Posterior circulation ischemia due to carotid artery dissection with hyperhomocysteinemia in a 51-year-old man

Ruo-Jun Wang, Zhi-Liang Guo, Zhi-Chao Huang, Zhi-Jie Ou, Xia Zhang, Shou-Jiang You, Yong-Jun Cao

Department of Neurology, The Second Affiliated Hospital of Soochow University, Suzhou, Jiangsu 215004, China.

To the Editor: Persistent trigeminal artery (PTA) is one of the most common surviving fetal anastomoses. During embryonic development, the link between the carotid and vertebral basilar circulation would normally regress. Carotid artery dissection (CAD) is a common cause of cerebral ischemia in young people, which can be caused by hyperhomocysteinemia (HHcy) possibly due to gene mutation. We report a case of posterior circulation ischemia caused by CAD.

A 51-year-old man was admitted to the emergency department after sudden onset of slurred speech and quadriplegia. He had no recorded medical conditions and no history of smoking and drinking. The positive of the blood tests including routine blood examination, liver and kidney function, blood coagulation, pre-transfusion examination, tumor markers, and thyroid function examinations were normal. The patient had low-density lipoprotein level of 3.22 mmol/L, homocysteine (Hcy) level of 70.6 μmol/L, while vitamin B12 level was normal. No bleeding or ischemic event recurred as yet. The level of homocysteine was at 17.9 μmol/L upon post-treatment review. The review of the CE-MRA 50 days later showed that the C1 segment of the left ICA dissection was mostly repaired [Figure 1H]. We report a patient with posterior circulation ischemia due to CAD and a PTA link the carotid and vertebral basilar circulation. When the left internal CAD occluded acutely, the right ICA system was unable to fully supply the left middle cerebral artery and posterior circulation, causing symptoms of left cortical infarction and posterior circulation ischemia. The pathogenic mechanisms of CAD are multi-factorial, which may be related to genetic predisposition and minor trauma. In 1995, Frosst et al.[1] showed that the substitution of C to T at nucleotide 677 in the MTHFR gene could lead to the replacement of encoded alanine by valine, thereby reducing the heat resistance and activity of the enzyme, affecting the methylation of Hcy and resulting in increased total Hcy level in plasma. Related studies have reported that HHcy is a risk factor for the development of CAD and stroke.[2] Our reported case is supportive for the finding of the previous studies.

Mural hematoma was shown as a hyper-intense crescent surrounding a compressed lumen on T1-weighted axial cervical MRI. Digital subtraction angiography could also

Access this article online

Quick Response Code:

Correspondence to: Dr. Shou-Jiang You, Department of Neurology, The Second Affiliated Hospital of Soochow University, Suzhou, Jiangsu 215004, China
E-Mail: 0319503013@163.com
Copyright © 2020 The Chinese Medical Association, produced by Wolters Kluwer, Inc. under the CC-BY-NC-ND license. This is an open access article distributed under the terms of the Creative Commons Attribution-Non Commercial-No Derivatives License 4.0 (CCBY-NC-ND), where it is permissible to download and share the work provided it is properly cited. The work cannot be changed in any way or used commercially without permission from the journal.

Chinese Medical Journal 2020;133(19)
Received: 09-03-2020 Edited by: Xiu-Yuan Hao and Xin Chen

2381
indicate signs of CAD, like the flame sign which could be seen in our patient. T1-weighted axial MRI with fat saturation is considered the gold standard for diagnosing CAD, compared to conventional angiography which is invasive, costly and unable to display mural hematoma. HR-MRA showed refined morphologic features of initial segment of the left internal CAD, thus establishing a specific diagnosis. The signal intensities of intramural hematoma vary over time. On days 1 and 2, the hematoma is usually hypointense in T1- and T2-weighted images. On day 2 to day 5, with the increase in methemoglobin content, the hematoma becomes hyperintense, especially in the T1 weighted images. These findings were consistent with the MRI of the patient 3 days after onset.

CADISS trial\(^{[4]}\) showed that there was no significant difference in clinical outcomes between anti-coagulation and anti-platelet therapy in patients with extra-cranial CAD, and the risk of recurrence of stroke is low. Research by Daou et al\(^{[5]}\) further suggested that both treatments could be used in intra-cranial, extra-cranial carotid, and vertebral artery dissection, with similar incidence of new or recurrent ischemic and hemorrhage events. Our case is consistent with the above findings that the patient has a favorable prognosis and a low risk of stroke recurrence, which may be due to self-healing of the CAD.

In conclusion, we have presented a special case of posterior circulation ischemia caused by acute occlusion of the left ICA dissection with right PTA. The CAD was presumably caused by HHcy due to homozygous mutation of MTHFR gene. The present report extends the clinical presentation spectrum of CAD-induced strokes.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

**Funding**

This work was supported by grants from the National Natural Science Foundation of China (No. 81901198) and Basic research of Suzhou Medical and health care (No. SYS201724).

**Conflicts of interest**

None.

**References**

1. Frostick P, Blom HJ, Milos R, Goyette P, Sheppard CA, Matthews RG, et al. A candidate genetic risk factor for vascular disease: a common mutation in methylenetetrafolate reductase. Nat Genet 1995;10:11–13. doi: 10.1038/ng0595-111.

2. Wang WM, Jin HZ. Homocysteine: a potential common route for cardiovascular risk and DNA methylation in psoriasis. Chin Med J 2017;130:1980–1986. doi: 10.4103/0366-6999.211895.

3. Bachmann R, Nassenstein I, Kosimman H, Dittrich R, Stehling C, Kugel H, et al. High-resolution magnetic resonance imaging (MRI) at 3.0 Tesla in the short-term follow-up of patients with proven cervical artery dissection. Invest Radiol 2007;42:460–466. doi: 10.1097/01.rli.0000262758.98098.d6.

4. Markus HS, Hayter E, Levi C, Feldman A, Venables G, Norris J. Antiplatelet treatment compared with anticoagulation treatment for cervical artery dissection (CADISS): a randomised trial. Lancet Neurol 2015;14:361–367. doi: 10.1016/S1474-4422(15)70018-9.

5. Daou B, Hammer C, Mouchtours N, Starke RM, Koduri S, Yang S, et al. Anticoagulation vs antiplatelet treatment in patients with carotid and vertebral artery dissection: a study of 370 patients and literature review. Neurosurgery 2017;80:368–379. doi: 10.1093/neuros/nyw086.

How to cite this article: Wang RJ, Guo ZL, Huang ZC, Ou ZJ, Zhang X, You SJ, Cao YJ. Posterior circulation ischemia due to carotid artery dissection with hyperhomocysteinemia in a 51-year-old man. Chin Med J 2020;133:2381–2382. doi: 10.1097/CM9.000000000001026