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Distribution characteristics of cerebral hemodynamic load in different brain regions of acute cerebral infarction patients undergoing acetazolamide PECT imaging

Abstract

Objective: To investigate the incidence of Cerebral Hypoperfusion Lesions (CHL) in patients with acute cerebral infarction and their distribution characteristics in different brain tissues.

Methods: A total of 177 patients with acute cerebral infarction were enrolled. Cerebral blood flow perfusion imaging was performed using acetazolamide stress testing, followed by SPECT scanning. PET image analysis software was utilized to convert analog-to-digital signals, and semi-quantitative data were employed to identify Regional Cerebral Blood Flow (RCBF) values. Regions with RCBF values ≥30% were defined as CHL. The positive rates of CHL in detrain tissues, including the parietal lobe, temporal lobe, frontal lobe, thalamus, basal ganglia, occipital lobe, and career compared.

Results: CHL occurred in all 177 patients with acute cerebral infarction, with a positive rate of 100%. Among these observed in different brain tissues, 675 sites were positive for CHL, with a positive rate of 27.24%. Among them, 408 sections of the left-brain tissue were CHL positive, with a positivity rate of 32.93%. Sections of the right brain tissue were CHL positive, with a positivity rate of 21.55%. The CHL positivity rate in the left-brain tissue was higher than in the right, and the difference was statistically significant (X^2=40.480, P<0.05). There were statistically significant differences in CHL pirates across different regions of the brain tissue (X^2=303.888, P<0.05). The positive rate of CHL in the left parietal lobe (X^2=44.308), temporal lobe (X^2=32.286), and frontal lobe (X^2=5.418) was higher than that on the right side, and the difference was statistically significant (P<0.05). There was no statistically significant difference in the positive rate of CHL in the left thalamus (X^2=0.111) alas ganglia (X^2=0.050), occipital lobe (X^2=3.347), and cerebellum (X^2=0.203) compared with the right side (P>0.05). There was no significant difference in the positive rate of CHL in different brain tissues between the malemute groups (P>0.05). There was no significant difference in the positive rate of CHL in different brain tissues between the group aged <60 years and T-group aged ≥60 years (P>0.05).

Conclusion: The positive rate of CHL in brain tissue is high in patients with acute cerebral infarction. The poise rate of CHL in the left-brain tissue is higher than that in the right, and the positive rate of CHL is not related to gender or age. Local cerebral blood flow passion insufficiency is a clinical feature of cerebral infarction, and cerebral blood flow perfusion imaging with acetazolamide stress test plays an important role in the diagnosis and assess mute cerebral infarction.

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Introduction

Acute cerebral infarction refers to a group of common neurological diseases characterized by clinical symptoms resulting from the degeneration and necrosis of brain tissue due to the sudden eruption of cerebral blood supply [1]. The majority of cases are caused by atherosclerosis or thrombus formation in the medium and small arteries supplying the brain, leading to stenosis occlusion of the lumen and subsequent focal acute cerebral ischemia. In some instances, the condition arises from the entry of solid, liquid, or gaseous foreign bodies into real arteries or the cervical arteries supplying the brain via the circulatory system, causing blood flow obstruction or a sudden reduction in blood flow, which results in the softening and noncorresponding brain tissue regions. According to data from 2022, there are over 7.6 million new cases of cerebral infarction globally each year, resulting in 3.3 million deaths. In China, the incidence and mortality rates of cerebral infarction rank first in the world. The primary principle of Cerebral Blood Flow Perfuse Imaging (CBFPI) involves intravenous injection of small-molecule, neutral, highly lipophilic amine Compounds or Tetra-Azacyclododecanaacetic Acid (DOTA) chelates. These agents can cross the normal blood-brain barrier to enter brain cells, where they generate secondary metabolites and emit gamma rays be tracked and displayed. When the Radiographic Contrast Medium (RCM) enters the brain cells, it is converted into water-soluble substances or decomposed into charged secondary producer the action of intracellular enzymes, remaining within the brain tissue. Through CBFPI imaging, the distribution of Regional Cerebral Blood Flow (RCBF) within the brain tissue is and subjected to quantitative analysis to assess the status of cerebral blood flow. The quantity (density) of Radiolabeled Cerebral Metabolic Tracers (RCM) entering brain tissue is directly proportional to the density of Regional Cerebra Blood Flow (RCBF). By measuring the radioactive signals generated by the tracer, local cerebral blood flow status can be indirectly assessed. Since RCM and RCBF are parallel ton functional metabolism, this examination can also reflect local brain functional status to a certain extent. When local cerebral blood flow increases or decreases, the corresponding brain functional regions receive a corresponding sups oxygen and nutrients, thereby modulating the metabolic level of brain cells. Conversely, a reduction in local cerebral blood flow leads to ischemic and hypoxic pathological changes in the corresponding brain functional regions [2,3]. Brain neuroscience represents the frontier of contemporary bioscience research; accurately determining the function, metabolism, and blood supply of diseased brain tissue provides valuable support for the diagnosis, treatment, and prognosis assessment of cerebrovascular diseases. Given the high number of acute cerebral infarction patients treated in our hospital in recent years, this study is conducted to precisely id early local cerebral blood flow and functional states, scientifically establish therapeutic time windows, mitigate brain tissue damage, enhance treatment efficacy, and reduce disability and mort.

