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Essential Hypertension and Neurovascular Compression at the Ventrolateral Medulla Oblongata: MR Evaluation

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PURPOSE: To investigate, using MR imaging, neurovascular compression at the ventrolateral medulla oblongata in patients with essential hypertension. **METHODS:** Thirty-two patients with essential hypertension (57.6 ± 7 years of age), 6 patients with secondary hypertension (56.7 ± 10.3 years of age), and 18 control subjects (50.5 ± 11 years of age) were evaluated. Transaxial three-dimensional fast low angle shot images were obtained (38/6/1 [repetition time/echo time/excitations]). The center of a 40-mm-thick slab (16 partitions) was placed at the pontomedullary junction. We evaluated the relationships between the upper ventrolateral medulla and the vertebral arteries and branches identified by their flow-related hyperintensities in each group. **RESULTS:** In the essential hypertension group, 29 (90.6%) of 32 cases showed neurovascular compression. Of those, 22 demonstrated neurovascular compression on the left side, 3 on both sides, and 4 on the right side. In the control group, 4 cases of 18 showed neurovascular compression. In the secondary hypertension group, 1 of 6 cases showed neurovascular compression. The rates of observed neurovascular compression between controls and essential hypertension group were statistically significant. **CONCLUSION:** We found a close correlation between essential hypertension and neurovascular compression at the ventrolateral medulla oblongata on the left side. MR with a 3-D fast low-angle shot sequence has acceptable spatial resolution and depicts blood vessels simultaneously by flow-related phenomena.

Index terms: Hypertension; Medulla oblongata; Brain stem, magnetic resonance

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Hyperactive cranial nerve dysfunction syndromes such as trigeminal neuralgia, hemifacial spasm, and glossopharyngeal neuralgia are currently treated by microvascular decompression with favorable results (1–4). The preoperative neuroradiologic examinations are vertebral angiogram, computed tomography (CT), and magnetic resonance (MR). Of those, MR has been one of the most important methods for preoperative evaluation of these hyperactive cranial nerve syndromes (5). But these examinations cannot always show the offending ves-

sels, so the surgical indications are determined mainly by the patients' clinical symptoms (6).

In 1978, Jannetta and his colleagues reported surgical cases of neurogenic hypertension and suggested that pulsatile compression of the left lateral medulla oblongata could cause arterial hypertension (7). Several clinical reports supporting this concept followed (8–12). In 1991, Yamamoto et al reported an experimental model of neurovascular compression at the ventrolateral medulla along with clinical cases of microvascular decompression for hypertension (13). In the same year, Kleineberg and his colleagues reported angiographic findings in patients with hypertension: in 80% of the angiograms of the hypertensive patients, an artery crossed the left root entry zone of the 9th and 10th cranial nerves (14). In 1992, Naraghi et al reported microanatomic studies of autopsy cases of essential hypertension and concluded that neurovascular compression at the left ventrolateral medulla is a cause of essential hypertension (15).

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TABLE 1: Hypertension characteristics in 56 cases

Characteristic	Essential Hypertension	Secondary Hypertension	Control Group
No. of cases	32	6	18
Sex, M/F	13/19	3/3	11/7
Age range, y	42–69	36–65	33–69
Average age, y	57.6 \pm 7	56.6 \pm 10.3	50.5 \pm 11
Systolic blood pressure, mm Hg	172.9 \pm 10.3	181.7 \pm 11.7	128.2 \pm 11
Diastolic blood pressure, mm Hg	96.8 \pm 10.5	106.3 \pm 8.5	70.3 \pm 11
Duration of hypertension, mo	35.8 \pm 31.2	92 \pm 31	

Note.—Average values are expressed as mean \pm standard deviation.

In this study, we evaluated neurovascular compression at the ventrolateral medulla in patients with essential hypertension using MR, which can provide us good spatial resolution and flow images simultaneously.

Materials and Methods

Fifty-six cases were evaluated (Table 1). The patients with essential and secondary hypertension all were referred from the department of cardiology of our hospitals for cerebrovascular disease. Of those, 38 patients who agreed to MR examination with additional sequence were evaluated. Patients over 70 years of age were excluded. Thirty-two patients were classified as having essential hypertension, and 6 patients as having secondary hypertension (5 renal hypertension, 1 primary hyperaldosteronism). The average age was 56.6 \pm 10.3 years. The essential hypertension group ranged from 42 to 69 years of age (57.6 \pm 7 years) and included 19 women and 13 men. In the essential hypertension group, the blood pressure before treatment ranged from 150/80 mm Hg (systolic blood pressure/diastolic blood pressure) to 210/120 mm Hg (172.9 \pm 10.3/96.8 \pm 10.5: mm Hg), and average duration of hypertension ranged from 3 to 120 months (35.8 \pm 31.2 months). In the secondary hypertension group, blood pressure before treatment ranged from 160/90 mm Hg to 220/120 mm Hg (181.7 \pm 11.7/106.3 \pm 8.5 mm Hg), and average duration of hypertension ranged from 12 to 144 months (92 \pm 31 months). The patients with hypertension all were on antihypertensive medication.

