Correlation between the imaging manifestations of cerebellar infarction and the clinical characteristics of patients: a retrospective analysis

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Abstract

Background

The aim of this study was to analyze the correlation between the imaging manifestations of cerebellar infarction and the clinical characteristics of patients.

Methods

A total of 83 patients with acute cerebellar infarction were included in this analysis. The NIHSS score, TOAST classification, CT and MRI examinations and serum laboratory tests were performed. The statistics of the data were carried out through descriptive statistics, T test and analysis of variance.

Results

Hyperlipidemia (66.3%), hypertension (77.1%) and carotid atherosclerosis (97.6%) were the main underlying causes of cerebellar infarction. Ataxia (67.5%) and brainstem syndrome (60.2%) were main clinical manifestations. Patients with NIHSS score ≤ 5 accounted for the majority (87.9%) and SAO (43.3%) and LAA (50.6%) were the main TOAST classifications. The cerebellar infarction caused by obstruction of SCA accounted for the majority (69.9%) and there was no statistical difference in the detection ability of CTA and MRA ($P > 0.05$). The incidence of left hemisphere cerebellar infarction was the highest (56.7%) and MRI showed an absolute advantage in showing the area of cerebellar infarction compared with CT ($P < 0.001$). The serum concentrations of D-dimer, LDL, and triglycerides were higher in patients with high NIHSS scores than in those with low scores ($P < 0.05$), while the serum HDL levels were just the opposite ($P < 0.05$). The serum D-dimer concentrations in patients classified as CE by TOAST were higher than that in patients classified as LAA and SAO ($P < 0.05$), while the serum concentrations of LDL were just the opposite ($P < 0.05$). The lesion sizes of cerebellar infarction were negatively related to the patient's serum HDL concentrations ($P < 0.05$) but it positively correlated with the NIHSS scores ($P < 0.05$). The patients classified as LAA had larger lesion sizes of cerebellar infarction than those classified as SAO and CE ($P < 0.001$).

Conclusions

CTA and MRA are comparable in showing the stenosis and obstruction of the cerebellar artery. However, MRI has an absolute advantage in showing the area of cerebellar infarction. The serum concentrations of D-dimer, HDL, LDL, and triglycerides are correlated with the patient's NIHSS scores, TOAST classification and infarct sizes, which are helpful in evaluating the condition of the disease.

Background

Cerebellar infarction is mainly caused by thrombosis or thromboembolism that leads to the blockage of the corresponding blood supply arteries of the cerebellum, which leads to a reduction or interruption of cerebellar blood flow and causes a series of symptoms and signs. Common risk factors for cerebellar infarction are hypertension, heart disease, diabetes, and atrial fibrillation [1]. Once the cerebellar infarction is formed, it causes a series of cell biochemical process disturbances caused by cerebral ischemia, leading to cytotoxic edema with increased intracellular water and opening or destruction of the blood-brain barrier. Cerebral edema is the pathological basis of local brain swelling signs and imaging low-density signs caused by cerebral infarction in the super-acute phase [2, 3]. Early diagnosis of cerebellar infarction is critical to the prognosis of patients. A physical examination is usually performed on the patient to get a preliminary understanding of the disease. However, its diagnosis often relies on head imaging examinations. In addition, examinations such as electrocardiogram, vascular ultrasound, blood lipids and coagulation function can further help clarify the cause of cerebellar infarction [4].

Computed tomography (CT) scan of the head is one of the most commonly used examination methods [5]. In the early stage of cerebral infarction (within three hours of onset), CT examination of the head can reveal some slight changes such as middle cerebral artery high density sign, unclear boundary between cortical edge and lenticular nucleus, and disappearance of brain sulci [6]. Magnetic resonance imaging (MRI) can find small lesions that cannot be detected by CT. It is also of great significance when reviewing the effect of treatment. One hour after the onset of cerebral infarction, MRI showed swelling of the local gyrus and narrowing of the brain sulcus, followed by abnormal long T1 and long T2 signals. In addition, MRI diffusion and perfusion imaging can detect early cerebral infarction more timely [4, 7, 8]. Based on the imaging examination data and epidemiological statistics of patients with cerebellar infarction, this study conducted an in-depth analysis of the correlation between the risk factors, etiology, distribution of blood vessels involved, infarct lesion size and scope, and clinical manifestations of cerebellar infarction.

Methods

Ethics statement

The study was approved by the Research Ethics Committee of Tongji Hospital, Tongji University, Shanghai, China.

This was an observational study and did not involve any form of active intervention in the diagnosis and treatment process of patients, the rights of patients were fully guaranteed.

All patients were informed of the detailed study process and signed a written informed consent form.

All research procedures and methods were strictly implemented in accordance with relevant guidelines and regulations.

Patients

From January 2017 to June 2020, a total of 83 patients with acute cerebellar infarction (Tongji Hospital, Tongji University, Shanghai, China) were included in this analysis. The clinical data of the patients was extracted, including gender, age, personal history, past medical history, clinical symptoms, laboratory

tests, National institute of Health Stroke Scale (NIHSS), Trial of org10172 in Acute Stroke Treatment (TOAST), and imaging manifestations. The demographic baseline data of the included patients are detailed in Table 1.

