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A study on lesion pattern of bilateral cerebellar infarct

P.-Q. WANG[1], Z.-G. DING[1], G.-B. ZHANG[1], Y. WANG[2], J.-Z. LIU[2]

[1]Department of Neurology, Xiangyang Hospital Affiliated to to Hubei University of Medicine, Xiangyang, Hubei, China
[2]CT Room, Xiangyang Hospital Affiliated to Hubei University of Medicine, Xiangyang, Hubei, China

Puqing Wang and *Zhigang Ding* contributed equally to this work

Abstract. – OBJECTIVE: To explore the lesion patterns and stroke mechanism of the acute bilateral cerebellar infarct.

PATIENTS AND METHODS: Patients admitted to Xiangyang Hospital with acute cerebellar infarcts, confirmed by diffusion-weighted imaging (DWI), were investigated. Patients were divided into two groups by lesions: unilateral cerebellar infarct (UCI) and bilateral cerebellar infarct (BCI). The demographic features, involved territories and concomitant lesions outside the cerebellum (CLOC). The causes were analyzed.

RESULTS: Amongst the 115 patients hospitalized with posterior circulation cerebral infarct due to acute stroke, 56 patients had cerebellar infarct. There were 36 (64.3%) cases of unilateral cerebellar infarct and 20 (35.7%) cases of the BCI. The baseline information shows that stroke history (p = 0.002), fibrinogen (p = 0.036) and admission NIHSS score (M) (p = 0.001) for the BCI group are higher than the unilateral cerebellar infarct group. The incidence rate of cerebellar infarct in a posterior inferior cerebellar artery (PICA) blood supplying territory is the highest by divisions of vascular distribution. Unilateral cerebellar infarct occurs more often (p = 0.006); BCI is more common in PICA+SCA blood supplying territory (p = 0.004). The incidence rate of BCI merged with CLOC is much higher than the unilateral cerebellar infarct (p = 0.002). Merged infratentorial lesions are more common (p = 0.022) than BCI with atherosclerosis (p = 0.041). Offending artery diseases are mainly in the V4 segment of the vertebral artery, and in the severe stenosis or occlusion of V4 and BA junction.

CONCLUSIONS: BCI was frequently involved in the PICA + SCA territory. Our results support the fact that embolism resulted from large artery atherosclerosis is the important stroke mechanism in the BCI.

Key Words:
Cerebellar infarct, Artery atherosclerosis, Cerebral infarction mode.

Abbreviations

NIHSS = National Institute of Health Stroke Scale; PICA = posterior inferior cerebellar artery; SCA = Superior cerebellar artery; posterior inferior cerebellar artery; BA = basilar artery; TOAST = Trial of Org 10172 in Acute Stroke Treatment; VBA = vertebrobasilar artery.

Introduction

The acute cerebellar infarct manifests itself as vertigo and clinical ataxia[1-3]. Previous studies focus on the unilateral cerebellar infarct, as cases of bilateral cerebellar infarct (BCI) are relatively rare. Studies on the features of BCI are rare and have limitations[4-6]. In order to define the clinic features of the cerebellar infarct, we compare the demographic characteristics, vascular distribution, concomitant lesion outside the cerebellum and the pathogeny and mechanism of cerebellar infarct between unilateral and BCI.

Patients and Methods

Selecting and Grouping of Study Objects

Patients hospitalized for acute cerebral infarct or TIA episode and declared in the acute phase infarct within 48h after MRI DWI from January 2009 to January 2011 were selected to participate in this study. Cerebrovascular imaging, blood chemistry, electrocardiogram and cardiac ultrasound inspections were conducted on all patients. Those who were found to have no acute phase infarct by magnetic resonance imaging (DWI), whose inspections were incomplete or who had cerebral hemorrhage are excluded.

Patients were divided into unilateral and BCI group[7] combined with DWI. Unilateral cerebellar

Corresponding Author: Guibin Zhang, MD; e-mail: zgb1969@126.com

infarct (UCI) is the appearance of one or more acute phase concomitant lesions on one cerebellar hemisphere and BCI is the appearance of an acute phase of concomitant lesions on two cerebellar hemispheres.