Materials and methods

Study population: A total of 177 patients with cerebral infarction admitted to our hopene January 2013 and December 2024 were selected. Among them, 129 were male, with an age range of 7-82 years (mean age: 59.88±12.30 years); 48 were female, with an age range of6-87 year (mean age: 63.56±10.13 years). There was no statistically significant difference in mean age between genders (t=.854, P=0.065). The study was approved by the Ethics Committee, and all participants provided informed consent. Inclusion criteria included: infestations: previous good health, first-onset acute illness, sudden onset of unilateral limb weakness or numbness, unilateral facial numbness or facial deviation, dysarthria, visual blurring, nausea, vomiting, and other focal neurological deficits. Some patients may have had impaired consciousness, with symptoms reaching peak severity within seconds to minutes and persisting without (2) Imaging examinations: A: ≥24 hours post-onset, SPECT/CT Cerebral Blood Flow Perfusion Imaging (CBFPI) or cranial CT showing definiypodense infarct lesions; B: ≥5 hours post-onset, cranial MRI Diffusion-Weighted Imaging (DWI) showing definite hyperintense infarct; C: Digital Subtraction Angiography (DSA), Computed Tomography Angiography (CTA), or Magnetic Resonance Angiography (MRA) showing definite vascular stenosis or occlusion. (3) History of hypertension, hyperlipidemia, diabetes, or atrial fibrillation, or presence of unhealthy lifestyle habits such as smoking, alcohol abuse, mental labor of exercise. (4) Laboratory examinations: possible abnormalities in coagulation function, blood glucose, lipid profile, liver function, or renal function, which aid in assessing disease severity identifying etiologies; Electrocardiogram (ECG) examination to rule out cerebral infarction caused by emboli from cardiac diseases. Exclusion criteria included conditions that could neurological deficits, such as intracerebral hemorrhage, intracranial tumors, brain abscesses, epilepsy, or hyping.

Clinical classification standards for cerebral infarction: (1) Mild Cerebral Infarction: The NIH Stroke Cale (NIHSS) score ranges from 1 to 4. Patients may present with mild symptoms, such as mild speech disorders, facial muscle weakness, or sensory abnormalities. (2) Mate Cerebral Infarction: The NIHSS score ranges from 5 to 15. Patients may present with moderate neurological deficits, such as obvious speech disorders, limb weeks, or hemiplegia [4]. (3) Severe Cerebral Infarction: The NIHSS score ranges from 16 to 20. Severe neurological deficit occurs, such as severe speech disorders or complete limb paralysis. (4) Very Severe Cerebral Infarction: The NIHSS score is greater than 20. Patients present with extremely severe neurological deficits, such as coma, aphasia, or complete paralysis of all four limbs.

Precautions for cerebral blood flow perfusion imaging: Prior to cerebral blood flow perfusion imaging, patients are required to fast fours, avoid strenuous exercise, maintain a state of rest, and remove all metallic objects from their person. Administration of sedatives, stimulants, and other drugs acting on the new system must be discontinued 24 hours before the examination. During the procedure, head movement must be strictly avoided to ensure the accuracy of the images. The examination must be conducted within 3 hours postprandial; if the patient is unable to eat beforehand, they must follow medical instructions to orally consume a 50g glucose solution or chive a 40ml intravenous injection of 50% glucose to prevent hypoglycemia from compromising the examination results.

SPECT/CT examination method: Subjects underwent SPECT/CT cerebral blood flow perfusion imaging using technetim-99m (99mTc)-labeled ethyl cysteine dimer (99mTc-ECD). (1) Equipment Model: Siemens Symbian T2 due-head SPECT/CT scanner. (2) Subjects underwent fasting examination upon waking. They orally ingested 400 mg of potassium perchlorate to block choroid pus uptake of $TcO4^-$. Subsequently, they underwent sensory deprivation for 30 minutes with eyes closed, eye masks, and earplugs inserted into the external Audi canals. (3) Intravenous injection of 740 MBq (20 mCi) 99mTc-ECD was administered, full a 15-minute interval. (4) SPECT/CT image

acquisition was performed in a quiet, light-free environment. Patients were positioned supine with head fixation. A 360° cerebral tomographic scan was conducted with the following parameters: 6° per frame, 32 frames total, 35 seconds per rime, 128×128 matrix, zoom factor 1.23, energy peak 140 keV, and window width 15%. Simultaneous CT tomographic acquisition and fusion were performed. (5) Dedicated cerebral blood flow perfusion processing software was used for image reconstruction, generating axial, coronal, and sagittal cerebral tomographic, CT images, and fused images. (6) All SPECT/CT cerebral blood flow perfusion images were interpreted by a unified team of trained nuclear medicine and neurology physic. Visual and semi-quantitative analyses were performed to evaluate the frontal, temporal, parietal, occipital lobes, thalamus, basal ganglia, a cerebellum (14 regions in total, including left and right sides). Region of Interest (ROI) radioactivity counts were measured in each region, and the ratio of lesion ROI co to cerebellar ROI counts was calculated to conduct semi-quantitative analysis of local cerebral blood flow in each region. Assessment of cerebral blood flow perfusion Typically Involves Multiple Parameters, Inclusive to Peak (TTP), Mean Transit Time (MTT), Relative Cerebral Blood Volume (RCBV), and Relative Cerebral Blood Flow (RCBF), to comprehensively evaluate changes following cerebral infarction.