Table 1
General clinical characteristics of the patients in this study (N = 83)

Items	Patients with cerebellar infarction	
Gender		
	Male	54(65.1%)
	Female	29(34.9%)
Ages		
	≤ 65	34(41%)
	> 65	49(69%)
Smoking		
	Yes	38(45.8%)
	No	45(54.2%)
Drinking alcohol		
	Yes	24(28.9%)
	No	59(71.1%)
Diabetes		
	Yes	29(34.9%)
	No	54(65.1%)
Coronary heart disease		
	Yes	10(12%)
	No	73(88%)
Hyperlipidemia		
	Yes	55(66.3%)
	No	28(33.7%)
Carotid atherosclerosis		
	Yes	81(97.6%)
	No	2(2.4%)
Atrial fibrillation		
	Yes	8(9.6%)

Items	Patients with cerebellar infarction	
Hypertension	No	75(90.4%)
	Yes	64(77.1%)
History of stroke	No	19(22.9%)
	Yes	65(21.7%)
	No	18(78.3%)

Inclusion and exclusion criteria for cerebellar infarction patients

Inclusion criteria are as follows: (1) diagnosed as cerebellar infarction; (2) either CT or MRI showed clear and measurable infarct lesion; (3) with etiological examination results, vascular imaging; and performing electrocardiogram and echocardiography to determine cardiogenic stroke; and (4) performed NIHSS score and had a clear TOAST classification. Exclusion criteria are as follows: (1) patients with malignant tumors; (2) intracranial space-occupying lesions before cerebellar infarction; (3) simultaneously with other infarcts, such as cerebral cortex and subcortex, brain stem, internal capsule, semi-oval center and other parts; and (4) with severe cognitive impairment.

Diagnosis of cerebellar infarction

The diagnosis of cerebellar infarction was based on the criteria of the American Heart Association/American Stroke Association Deleters Sections from 2018 Stroke Guidelines [9] and was considered with clinical data and auxiliary examination. Clinical history and symptoms included: (1) suffered from diseases prone to thrombosis, such as heart disease, atrial fibrillation, cardiomyopathy, myocardial infarction, etc.; (2) sudden onset, reaching the peak of disease in a short time; (3) had signs or symptoms of the nervous system; and (4) severe patients experienced coma, gastrointestinal bleeding, brain herniation, and even died soon. CT and MRI examinations: (1) showed changes in ischemic infarction or hemorrhagic infarction; (2) CT and MRI of the brain showed infarcts at the embolization site, the boundary was not clear, and there was a certain space-occupying effect. Auxiliary examinations: (1) increased brain pressure and corresponding changes in cerebrospinal fluid cytology; (2) the electrocardiogram showed abnormalities such as myocardial infarction or arrhythmia; and (3) carotid artery ultrasound examination evaluated the degree of lumen stenosis and atherosclerotic plaque and helped confirm carotid artery embolism.

NIHSS score

According to the National Institute of Health stroke scale (NIHSS) [10], the patients were scored for clinical prognosis assessment of patients. The evaluation includes consciousness, language, motor

function, sensory loss, visual field defect, eye movement, coordinated movement, neglect and articulation. The scoring area is 0–42 points. In this study, the highest score was 15 points. We divided the patients according to NIHSS score into three groups (< 1 , $> 1 \leq 5$, and > 5 points).

TOAST classification

Based on the clinical data, the patients were classified by the standard of Trial of org10172 in Acute Stroke Treatment (TOAST) [11]. The concepts used in this classification are mainly derived from the Harvard stroke registration classification and the National Institute of Neurological Diseases and Stroke Database of the United States [11–13]. According to the criteria, the patients included in this study were mainly divided into the following types: small-artery occlusion (SAO), large-artery atherosclerosis (LAA) and cardioembolism (CE).

CT and MRI examination

All patients underwent head CT scan. The cross-sectional scan took the canthus ear line as the baseline, and scanned 8 to 10 slices upward in sequence, with a thickness of 5 mm, and a coronal scanning was also performed. All patients underwent cranial magnetic resonance (MRI) examination, including horizontal FSE, T1 FLAIR, T2 FLAIR and DWI sequence scans and sagittal FSE sequence. In order to further clarify the cause and scope of the disease, all patients received carotid/vertebral artery computed tomography angiography (CTA)/magnetic resonance angiography (MRA) and whole cerebral angiography [1, 4, 7, 9].

Identification of the involved artery

With reference to the methods reported in the previous literature [14], the location of the infarct was confirmed by imaging examination. The blood supply arteries of the cerebellum were divided into three branches. Posterior inferior cerebellar artery (PICA): the blood supply area for the inferior vermis and the lower back of the cerebellar hemisphere. Anterior inferior cerebellar artery (AICA): the blood supply area for the midfoot, the cerebellar pompons, and the anterior lower part of the cerebellum. Superior cerebellar artery (SCA): the blood supply area for the upper half of the cerebellar hemisphere, the oral vermis and the dentate nucleus.

Blood laboratory tests

In the early morning on an empty stomach, 5 mL of venous blood was collected from the patient's elbow vein. The blood was kept in a centrifuge tube without anticoagulant. After the blood had clotted at room temperature, it was centrifuged for 8 minutes (at 3,000 rpm). The supernatant in the centrifuge tube was carefully aspirated, placed in a new EP tube, and stored in a refrigerator at -20°C for testing. The following biochemical indicators were detected on the automatic biochemical analyzer, which included D-dimer, blood sugar, high density lipoprotein (HDL), low-density lipoprotein (LDL), triglycerides and cholesterol.