Radiographic Evaluation

The MRI examination was conducted using the 1.5T NOVUS magnetic resonance image scanner produced by Siemens (Erlangen, Germany). The scanning parameters were as follows: collimation was 5 mm, interval was 1 mm, matrix was 256 × 256; the SE-EPI axis view was TR 3400 ms, TE 98 ms of DWI and the single-shot echo planar imaging to exert diffusion sensitive gradient pulse was applied in the X, Y, Z directions. The diffusion sensitive coefficient (the value of b) is 0 and 1000 s/mm^2 respectively.

Computed tomography angiography (CTA) examination was conducted as follows: Apply the CT with 64 rows produced by GE (Fairfield, CT, USA), head and neck joint CTA (the superior border of aortic arch reaches calvarium). Post-processing techniques include volume reappearance, VR; dimensions-multiplanar reconstruction, 3D-MPR; curved planar reconstruction, CPR and three dimensions-maximum intensity projection, 3D-MIP. Combine the original axial images to observe features such as stenosis degree and occlusion of the coronary artery and conduct quantitative measurement on the tracheal constriction[8-11].

Image Analysis

The MRI and CTA images were measured and evaluated by two associate chief physicians of imaging department respectively with the application of double-blind methods. It is subject to jointly confirmed diagnosis. The positioning of MRI-DWI lesions was referred to in the literature[12]. According to North American Symptomatic Carotid Endarterectomy Trial (NASCET)[13] count the artery stenosis rate [degree of stenosis] = (the diameter of the original tube-the left diameter of tube in the narrow area)/the diameter of the original tube × 100%]. If the stenosis rate is more than 50%, it is designated as collusive disease. The division of territories for blood supplying is referred to methods of Vuillier: (1) The PICA gives out two branches: inferior vermis of blood supplying and the down-back of cerebellar hemispheres; (2) Anterior inferior cerebellar artery(AICA) is a single feeding leaf, upper leaf and front of the inferior semilunar lobule and flocculus and cerebellar peduncle; (3) Superior cerebellar artery can be divided into medium-ramus external, the upper half of cerebellar hemispheres for blood supplying to part of the vermis and dental nucleus.

To analyze the concomitant lesions outside cerebrum, divide the lesions into three anatomic levels: (1) Subtentorial (brainstem); (2) Supratentorial (dorsal thalamus, blood supplying territory of posterior cerebral artery, infarct lesions of anterior circulation); (3) Both.

Types of Stoke Causes and Mechanisms

The causes of disease were diagnosed by clinical and auxiliary examination data with reference to the TOAST classification and reported literature[14]. The judging criteria for the causes of cerebral infarct were. As follows: (1) Large artery atherosclerosis (LAA) with a clinical manifestation of disordered brain function. The image showed that the corresponding atherosclerotic stenosis ≥50% or had occlusion, but excluded cardiac disease. (2) Small vessel disease (SVD) with a risk factor of cerebrovascular disease, the largest diameter of lesion is < 1.5 cm; there is no atherosclerotic stenosis, excluding the source of cardiac embolus; (3) Cardio embolism (CE), there is no atherosclerotic stenosis, and there is a possibility that there exists a potential source of cardiac embolus in the patient; (4) Other causes, including aortic dissection, Moya moya disease, arterial aneurysm, arteritis, ischemia and other specific vascular diseases; (5) Unclear causes, after comprehensive inspection, no possible causes are found; (6) Two or more than two causes (≥ 2E). If the patient has two or more than two cause of stroke, and after evaluation by two neurologists, no agreement can be reached about the cause, it can be divided into this type; (7) Dysplasia of vertebral artery

Statistical Analysis

Apply SPSS18.0 software (SPSS Inc., Chicago, IL, USA) to describe and analyze data. The statistical description of the two groups of measurement data: if it meets the conditions of normal distribution, use $\bar{x} \pm$ SD to represent; if it does not meet the conditions, use median (M, 25%-75% percentile) to represent. Hypothesis testing for two groups of measurement data: if it meets the conditions of normal distribution and homoscedasticity, use the *t*-test. If there is the heterogeneity of variance for the normal distrib-

ution, use the *t*-test. If it meets neither, use the Manne Whitney U test. Use frequency to represent the statistical description for the two groups of enumeration data. Hypothesis testing for two groups of enumeration data: if theoretical frequency ≥ 5, use Mann-Whitney U test; if 1 ≤ theoretical frequency < 5, use Mann-Whitney U correction test; if the theoretical frequency < 1, use Fisher's exact test. All statistics tests were two-sided. The *p*-value was < 0.05 showing that the difference was statistically significant.

