, 19 who presented 32 sides of radiological RMCV A and could be followed for 5.7 ± 1.9 years (range: 3.0–10.3 years) with periodical physical examination and noncontrast surveillance MRIs were analyzed in detail. This patient population comprised 11 men and eight women, with a mean age of 47.8 ± 14.7 years (range: 24–71 years), including four patients with hypertension, one with diabetes mellitus, two with dyslipidemia, and one with smoking history.

Two patients in the latter group visited our hospital, were diagnosed with VBDE, and were followed from June 2010 to July 2013 and from September 2015 to March 2021.

All imaging sequences were performed using a 3.0 T MR scanner (Achieva R2.6; Philips Medical Systems, Best, The Netherlands). Imaging data were then transferred to a workstation (Virtual Place Lexus 64, 64th edition; AZE, Tokyo, Japan) and independently analyzed by three of the authors (S.T., S.N., and H.I.). Afterward, the Wilcoxon signed-rank test was used for statistical analyses.

The present study was performed in accordance with the guidelines of our institution for human research, and written informed consent was obtained from all the patients before the start of the study.

RESULTS

On axial T2- and postcontrast T1-weighted axial and coronal MRI images, RMCV A was well delineated in all 57 patients [Figure 2]. The offending VAs were commonly tortuous with variable diameters, and all the medullary compression sites were solely identified in the rostral part. Among these cases of RMCVA, 48 (84.2%) were mild compressions, with 37 sides on the right and 39 on the left [Figure 3]. Meanwhile, the remaining 9 (15.8%) showed considerable compression with tortuous VA; eight sides were on the right and two on the left [Figure 4 and Table 1]. In the selected 19 patients, the RMCVA was found on 17 sides on the right and 15 on the left. Furthermore, these were identified at the following sites: V on one side (3%), VL in 22 (69%), and L in 9 (28%). Of the identified cases of RMCVA, 30 (94%) were mild compressions and 2 (6%) were considerable compressions. During the follow-up period, none of the patients showed neurological deterioration or radiological progression of RMCVA [Table 2]. For example, serial MRI images of a 69-year-old man who initially presented with dull headache and was followed for 7.7 years are shown in [Figure 5 and Table 2, No 19]. In addition, statistical analysis showed that male
Figure 3: Postcontrast axial T1-weighted magnetic resonance images of different patients showing mild vertebral artery compression of the rostral medulla oblongata (a-f, arrows) in the ventral (a and b), ventrolateral (c and d), and lateral (e and f) aspects. CeT: Cerebellar tonsil, Med: Medulla oblongata, VA: Vertebral artery.

Figure 4: Postcontrast axial T1-weighted magnetic resonance images of different patients showing the vertebral arteries with variable diameters considerably compressing the rostral medulla oblongata (a-i, arrows) on the right (a, c-f, i), left (g), and both sides (b). CeT: Cerebellar tonsil, Med: Medulla oblongata, VA: Vertebral artery.
sex, aging, and an increase in BMI significantly contributed to a considerable compression. In contrast, hypertension, diabetes mellitus, dyslipidemia, and smoking history were statistically unevaluable due to the small number of participants [Table 3].

**Table 2: Analyzed factor and statistical difference between mild and considerable compression.**

| S. No. | Age (y) | Sex | HT | Side, mode | Aspect on Med | FUP (y) | ND | RP |
|-------|---------|-----|----|------------|---------------|---------|----|----|
| 1.    | 40      | M   | (−) | L, mc      | Lat           | 10.3    | (−) | (−) |
| 2.    | 51      | M   | (−) | R, mc; L, mc | R, VL; L, VL  | 7.7     | (−) | (−) |
| 3.    | 26      | M   | (−) | R, mc      | V             | 5.5     | (−) | (−) |
| 4.    | 64      | W   | (−) | R, mc; L, mc | R, Lat; L, Lat | 3.0     | (−) | (−) |
| 5.    | 32      | M   | (−) | R, mc      | Lat           | 4.2     | (−) | (−) |
| 6.    | 24      | M   | (−) | R, mc; L, mc | R, VL; L, VL  | 6.2     | (−) | (−) |
| 7.    | 71      | W   | (+) | R, mc; L, mc | R, Lat; L, Lat | 4.0     | (−) | (−) |
| 8.    | 43      | W   | (−) | R, mc; L, mc | R, VL; L, VL  | 5.7     | (−) | (−) |
| 9.    | 48      | W   | (−) | R, mc; L, mc | R, VL; L, VL  | 6.8     | (−) | (−) |
| 10.   | 52      | M   | (−) | R, mc; L, mc | R, Lat; L, VL | 6.9     | (−) | (−) |
| 11.   | 38      | M   | (−) | R, mc; L, mc | R, VL; L, VL  | 7.0     | (−) | (−) |
| 12.   | 25      | M   | (−) | R, mc; L, mc | R, VL; L, VL  | 7.0     | (−) | (−) |
| 13.   | 63      | W   | (−) | R, mc      | Lat           | 3.8     | (−) | (−) |
| 14.   | 47      | W   | (−) | R, mc; L, mc | R, VL; L, Lat | 6.9     | (−) | (−) |
| 15.   | 50      | W   | (+) | R, cc      | VL            | 3.4     | (−) | (−) |
| 16.   | 64      | M   | (−) | L, mc      | VL            | 3.3     | (−) | (−) |
| 17.   | 63      | W   | (+) | R, mc; L, mc | R, VL; L, VL  | 3.8     | (−) | (−) |
| 18.   | 38      | M   | (+) | R, mc; L, mc | R, VL; L, VL  | 5.2     | (−) | (−) |
| 19.   | 69      | M   | (−) | R, cc; L, mc | R, VL; L, VL  | 7.7     | (−) | (−) |
| 20.   | 47.8±14.7 | (Range, 24−71) | W8 | M: 11; 4/19 | R: 17; L: 15 | (Range, 3.0−10.3) | 5.7±1.9 | 0 | 0 |