Statistical processing of data

The data collected from this study included counting data and measurement data. The counting data was analyzed by the fourfold table and the R×C table, including chi-square test for comparing two rates or two constituent ratios, chi-square test for comparing multiple rates or multiple constituent ratios, correlation analysis for classification data and continuous adjusted chi-square test. The measurement data was analyzed by T test and analysis of variance (ANOVA). The T test was used for comparing of means between two groups and the analysis of variance was used for comparing the means of more than two groups. For the analysis of variance, each sample was required to be from a normal population and the overall variance of each group was homogeneous. The SPSS 22.0 software was hired to complete the data analysis of this study, a P value less than 0.05 was considered statistically significant.

Results

Demographic characteristics and underlying diseases of patients

Eighty-three patients with cerebellar infarction were included in this study (Table 1), of which 54 were men (65.1%) and 29 were women (34.9%). There were 38 patients (45.8%) with smoking history and 24 patients (28.9%) with drinking history. The underlying diseases of 83 patients were as follows: 29 with diabetes (34.9%), 10 with coronary heart disease (12%), 55 with hyperlipidemia (66.3%), 81 with carotid atherosclerosis (97.6%), 8 with atrial fibrillation (9.6%), 64 with hypertension (77.1%), and 65 (21.7%) with history of stroke. It is seemed that hyperlipidemia (66.3%), hypertension (77.1%) and carotid atherosclerosis (97.6%) were the main underlying causes of cerebellar infarction. Especially, there was the most obvious correlation between carotid atherosclerosis and cerebellar infarction.

Clinical manifestations of patients

In terms of clinical manifestations (Table 2), the incidence of ataxia was 67.5% (56/83), the incidence of brainstem syndrome was 60.2% (50/83), and the incidence of vestibular syndrome was 10.9% (9/83%). The patient's NIHSS scores were divided into three sections (Table 2) (≤ 1 , $> 1 \leq 5$, and > 5 points), patients of ≤ 1 accounted for 54.2% (45/83%), those of $> 1 \leq 5$ points accounted for 33.7% (28/83%), those of > 5 points accounted for 12.1% (10/83%). In terms of the TOAST classification (Table 2), patients classified as SAO accounted for 43.4% (36/83), those as LAA accounted for 50.6% (42/83), and those as CE accounted for 6% (5/83).

Table 2
Clinical symptoms and NIHSS scores of patients (N = 83)

Items	Patients with cerebellar infarction	
Ataxia		
	Yes	56(67.5%)
	No	27(32.5%)
Brainstem syndrome		
	Yes	50(60.2%)
	No	33(39.8%)
Vestibular syndrome		
	Yes	9(10.8%)
	No	74(89.2%)
NIHSS score		
	≤ 1	45(54.2%)
	> 1 ≤ 5	28(33.7%)
	> 5	10(12.1%)
TOAST classification		
	Small-artery occlusion	36(43.4%)
	Large-artery atherosclerosis	42(50.6%)
	Cardioembolism	5(6%)
NIHSS, National institute of Health Stroke Scale. TOAST, Trial of org10172 in Acute Stroke Treatment.		

Comparison of the efficacy of CTA and MRA in displaying the arterial blood vessels involved in cerebellar infarction

Judging from the CTA (Table 3), the conditions of cerebellar blood supply artery involved were as follows: SCA (58/83, 69.9%), PICA (19/83, 22.9%), AICA (16/83, 19.3%), SCA + PICA (12/83, 14.5%), SCA + AICA (12/83, 14.5%), and PICA + AICA (1/83, 1.2%). Judging from the MRA (Table 3), the conditions of cerebellar blood supply artery involved were as follows: SCA (47/83, 55.6%), PICA (22/83, 22.5%), AICA (19/83, 22.9%), SCA + PICA (14/83, 16.9%), SCA + AICA (16/83, 19.3%), PICA + AICA (5/83, 6%), and SCA + PICA + AICA (3/83, 3.6%). Whether it was CTA or MRA, the incidence of cerebellar infarction by implicating in SCA was the highest (more than 55%). In terms of the detection rate, there was no statistical difference between CTA and MRA ($P < 0.05$) (Figs. 1A - I).

Table 3
Comparison of CTA and MRA in the involvement of cerebellar artery (N = 83)

Incidence of involvement of different cerebellar artery									
Items	CTA				MRA				P value
	Yes		No		Yes		No		
	N	%	N	%	N	%	N	%	
Superior cerebellar artery (SCA)	58	69.9	25	30.1	47	55.6	36	43.4	0.097
Posterior inferior cerebellar artery (PICA)	19	22.9	64	77.1	22	26.5	61	73.5	0.589
Anterior inferior cerebellar artery (AICA)	16	19.3	67	80.7	19	22.9	64	77.1	0.568
SCA + PICA	12	14.5	71	85.5	14	16.9	69	83.1	0.669
SCA + AICA	12	14.5	70	85.5	16	19.3	67	80.7	0.427
PICA + AICA	1	1.2	82	98.8	5	6	78	94	0.210
SCA + PICA + AICA	0	0	0	0	3	3.6	80	96.4	-
Total	118	23.7	379	76.3	126	21.7	455	78.3	0.421
N, number of cases; CTA, computed tomography angiography; MRA, magnetic resonance angiography; SCA, superior cerebellar artery; PICA, posterior inferior cerebellar artery; AICA, anterior inferior cerebellar artery.									

