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Case Report

Hyperperfusion complications after successful cerebral arterial recanalization: a case report and literature reviews

Yu-Cheng Lin[1,2], Chun-Hung Su[3], Hsin-Yi Chi[1,2*]

[1] *Department of Neurology, Chung Shan Medical University Hospital, Taichung City, Taiwan*
[2] *Department of Neurology, Chung Shan Medical University, Taichung City, Taiwan*
[3] *Department of Internal medicine, Chung Shan Medical University Hospital, Taichung City, Taiwan*

Cerebral hyperperfusion syndrome (CHS) is a rare but severe complication after carotid endarterectomy (CEA), carotid angioplasty and stenting (CAS), and mechanical thrombectomy (MT). This syndrome results from a sudden increase of blood flow in a chronic low-flow artery after recanalization .Cerebral vessel dysautoregulation is the most adapted theory to explain CHS. Herein, we describe one case of CHS, who experienced the dramatic changes from coma to total recovery. We discussed the possible differential diagnosis, risk factors, preventive measurements and treatment based on our case and series of literature reviews.

Keywords: cerebral hyperperfusion syndrome, carotid angioplasty and stenting, cerebral autoregulation.

1. Introduction

Since the first successful carotid endarterectomy(CEA) in 1954, carotid artery stenting (CAS)in 1989(2), and mechanical thrombectomy (MT) in the 1980s, these revolutionary therapeutic approaches have become increasingly popular worldwide (3). CEA and CAS are aimed at treating high-grade asymptomatic or symptomatic carotid artery stenosis in order to restore patency and reduce the long-term risk of stroke (4). With the strong evidence of improving functional outcomes and survival, MT has become the standard of care for patients with acute ischemic stroke due to a large vessel occlusion (5). Although these treatments rebuild the cerebral flow, abundant perfusion may not always contribute to a fair neurological outcome (6), especially when the cerebrum is in a state of chronic hypoperfusion secondary to vascular stenosis. In these cases, long-standing vasculopathy impairs cerebral autoregulation and results to limit capacity to augmentation of the cerebral blood flow (CBF) after successful reperfusions (7).

During restored blood flow status, one may experience various neurological symptoms over the treated hemisphere, including visual disturbances, severe headache, seizures, and conscious deterioration, as defined by cerebral hyperperfusion syndrome (CHS). Severe intracranial hemorrhage (ICH) and subarachnoid hemorrhage may occur in some cases, leading to significant permanent neurological damage. Herein, we present one patient met this critical challenge of CHS.

2. Case Presentation

A 90-year-old man had ischemic heart disease, hypertension, diabetes mellitus, hyperlipidemia, and 2 episodes of right hemisphere ischemic stroke. However, he could perform his daily life with partial assistance. He was admitted for stenting because of symptomatic right internal carotid artery

* Corresponding Author: Hsin-Yi Chi
Address: No. 110, Sec. 1, Jianguo N. Rd., Taichung City, 40201, Taiwan
Tel: 886-4-24739595 ext. 34836
E-mail: ann.jih@msa.hinet.net

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stenosis (Figure 1). The treatment was successfully administered. However, a decreased consciousness level accompanied by total paralysis of the left limb developed a few hours after this procedure. Initial National Institute of Health Stroke Scale (NIHSS) recorded by the neurologist was 24 (4 point is the old sequalae). Emergent computed tomography angiography and computed tomography perfusion did not reveal any signs of intracranial hemorrhage, perfusion abnormalities or large-artery occlusion. Edematous changes over the right hemisphere presented in non-contrast CT, suggesting a hyperemic brain (Figure. 2). During the bedside approach , high systolic blood pressure (SBP) ranged from 171 mmHg to 204mmHg was checked. Aggravated blood pressure control was conducted to the target blood pressure below 140/90mmHg. His consciousness and muscle power gradually regained and completely recovered to his baseline. This patient was discharged from hospital smoothly 5 days later.

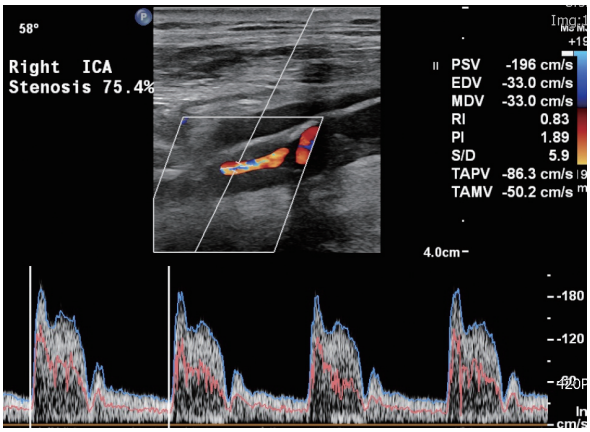


Figure 1. Carotid sonography revealed high degree stenosis with increased flow velocity in right internal carotid artery

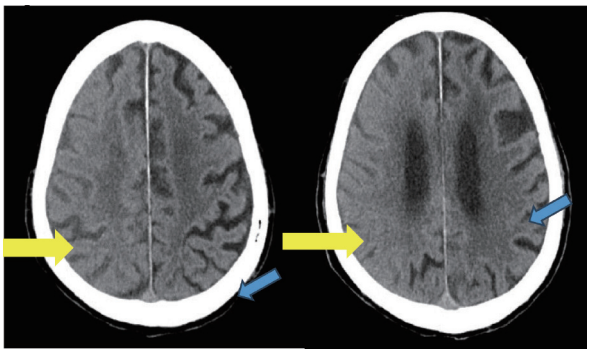


Figure 2. Brain computed tomography displayed swollen brain with diffuse hyperattenuating sulci in the right hemisphere (marked by yellow arrows) compared with the deep sulci in the left hemisphere (marked by blue arrows)