Results

Comparison of Two Groups on Demographic Baseline Information (Table I)

Amongst the 115 hospitalized patients with posterior circulation cerebral infarct due to acute stoke, there were 56 patients (48.7%) that had cerebellar infarct with infarcts of other sites. There were 46 male patients, and the average age was 60. 83 ± 12. 34; and ten female patients with an average age of 65.2 ± 10.8. There were 36 (64.3%) cases of UCI, 20 (35.7%) cases of the BCI. The baseline information shows that stroke history (p = 0.002), fibrinogen (p = 0.036) and admission NIHSS score (M) (p = 0.001) for group of BCI are much higher that those for group of UCI.

Distribution Pattern of Lesions on Cerebellum and Concomitant Lesions Outside Cerebellum (Table II)

The incidence rate of cerebellar infarct in the PICA blood supplying territory is the highest by divisions of vascular distribution and UCI occurs more often (p = 0.006); BCI is more common in the PICA+SCA blood supplying territory (p = 0.012). There is no difference in other blood supplying territories between the two groups.

The incidence rate of the BCI merged with CLOC is much higher than UCI (p = 0.002). Also, the merged infratentorial lesions are more common (p = 0.022).

Analysis of the Causes of BCI

Bilateral cerebellar infarct with LAA is common (p = 0.041). Offending artery diseases are mainly in the V4 segment of the vertebral artery, and in the severe stenosis or occlusion of V4 and BA junction.

In LAA, the most common artery distribution areas of BCI are PICA + SCA (8/17), followed by the SCA (6/17), and in the UCI group. The most common artery distribution area is the PICA (10/21).

Figure 1 shows one case of a 66-year-old male patient. He was hospitalized due to sudden dizziness with limb weakness. After a physical examination, we found out that he got ataxia, with vi-

Table I. Demographic baseline information of cerebellar infarct.

	UCI (n = 36)	BCI (n = 20)	*p*
Age (x ± s)	61.28 ± 13.02	62.20 ± 10.54	0.79[a]
Male (n, %)	29 (80.6)	17 (85)	0.96[b]
Risk factors (n, %)			
Hypertension	25 (69.4)	14 (70.0)	0.97[c]
Diabetes mellitus	8 (22.2)	4 (20.0)	1.00[b]
Hyperlipidaemia	8 (22.2)	8 (40.0)	0.16[c]
Stroke history	10 (27.8)	14 (70.0)	0.00[c]
Smoking	12 (33.3)	7 (35.0)	0.90[c]
Drinking	10 (27.8	4 (20.0)	0.52[c]
Cardiac source of embolus	3 (8.3)	2 (10.0)	1.00[b]
Low density lipoprotein	2.78 ± 0.77	3.14 ± 0.63	0.15[a]
Fibrinogen	2.55 ± 0.62	3.29 ± 1.09	0.04[e]
Fasting blood glucose	5.52 (4.62-7.61)	6.92 (5.79-8.36)	0.14[d]
Admission NIHSS score (M) (M, 25%-75% percentile)	2 (2-2)	22 (3-34)	0.00[d]

Note: UCI means unilateral cerebellar infarct; BCI means bilateral cerebellar infarct. a means test of two independent samples; b means Pearson χ^2 correction test; c means Pearson^2-test; d means Mann-Whitney U test and e means *t*-test. Fasting blood glucose (normal reference value: 3.89-6.11 mmoL/L); Low-density lipoprotein (normal reference value: 1.2-3.6 mmoL/L); Fibrinogen (normal reference value: 2-4 g/L).

Table II. Comparison of the distribution of feeding arteries, combined lesions and the causes of infarcts between the two groups.