Clinical diagnostic criteria for Brain Hypoperfusion Area (BHA) tomographic imaging

(1) Normal results: l brain perfusion tomographic imaging with no definite abnormal perfusion areas observed on axial, coronal, and sagittal planes. Under normal conditions, the distribution of radioactivity in the cerebral cortex, basal ganglia, and thalamus is uniform and symmetrical, with the uptake ratio between the left and right hemispheres ranging between 0.9 and 1.1. **(2) Abnormal results:** Includes local hyper perfusion or hypoperfusion in the brain parenchyma. Hyper perfusion may indicate seizure activity, traumatic brain injury, or post-surgical status, or brain tumors; hypoperfusion may indicate transient ischemic attack, cerebral infarction, anxiety, depression, or Alzheimer's disease. (3) Diagnostic Criteria for Brain Perfusion Lesions: ① Radioactivity Distribution: Local radioactivity sparsity or defect suggests insufficient perfusion, possibly caused by cerebral infarctions hemic cerebrovascular disease; local radioactivity concentration may indicate seizure activity, vascular malformations, or hypermetabolic tumors. ② Symmetry Comparison: Perfusion in bilateral ce hemispheres should be symmetrical, with particular attention to the frontal, temporal, parietal lobes, and basal ganglia. Asymmetry exceeding 15% is clinically significant; reduced perfusion on the left side is common in post-stroke sequelae, while abnormalities on the right side may affect spatial cognitive function. ③ Brain Region Specie: Perfusion abnormalities in different brain regions correspond to specific functional impairments. For example, frontal lobe hypoperfusion is associated with decreased executive function, temporal lobe abnormalities affecter, and occipital lobe changes often lead to visual impairments. Alzheimer's disease typically manifests as reduced blood flow in the bilateral parietal lobes and posterior conflate gyrus. ④ Dynamic Changes: Comparing results from rest and stress tests can assess cerebrovascular reserve function. No increase in blood flow after stress suggests vascular dysregulation, common in chronic cerebral ischemia. Epileptic foci show hypoperfusion during the interictal period and hyper perfusion during the ictal period. ⑤ Artifact Discriminatorily factors causing false positives must be excluded, such as image blurring due to patient movement or artifacts caused by improper attenuation correction. Certain medications, such as sedatives and vasodilators, may affect imaging results and should be analyzed in conjunction with the medication history.

Statistical methods

Acetazolamide loading test was performed for cerebral blood flow perfusion imaging, followed by SPECT scanning. PET image analysis it was used for analog-to-digital conversion, and semi-quantitative data were employed to identify Regional Cerebral Blood Flow (RCBF) values. Regions with local RCBFES less than 30% of the mean were defined as Brain Hypoperfusion Areas (BHA). The incidence of blood flow hypoperfusion lesions in different brain regions, unclog the parietal lobe, temporal lobe, thalamus, basal ganglia, frontal lobe, occipital lobe, and cerebellum, was compared. Statist analysis was conducted using SPSS 26 software. The BHA incidence was calculated as the number of brain tissue hypoperfusion lesions divided by the number of brain tissue perf, multiplied by 100%. Proportions were expressed as percentages (%). For normally distributed count data, inter-group comparisons of proportions were performed using the Chore (χ^2) test, with a two-sided $P < 0.05$ indicating statistical significance.

Results

Comparison of CHL incidence in different regions among patients with acute cerebral infarction

CHL occurred in all 177 patients with acute reburial infarction, with an incidence rate of 100%, as shown in (Figures 1-3). A total of 2478 brain tissue sections from different regions were examined, with 675 sections showing CHL, resulting in a CHL incidence rate of 27.24%. Among them, there were 408 CHL slices in the left-brain tissue with an incidence rate of 32.93%. Th were 267 CHL slices in the right brain tissue with an incidence rate of 21.55%. The incidence rate of CHL in the left-brain tissue was higher than in the right brain tissue, and the difference was statistically significant (X^2=40.480, P<0.05). Among them, there were 137 CHLs in the parietal lobe, with an incidence rate of 38.7%. Of, there were 99 CHLs in the left parietal lobe, with an incidence rate of 55.93%. There were CHLs in the right parietal lobe, with an incidence rate of 21.47%. There were 136 CHLs in the temporal land incidence rate of 38.42%. Among them, there were 94 CHL slices in the left temporal lobe, with an incidence rate of 53.11%. The was 42 CHL slices in the right temporal lobe, with an incidence rate of 23.73%. There were 89 BHA slices in the front lobe, with a CHL incidence rate of 25.14%. Among them, there were 54 CHL slices in the left frontal lobe, with an incidence rate of 30.51%. Right frontal lobe CHL: 35 slices, CHL incidence 19.77%. Thalamic CHL 125 slices, CHL incidence 35.31%. Among them, left thalamic CHL: 61 slices, CHL incidence 36%. Right thalamic CHL: 64 slices, CHL incidence 36.16%. Basal CHL: 122 slices, CHL incidence 34.46%. Among them, left basal CHL: 60 slices, CHL incidence 33.90%. Right basal CHL: 62 slices, incidence rate 35.03%. Occipital lobe CHL: 61 slices, incidence rate 17.23%. Among them, left occipital lobe CHL: 37 slices, incidence rate 20.90%. Rig occipital lobe CHL: 24 slices, incidence rate 13.56%. Cerebellar CHL: 5 slices, incident rate 1.41%. Among them, left cerebellar CHL: 3 slices, incidence rate 1.69%. There were 2 cases of CHL in the right cerebellum, with an incidence rate of 1.13%. The incidence rates of BHATHE left parietal lobe (X^2=44.308, P<0.05), temporal lobe X^2=32.286, P<0.05), and frontal lobe (X^2=5.418, P<0.05) were higher than those on the right side, and the differences were statistically