cc: Considerable compression, FUP: Follow-up period, HT: Hypertension, L: Left, Lat: Lateral, mc: Mild compression, M: Man, Med: Medulla oblongata, ND: Neurological deterioration, R: Right, RP: Radiological progression, V: Ventral, VL: Ventrolateral, W: Woman

**Figure 5:** (a-e) Serial axial T2-weighted magnetic resonance images showing stable findings of vertebral artery compressions on both sides of the rostral medulla oblongata (arrows) for 7.7 years. Med: Medulla oblongata, VA: Vertebral artery.

**VBDE cases**

**Case 1**

A 70-year-old hypertensive man suffering from cerebral infarction presented with motor paresis in the left
upper extremity and dysarthria. Cerebral MRI showed a VBDE mildly compressing the rostroventral medulla [Figures 6a and b]. Two years later, the patient sustained subarachnoid hemorrhage due to VBDE rupture, which was conservatively managed, followed by ventriculoperitoneal shunting for delayed communicating hydrocephalus. One year later, the patient presented with dyspnea and aspiration pneumonia. Cerebral MRI revealed a remarkable enlargement of the VBDE with marked compression of the rostroventral medulla [Figures 6c and d]. The patient was then transferred to another hospital in Grade 5 on the modified Rankin scale and lost to follow-up.

Case 2

A 75-year-old asymptomatic man underwent cerebral MRI for physical check-up and was diagnosed with VBDE and mild pontine compression [Figures 7a-c]. The patient had a medical history of hypertension and dyslipidemia, in addition to habitual smoking. Subsequently, the patient was placed under observation as an outpatient, showing no signs of neurological deterioration for 5.4 years, when, the patient presented with intolerable nausea. Cerebral MRI then performed revealed marked VBDE enlargement with considerable compression to the rostral ventrolateral medulla and pons [Figures 7d-f]. Detailed examinations did not identify any causative pathologies for his nausea other than the VBDE. One month later, the patient was emergently transported to our hospital for cardiopulmonary arrest. Autopsy imaging showed diffuse subarachnoid hemorrhage, predominantly distributed in the cerebral cisterns of the posterior fossa.

DISCUSSION

In the present study, there were no patients with RMCVA presenting with neurological deterioration or radiological progression, even when with considerable degrees of compression. In contrast, as with the previous reports, patients with VBDE showed both neurological and radiological progressions.\[8,23\] This means that these two entities may have different etiologies, although requiring further considerations. Initially, the population size of asymptomatic RMCVA was small, with only a few patients with hypertension, diabetes mellitus, dyslipidemia, and smoking history that may contribute to development of arteriosclerosis and consequent VA elongation. Moreover, in our study, patients with these comorbidities were well controlled by medical treatment. Next, the most identified cases of RMCVA showed a mild compression, which could in part explain a benign natural history of asymptomatic

| Analyzed factor   | Statistical difference |
|-------------------|------------------------|
| Male Sex          | MC<CC                  |
| Aging             | MC<CC                  |
| Body mass index   | MC<CC                  |
| Hypertension      | SUE                    |
| Diabetes mellitus | SUE                    |
| Dyslipidemia      | SUE                    |
| Smoking history   | SUE                    |

CC: Considerable compression, MC: Mild compression, SUE: Statistically unevaluable

**Table 3:** Profile of 19 patients (32 sides) with neurological and radiological follow-ups.

**Figure 6:** (a-d) Axial T2-weighted magnetic resonance images of a 70-year-old man showing a remarkable vertebrobasilar dolichoectasia enlargement in 3.2 years with marked compression of the rostroventral medulla oblongata (a, c, d: asterisk).

