International Journal of Medical Science and Clinical Invention 9(4): 6036-6039, 2022
DOI:10.18535/ijmsci/v9i04.01
https://valleyinternational.net/index.php/ijmsci

Case Report,

Lacunar Infarction In Old Stroke: A Case Report

Mohammad Ridho Devantoro[1*], Wilson Saputra Wijaya[2], Muhammad Iqbal Arigi Putra[1], Raden Yogaswara[3]

[1]Department of Neurology, Trisakti University, Jakarta, Indonesia
[2]General Practicioner, Tangerang General Hospital, Banten, Indonesia
[3]Department of Neurology, Mintohardjo Naval Hospital, Jakarta, Indonesia
E-mail Address: rdevantoro@gmail.com

Abstract:

Introduction: Lacunar stroke accounts for nearly a quarter of all ischemic strokes and is an important cause of vascular cognitive impairment and dementia.
Case Presentation: A 52-year-old man with lacunar infarct presented with weakness in his left thumb and forefinger since one day ago when he was sitting down. The patient's fingers appear bent as if holding a pencil and could not be straightened. Physical examination showed an high blood pressure, and paresis of the right facial and hypoglossal nerve. A non-contrast CT scan of head using the parenchymal window showed a hypodense lesion on the left pons, interpreted as a lacunar infarct.
Discussion: Lacunar strokes represent approximately 25% of ischemic strokes and most are associated with good outcomes. However, in 20% to 30% of patients with lacunar stroke, early neurologic damage occurs within the first days after stroke onset. Lacunar stroke was defined as acute lacunar syndrome with infarction <15 mm in diameter in the basal ganglia, deep white matter, thalamus or pons that is not associated with large vessel atherosclerosis or a cardioembolic source.
Conclusion: Lacunar infarction is a potentially reversible. These conditions can potentially be well managed with adequate treatment.

Keywords: Hypertension, Lacunar Infarction, Multiple Artery Stenosis, Monoparesis

Introduction:

Lacunar stroke accounts for nearly a quarter of all ischemic strokes and is an important cause of vascular cognitive impairment and dementia. Despite of its importance, the pathogenesis of lacunar stroke is less understood than that of other stroke subtypes, such as large artery disease and cardioembolism. This is partly because lacunar stroke is rarely fatal during its acute phase, and therefore, there is a lack of pathological data to regard this condition as dangerous. Moreover, lacunar infarcts are often not clearly visible on computed tomography scans. Accurate phenotyping requires magnetic resonance imaging (MRI). It has been shown that computed tomography-based methods combined with the lack of detailed investigation of the source of emboli, as used in many epidemiological studies and clinical trials, can overdiagnose small vessel disease; as many as 50% of cases diagnosed as lacunar stroke turn out to be due to other causes (such as great artery disease) when more detailed imaging, including the use of the MRI, is performed. However, there are very few epidemiological studies of MRI-based subtypes

Case Presentation:

A man, 52 years of age, was brought to Mintohardjo Hospital. He complained of weakness on his left thumb and index finger since the day before, when he was sitting down. Due to these complaints, his fingers cannot be straightened and appear bent as if holding a pencil. The man also complained of slurred speech since the day before, which still seemed coherent, but difficult to comprehend. The patient had no difficulty swallowing, and was still able to eat and drink on his own. The patient complained of a pulsing headache in all areas of the head, he gave his pain a 5 out of 10 in the pain scale. The patient did not complain of nausea, syncope, or seizures. His ability to urinate and defecate remained unchanged.

The patient has history of right-sided weakness after a stroke 3 years ago; the patient took 80 mg aspirin, 250 mg citicoline, and 10 mg simvastatin daily. The patient has a history of hypertension and has been taking 10 mg amlodipine every morning. The patient's family said the patient routinely goes to the doctor to keep his hypertension under control. The patient used to smoke but has now stopped. The patient now mostly remains at home.

The vital signs on admission are as follows: blood pressure 169/90 mmHg, pulse rate of 82 beats per minute, respiratory rate of 20 breaths per minute, oxygen saturation of 98%, temperature 37°C. On physical examination, there appeared to be paresis of the right facial nerve and hypoglossus nerve. Motor strength in the right hand and foot, as well as left hand is 3, while the left leg had normal motor strength. The man had normal sensory perception. Physiological reflexes of the biceps, triceps, patellar seemed increased on the right extremity. The left patellar reflex is not visible. Babinski and Chaddock's pathological reflexes are seen in the right extremity.