significant. There were no statistically significant differences in the incidence rates of BHA between the left and right thalamus (X^2=0.111, P>0.05), basal ganglia (X^2=0.050, P>0.05), occipital lobe (X^2=3.347, P>0.05), and cerebellum (X^2=0.203, P>0.05). (Table 1 and Figure 4).

Table 1: Distribution of CHL in different regions among 177 patients with acute cerebral infarction.

Group	CHL(n=177)	Incidence (%)	X^2	P
Left parietal lobe	99	55.93	44.308	0.000
Left temporal lobe	94	53.11	32.286	0.000
Left frontal lobe	54	30.51	5.418	0.020
Left thalamus	61	34.46	0.111	0.739
Left basal ganglia	60	33.9	0.088	0.767
Left occipital lobe	37	20.9	3.347	0.067
Left cerebellum	3	1.69	0.203	0.652
Left brain total	408	32.93	40.480	0.000
Right parietal lobe	38	21.47		
Right temporal lobe	42	23.73		
Right frontal lobe	35	19.77		
Right thalamus	64	36.16		
Right basal ganglia	62	35.03		
Right occipital lobe	24	13.56		
Right cerebellum	2	1.13		
Right brain total	267	21.55		
Total	675	27.24		
Statistical analysis			303.888	0.000

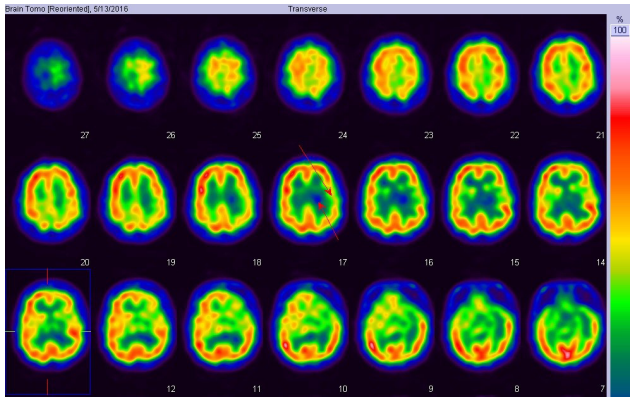

Figure 1: SPECT transverse section image shows a radiotracer defect in the left basal ganglia and corona radiata; reduced cerebral blood flow perfusion the left parietal and temporal lobes.

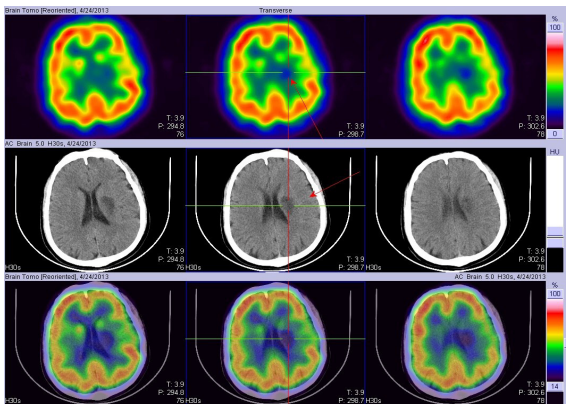

Figure 2: SPECT shows a radiotracer defect in the left basal ganglia and corona radiata, while the simultaneous CT shows reduced density in this SPECT shows reduced cerebral blood flow perfusion in the left parietal and temporal lobes, whereas CT shows no obvious abnormal density changes in this area.