Eighteen normotensive volunteers were evaluated as the control group. They had a systolic blood pressure less than 140 mm Hg and a diastolic pressure less than 90 mm Hg at their office visit (128.2 \pm 11/70.3 \pm 11 mm Hg). The controls ranged from 33 to 69 years of age (50.5 \pm 11 years) and included seven women and 11 men.

MR was performed on a 1.5-T superconducting magnet and a circularly polarized head coil. A 50-mm-thick transverse saturation band was placed at the parietal region to reduce signal from venous blood. After obtaining a initial T1-weighted sagittal section, we obtained transaxial three-

dimensional fast low-angle shot images using the following parameters: 38/6/1 (repetition time/echo time/excitations), 250 mm field of view, 45° flip angle, matrix 192 \times 256. The slab thickness was 40 mm, and 16 partitions were obtained. Scanning time was 5 minutes, 13 seconds. The center of the slab was placed at the pontomedullary junction, and the slab was placed parallel to the hard palate.

Critical evaluation is limited to the area extending 15 mm caudal to the pontomedullary junction (16, 17). In that section, we could see the pyramid, olive, flocculus, and inferior cerebellar peduncle. We could recognize the 9th and 10th cranial nerve rootlets in most cases. In this area, we evaluated the relationships between the medulla and the vertebral arteries and branches identified by their flow-related hyperintensities. "Neurovascular compression" was diagnosed when the hyperintensities were obviously contiguous with the medulla or the nerve root entry zone. The deformity of medulla was not necessary to define neurovascular compression. The MR scans were evaluated, reviewed, and discussed by three readers without blinding.

No patients were treated surgically in our series.

The significance of the correlation between neurovascular compression and essential hypertension was statistically analyzed using χ^2 test.

Results

Essential Hypertension Group ($n = 32$)

Twenty-nine cases (90.6%) of neurovascular compression were depicted in the essential hypertension group (Table 2). Four cases were seen on the right side, 22 on the left side, and three on both sides. Neurovascular compression was absent in three cases. In 16 cases, the medulla was apparently deformed by an artery (Fig 1). The vertebral artery alone caused neurovascular compression at the left ventrolateral medulla in 18 cases. The vertebral artery combined with smaller branches (posterior inferior

TABLE 2: Findings of neurovascular compression in 56 MR studies

Factor	Essential Hypertension	Secondary Hypertension	Control Group
No. of cases	32	6	18
Right side	4	0	1
Left side	22	1	2
Bilateral	3	0	1
Total	29	1	4

cerebellar artery in most cases) compressed the medulla and the root entry zone in 7 cases (Fig 2).

Secondary Hypertension Group (n = 6)

Neurovascular compression was noticed in one patient with renal hypertension. The compressing artery was the left vertebral artery and the left posterior inferior cerebellar artery.

Control Group (n = 18)

Four cases (22.2%) showed neurovascular compression: 1 on the right side, 2 on the left side, and 1 on both sides. None of 18 cases had definitely compressed and deformed medulla by the arteries (Fig 3). The rates of observed neurovascular compression between control subjects and the essential hypertension group were statistically significant ($\chi^2 = 21.1$, $P < .01$).

Discussion

In the essential hypertension group, more than 90% of the cases showed neurovascular compression, especially on the left side. This rate was significantly higher than that of the control group ($P < .01$). Our findings of a close relationship between essential hypertension and neurovascular compression at the ventrolateral medulla on the left side support the theory of