3. Discussion

CHS was first described as a clinical syndrome following CEA (8). According to Bouri et al (9), criteria of post-CEA CHS are as follows: (1) Within 30 days post procedure, (2) Evidence of hyperperfusion in transcranial Doppler (TCD) or brain imaging, or SBP > 180mmHg, (3) clinical neurologic features exist, decreased conscious level, and radiological features as cerebral edema or ICH (4) Exclude other possible causes, such as new ischemia, metabolic or pharmacologic cause. Based on literature reviews , CHS occurred with an incidence of 0.3-3% after CEA (6, 10). The incidence of CHS after CAS ranged from 1.2-4.6% depending on the variation in research design and disease definition (8). However, the incidence of CHS in post-MT remained unclear due to mixed clinical changes in acute stroke with large vessel occlusion, including reperfusion injury, hyperperfusion and tissue ischemia (8).

The clinical features of CHS include neurologic disorders, such as ICH, visual disturbances, severe headache, seizures, and conscious deterioration. Symptomatic CHS can emerge from a moderately increasing cerebral blood flow(CBF). According to previous studies, 20%-40% of increasing CBF above the baseline could cause CHS (10, 11). Series literatures pointed out the pathophysiology of this syndrome is brain tissue damage following cerebral vessel dysautoregulation (11).

During hypoperfusion, compensatory mechanisms, such as wall construction changes or dilatation (7) initiate to maintain normal CBF. Autoregulation in cerebral vessels, especially arterioles is conducted by myogenic and neurogenic components (7). In chronically dilated vessels, as mentioned earlier, normal regulation would be impaired and fail to adapt to fluctuating blood pressure (7), as we called dysautoregulation. Therefore, once recanalization is successful, a surge in CBF and blood pressure occurs in the treated artery, and tissue injury emerges due to autoregulation failure in the corresponding territory(7). In patients with MT, the vessel wall may

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become vulnerable with increased permeability under an ischemic insult (8). Compared with CEA and CAS, edema and tissue damage may have been worse in the MT group considering the vulnerable ischemic area and inflammatory reaction (6). Additionally, early venous drainage can occur after reperfusion therapy for acute ischemic stroke (12, 13), caused by capillary vasodilation, which may subsequently result in hemorrhagic transformation and ICH.

Identifying potentially predictive factors and stratifying risks are crucial for preventing and managing CHS. Large clinical studies pointed out the four major risk factors associated with CHS were older age, critical intracranial atherosclerotic stenosis ≥90%, severe contralateral intracranial atherosclerotic disease, and longstanding pre-existing hypertension (8, 14). Other reported risk factors included poor collateral supply, history of stroke or transient ischemic attacks, white matter hyperintensities, cerebral microbleeds on magnetic resonance imaging (MRI), chronic kidney disease, diabetes mellitus, and intraoperative or persistent postoperative hypertension (7, 8, 13, 15). Except the common risks mentioned above, embolic debris during CAS procedure, prolonged ischemia and reperfusion injury in MT patients were additional conditions (6, 8). Factors predictive of CHS after carotid revascularization included patient age(8, 16, 17) , pre-treatment cerebral vasoreactivity (17), pre-treatment asymmetric index of CBF ([ipsilateral resting CBF/contralateral resting CBF]×100) (17), and ratio of CBF increase after operation (measured by perfusion-weighted MRI or single-photon emission CT) (8, 16, 17).

Form the viewpoints of CHS treatment, intensive blood pressure(BP) control should be conducted after CAS/CEA procedure (8, 13, 14). Optimal blood pressure is recommended to maintain under 140/90mmHg. For high-risk patients (e.g. long standing hypertension, severe carotid stenosis over 90%, or poor collateral blood flow) conducting more strictly control as BP below 120/80mmHg is suggested (8, 14). Duration of close BP monitor and control is at least 24 hours post the procedure. TCD is recommended to follow hyperperfusion status in this duration (8).

Recently, staged angioplasty(SAP) has been proposed to gradually adapt to increasing CBF and avoid CHS (8, 18, 19). SAP divides traditional CAS into two steps. First, a carotid angioplasty with a balloon is performed. After cerebral hemodynamic stability, a carotid stenting is implanted. This innovative strategy is proven to reduce CHS and cerebral hyperperfusion complications (19). However, the interval between the angioplasty and the stenting and inter-procedure risk is undetermined (18).

In MT patients, perioperative management centered on blood pressure control is important for preventing hyperperfusion(6).After MT, blood pressure control according to the degree of recanalization is recommend(8).Once harboring hyperperfusion diagnosed, strict management of blood pressure under 120/80 mmHg was suggested (6).Meanwhile, intensive care at least 24 hours with close monitor neurological conditions and following-up images would be necessary (8).

4. Conclusion

We presented one classic CHS after standard CAS procedure. A patient with comatose response and hemiplegia recovered dramatically after aggressive blood pressure control. In deal with recanalization therapy for chronic carotid arterial stenosis, more investigation of the underling diseases, the mechanism of large arterial stenosis, the collateral vascular distribution and familiar with measurements to reduce CHS are necessary before our aggressive procedures.

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