**Figure 7:** (a-f) Axial T2-weighted magnetic resonance images of a 75-year-old man showing marked vertebrobasilar dolichoectasia enlargement in 5.4 years with considerable compression of the rostral ventrolateral medulla oblongata (f: asterisk).
RMCA. Furthermore, in the present study, nearly 30% of the identified VA compression sites were located in the lateral part of the medulla, instead of the ventrolateral part. The lateral location might associate with an asymptomatic presentation of RMCA since it is less likely compress the central vasomotor center and pyramidal tracts in a direct manner. In contrast, a fraction of VBDE cases has been documented to be asymptomatic, but the natural history of this condition has yet to be described and is still unclear. Formation of RMCA and VBDE may be multifactorial, as it could be influenced by age, sex, BMI, underlying diseases, race, and social life. In addition, some excessive dynamics exerted to the head-and-neck regions involving the VA may contribute to their formation. Thus, comprehensive investigations are needed to better understand RMCA and VBDE with their similarities and differences. In our study, male sex, aging, and an increase in BMI significantly contributed to considerable RMCA presentation. These factors may promote VA elongation and medullary compression through consequent arteriosclerosis and hypertension.

This study had several limitations. First, it was performed retrospectively in small populations, and the participants were not randomly assigned. In addition, the study population consisted of patients with inhomogeneous sex and age distributions. Finally, there were few patients with hypertension, diabetes mellitus, dyslipidemia, and smoking history in the RMCA population, which made it difficult to evaluate them using a statistical approach. Even with these limitations, we believe that the findings of this study can help to better understand the characteristics and natural history of RMCA.

CONCLUSION

Unlike VBDE, RMCA seems to be a benign condition without progression even when with a considerably tortuous appearance. Degree of the compression may not be relevant to the patient's neurological status. RMCA conditions may be formed in association with aging and obesity.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Akaishi T, Kiyomoto H, Abe M, Okuda H, Ishikawa K, Endo T, et al. A 29-year-old woman with recurrent pregnancy-induced hypertension based on vascular compression of the medulla oblongata. Intern Med 2019;58:2257-61.
2. Carlson EJ, Tominaga Y, Ivancic PC, Panjabi MM. Dynamic vertebral artery elongation during frontal and side impacts. Spine J 2007;7:222-8.
3. Cravo SL, Possas OS, Ferreira-Neto ML. Rostral ventrolateral medulla: An integrative site for muscle vasodilation during defense-altering reactions. Cell Mol Neurobiol 2003;23:579-95.
4. Deistung A, Schäfer A, Schweser F, Biedermann U, Güllmar D, Trampel R, Turner R, et al. High-resolution MR imaging of the human brainstem in vivo at 7 Tesla. Front Hum Neurosci 2013;7:710.
5. DelRosso L, Gonzalez-Toledo E, Chesson AL Jr., Hoque R. Positional central apnea and vascular medullar compression. Neurology 2012;79:2156-7.
6. Ding S, Yan X, Guo H, Yin F, Sun X, Yang A, et al. Morphological characteristics of the vertebrobasilar artery system in patients with hemifacial spasm and measurement of bending length for evaluation of tortuosity. Clin Neurol Neurosurg 2020;198:106144.
7. Fontes MA, Silva LC, Campagnole-Santos MJ, Khosla MC, Guertzenstein PG, Santos RA. Evidence that angiotensin-(1-7) plays a role in the central control of blood pressure at the ventro-lateral medulla acting through specific receptors. Brain Res 1994;665:175-80.
8. Gutierrez J, Sacco RL, Wright CB. Dolichoectasia-an evolving arterial disease. Nat Rev Neurol 2011;7:41-50.
9. Ikeda K, Nakamura Y, Hirayama T, Sekine T, Nagata R, Kano O, et al. Cardiovascular risk and neuroradiological profiles in asymptomatic vertebrobasilar dolichoectasia. Cerebrovasc Dis 2010;30:23-8.
10. Ivancic PC, Ito S, Tominaga Y, Carlson EJ, Rubin W, Panjabi MM. Effect of rotated head posture on dynamic vertebral artery elongation during simulated rear impact. Clin Biomech (Bristol, Avon) 2006;21:213-20.
11. Kutty RK, Yamada Y, Takizawa K, Kato Y. Medullary compression due to ectatic vertebral artery-case report and review of literature. J Stroke Cerebrovasc Dis 2020;29:104460.
12. Li Q, Xie P, Yang WS, Yan B, Davis S, Caplan LR. Vertebral artery compression syndrome. Front Neurol 2019;10:1075.
13. Manava P, Naraghi R, Schmieder R, Fahlbusch R, Doerrler A, Lell MM, et al. 3D-visualization of neurovascular compression at the ventrolateral medulla in patients with arterial hypertension. Clin Neuroradiol 2021;31:335-45.
14. Middlebrooks EH, Yamamura K, Bennett JA, Bidari S. Normal relationship of the cervicomedullary junction with the obex and olivary bodies: A comparison of cadaveric dissection and in vivo diffusion tensor imaging. Surg Radiol Anat 2015;37:493-7.
15. Miyazaki M, Hashimoto T, Sakurama N, Yoshimoto T, Tayama M, Kuroda Y. Central sleep apnea and arterial compression of the medulla. Ann Neurol 1991;29:564-5.
16. Naraghi R, Gaab MR, Walter GF, Kleinberg B. Arterial hypertension and neurovascular compression at the
ventrolateral medulla. A comparative microanatomical and pathological study. J Neurosurg 1992;77:103-12.