Comparison of CT and MRI in identifying the size and location of of cerebellar infarction lesion

Compared with CT (37/83, 44.6%), MRI (83/83, 100%) showed an absolute advantage in detecting cerebellar infarction lesions ($P < 0.0001$). CT and MRI examinations revealed that the locations of cerebellar infarction were as follows: left hemisphere (56.7% and 40.9%), right hemisphere (16.2% and 19.3%), vermis (5.4% and 2.4%), and multiple sites (21.6% and 38.6%). The incidence of left hemisphere cerebellar infarction (> 40%) accounted for the majority. CT and MRI examinations showed that the scopes of cerebellar infarction were as follows: unilateral cerebellar infarction (83.8% and 79.5%) and bilateral infarction (16.2% and 20.5%). The unilateral infarction accounted for the majority (> 79%) (Figs. 2A - I). Comparing CT or MRI to measure the maximum diameter of infarct lesions, we found that there was no statistical difference between the number of cases with infarct diameter < 20 mm and ≥ 20 mm ($P = 0.337$). The statistical data are shown in Table 4.

Table 4
Detection efficiency of cerebellar infarct area for CT and MRI (N = 83)

Items	Performance	CT examination		MRI examination		Statistical value	P value
		N	%	N	%		
Display of infarct							
	Yes	37	44.6	83	100	63.63	< 0.0001
	No	46	55.4	0	0		
Location of infarct							
	Left hemisphere	21	56.7	34	40.9	5.531	0.137
	Right hemisphere	6	16.3	16	19.3		
	Vermis	2	5.4	1	1.2		
	Multiple sites	8	21.6	32	38.6		
	Not showing	46 [□]	-	0	0		
Scope of infarction							
	Unilateral	31	83.8	66	79.5	0.301	0.586
	Bilateral	6	16.2	17	20.5		
	Not show	46 [□]	-	0	0		
Maximum diameter of infarct size							
	< 20 mm	20	54.1	37	44.6	0.921	0.337
	≥ 20 mm	17	45.9	46	55.4		
	Not show	46 [□]	-	0	0		
CT, computed tomography; MRI, magnetic resonance imaging; [□] , In CT examination, 46 patients could not see measurable infarct lesions.							

Correlations between the changes of D-dimer and blood lipids and NIHSS scores in patients with cerebellar infarction

The patients with NIHSS scores of > 5 (1.38 ± 0.76 mg/L) had higher serum D-dimer concentrations than patients with ≤ 1 (0.51 ± 0.26 mg/L) and $> 1 \leq 5$ scores (0.55 ± 0.23 mg/L) ($P = 0.003$) (Fig. 3A). The

serum HDL concentrations in patients with NIHSS scores of ≤ 1 (1.28 ± 0.38 mmol/L) were higher than those with > 5 (1.04 ± 0.23 mmol/L) and $> 1 \leq 5$ scores (1.08 ± 0.20 mmol/L) ($P = 0.046$) (Fig. 3B). The serum LDL concentrations were higher in patients with > 5 scores (3.71 ± 1.10 mmol/L) than those with $> 1 \leq 5$ (3.48 ± 0.98 mmol/L) and ≤ 1 scores (2.61 ± 1.17 mmol/L) ($P = 0.024$) (Fig. 3C). The serum triglycerides concentrations in patients with NIHSS scores of > 5 (1.73 ± 0.93 mmol/L) and $> 1 \leq 5$ (1.85 ± 0.73 mmol/L) were higher than those with NIHSS scores of ≤ 1 (1.05 ± 0.57 mmol/L) ($P = 0.041$) (Fig. 3D). The statistical data are shown in Table 5.

Table 5

The relationship between the changes of D-dimer and blood lipids and NIHSS score in patients with cerebellar infarction (N = 83)

Items	N	NIHSS scores			F value	P value
		≤ 1	$> 1 \leq 5$	> 5		
D-dimer (mg/L)	83	0.51 ± 0.26	0.55 ± 0.23	1.38 ± 0.76	5.246	0.003
Blood sugar (mmol/L)	83	6.59 ± 2.02	7.28 ± 3.29	7.58 ± 2.24	0.911	0.407
HDL (mmol/L)	83	1.28 ± 0.38	1.08 ± 0.20	1.04 ± 0.23	3.219	0.046
LDL (mmol/L)	83	2.61 ± 1.17	3.48 ± 0.98	3.71 ± 1.10	3.909	0.024
Triglycerides (mmol/L)	83	1.05 ± 0.57	1.85 ± 0.73	1.73 ± 0.93	2.938	0.041
Cholesterol (mmol/L)	83	4.17 ± 0.50	5.07 ± 1.38	5.29 ± 1.52	2.160	0.122
N, number, NIHSS, National institute of Health Stroke Scale; HDL, high density lipoprotein; LDL, low density lipoprotein.						

Correlations between the changes of D-dimer and blood lipids and TOAST classification in patients with cerebellar infarction

The concentrations of serum D-dimer in patients classified as CE (1.12 ± 0.56 mg/L) were higher than that in patients classified as LAA (0.58 ± 0.25 mg/L) and SAO (0.56 ± 0.22 mg/L) ($P = 0.032$) (Fig. 3E). The patients classified as LAA (3.58 ± 1.33 mmol/L) and SAO (3.76 ± 1.48 mmol/L) had higher serum concentrations of LDL than those classified as CE (2.85 ± 1.22 mmol/L) ($P = 0.042$) (Fig. 3F). However, the concentrations of blood glucose, HDL, triglycerides, and cholesterol did not show statistical significance among the three different types of patients ($P > 0.05$). The statistical data are shown in Table 6.