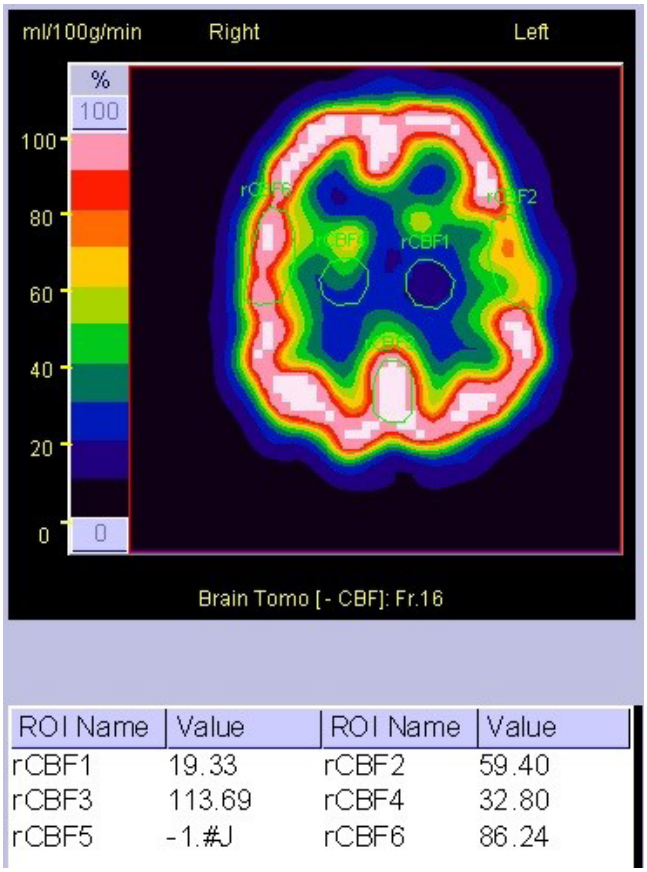

Figure 3: Semi-quantitative analysis of a SPECT transverse section image, outlining the ROI counts of each lesion site and the corresponding contralateral s, as well as the ROI counts of the cerebellar region.

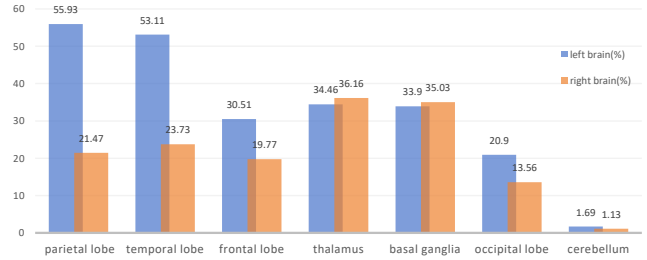

Figure 4: Distribution of CHL in different regions among 177 patients with cerebral infarction.

Comparison of CHL incidence in acute ischemic stroke patients by gender.

Among the 177 patients with acute ischemic stroke there were 129 males and 48 females. In the male group, a total of 505 cerebral tissue CHLs were identified, with an incidence rate of 27.96%; specifically, 296 CHLs were found in the left cerebral tissue (incidence rate: 3.78%) and 209 in the right cerebral tissue (incidence rate: 23.15%). In the female group, a total of 170 CHLs certified, with an incidence rate of 25.30%; specifically, 122 CHLs were found in the left cerebral tissue (incidence rate: 33.33%) and 58 in the right cerebral tissue (incidence rate: 17.26%). There was no statistically significant difference in the incidence rs of CHLs in different cerebral tissues between the male and female groups (P>0.05) (Table 2 and Figure 5).

Table 2: Comparison of CHL incidence between different genders in 177 patients with cerebral infarction.

Group	Male (n=129)	Incidence (%)	Women (n=48)	Incidence (%)	X^2	P
Left parietal lobe	73	56.59	26	54.17	0.083	0.773
Left temporal lobe	71	55.04	23	47.92	0.713	0.399
Left frontal lobe	39	30.23	15	31.25	0.017	0.896
Left thalamus	40	31.01	21	43.75	2.515	0.113
Left basal ganglia	40	31.01	20	41.67	1.774	0.183
Left occipital lobe	30	23.26	7	14.58	1.591	0.207
Left cerebellum	3	2.33	0	-	1.136	0.287
Left brain total	296	32.78	112	33.33	0.007	0.931
Right parietal lobe	31	24.03	7	14.58	1.852	0.174
Right temporal lobe	34	26.36	8	16.67	1.815	0.178
Right frontal lobe	27	20.93	8	16.67	0.401	0.527
Right thalamus	47	36.43	17	35.42	0.016	0.900
Right basal ganglia	47	36.43	15	31.25	0.413	0.520
Right occipital lobe	21	16.28	3	6.25	3.002	0.083
Right cerebellum	2	1.55	0	-	0.753	0.386
Right brain total	209	23.15	58	17.26	1.125	0.289
Total	505	27.96	170	25.30	1.755	0.185

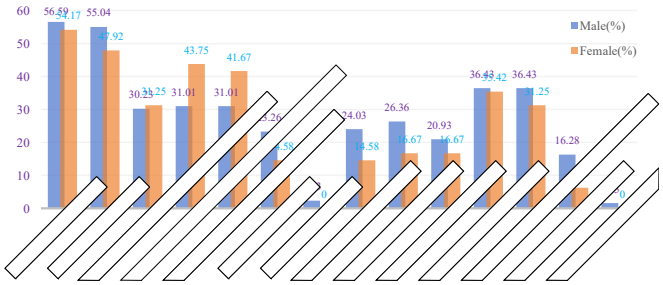

Figure 5: Comparison of CHL incidence in 177 patients with acute cerebral infarction by gender.
Comparison of CHL incidence in patients with acute cerebral infarction of different ages.

Among the 177 patientive cerebral infarction, 77 were <60 years old and 100 were ≥60 years old. In the <60 years group a total of 296 CHLs were detected, with an incidence of 27.46%; among them, 174 CHLs were detected in brain tissue (incidence 32.28%) and 122 in the right brain tissue (incidence 22.63%). In t years group, a total of 379 CHLs were detected, with an incidence of 27.07%; among them, 234 CHLs w detected in the left-brain tissue (incidence 33.43%) and 145 in the right brain tissue (incidence 20.71%There was no significant difference in the CHL incidence of different brain tissues between the <60 years group and the ≥60 years group (P>0.05) See (Table 3 and Figure 6).

