Journal of the Neurological Sciences 287 (2009) 227–235

Contents lists available at ScienceDirect

Journal of the Neurological Sciences

journal homepage: www.elsevier.com/locate/jns

Ischemic stroke patterns and hemodynamic features in patients with small vertebrobasilar artery

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ARTICLE INFO

Article history:
Received 31 December 2008
Received in revised form 15 June 2009
Accepted 7 July 2009
Available online 13 August 2009

Keywords:
Small vertebrobasilar artery
Long circumferential artery
Ischemic pattern
MR imaging
Angiography

ABSTRACT

Background: To determine the role of small vertebrobasilar artery (SVBA) in patients with posterior circulation stroke (PCS), we evaluated the ischemic patterns, collateral features, and stroke mechanisms in PCS patients with SVBA.
Methods: Ischemic findings on magnetic resonance (MR) imaging were correlated with 3D time-of-flight/contrast-enhanced MR angiography and/or catheter angiography in 18 patients (mean age, 68.0 ± 11.8 years; 9 males). SVBA (lumen diameter of <3 mm) was compared with stenotic normal-sized VBA (NVBA) in 14 PCS patients.
Results: Ischemic lesions were predominantly observed in the cerebellum and/or medulla (vertebral artery (VA) territory). All subjects had fetal posterior circulation (FPC) from the internal carotid artery to the posterior cerebral artery. Sixteen patients (88.9%) had distal or diffuse VA stenosis/occlusion. In 14 patients (77.8%), long circumferential artery (LCA) was prominently observed. In atherothrombotic patients, infratentorial PCS might occur following artery-to-artery embolism from the low-flowed or stenotic VA to LCA. Ischemic patterns between subjects with and without VA disease were almost similar. As the degree of VA disease increased, the frequency of LCA prominence showed an increased tendency ($P = 0.003$). Relatively small, scattered infarcts were observed in patients with SVBA than in those with stenotic NVBA.
Conclusions: FPC does not protect against infratentorial PCS. Regardless of extensive arterial lesions, relatively small infarcts may be due to previously established collaterals from the LCA, which could compensate for the defects in the infratentorial area.

1. Introduction

A small vertebrobasilar artery (SVBA) is infrequently observed in stroke patients, regardless of whether it is directly related to ischemic stroke. It may not be possible to adequately visualize an SVBA on three-dimensional (3D) time-of-flight (TOF) magnetic resonance (MR) angiography (MRA); moreover, because its exact nature is unclear, it attracts little attention until posterior circulation stroke (PCS) occurs. Ischemic patterns in patients with SVBA appear to be different from those in subjects with a normal-sized VBA (NVBA). Although it has been reported that SVBA might induce PCS [1], the collaterals that would compensate for the posterior circulation system in the case of ischemic stroke have not been well established. Furthermore, a correlation of neuroimaging findings with vascular pathologies has not been attempted in PCS patients with SVBA. Therefore, we evaluated the ischemic pattern of the vascular lesions by magnetic resonance (MR) imaging and the hemodynamic pattern (collateral circulation) by MR angiography (MRA) and/or transfemoral cerebral angiography (TFCA) and assessed the stroke mechanism in PCS patients with SVBA by using the above-mentioned techniques together with transcranial Doppler (TCD).

2. Methods

2.1. Patient selection

The present study is a case series from 2 academic centers (Seoul National University Hospital (SNUH) and Myongji Hospital (MJH)). We assessed 1959 acute ischemic stroke patients (SNUH: $n = 1604$, between September 2003 and February 2006; MJH: $n = 355$, between March 2006 and September 2007). Within 1 week of symptom onset, patients underwent brain MRI and 3D TOF MRA by a 1.5-Tesla MR system (SNUH: Siemens 1.5 Vision, Erlangen, Germany; MJH: Intera 1.5 T 10.3 version, Eindhoven, Netherlands). The stroke types were categorized as follows: anterior circulation stroke (ACS) = 1120 (57.2%), PCS = 640 (32.7%), ACS plus PCS = 33 (1.7%), transient ischemic attack (TIA) = 162 (8.3%), and venous sinus thrombosis = 4 (0.2%).

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doi:10.1016/j.jns.2009.07.007

Table 1
Summary of 18 PCS patients with small vertebrobasilar artery.

No	Sex/age	Risk factor(s)	VA lesion	BA lesion	Prominence	FPC	TCD
1	F/53	HT	–	–	PICA (R)	B	–
2[a]	M/69	HT	–	–	–	B	Decreased MFV and turbulent waves of (B) VBAs (<30 cm/s)
3[a]	F/69	HT, DM, Af	L (diffuse)	D	PICA (B), AICA (R)	B	–
4	F/76	HT	L (V_4)	–	AICA (L)	B	–
5[b]	M/87	HT	L (V_3 and V_4)	–	PICA (L)	B	Stenosis of BA (>80 cm/s)
6[a]	F/76	HT	L (V_4)	M	PICA (B)	B	Stenosis of (B) VBAs (>80 cm/s)
7[a]	M/69	Af	R (diffuse)	–	–	U	Dampened signal of (B) VBAs
8[a]	M/73	HT, Af	R (V_4)	–	–	B	–
9[a]	M/70	HT, DM, smoking	R (diffuse)	–	–	U	Dampened signal of (L) VA and BA Stenosis of (R) VA (> 110 cm/s)
10[a]	F/77	HT, DM	R (V_4), L (diffuse)	–	PICA (R)	B	Blunted signal of BA
11[b]	F/53	HT	B (VBA junction)	P	PICA (B)	B	–
12[b,c]	F/58	HT, DM	B (VBA junction)	–	PICA (B)	B	Blunted signal of (B) VBAs
13[a,b,c]	F/65	HT	B (V_4)	–	PICA (B)	B	Dampened signal of (L) VA and BA bidirectional flow of (R) VA
14[b,c]	M/53	HT	B (V_4)	P	PICA (B)	B	Stenosis of BA (>140 cm/s) Dampened signal of (L) VA
15[a,c]	M/87	HT	B (V_4)	–	PICA (B)	B	Dampened signal of (L) VA Absent signal of (R) VA and BA
16[a,b,c]	M/46	–	R (diffuse) L (V_4)	P	PICA (L), AICA (L)	B	–
17[a,b]	F/64	HT	B (V_4)	–	AICA (B)	B	Stenosis of BA (>120 cm/s) Dampened signal of (B) VAs
18[a,b,c]	M/79	HT	B (diffuse)	P	AICA (B), SCA (B)	B	–