Table 6

The relationship between the changes of D-dimer and blood lipids and TOAST typing in patients with cerebellar infarction (N = 83)

Items	N	TOAST typing			F value	P value
		LAA	SAO	CE		
D-dimer (mg/L)	83	0.58 ± 0.25	0.56 ± 0.22	1.12 ± 0.56	3.668	0.032
Blood sugar (mmol/L)	83	7.36 ± 2.23	6.82 ± 2.87	7.18 ± 2.03	0.785	0.398
HDL (mmol/L)	83	1.08 ± 0.23	1.05 ± 0.26	1.31 ± 0.35	3.094	0.051
LDL (mmol/L)	83	3.58 ± 1.33	3.76 ± 1.48	2.85 ± 1.22	3.984	0.042
Triglycerides (mmol/L)	83	1.39 ± 0.86	1.65 ± 1.19	1.49 ± 1.17	0.525	0.665
Cholesterol (mmol/L)	83	4.91 ± 0.86	4.78 ± 1.49	5.01 ± 1.62	2.981	0.203
N, number, TOAST, Trial of org10172 in Acute Stroke Treatment; HDL, high density lipoprotein; LDL, low density lipoprotein; LAA, large artery atherosclerosis; CE, cardioembolism; SAO, small-artery occlusion.						

Correlations between the changes of D-dimer and blood lipids and the infarct sizes of cerebellar infarction (CT and MRI)

From the perspective of CT measurement, the serum concentrations of HDL in patients with the infarct diameter of < 20mm (1.27 ± 0.26 mmol/L) were higher than that in patients with ≥ 20 mm (1.30 ± 0.28 mmol/L) ($P = 0.019$) (Fig. 4A). In terms of MRI, the serum concentrations of HDL in patients with the infarct diameter of < 20mm (1.05 ± 0.25 mmol/L) were higher than that in patients with ≥ 20 mm (1.02 ± 0.36 mmol/L) ($P = 0.013$) (Fig. 4B). However, the concentrations of D-dimer, blood glucose, LDL, triglycerides, and cholesterol did not show statistical significance on the different infarct size in patients with cerebellar infarction ($P > 0.05$). The statistical data are shown in detail in Table 7.

Table 7

The relationship between the changes of D-dimer and blood lipids and the maximum diameter of infarct size in patients with cerebellar infarction

Items	The diameter of infarct size (CT)		T value	P value
	< 20 mm	≥ 20 mm		
D-dimer (mg/L)	0.48 ± 0.35	0.58 ± 0.56	0.392	0.535
Blood sugar (mmol/L)	7.22 ± 2.36	7.66 ± 2.49	0.279	0.601
HDL (mmol/L)	1.27 ± 0.26	1.05 ± 0.25	6.085	0.019
LDL (mmol/L)	4.01 ± 1.25	3.97 ± 1.36	0.003	0.957
Triglycerides (mmol/L)	1.64 ± 0.77	1.80 ± 0.82	0.373	0.545
Cholesterol (mmol/L)	5.71 ± 1.60	5.58 ± 1.68	0.058	0.811
Items	The diameter of infarct size (MRI)		T value	P value
	< 20 mm	≥ 20 mm		
D-dimer (mg/L)	0.62 ± 0.25	0.51 ± 0.35	0.635	0.428
Blood sugar (mmol/L)	6.78 ± 2.42	7.22 ± 2.71	0.001	0.982
HDL (mmol/L)	1.30 ± 0.28	1.02 ± 0.36	6.397	0.013
LDL (mmol/L)	3.48 ± 1.15	3.44 ± 1.09	0.031	0.862
Triglycerides (mmol/L)	1.68 ± 0.96	1.75 ± 1.12	0.001	0.987
Cholesterol (mmol/L)	5.11 ± 1.51	5.04 ± 1.49	0.050	0.824
N, number; CT, computed tomography; MRI, magnetic resonance imaging; HDL, high density lipoprotein; LDL, low density lipoprotein.				

Correlations between the NIHSS scores and TOAST typing and the diameter of infarct sizes in patients with cerebellar infarction

From the perspective of CT measurement, the infarct sizes of cerebellar infarction in patients with NIHSS scores of > 1 (31.86 ± 16.08 mm) were larger than that in those with NIHSS scores of ≤ 1 (18.98 ± 10.81 mm) ($P = 0.028$) (Fig. 4C). The patients classified as LAA (33.26 ± 13.68 mm) had larger infarct sizes of cerebellar infarction than those classified as SAO (9.31 ± 4.23 mm) and CE (7.16 ± 3.88 mm) ($P < 0.0001$) (Fig. 4D). From the perspective of MRI measurement, the infarct sizes of cerebellar infarction in patients with NIHSS scores of > 1 (33.98 ± 16.63 mm) were larger than that in those with NIHSS scores of ≤ 1 (21.87 ± 15.56 mm) ($P = 0.035$) (Fig. 4E). The infarct sizes in patients classified as LAA (40.05 ± 17.23 mm) were larger than that in patients classified as SAO (13.69 ± 5.68 mm) and CE (15.96 ± 8.65 mm) ($P < 0.0001$) (Fig. 4F). The statistical data are shown in Table 8.