The laboratory findings showed SARS-CoV-2 rapid test antigen was negative. A non-contrast CT scan of head using the parenchymal window showed a hypodense lesion on the left pons, interpreted as a lacunar infarct. (**Figure.1**),

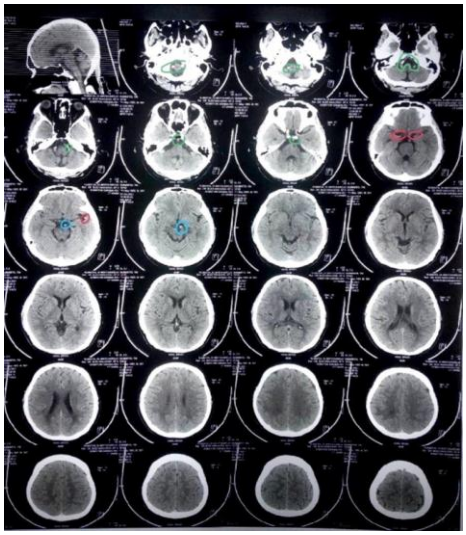

Figure 1: Non- contrast CT scan parenchymal window. Atherosclerosis features, green = vertebrobasilar (+ suspected left vertebral stenosis), red = middle cerebral artery, blue = posterior cerebral artery

Based on these findings the patient was diagnosed with lacunar infarction in old stroke. The patient was treated in hospital and was given an aspirin tablet once a day, Citicoline 250 mg tablets twice a day, Cilostazol 100 mg tablets twice a day, Simvastatin 10 mg tablets once a day, and Amlodipine 10 mg tablets once a day. The patient also had his lipid profile checked. On the second day of treatment, the patient only complained of weakness of the thumb and forefinger and spoke slightly sluggishly; there was no complaint of headache. Vital signs are normal. There was no change in therapy. The third day of treatment, the patient was discharged and prescribed Citicoline 250 mg tablets 2 times a day, Cilostazol 100 mg tablets 2 times a day, Simvastatin 10 mg tablets once a day, Folic acid tablets 2 times a day, Neurodex 2 times a day, and Amlodipine 10 mg tablets 1 times a day

Discussion:

Lacunar strokes represent approximately 25% of ischemic strokes and most are associated with good outcomes. However, in 20% to 30% of patients with lacunar stroke, early neurologic damage occurs within the first days after stroke onset.[2]

Lacunar stroke was defined as acute lacunar syndrome with infarction <15 mm in diameter in the basal ganglia, deep white matter, thalamus or pons that is not associated with large vessel

atherosclerosis or a cardioembolic source. CCL2, CCL3, CCL4, and OASL were more expressed in lacunar stroke than in non-lacunar stroke. The neutrophil chemoattractant cytokine IL8 is more expressed in non-lacunar (large artery and heart) strokes than in lacunar strokes. Lacunar stroke also increases ERBB2. ERBB2 (HER2) affects endothelial cell function and modulates the pathway and proliferation of MAPK, PI3K/Akt, Phospolipase C, Protein Kinase C and STAT. ERBB2 activation may contribute to small vessel lipohyalinosis in lacunar stroke.[3]

According to the Oxfordshire Community Stroke Project (OCSP) classification, lacunar syndromes can be diagnosed by a combination of motor and sensory deficits, or by a characteristic clinical syndrome (eg, dysarthria/clumsy hands). The OCSP classification has good interobserver reliability, can provide rapid information about the etiology and prognosis of acute stroke, and is easy to communicate between doctors. However, studies show that though the OCSP classification correctly identifies nonlacunar stroke subtypes, the sensitivity and specificity in identifying imaging-confirmed lacunar infarcts are inconsistent, particularly in the early hours after stroke onset.[4]

IST-3 (Third International Stroke Trial) is a large multicenter trial of r-tPA (recombinant tissue-type plasminogen activator) in patients over 18 years of age with any subtype of acute ischemic stroke. In the subgroup of the IST-3 trial, we tested for the following: (1) factors associated with lacunar infarction on 24- to 48-hour CT scans; (2) OCSP classification in identifying subsequent lacunar infarcts on CT scans at 24 to 48 hours; and (3) combined OCSP and National Institutes of Health Stroke Scale (NIHSS) in identifying lacunar infarcts on follow up CT scans at 24 to 48 hours.[4]

In our patient, the risk factors were hypertension, dyslipidemia and a history of smoking. Based on a study in Indonesia, patients have a high risk of lacunar stroke by being a male, with history of hypertension and dyslipidemia. History of smoking did not significantly influence the risk of lacunar stroke (95% CI, p=0.325).[4] in addition, Asian countries are known to have a high prevalence of lacunar infarction. In China, lacunar ischemic stroke is second only to large artery atherosclerosis. Hypertension remains a major risk in lacunar stroke due to the development of atherosclerosis from tear stress. This is proven in this study, it appears that the correlation of hypertension to lacunar stroke is higher, while the correlation of dyslipidemia is higher to atherosclerosis of large arteries.[5,6,7] This risk factor is independent of age, increasing age to <60 years is known to increase the risk of lacunar stroke.[7]

The results of a non-contrast CT scan of the head showed a hypodense lesion in the pons, measuring about 11 millimeters in size and not extending anteriorly, meeting the features of a lacunar pons stroke in the study.[8] Not only that, atherosclerosis along the vertebrobasilar arteries, both middle cerebral arteries to the left posterior cerebral artery can be seen.