17. Nezu T, Hosomi N, Kuzume D, Naito H, Aoki S, Morimoto Y, et al. Effects of vascular compression on the rostral ventrolateral medulla for blood pressure variability in stroke patients. J Hypertens 2020;38:2443-50.

18. Nibu N, Cholewicki J, Panjabi MM, Babat LB, Grauer JN, Kotha R, et al. Dynamic elongation of the vertebral artery during an in vivo whiplash simulation. Eur Spine J 1997;6:286-9.

19. Nicholas JS, D’Agostino SJ, Patel SJ. Arterial compression of the retro-olivary sulcus of the ventrolateral medulla in essential hypertension and diabetes. Hypertension 2005;46:982-5.

20. Nishitaka M, Hirashima Y, Tomita T, Futatsuya R, Horie Y, Endo S. Measurement of basilar artery bending and elongation by magnetic resonance cerebral angiography: Relationship to age, sex and vertebral artery dominance. Arch Gerontol Geriatr 2004;38:251-9.

21. Passero S, Rossi S, Giannini F, Nuti D. Brain-stem compression in vertebrobasilar dolichoectasia. A multimodal electrophysiological study. Clin Neurophysiol 2001;112:1531-9.

22. Quisling RG, Quisling SG, Mickle JP. Obex/nucleus gracilis position: Its role as a marker for the cervicomedullary junction. Pediatr Neurosurg 1993;19:143-50.

23. Sadashiva N, Shukla D, Bhat DI, Devi BI. Vertebral artery dolichoectasia with brainstem compression: Role of microvascular decompression in relieving pyramidal weakness. Acta Neurochir (Wien) 2016;158:797-801.

24. Salvi F, Mascalchi M, Bortolotti C, Meletti S, Plasmati R, Rubboli G, et al. Hypertension, hyperekplexia, and pyramidal paresis due to vascular compression of the medulla. Neurology 2000;55:1381-4.

25. Savitz SI, Ronthal M, Caplan LR. Vertebral artery compression of the medulla. Arch Neurol 2006;63:234-41.

26. Schulz R, Fegbeutel C, Althoff A, Traupe H, Grimminger F, Seeger W. Central sleep apnoea and unilateral diaphragmatic paralysis associated with vertebral artery compression of the medulla oblongata. J Neurol 2003;250:503-5.

27. Sendeski MM, Consolim-Colombo FM, Leite CC, Rubira MC, Lessa P, Krieger EM. Increased sympathetic nerve activity correlates with neurovascular compression at the rostral ventrolateral medulla. Hypertension 2006;47:988-95.

28. Tomasello F, Alafaci C, Salpietro FM, Longo M. Bulbar compression by an ectatic vertebral artery: A novel neurovascular construct relieved by microsurgical decompression. Neurosurgery 2005;56 Suppl 1:117-24.

29. Watters MR, Burton BS, Turner GE, Cannard KR. MR screening for brain stem compression in hypertension. AJNR Am J Neuroradiol 1996;17:217-21.

30. Zizka J, Ceral J, Elias P, Tintera J, Klzo L, Solar M, et al. Vascular compression of rostral medulla oblongata: Prospective MR imaging study in hypertensive and normotensive subjects. Radiology 2004;230:65-9.

How to cite this article: Tsutsumi S, Nonaka S, Ono H, Ishii H, Vertebral artery compression of the medulla oblongata: A benign radiological finding? Surg Neurol Int 2022;13:36.