Table 3: Comparison of CHL incidence in 177 patients with acute cerebral infarction of different ages.

Group	<60 years old (n=77)	Incidence (%)	≥60 years old (n=100)	Incidence (%)	X^2	P
Left parietal lobe	43	55.84	56	56.00	0.983	0.552
Left temporal lobe	41	53.25	53	53.00	0.001	0.974
Left frontal lobe	23	29.87	31	31.00	0.026	0.871
Left thalamus	26	33.77	35	35.00	0.029	0.864
Left basal ganglia	28	36.36	32	32.00	0.37	0.543
Left occipital lobe	12	15.58	25	25.00	2.333	0.127
Left cerebellum	1	1.3	2	2.00	0.128	0.72
Left brain total	174	32.28	234	33.43	0.181	0.67
Right parietal lobe	15	19.48	23	23.00	0.306	0.58
Right temporal lobe	18	23.38	24	24.00	0.009	0.923
Right frontal lobe	18	23.38	17	17.00	1.115	0.291
Right thalamus	32	41.56	32	32.00	1.722	0.189
Right basal ganglia	30	38.96	32	32.00	0.926	0.336
Right occipital lobe	9	11.69	15	15.00	0.407	0.523
Right cerebellum	0	0	2	2.00	1.558	0.212
Right brain total	122	22.63	145	20.71	0.664	0.415
Total	296	27.46	379	27.07	0.147	0.702

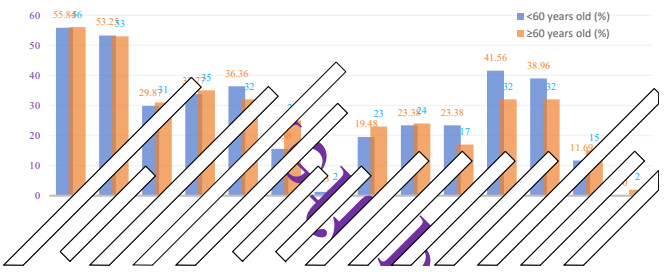

Figure 6: Comparison of CHL incidence in 177 patients with acute cerebral infarction across different age groups.

Discussion

Cerebral infarction primarily refers to acute brain tissue injury caused by vascular occlusion or rupture. Ischemic cerebral infarction I by vascular occlusion, while hemorrhagic cerebral infarction results from vascular rupture. Clinically, acute cerebral infarction accounts for 80% of cerebrovascular des. The core pathology of acute cerebral infarction is vascular obstruction, with the most common etiologies including atherosclerosis, embolism secondary to atrial fibrillation thrombosis due to intrinsic blood abnormalities. Consequently, acute cerebral infarction constitutes one of the fundamental pathological bases of cerebral infarction [5].

According to the Global Burden of Disease analysis, ischemic stroke remains one of the leading causes of death and long-term disability worldwide, with ice accounting for approximately 70% or more of all stroke cases. Although age-standardized incidence and mortality rates have declined in some High-Income Countries (HICs), the baste number of patients affected by stroke continues to increase due to population aging and the cumulative burden of vascular risk factors. Since the second half of the 20th century, the conceptual rake of stroke has undergone significant changes, evolving from a simple model of arterial occlusion to the ischemic cascade, and more recently to a comprehensive paradigm emphasizing the neurovascular unit and systemic regulatory networks. These changes have profoundly influenced experimental research and clinical practice [6].

Cerebral blood flow perfusion refers to the process of blood flow to tissues and is an important indicator for assessing cerebral oxygen supply and metabolic status. In patients with acute cerebral infarction, irreversible focal brain damage occurs due to a sudden or sustained reduction in Cerebral Blood Flow (CBF). Cerebral blood fusion imaging is an examination method that utilizes radionuclide imaging technology to observe cerebral blood flow perfusion, playing a significant role in the diagnosis, treatment, and assessment of vain diseases. The principle of cerebral blood flow perfusion imaging is based on the fact that when the activity of neurons in a specific brain region increases, the blood flow to that region al accordingly to meet its metabolic demands. By injecting radiolabeled compounds capable of emitting gamma rays, such as 99mTc-ECor 99mTc-HMPAO, into the body, these compounds can cross the blood-brain barrier to enter brain tissue and remain there for a period of time. Using a Single Photon Miscomputed Tomography (SPECT) scanner, also known as a gamma camera, imaging is performed to visualize the distribution of the radiotracer within the brain tissue, generating images thatch the blood flow perfusion status of different brain regions. Regions with abundant blood flow exhibit high tracer accumulation and concentration, appearing as high signals on the image; conversely, regions with red blood flow show low tracer accumulation, appearing as low signals. This technique can be used to observe various brain diseases, including cerebrovascular diseases such as transient ischemic attacks, cell infarction, and cerebral arteriosclerosis, as well as epilepsy, Alzheimer's disease, and brain tumors. It can also be used to evaluate cerebral blood flow perfusion and function head injury or surgery, and to assess and differentiate between recurrence and necrosis following brain tumor surgery and radiotherapy [7].