In a study by Pikija S et al, it is known that ischemic stroke at the site supplied by the vertebrobasilar artery is more likely to be caused by atherosclerosis. Of the 245 patients, most had left vertebral artery calcification through CT imaging (75.9%).[9] another study also supported that 73% of small/lacunar pontine strokes had basilar artery plaque. This supports the patient's history of recurrent stroke, since large artery atherosclerosis is known to increase the risk of small vessel disease in penetrating arteries through thickening, lipohyalinosis and fibrinoid degeneration.[10] Stroke recurrence in vertebrobasilar stenosis >50% has a three times higher risk at 90 days after the first stroke.[11] In the case of middle cerebral artery atherosclerosis, it is known that there is a higher risk of branch atheromatous disease (larger infarct) than lacunar infarction so the patient has a higher risk of recurrence and wider infarction in later life.

Conclusion:

A man, 52 years of age, presents with weakness of the left thumb and index finger since the day before. His fingers cannot be straightened and appear bent as if holding a pencil. He complained of slurred speech since the day before, which still seemed coherent, but difficult to comprehend. The patient complained of a pulsing headache in all areas of the head, and gave a scale of 5 out of 10 in the pain scale. On physical examination, there appeared to be paresis of the right facial nerve and hypoglossus nerve, as well as slight weakness on the right and left sided extremities. The patient was treated in hospital and was given Aspirin, Citicoline, Cilostazol, Simvastatin and Amlodipine. He was discharged at day three and was prescribed the same medication as was given during his stay in the hospital with an addition of

folate.

Conflict of Interest:
None declared

Funding:
No funding sources

Author Contributions:
All authors have a balanced contribution to this paper

References:

[1] Rutten-Jacobs LC, Markus HS. Vascular risk factor profiles differ between magnetic resonance imaging-defined subtypes of younger-onset lacunar stroke. Stroke. 2017 Sep;48(9):2405-11.

[2] Barberick A, Schneider C, Reiff T, Gumbinger C, Ringleb PA. Dual antiplatelet therapy improves functional outcome in patients with progressive lacunar strokes. AHA. 2019;50:1007-9.

[3] Falcione S, Kamtchum J, Sykes G, Jikling G. RNA expression studies in stroke: what can they tell us about stroke mechanism?. HHS. 2021;33:24-9.Doi: 10.1097/WCO.0000000000000786.

[4] Arba F, Mair G, Phillips S, Sandercock P, Wardlaw JM. Improving clinical detection of acute lacunar stroke. AHA. 2020;51:1411-18. doi: https://doi.org/10.1161/STROKEAHA.119.028402.

[5] Harris S, Kurniawan M, Rasyid A, Mesiano T, Hidayat R. Cerebral small vessel disease in Indonesia: Lacunar infarction study from Indonesian Stroke Registry 2012–2014. SAGE Open Med. 2018;6:205031211878431.

[6] Lv P, Jin H, Liu Y, Cui W, Peng Q, Liu R, et al. Comparison of risk factor between lacunar stroke and large artery atherosclerosis stroke: A cross-sectional study in China. PLoS One. 2016;11(3):1–9.

[7] Cai Z, He W, Peng CY, Zhou J, Xu QL, Wu ZS. The prevalence of lacunar infarct decreases with aging in the elderly: A case-controlled analysis. Clin Interv Aging. 2016;11:733–8..

[8] Yang L, Qin W, Li Y, Yang S, Gu H, Hu W. Differentiation of pontine infarction by size. Open Med. 2020;15(1):160–6.

[9] Pikija S, Magdič J, Hojs-Fabjan T. Calcifications of vertebrobasilar arteries on CT: Detailed distribution and relation to risk factors in 245 ischemic stroke patients. Biomed Res Int. 2013;2013.

[10] Klein IF, Lavallée PC, Mazighi M, Schouman-Claeys E, Labreuche J, Amarenco P. Basilar artery atherosclerotic plaques in paramedian and lacunar pontine infarctions: A high-resolution MRI study. Stroke. 2010;41(7):1405–9.

[11] Nouh A, Remke J, Ruland S. Ischemic posterior circulation stroke: A review of anatomy, clinical presentations, diagnosis, and current management. Front Neurol. 2014;5 APR(April):1–16.