Table 8

The relationships between the NIHSS scores and TOAST typing and the diameter of infarct area in patients with cerebellar infarction

Items	Groups	The diameter of infarct area (CT scan) (mm)	F value	P value
NIHSS scores	≤ 1 (18 cases)	18.98 ± 10.81	4.456	0.028
	> 1 ≤ 5 (14 cases)	31.86 ± 16.08		
	> 5 (5 cases) [□]			
TOAST typing	LAA	33.26 ± 13.68	20.297	< 0.0001
	SAO	9.31 ± 4.23		
	CE	7.16 ± 3.88		
Items	Groups	The diameter of infarct area (MRI) (mm)	F value	P value
NIHSS scores	≤ 1 (45 cases)	21.87 ± 15.56	5.752	0.035
	> 1 ≤ 5 (29 cases)	33.98 ± 16.63		
	> 5 (9 cases) [□]			
TOAST typing	LAA	40.05 ± 17.23	83.659	< 0.0001
	SAO	13.69 ± 5.68		
	CE	15.96 ± 8.65		
[□] , because the number of cases in this group was too small, the two groups with similar clinical significance were combined for statistical analysis. N, number; TOAST, Trial of org10172 in Acute Stroke Treatment; NIHSS, National institute of Health Stroke Scale; MRI, magnetic resonance imaging; LAA, large artery atherosclerosis; CE, cardioembolism; SAO, small-artery occlusion.				

Discussion

Cerebellar ischemia and hypoxia cause ischemic necrosis of local tissues, called cerebellar infarction. It is mainly caused by atherosclerosis, thrombosis or embolism in the posterior circulation (also known as the vertebrobasilar system) that supplies blood to the brain [15]. High-risk factors mainly include high blood pressure, diabetes, dyslipidemia, smoking, lack of exercise, abdominal obesity, heart disease, and poor eating habits [2, 11, 12, 14, 15]. Because of the extensive anastomosis between the cerebellar blood supply arteries, the incidence of cerebellar infarction is low, accounting for about 1.5–3% of ischemic stroke [16]. However, larger cerebellar infarcts are likely to cause hydrocephalus and lower brainstem compression, with severe clinical symptoms and higher mortality. So improving the understanding of the

clinical characteristics of cerebellar infarction is essential for early diagnosis, treatment, prevention and assessment of prognosis.

Our research showed that the incidence of cerebellar infarction in men and women was close to 2:1 and there were more patients older than 65 years old. However, smoking and drinking did not seem to be associated with cerebellar infarction. We found that the included patients with cerebellar infarction had multiple underlying diseases, including diabetes, coronary heart disease, hyperlipidemia, carotid atherosclerosis, atrial fibrillation and hypertension. It is showed that hyperlipidemia, hypertension and carotid atherosclerosis were the main underlying causes. Especially, there was the most obvious correlation between carotid atherosclerosis and cerebellar infarction. It is generally believed that the risk factors of cerebellar infarction are consistent with the risk factors of cerebrovascular diseases, and the latter significantly increases the incidence of cerebellar infarction [11, 12, 17]. In terms of clinical manifestations, the incidence of ataxia and brainstem syndrome was relatively high, while the incidence of vestibular syndrome was relatively low. Common clinical manifestations of cerebellar infarction include dizziness/vertigo, nystagmus, limb and gait ataxia, cerebellar dysarthria, etc. Previous studies have found that infarcts in the blood supply area of PICA and AICA are more likely to cause dizziness in patients than infarcts in the SCA area. This is because the structure of the vestibular cerebellar nodules and floccules are mainly supplied by PICA and AICA [18]. The prominent clinical manifestations of SCA are limb and gait ataxia and dysarthria [15]. Due to the limited sample size in this study, it is difficult to clarify the differences in clinical manifestations caused by infarctions in different vascular regions, which needs to be further explored in future. The NIHSS scores of the patients included in this study were mostly less than one point, and the TOAST classification indicated that the SAO and LAA were mostly. Studies have shown that most patients with cerebellar infarction have lower NIHSS scores, and the TOAST etiological classification of cerebellar infarction has a certain relationship with the involved arteries [1, 2, 7, 15, 18].

We analyzed the vascular imaging results of patients with cerebellar infarction and found that the incidence of cerebellar infarction caused by SCA involvement was the highest regardless of whether it was performed by CTA or MRA. In addition to SCA, the probability of PICA involvement is also significantly greater than that of AICA. The involvement of different cerebellar blood vessels will produce different clinical symptoms, which is helpful for the diagnosis and prognosis of the disease. Obstruction of the PICA can lead to a headache and AICA territory infarction often leads to dysmetria, unilateral hearing loss and ipsilateral facial paralysis, and obstruction of the SCA tends to produce more ataxia, dysarthria and nystagmus [15]. By calculating the detection rate, we found that there was no statistical difference in the detection rate of different blood vessels between CTA and MRA. This indicates that if MRA cannot be performed for the patient, enhanced CT angiography and perfusion imaging will provide a good reference for the display of cerebellar infarcted blood vessels [7, 15, 16]. In this study, both CT and MRI examinations showed that the incidence of unilateral cerebellar infarction accounted for more than 80%; and the left hemisphere cerebellar infarction was the main one. This was a sure finding that MRI had an absolute advantage in detecting cerebellar infarction compared with CT. Brain CT scan is the most commonly used examination for cerebral infarction, but it is not sensitive to ultra-early ischemic

lesions and small infarcts, especially brain stem. However, MRI can detect cerebral infarction early and has a high sensitivity [15, 16]. One hour after the onset, local gyrus swelling, narrowing of the brain sulcus and abnormal long T1 and long T2 signals can be seen. In particular, MRI is sensitive to lacunar infarcts in the thalamus, cerebellum, and brainstem [7].