Cerebral Hypoperfusion Lesions (CHL) refer to a series of clinical manifestations caused by local tissue hypoxia and ischemic blood flow in the vascular lumen significantly decreases and flow velocity slows down due to excessive blood pressure decline or significant blood pressure fluctuation, based on atherosclerosis of the cerebral volar wall or luminal stenosis. The clinical symptoms vary depending on the pathological manifestations caused by hypoperfusion of brain tissue resulting from different vascular stenoses. The relationship between the sing cerebral vessels and clinical manifestations is roughly as follows: 1. Internal carotid artery stenosis: may present with contralateral hemiplegia, sensory dist, homonymous hemianopia, aphasia, etc. 2. Middle cerebral artery stenosis: may manifest as aphasia, hemiplegia, hemisensory nice, central facial paralysis, hemianopia, aphasia, alexia, etc., and may even lead to death due to brain herniation. 3. Anterior cerebrally stenosis: may cause expressive aphasia (also known as Broca's aphasia or motor aphasia), sensory disturbance, grasp reflex, etc., due to lesions in the poste part of the inferior frontal gyrus of the dominant hemisphere (Broca's area), and may even lead to contralateral facial, tongue, and upper limb paralysis. ion cerebral artery stenosis: may present with contralateral hemianopia, sensory disturbance, hemiplegia, dyslexia, metamorphopsia, and occlusion of the upper end of the basilar artery manifests as complete blindness. 5. Vertebral artery stenosis: may be life-threatening; lateral medullary may present with nausea, nystagmus, ataxia, etc. In addition, hypoperfusion cerebral infarction is a type of cerebral ischemia; when hematic hypoperfusion occurs, patients with cerebral artery stenosis are prone to local cerebral ischemia at the border zones or watershed areas of adjacent vascular branches, or even large-scale care ischemia in the territory supplied by the stenotic artery, manifesting as watershed infarct lesions [8].

The data from this group indicate that all 177 patients with acute cerebral infarction exhibited Cerebral Hypoperfusion Lesions (CHL), dipositive rate of 100%. Among the 2478 different sites of brain tissue observed, 675 sites were positive for CHL, resulting in a privet rate of 27.24%. Specifically, 408 sites in the left-brain tissue were positive, with a positive rate of 32.3%, while 267 sites in the right brain tissue were positive, with a positive rate of 21.55%. The positive rate of CHL in the left-brain tissue was signific higher than that in the right brain tissue ($P<0.05$). There were statistically significant differences in the positive rates of CHL across different regions of the brain tissue ($P<0.05$).

The results suggest that cerebral hypoperfusion lesions are relatively common in patients with acute cerebral infarction, with a higher positivity rate o in the left hemisphere than in the right. The data from this study indicate that in patients with acute cerebral infarction, the positivity rate of CHL in the left parietal, temporal, and frontal lobe brain tissues was higher than that on the right side, with statistically significant differences ($P<0.05$). However, there were no statistically significantfferences in the

positivity rate of CHL in the left thalamus, basal ganglia, occipital lobe, and cerebellum compared to the right side (P>0.05). There were no significant differences in the positivity rate of CHL in different brain tissues between the male and female groups (P>0.05. Similarly, there were no significant differences in the positivity rate of CHL in different brain tissues between the group aged <60 years and the group aged ≥60 years (P>0.05). These findings suggest that the positivity rate of CHL varies across different brain regions in patients with acute cerebral infarction, while there is no significant different positivity rate of CHL in different brain tissues among patients of different genders or ages.

The research group believes that patients with acute cerebral infarction are highly susceptible to hypoperfusion cerebral infarction due to cerebral hemodynamic discovers, microthrombus formation, decreased blood purification function, and collateral circulation disorders. Local cerebral blood flow perfusion insufficiency is a clinical characteristic of cerebral infarction Clinically, the acetazolamide stress test cerebral blood flow perfusion imaging plays an important role in the diagnosis and assessment of acute cerebral infarction. Nuclear medicine techniques Such Ingle-Photon Emission Computed Tomography (SPECT) and Positron Emission Tomography (PET) can easily capture patients' CHL information. In this study, the positives' of CHL in the left parietal, temporal, and frontal lobe brain tissues was relatively high; therefore, paying special attention to the CHL status of the left-brain tissue can improvs tic quality, and the positive rate of CHL is not related to gender or age. Single-photon emission computed tomography is a safe and effective imaging examination method that has signifiable for the diagnosis, treatment, and assessment of various brain diseases.

For cerebral blood perfusion examination, common methods include

Emission Computed Tomography (SPECT and): SPECT can be used to measure local cerebral blood flow in patients with acute cerebrovascular disease, early displaying the location, extent, and changes in local cerebral blood flow of cerebra infarction; PET can display local cerebral blood flow, oxygen metabolism, and glucose metabolism in cerebral infarction lesions, and monitor the ischemic penumbra and the metabolic effects on distant regions. However, due to high costs, it is mainly used for clinical and basic research on cerebrovascular diseases.

MR Diffusion-Weighted Image (DWI) and MR Perfusion-Weighted Imaging (PWI): These two techniques can diagnose acute cerebral infarction in the ultra-early stage, detecting ischemic changes within minuteness, diagnosing acute cerebral infarction earlier than conventional MRI.