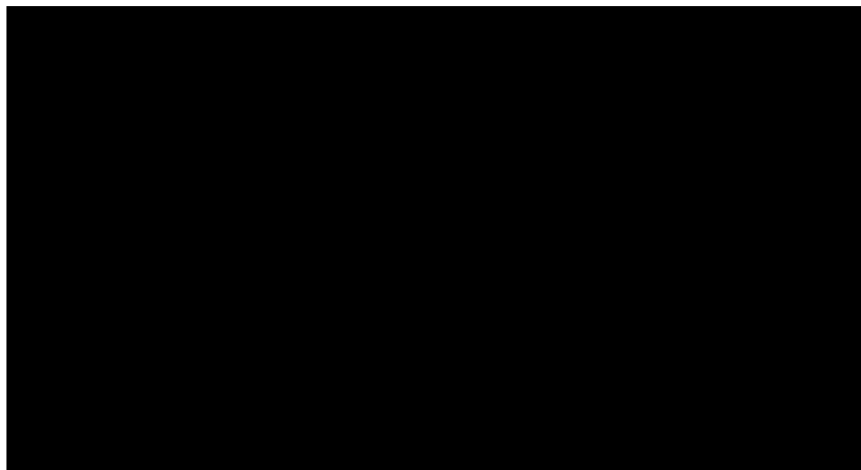


Fig 1. 60-year-old woman with long-standing essential hypertension. The medulla is compressed and deformed by the left vertebral artery (arrow) at the left retroolivary sulcus.

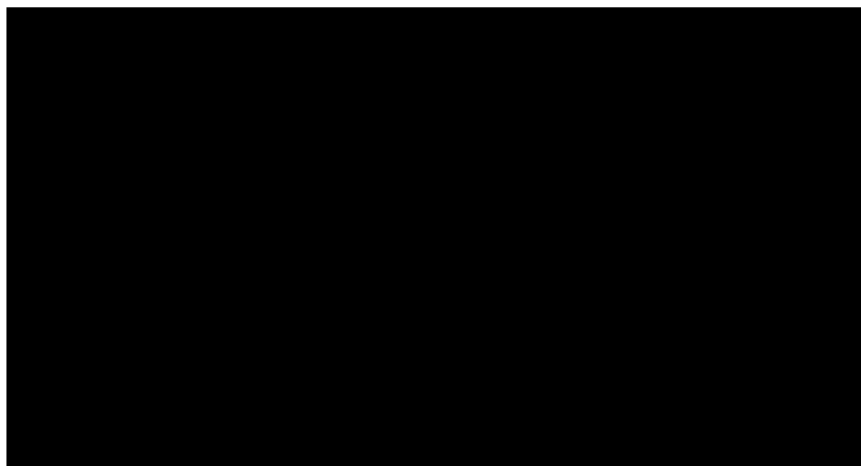
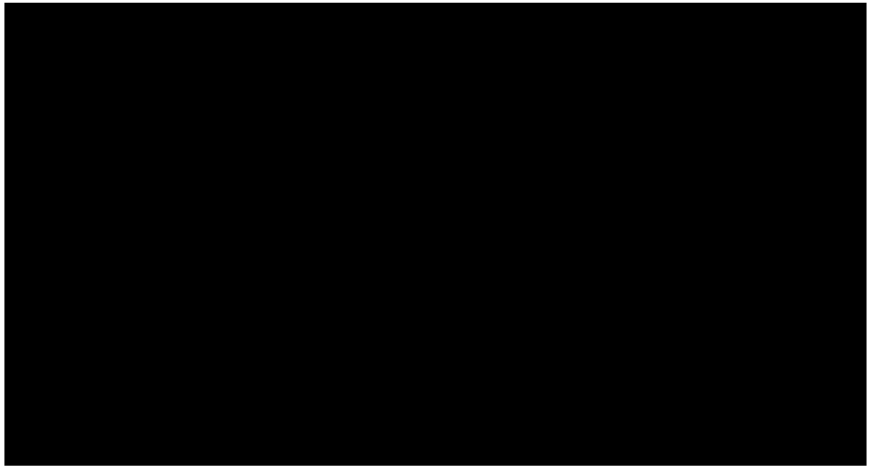


Fig 2. 68-year-old woman with essential hypertension. The left vertebral artery (arrow) and its branch (arrowhead) compress the medulla at the left retroolivary sulcus.

Fig 3. 58-year-old normotensive man (control subject). Vertebral arteries are shown as hyperintensities (*arrowheads*). The 9th and 10th cranial nerve rootlets can be recognized (*arrows*). No vascular structure apparently compresses the medulla.



Jannetta and the observation of Kleineberg and Naraghi et al. Jannetta stated that the offending artery was seen between the inferior olive and the 9th and 10th cranial nerves with the loop of the artery compressing the medulla (10). Naraghi reported that the neurovascular compression was recognized at the rostral medulla just caudal to the pontomedullary junction and lateral to the olive in the retroolivary sulcus (15). The results of our observations were consistent with those findings.