Our study found that patients with high NIHSS scores had higher serum concentrations of D-dimer, LDL, and triglycerides than those with low scores, while HDL levels were just the opposite. The increase of D-dimer suggests that it is related to thrombotic diseases caused, and it illustrates the enhancement of fibrinolytic activity. Existing studies generally support that the increase of D-dimer is a risk factor for cerebral infarction, and it has predictive significance for the course of the disease [19–21]. Studies have shown that cholesterol carried by LDL is prone to accumulate on the walls of blood vessels and arteries, forming unstable soft plaques, which blocks the cerebral artery and causes a cerebral infarction [22–24]. The main harm of triglycerides to the human body is mainly to cause atherosclerosis, block blood vessels and form thrombus. It is closely related to the occurrence of cerebral infarction [23, 24]. Our research suggests that the D-dimer, LDL, HDL, and triglycerides can be used as indicators to predict the severity of the NIHSS scores in patients with cerebellar infarction [25]. The TOAST subtype classification standard focuses on the etiological classification, which has good credibility in clinical application [26]. We found that the serum D-dimer concentrations in patients classified as CE was higher than that in patients classified as LAA and SAO. The cardiogenic embolus is detached and embolized in the corresponding cerebral artery with the blood flow, causing ischemic cerebral infarction in the corresponding blood supply area. Among them, atrial fibrillation-related infarctions account for more than 79% of all cardiogenic infarctions [11, 26]. Previous studies have also shown that D-dimer is intrinsically related to cardiogenic cerebral infarction [27, 28]. Our research suggests that patients with cerebellar infarction with elevated D dimer concentrations should focus on the investigation of the possibility of cardiogenic embolism. Our study showed that patients classified as LAA and SAO had higher serum concentrations of LDL than those classified as CE, suggesting that LDL plays a prominent role in vascular embolism caused by atherosclerosis. LDL receptors are distributed on the cell membrane surface of various tissues throughout the body, such as arterial wall cells, and abnormally increased levels can cause atherosclerosis [29].

In the study, all patients with cerebellar infarction underwent cranial CT and MRI. We found that both CT and MRI showed that the serum HDL concentrations of patients with a cerebellar infarct area of less than 20mm in diameter was significantly higher than that of patients with an infarct area greater than or equal to 20mm in diameter. It is known that the high concentrations of HDL has a negative correlation with the occurrence of cardio-cerebral vascular embolism and infarction [30, 31]. Our study suggests that HDL is a protective factor against cerebellar infarction and its high levels can reduce the occurrence of cerebellar infarction, which can also be understood that patients with higher HDL indicate a lower degree of cerebellar infarction and a better prognosis. Studies showed that HDL can bind to lipids attached to the walls of blood vessels to dissolve them. The dissolved products are transformed into excretable metabolites through the liver's biochemical reaction, thereby reducing the occurrence of vascular sclerosis and embolism [32, 33]. Regardless of the result of CT or MRI, we found that the diameter of the cerebellar infarct area of patients with NIHSS scores more than one point was significantly larger than that of

patients with NIHSS scores less or equal to 1 point. The tool of NIHSS scores is mainly used to evaluate the neurological status of stroke patients, and has important significance in individualized treatment, prognosis prediction and correct health education. The higher the scores are, the more severe the nerves damage [10, 25]. Our research suggests that the NIHSS scores are positively correlated with the sizes of the cerebellar infarction area, which can indirectly reflect the severity of the infarction. In addition, we found that the cerebellar infarct area of patients classified as LAA by TOAST classification was significantly larger than that of patients classified as SAO and CE, and the measurement results from CT and MRI showed the same trend. The LAA patients often are found with significant stenosis in the carotid artery, anterior cerebral artery, middle cerebral artery, posterior cerebral artery, and vertebrobasilar artery during retrospective examination. Cerebral infarction caused by LAA often manifests as large area, rapid onset and high mortality rate [34, 35].

Conclusions

The findings of this study are summarized as follows. Hyperlipidemia, hypertension and carotid atherosclerosis are the main causes of cerebellar infarction. Ataxia and brainstem syndrome are the main clinical manifestations. SAO and LAA patients account for the majority. Cerebellar infarction caused by obstruction of SCA accounts for the majority, but there is no statistical difference in the detection ability of CTA and MRA. Unilateral infarction is the most and the incidence of left hemisphere cerebellar infarction is the highest. However, compared with CT, MRI has an absolute advantage in showing the area of cerebellar infarction. The serum concentrations of D-dimer, LDL, and triglycerides are higher in patients with high NIHSS scores than in those with low scores, while the serum HDL levels are just the opposite. The serum D-dimer concentrations in patients classified as CE by TOAST are higher than that in patients classified as LAA and SAO, while the serum concentrations of LDL are just the opposite. The lesion sizes of cerebellar infarction are negatively correlated to the patient's serum HDL concentrations but it positively correlated with the NIHSS scores. The patients classified as LAA had larger lesion sizes of cerebellar infarction than those classified as SAO and CE.

Abbreviations

AICA, anterior inferior cerebellar artery; CE, cardioembolism; CT, computed tomography; CTA, computed tomography angiography; HDL, high density lipoprotein; LAA, large artery atherosclerosis; LDL, low density lipoprotein; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; NIHSS, National institute of Health Stroke Scale; PICA, posterior inferior cerebellar artery; SAO, small-artery occlusion; SCA, superior cerebellar artery; TOAST, Trial of org10172 in Acute Stroke Treatment.

Declarations

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Contributions

PJ W designed the project. L W, KW Z, JQ M and JN participated in the whole study, extracted the data, analyzed the data and wrote the manuscript. All authors read and approved the final manuscript.