PCS, posterior circulation stroke; VA, vertebral artery; BA, basilar artery; PICA, posterior inferior cerebellar artery; AICA, anterior inferior cerebellar artery; SCA, superior cerebellar artery; FPC, fetal posterior circulation; HT, hypertension; DM, diabetes mellitus; Af, atrial fibrillation; L, left; R, right; U, unilateral; B, bilateral; P, proximal; M, middle; D, distal; TFCA, transfemoral cerebral angiography; MFV, mean flow velocity.
[a] Contrast-enhanced MR angiography performed.
[b] TFCA performed.
[c] Retrograde filling of BA from anterior circulation.

2.2. SVBA definition

The mean diameter of the normal basilar artery (BA) has been reported to be 3.17 mm [2] and the hypoplastic vertebral artery (VA) was defined to have a lumen diameter of less than 2–3 mm [3,4]. Because there is no consensus on the value of SVBA, we defined SVBA as a diameter of less than 3 mm. For defining the diameter of SVBA, we examined the mid-portion level of the BA and the V_2 of the largest VA by using magnified images of MRA on a picture archiving and communication system (PACS). The location of the VA was defined according to standard criteria [5,6] (V_1; prevertebral portion, V_2; the portion within the vertebral columns, V_3; atlantoaxial portion, V_4; intracranial portion).

2.3. Study patients

We assessed 37 acute stroke patients with SVBA. MRI and MRA were obtained at 2.3 ± 1.1 days after stroke onset. We included patients whose configuration of SVBA was visible on 3D TOF MRA. We also included those who had undergone angiographic work-up (e.g., contrast-enhanced MRA or TFCA) even if their SVBA was not or only faintly visible on routine 3D TOF MRA. Patients were excluded if they showed no visible VBA ($n=7$) or only a rudimentary VBA ($n=3$) on 3D TOF MRA; moreover, these 10 patients had not undergone detailed angiographic work-up that could enable a diagnosis of SVBA. Among the excluded 10 patients, 9 had been afflicted by PCS and the remaining one by ACS. Those who showed no relevant lesions on diffusion-weighted images (DWI; $n=2$) were also excluded. Of the 25 stroke patients with SVBA (7 with ACS and 18 with PCS), 18 PCS patients who underwent MRI and MRA at 2.2 ± 1.2 days after the onset of symptoms were eventually selected for the study. Acute ischemic lesion was defined by high-signal intensity on DWI with low-signal intensity on the apparent diffusion coefficient map (only 2 patients [nos. 4 and 18] by T2-weighted image; T2-WI). Of these 18 patients, 12 patients (66.7%; nos. 2, 3, 6–10, 13, and 15–18) underwent contrast-enhanced MRA with the same TOF MR sequence to evaluate the course of the VBA.

We assessed 30 acute PCS patients with NVBA between March 2007 and September 2007 at MJH and considered them as the control group. We regarded NVBA as a diameter of more than 3 mm, except in the case of unilateral hypoplastic VA. We obtained MRI and 3D contrast-enhanced MRA scans for the control subjects at 2.2 ± 1.4 days after the onset of symptoms.

The topography of the infarcts was determined with reference to the maps, establishing an anatomical correspondence with the dominant arterial territories of the brainstem and cerebellum [7]. TFCA was performed in 8 patients with SVBA (nos. 5, 11–14, and 16–18) and 2 patients with NVBA (nos. 7 and 14) after an informed consent was obtained from each patient. The possibility of VBA dissection could be excluded by the findings of TFCA (string sign) or source view of MRA (double lumen) [8].

The long circumferential artery (LCA) (e.g., posterior inferior [PICA], anterior inferior [AICA], or superior cerebellar artery [SCA]) was regarded to be prominent if it was well depicted on MRA or TFCA compared to its parent SVBA. In patients with NVBA, LCA was regarded to be present if it was adequately visible on MRA or TFCA.

The circle of Willis was graded as fetal posterior circulation (FPC) when one of the P2 segments of the posterior cerebral artery was supplied by the internal carotid artery via the posterior communicating artery (absent or hypoplastic P1 segment of the posterior cerebral

Fig. 1. Ischemic stroke patterns according to the various vascular lesions observed in 18 patients with small VBA. Group I: no VA disease, with or without LCA prominence ($n=2$), Group II: unilateral VA disease, with (upper) or without (low) LCA prominence ($n=7$), and Group III: bilateral VA disease, with LCA prominence VA, vertebral artery; BA, basilar artery; LCA, long circumferential artery; PICA, posterior inferior cerebellar artery; AICA, anterior inferior cerebellar artery; SCA, superior cerebellar artery; LAA, large artery atherosclerosis; SAO, small artery occlusion; CE, cardiac embolism.