	UCI	BCI n = 36	*p* n = 20
Arterial territories			
PICA	19 (52.8)	3 (15.0)	0.01[c]
AICA	4 (11.1)	0 (0)	0.29[f]
SCA	6 (16.7)	8 (40.0)	0.05[c]
PICA+SCA	3 (8.3)	8 (40.0)	0.01[b]
PICA+AICA	1 (2.8)	0 (0)	1.00[f]
AICA+SCA	1 (2.8)	0 (0)	1.00[f]
PICA+AICA+SCA	2 (5.6)	1 (5.0)	1.00[b]
Concomitant lesions outside cerebellum			
Null	21 (58.3)	3 (15.0)	0.00[c]
Infratentorial (brainstem)	6 (16.7)	9 (45.0)	0.02[c]
Supratentorial	7 (19.4)	5 (25.0)	0.88[b]
Infratentorial + supratentorial	2 (5.6)	3 (15.0)	0.49[b]
Causes of stroke			
Disease of main artery	21 (58.3)	17 (85.0)	0.04[c]
Cardio embolus	4 (11.1)	2 (10.0)	1.00[b]
Disease of arterioles	4 (11.1)	0 (0)	0.29[f]
Other types of clear causes	1 (2.8)	0 (0)	1.00[f]
Types of unclear causes	2 (5.6)	1 (5.0)	1.00[b]
≥ two causes	1 (2.8)	0 (0)	1.00[f]
Dysplasia of VA	3 (8.3)	0 (0)	0.55[f]

Note: [b]Means Pearson Chi-square correction test; [c]Means Pearson Chi-square test; [f]Means Fisher's exact test.

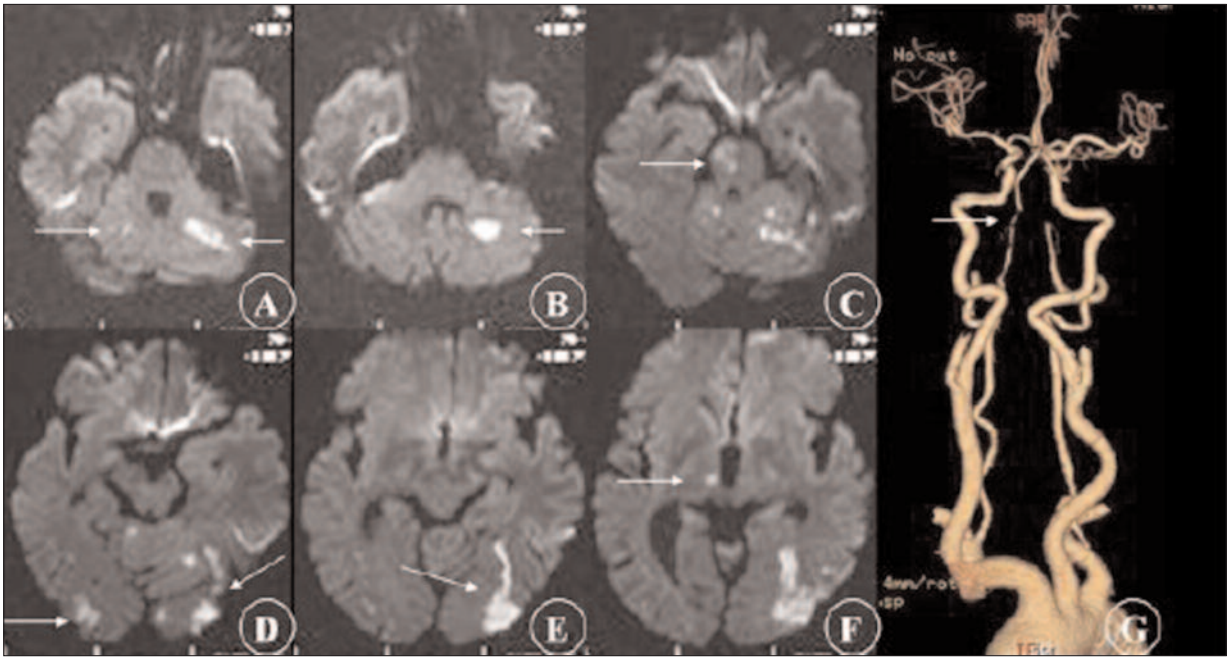

Figure 1. The arrows in ***A, B,*** shows the BCI; ***C, D, E, F,*** arrows are right pons, bilateral occipital lobes, and right thalamus infarct merged outside the cerebellum; G is CTA map, and the arrows refer to the V4 segment of the vertebral artery and the BA beginning part of narrow occlusion.

sual field defects and hemiplegia on left limbs. The MRI of his head showed multiple cerebral infarcts. The stenosis of the offending artery was shown in the CTA images.