Brain Contrast-Enhanced CT (CT Perfusion Imaging): By intravenous injection of contrast agent technology reflects blood flow perfusion in brain tissue, used to diagnose cerebral embolism, brain tumors, craniocerebral trauma, etc. Its advantages lie in high targeting ability, precise detection of small lesions, and clear observation of vascular structures; however, it involves high radiation and is not suitable for individuals with sensitive constitutions. Siemens Dual-Scout Brain Perfusion Imaging (CTP): Fast scanning speed, clear imaging, and low radiation allow for the rapid and precise assessment of whole-brain blood flow, distinguishing the core infarct area" from "salvageable ischemic brain tissue." It is suitable for patients suspected of acute stroke, patients with Transient Ischemic Attack (Totients with vascular diseases such as carotid stenosis, and patients with brain tumors.

One-Stop Head and Neck CTA+CTP Scanning Technote a single intravenous injection of contrast agent, rapid scanning is performed to simultaneously complete Head and Neck CT Angiography (CTA) and CT Perfusion Imaging (CTP), copy evaluating the vascular anatomy of the head and neck and cerebral tissue blood perfusion function. It is applicable to multiple fields including Neurology, Emergency Medicine, Neurosurgery, habilitation Medicine, and health check-ups.

Acute ischemic stroke is characterized by high incidence, high disability rates, and high mortality rates, necessitating comprehensive societal awareness. Proactive comprehensive prevention and commeasures must be implemented, with particular emphasis on public education, screening, diagnosis, treatment, and daily prevention. Current evidence identifies key risk factors, including a history of hypertension, ds, and hyperlipidemia, as well as lifestyle factors such as high-salt, high-fat, and high-sugar diets, smoking, alcohol consumption, sedentary behavior, massive mental exertion. A prevention-oriented approach is critical, requiring increased public awareness, widespread screening for risk factors, and universal promotion of blood pressure and glucose control, lipid management, smoking cessation, alcohol moderation, healthy dietary habits, and regular physical activity. For patients with acute ischemic stroke, underlying etiologies must be actively addressed. In cases of confidant carotid artery stenosis, consideration should be given to carotid endarterectomy or carotid artery stenting [9-11].

Review the prevention and treatment knowledge of acute cerebral infarction again

Control hypertension: Hypertension is one the main risk factors for cerebral infarction. Keeping blood pressure within the normal range can effectively reduce the risk of cerebral infarction. Monitor blood pressure regularly, take antic medication as prescribed by a doctor, and pay attention to dietary and lifestyle adjustments.

Maintain a healthy weight: Being overweight and obese increase the risk of cerebral infarction. Maintaining a healthy weight through a balanced diet and increased exercise can reduce the occurrence of cardiovascular diseases, indirectly lowering the risk of cerebral infarction.

Quit smoking and limit alcohol: Smoking alive drinking increase the burden on blood vessels, leading to vascular lesions and subsequently triggering cerebral infarction. Therefore, quitting smoking and limiting alcohol consumption is very important for preventing cell infarction.

Healthy diet: The diet should be low in salt and fat, high in fiber, with increased consumption of fruits, vegetables, whole grains, and nuts, and reduce of animal fats and trans fats. This helps maintain vascular health and prevent cerebral infarction.

Moderate exercise: Regular aerobic exercise, such as walking, jogging, and swimming, improve blood circulation, enhance cardiovascular health, and lower the risk of cerebral infarction. It is recommended to engage in at least 150 minutes of moderate-intensity exerted per week.

Regular health check-ups: Regular health examinations, especially checking indicators such as blood lipids, blood glucose, and blood pressure, help detect and control risk factors

for cerebration early.

Relieve stress: Excessive long-term stress may burden the cardiovascular system and increase the risk of cerebral infarction. Maintaining psychological balance and reducing stress throughtation, deep breathing, and relaxation training helps physical health.

Medication prevention: For patients who have experienced a cerebral infarction or have high-risk factors, doctors may recommend long-teal antiplatelet drugs such as aspirin and clopidogrel, as well as statins like simvastatin and rosuvastatin to regulate lipids and stabilizes.

Control blood glucose: Patients with diabetes should strictly control their blood glucose levels to reduce the risk of cerebral infarction. This includes a balanced diet, regular exercise, ani use of hypoglycemic drugs when necessary.

Rehabilitation treatment: For patients with post-cerebral infarction sequelae. Timely rehabilitation treatments such as physical therapy, oral therapy, and speech therapy aim to help patients recover limb function, language ability, and daily living skills.

Abbreviations: ACI: Acute Cerebral Infarction; SPECT: Single-Photon Emission Computed Tomography; CBFPI: Cerebral Blood Flow Perfusion Imaging; CHL: Cerebral Hypoperfusion Lesions; ASCBFPI: Acetazolamide Stress Cerebral Blood Flow Perfusion Imaging.

Declarations

Author contribution statement: The authorship order of this paper was determined by all authors through joint discussion prior to submission and will not be altered after submission. Any necessary changes must be accompanied by an institutional certificate and a written statement signed personally by all authors confirming their agreement with the authorship.

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