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Vertebral artery stump syndrome in acute ischemic stroke

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ABSTRACT

Although the carotid artery stump as an embolic source for ischemic stroke has been well described, there have been few systematic reports of a similar syndrome in the posterior circulation (PC) after vertebral artery (VA) origin occlusion. The aim of this study was to identify the incidence and characteristics of acute ischemic stroke with VA stump syndrome. Of 3463 consecutive patients who were admitted within 7 days after onset, 865 patients with acute ischemic stroke in the PC were enrolled. The diagnostic criteria of VA stump syndrome included: (1) acute ischemic stroke in the posterior circulation; (2) the VA origin occlusion identified on MRA, duplex ultrasound, CT angiography, and/or conventional angiography; (3) presence of distal antegrade flow in the ipsilateral VA; and (4) absence of other causes of ischemic stroke. Of the 865 patients with PC stroke, 12 (1.4%) were diagnosed as having VA stump syndrome. The ischemic lesions included the cerebellum in all patients. Nine patients had multiple ischemic lesions in the brain stem, thalamus, or posterior lobe other than cerebellum. On duplex ultrasound, a to-and-fro flow pattern was observed in the culprit VA in 10 patients. Three patients had recurrences of ischemic stroke in the PC during the acute phase. VA stump syndrome was not a rare mechanism of PC stroke, and there was a high rate of stroke recurrence during the acute phase. Vascular assessment by a multimodality approach can be used to promptly detect VA stump syndrome.

1. Introduction

The role of the carotid artery stump as an embolic source for ischemic stroke has been well described [1,2]. Ischemic stroke following carotid artery occlusion can be caused by embolism from the contralateral carotid artery via the circle of Willis, the distal limit of the propagated thrombus, and an embolism from the carotid bifurcation via the external carotid artery [3].

Conversely, there have been few reports of a similar syndrome causing stroke in the posterior circulation after vertebral artery origin occlusion, which has been described as "vertebral artery (VA) stump syndrome" [4,5]. This syndrome was first described in 2008, in which two patients with posterior circulation strokes after VA origin occlusion were treated with endovascular intervention due to failed medical therapy [4]. A similar syndrome was reported in 1992, in which angiographically documented VA origin occlusion caused posterior circulation stroke in 7 patients, before diffusion-weighted MRI (DWI) had been widely applied to detect acute stroke [6]. Because these patients were selected from those who had angiography [6], the incidence of VA stump syndrome has not been determined. Since recent advances in neuroimaging, VA stump syndrome has not been systematically examined.

The goal of the present study was to identify the incidence and clinical characteristics of acute ischemic stroke with VA stump syndrome after DWI had become a routine modality for diagnosing acute stroke.

2. Methods

Between April 2007 and November 2011, 3463 consecutive patients were admitted to our hospital within 7 days of onset of acute ischemic stroke (2258 anterior circulation, 135 anterior and posterior circulation, 865 posterior circulation, and 205 unidentified responsible territory including incomplete imaging study or transient ischemic attack without ischemic lesions). Written informed consent was obtained from all patients or their next of kin. All patients without contraindications underwent both DWI and CT. Cephalocervical arterial lesions were assessed using three-dimensional time-of-flight MR angiography (MRA) unless contraindicated and duplex ultrasound in all patients. All patients underwent transthoracic echocardiography and 24-hour Holter electrocardiography. Three-dimensional CT angiography (3D-CTA) and conventional angiography were performed if possible. The term VA stump syndrome has previously been used without clear definition [4,5]. In the present study, the diagnostic criteria of VA stump syndrome included: (1) acute ischemic stroke in the posterior circulation; (2) VA origin occlusion, which can

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Table 1
Characteristics, stroke features, and treatment for 12 patients.