Discussion

In order to analyze the characteristics of the cerebellar infarct, we divided the cerebellar infarct into the unilateral and BCI, and compared the clinical and radiographic features of the two groups. The results show that amongst the 115 patients hospitalized with posterior circulation cerebral infarct due to acute stroke, 48.7% had cerebellar infarct combined with other parts, in which BCI accounts for 35.7%. Bilateral cerebellar infarct accounts for 17.4% of all ischemic stroke, suggesting that the incidence rate of the BCI is relatively high. In past CT and conventional sequence MRI, the incidence rate of the BCI was no more than 12%[15]. Recent MR-DWI studies report that the incidence rate of BCI reaches 23-31% 1-2, 4, which is close to this study's results.

After comparing the baseline data of the two groups, we find that only stroke history, fibrinogen level, and admission NIHSS score for a group of BCI is much higher those for a group of the UCI. This indicates that patients with a history of stroke and increased fibrinogen are more likely to get BCI and patients with BCI becomes sicker on admission. The research results by Hong et al[7] of 50 patients with BCI show that patients with diabetes mellitus are more likely to have BCI. However, research by Lee et al[16] on the demographics of 591 patients with posterior circulation stroke displays that hypertension is the most common risk factor.

To analyze the correlation between cerebellar infarct and cerebellar infarct artery, we divided the artery distribution area by cerebellar infarct. We found that the incidence rate of the cerebellar infarct was the highest in the PICA supply area. The UCI occurred more often, and the BCI was more common in the PICA + SCA supply area, which is consistent with the literature[17-19].

We further observed that the BCI with large artery atherosclerotic stenosis is more common, followed by cardiogenic embolism. The CTA angiography were further analyzed and we found that the offending artery disease were mainly in the V4 segment of the vertebral artery, and at the junction of the V4 and BA with severe stenosis or occlusion, which are consistent with the findings of Hong et al[7]. We also found in the LAA, the most common arterial distribution of the BCI was PICA + SCA, followed by the SCA. The most common artery distribution of UCI group was PICA, which suggested that there may be differences in the pathophysiology of stroke. We analyzed and found the reasons may be that atherosclerosis probably causes unilateral infarct, but atherosclerosis of upper-level 20-22 causes the bilateral infarct.

We also analyzed the concomitant lesions distribution outside the cerebellum and found that the incidence rate of a combination of BCI and infarcts outside the cerebellum was significantly higher than the UCI group, and that merged infratentorial lesions were common. These results suggested that, in addition to causing site infarction, atherosclerosis may also be the emboli source of infarct on artery distal site. Cho et al[23-26] detected microembolic signals (MES) in the VBA circulating blood in 12.9% patients with acute posterior circulation infarct using TCD, and also found that frequency of MES occurred more often in patients with severe VBA stenosis. The most manifested as multiple infarct modes, further supporting the role arterial embolization played in the cerebellar endogenous infarction.

The concept of acute BCI also includes criticisms, for acute and sub-acute damage on DWI MRI both show high signal. The infarct can not be determined in this short period to have occurred simultaneously or successively, that is, that on DWI bilateral lesions is caused by a plurality of pieces decomposed by emboli[27-30] or multiple emboli remains unclear. Therefore, the DWI inspection of the selected cases in this study is completed within 48h so as to reduce the proportion of repeated embolization. Meanwhile, the study sample is composed of hospitalized patients, which may not be representative of all patients with atherosclerotic stenosis, needing further studies of large-scale samples.

Conclusions

These results suggest that the BCI is not uncommon. In the infarct mode, UCI mainly occurs in PICA supply area, and BCI is more common in the PICA + SCA supply area. Arterial embolization induced by artery atherosclerosis is one of the important reasons for stroke.

Understanding the patterns and causes of BCI-ion can play an important guiding role in helping clinicians select proper and timely treatment measures. In addition to anticoagulation or antiplatelet, and expanding to improve infusion therapy, intervention treatment is also needed against the main artery stenosis and plaque instability.

Conflict of Interest

The Authors declare that there are no conflicts of interest.

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