Based on the intraoperative observations, Jannetta and coworkers concluded that pulsatile compression of the left lateral medulla could cause arterial hypertension (7, 8, 10, 11). Neurovascular compression at the ventrolateral medulla can cause permanent irritation and activation of the vasopressor neurons (18–21). Neurovascular compression at the 10th cranial nerve can cause a blockade of cardiac C-fibers of the vagus nerve, which results in a rise in blood pressure (22). There is no reasonable explanation for the left-sided dominance of neurovascular compression.

Neurovascular compression is a physiologic phenomena. It is difficult to obtain true “live pictures” from the cadaver specimen even with the perfusion method (15, 23). In a control group, we clearly cannot explore the left ventrolateral medulla by surgery. Angiograms do not show the medullary structures. On the other hand, MR provides good spatial resolution and flow images simultaneously. Thus, it is a suitable method for evaluation of neurovascular compression.

We used 3-D fast low-angle shot imaging for detection of cranial nerve and offending arteries. Compared with the two dimensional spin

echo imaging, this 3-D gradient-echo sequence has a better spatial resolution as well as the ability to show the flow-related phenomena (24–26). The anatomic information provided was acceptable considering the short acquisition time: the roots of the 9th and 10th cranial nerves were recognized in most cases. This sequence can easily be added to the routine examination for screening purpose.

According to the findings of Jannetta and Naraghi, the presence of an arterial loop caused by arteriosclerosis was the important finding in patients with neurovascular compression (10, 15). In MR, such a segment can be missed with time of flight MR angiography, because there can be signal loss in the segment of inferior flow direction in the looping segments. Pulsation and complex flow within the vascular loops may also contribute to signal loss (27). We chose a short echo time (6 milliseconds) to reduce the in-plane flow effect, but an even shorter echo time will be necessary in future.

With prolonged hypertension, arteries become elongated and tortuous. So the possibility arises that neurovascular compression could be a result of hypertension. Naraghi et al found no compressing vascular loop in cases of hypertension (13). In our secondary hypertension group, only one of six patient showed vascular compression. Because the duration of secondary hypertension was longer than that of essential hypertension in our series, it is not probable that neurovascular compression is a secondary phenomena caused by long-standing hypertension.

We did not find constant neurovascular compression in the cases of essential hypertension. This can be attributed to the limit of the spatial

resolution as well as to detection of flow images with MR. Failure to depict the root entry zone and looping of blood vessels may lead to misinterpretation. Indeed, there may be a significant number of patients with "essential hypertension" not caused by neurovascular compression of the ventrolateral medulla. For neurogenic hypertension only, there are several other pathophysiologic factors such as decreased beta-receptor sensitivity, structural vascular changes (28), sympathetic overactivity (29), obesity (30), and so on.

In four cases (22.2%) of the control group, MR showed the vessels apparently contiguous to the medulla. Tash et al reported that 21% of asymptomatic patients had contact by a vascular structure at the root entry zone of the seventh nerve (6). The presence of asymptomatic cases with neurovascular compression makes it difficult to confirm definite symptoms. As they stated, larger prospective studies with surgical correlation are necessary to confirm the MR findings.

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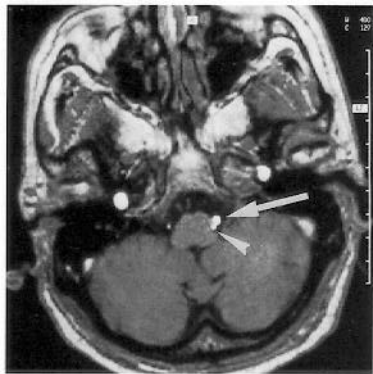
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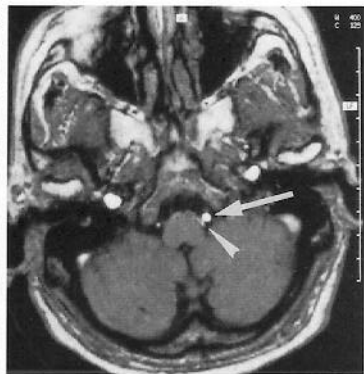
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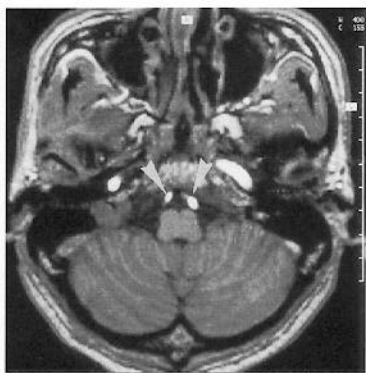
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