Patient No.	Sex	Age, years	History of stroke	Vascular risk factors	Stroke subtype	Site of Infarct	Arterial lesion	Major Neurological signs
1	F	66	None	HT, DM	LAA	R PICA	Occlusion at R VA origin	Vertigo, nystagmus R limb ataxia, headache
2	F	54	None	HT, DM, DL	LAA	R PICA	Occlusion at R VA origin	Vertigo
3	M	78	None	HT, DL	LAA	L SCA pons	Occlusion at L VA origin	Vertigo L limb ataxia
4	M	64	None	HT, DL smoking	LAA	Bil SCA Bil thalami Bil occipital lobes	Occlusion at L VA origin*	Semicoma Anisocoria Tetraparesis
5	F	46	None	None	LAA	R PICA R thalamus	Occlusion at R VA origin	Dizziness, nystagmus diplopia
6	F	66	None	HT	LAA	L PICA R occipital lobe	Occlusion at L VA origin	Dizziness L inferior quadrianopsia
7	M	63	None	HT, DM, DL	LAA	L PICA	Occlusion at L VA origin Severe stenosis at R VA origin	Vertigo L limb ataxia
8	M	65	None	HT, DL	LAA	Bil PICA R occipital lobe	Occlusion at Bil VA origin	Diplopia L superior quadrianopsia Headache
9	M	55	None	HT	LAA	L PICA Bil occipital lobes	Occlusion at L VA origin	Vertigo, nystagmus Headache
10	M	56	None	HT smoking	LAA	R PICA L SCA Bil occipital lobes	Occlusion at R VA origin Severe stenosis at L VA origin	Dizziness L limb ataxia, Headache
11	M	55	None	HT smoking	LAA	L SCA R occipital lobe	Occlusion at L VA origin	Dizziness, nystagmus, L limb ataxia R sensory disturbance Headache
12	M	65	None	HT	LAA	L PICA L SCA pons	Occlusion at L VA origin	Dizziness, nystagmus Dysarthria R hemiparesis R limb ataxia

Patient No.	Initial treatment	Secondary prevention	Recurrence of ischemic stroke (follow-up period)	NIHSS score on admission	NIHSS score at discharge	mRS score at discharge
1	UFH + aspirin	Clopidogrel	None (3 years)	2	2	4
2	UFH + warfarin	Warfarin	None (6 months)	0	0	2
3	UFH + aspirin	Aspirin	None (6 months)	2	0	3
4	IV-tPA → ARG + aspirin + warfarin	Aspirin + warfarin	None (5 years)	33	0	0
5	UFH + aspirin → UFH + warfarin	Warfarin	None (1 year)	0	0	0
6	UFH + warfarin	Warfarin	None (3 years)	1	1	0
7	UFH + aspirin → UFH + aspirin + warfarin	Aspirin + warfarin	None (2 years)	3	0	0
8	UFH + aspirin → UFH + aspirin + warfarin	Aspirin+ warfarin	None (8 months)	1	1	1
9	UFH + warfarin	Warfarin	None (6 months)	2	0	1
10	UFH + aspirin → cilostazol → UFH + aspirin + warfarin	Aspirin + warfarin	Recurrence during cilostazol (no recurrence over the following 3 years)	2	1	2
11	UFH + aspirin → UFH + warfarin	Warfarin	Recurrence during UFH + aspirin (no recurrence over the following 2 years)	3	2	2
12	ARG + UFH + aspirin	Aspirin	Recurrence during UFH + aspirin (no recurrence over the following 6 months)	15	13	4

HT = hypertension; DM = diabetes mellitus; DL = dyslipidemia; LAA = large artery atherosclerosis; SCA = superior cerebellar artery; VA = vertebral artery; PICA = posterior inferior cerebellar artery; IV-tPA = intravenous tissue plasminogen activator; UFH = unfractionated heparin; ARG = argatroban.
* Detected by conventional angiography after IV-tPA.

cause acute ischemic stroke, identified on brain MRA, basi-parallel anatomical scanning (BPAS)-MRI [7], duplex ultrasound, 3D-CTA, and/or conventional angiography; (3) presence of distal antegrade flow in the ipsilateral VA despite its origin occlusion identified on ultrasound, 3D-CTA, and/or conventional angiography; and (4) absence of other causes of ischemic stroke, including intracranial vertebrobasilar artery lesions or emboligenic diseases. The presence of pre-existing VA origin occlusion before stroke onset was not included in the diagnostic criteria because patients had not undergone examinations for VA just before stroke onset, and it is difficult