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VASCULAR RISK FACTORS FOR CEREBRAL SMALL VESSEL DISEASE

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Abstract: Cerebral small vessel disease (SVD) refers to the syndrome of clinical, cognitive, imaging and pathological manifestations induced by lesions of the small cerebral perforating arteries and arterioles (diameter, 40-200 μm), capillaries, and venules [2]. It is customary to refer to the clinical and imaging findings induced by lesions of the small perforating arteries and arterioles and the resulting brain damage in the cerebral white and deep grey matter [1]. These perforating vessels are essential to maintain optimum functioning of the brain's most metabolically active nuclei and complex white matter networks [3]. The imaging features of ischemic SVD are lacunes, white matter hyperintensity (WMH), cerebral microbleeds (CMB), enlarged perivascular spaces (EPVS) and brain atrophy (BA) [1].

Key words:cognitive, vessels, brain atrophy, lacunes, stroke , hypertension.

Cerebral SVD accounts for approximately one quarter of all ischemic strokes, which is more than twice the risk of recurrent stroke [2]. Studies have shown that SVD is more common in the Chinese than in the Westerners [4]. Cerebral SVD is not only a leading cause of lacunar stroke, cognitive dysfunction, dizziness, unstable gait and urination disorders, but it can also lead to senile depression, Parkinsonism, and accounts for up to 45% of dementias [7]. The most common types of cerebral SVD are age-related cerebral SVD and hypertension-related cerebral SVD, which are considered as the risk factors for ischemic stroke [5].

Material and methods: This was a retrospective observational cohort study. A total of 61 patients admitted to our hospital with first-ever acute ischemic stroke confirmed by cranial magnetic resonance imaging (MRI) were consecutively enrolled between January 2021 and December 2022.
The inclusion criteria were: 1) acute ischemic first-ever stroke within 7 days of admission; 2) age between 35 and 90 years old; 3) classified as LAA or cerebral SVD (lacunar infarction or lacunar infarction with evidence of white matter lesions). The aetiology of acute ischemic stroke was classified in accordance with the Trial of ORG 10172 in Acute Stroke Treatment (TOAST) classification. The TOAST classification system is a widely used method for classifying ischemic stroke and involves the division of strokes into different subtypes according to clinical and neuroimaging information [6].
The exclusion criteria were: 1) cardioembolism, stroke of other undetermined aetiology, acute stroke of other determined aetiology; 2) potential cardiogenic emboli, infections, malignant diseases, severe heart disease, heart, liver, or kidney failure, poisoning, specific white matter diseases, multiple sclerosis, hypercoagulability, hydrocephalus, acute disseminated encephalomyelitis, hypoxia, or adrenal white matter malnutrition; 3) stroke history, mixed strokes (haemorrhage and then infarction or infarction and then haemorrhage), arteritis-induced cerebral infarctions, malignant tumours, alimentary tract haemorrhage, subarachnoid haemorrhage, cerebral haemorrhage, or pregnancy and lactation; 4) patients who had insufficient examinations after admission in whom an etiological analysis.
Results: Demographic data of the studied patient population
There were 61 hospitalized patients with first-ever ischemic stroke, we excluded 36 patients with cardioembolism, 14 patients with stroke of other undetermined aetiology, 4 patients with acute stroke of other determined aetiology, 4 patients with renal insufficiency.. There were 22 male and 39 female patients aged from 35 to 89 years. Based on their clinical symptoms, signs, and imaging results, all of the patients were divided into an ischemic cerebral SVD group and an LAA group according to the

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TOAST criteria. The LAA group (n = 171) contained 22 men and 39 women (61.0 ±10.1 years). The ischemic cerebral SVD group (n = 182) comprised 12men and 49women (62.3 ±9.8 years). A total of 70 cranial MRI-confirmed non-stroke patients older than 35 years who were admitted to the hospital during the same period served as the control group (42 men and 28 women, 54.6 ±8.9 years). Controls had no history of stroke, and were matched with cases for age and sex. These patients had migraine, hypokalemic periodic paralysis, idiopathic facial paralysis, hypertension, or trigeminal neuralgia. Control participants did not have bleeding tendencies, cerebral aneurysms, arteriovenous malformations, mental illness, severe liver, kidney, heart, or lung diseases, autoimmune diseases, or cerebral SVD. There was no significant difference in the general information between the two groups ($p > 0.05$). A flow chart of the study selection process is shown.

The presence of cerebral SVD has a major impact on some conventional therapies. Therefore, early detection and treatment of vascular risk factors for cerebral SVD are of great significance. This study aimed to determine the risk factors for arteriolosclerosis-induced ischemic cerebral SVD and to compare them with those of LAA.

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