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Ethics declarations

Ethics approval and consent to participate

The study was approved by the Research Ethics Committee of Tongji Hospital, Tongji University, Shanghai, China. This was an observational study and did not involve any form of active intervention in the diagnosis and treatment process of patients, the rights of patients were fully guaranteed. All patients were informed of the detailed study process and signed a written informed consent form. All research procedures and methods were strictly implemented in accordance with relevant guidelines and regulations.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interest.

Availability of data and materials

The datasets supporting the conclusions of this article are included within the article. The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable

request.

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Figures

Figure 1

Cerebellar infarction caused by involvement of the vertebrobasilar artery system. (A) CTA showed severe stenosis of the initial segment of the basilar artery and the right vertebral artery (marked with red arrows); (B) CTA oblique presentation of severe stenosis of the initial segment of the basilar artery and the right vertebral artery (marked with red arrows); (C) CTA lateral presentation of severe stenosis of the initial segment of the basilar artery (marked with a red arrow); (D) MRA showed severe stenosis in the middle of

the basilar artery (marked with a red arrow); (E) MRI showed a large infarct area on the right side of the cerebellum (marked with a red arrow); (F) CT showed a large infarct area on the right side of the cerebellum (marked with a red arrow); (G) MRI showed a large infarct area on the the left side of the cerebellum; (H) MRI showed a small infarct area scattered on the left side of the cerebellum and vermis (marked with a red arrow); (I) CT showed a same infarct area on the left side of the cerebellum (marked with a red arrow). CTA, computed tomography angiography; MRA, magnetic resonance angiography; CT, computed tomography; MRI, magnetic resonance imaging.

Figure 2

Cerebellar infarction caused by the involvement of the cerebellar artery. (A) MRA found that the left anterior inferior cerebellar artery was not displayed, suggesting that the left anterior inferior cerebellar artery was infarcted (marked with a red arrow). (B) MRI showed a large area of infarction in the left cerebellum (marked with a red arrow). (C) CT showed a large area of infarction in the same area left cerebellum (marked with a red arrow). (D) CTA found that the left anterior inferior cerebellar artery was not displayed, suggesting that the left anterior inferior cerebellar artery was infarcted (marked with a red arrow). (E) CT showed a large area of infarction in the left cerebellum (marked with a red arrow). (F) MRI showed a large area of infarction in the same area left cerebellum (marked with a red arrow). (G) MRA found that the left posterior inferior cerebellar artery was not displayed, suggesting that the left posterior inferior cerebellar artery was infarcted (marked with a red arrow). (H) MRI showed a small area of infarction in the left cerebellum (marked with a red arrow). (I) CT showed a small area of infarction in the same area left cerebellum (marked with a red arrow). CTA, computed tomography angiography; MRA, magnetic resonance angiography; CT, computed tomography; MRI, magnetic resonance imaging.

Figure 3

Relationships between the changes of D-dimer and blood lipids and NIHSS scores and TOAST typing in patients with cerebellar infarction. (A) The patients with NIHSS scores of >5 had higher serum D-dimer concentrations than patients with ≤ 1 and $>1 \leq 5$ scores ($P=0.003$). (B) The serum HDL concentrations in patients with NIHSS scores of ≤ 1 were higher than those with >5 and $>1 \leq 5$ scores ($P=0.046$). (C) The serum LDL concentrations were higher in patients with >5 scores than those with $>1 \leq 5$ and ≤ 1 scores ($P=0.024$). (D) The serum triglycerides concentrations in patients with >5 and $>1 \leq 5$ NIHSS scores were higher than those with ≤ 1 NIHSS scores ($P=0.041$). (E) The concentrations of serum D-dimer in patients

classified as CE was higher than that in patients classified as LAA and SAO ($P=0.032$). (F) The patients classified as LAA and SAO had higher serum concentrations of LDL than those classified as CE ($P=0.042$). NIHSS, National institute of Health Stroke Scale; TOAST, Trial of org10172 in Acute Stroke Treatment; HDL, high density lipoprotein; LDL, low density lipoprotein; LAA, large artery atherosclerosis; CE, cardioembolism; SAO, small-artery occlusion.

Figure 4

Relationships between the infarct size of cerebellar infarction and blood indicators, NIHSS scores and TOAST typing. (A) CT showed that the serum concentrations of HDL in patients with the infarct maximum diameter of < 20mm was higher than that in patients with ≥ 20 mm ($P=0.019$). (B) MRI showed that the serum concentrations of HDL in patients with the infarct maximum diameter of < 20mm was higher than

that in patients with $\geq 20\text{mm}$ ($P=0.013$). (C) CT showed that the infarct sizes of cerebellar infarction in patients with NIHSS scores of >1 were larger than that in those with NIHSS scores ≤ 1 ($P=0.028$). (D) CT showed that the patients classified as LAA showed larger infarct sizes of cerebellar infarction than those classified as SAO and CE ($P<0.0001$). (E) MRI showed that the infarct sizes of cerebellar infarction in patients with NIHSS scores of >1 were larger than that in those with NIHSS scores ≤ 1 ($P=0.035$). (F) MRI showed that the patients classified as LAA had larger infarct sizes of cerebellar infarction than those classified as SAO and CE ($P<0.001$). NIHSS, National institute of Health Stroke Scale; TOAST, Trial of org10172 in Acute Stroke Treatment; HDL, high density lipoprotein; LDL, low density lipoprotein; LAA, large artery atherosclerosis; CE, cardioembolism; SAO, small-artery occlusion.