Improvement of Cerebral Perfusion in Hemodynamic Stroke Patients after Carotid Stenting: Clinical, Cerebral TCD, and Cerebral DSA Evaluations

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Abstract---Hemodynamic stroke is caused by hypoperfusion, instead of infarction or embolism, with a prevalence of approximately 10% of all cerebral infarctions. Introducing hypoperfusion as the causative factor for ischemic stroke is crucial in patient care and management. It can be demonstrated through clinical symptoms, physical examination, and diagnostic imaging. Transcranial Doppler (TCD) and Digital Subtraction Angiography (DSA) examinations to determine cerebral blood flow are very helpful in assessing cerebral perfusion improvement in hemodynamic stroke. A 49-year-old male patient complained of sudden weakness in the right half of the body after waking up, accompanied by a headache and communication difficulties. He also experienced weakness of the right upper and lower limb 1 week before hospital admission and improved 10 hours afterward. The results of the physical examination indicated differences in right and left arm systolic blood pressure of more than 20 mmHg, severe dysarthria, central right facial and lingual palsy, right hemiparesis, and right hemihypesthesia. The results of the CT scan of the head without contrast revealed a subacute cerebral ischemia infarct in the cortical/subcortical left parietal lobe. The results of the TCD examination revealed that the Mean Flow Velocity (MFV) of the right ophthalmic artery and the right siphon artery was 50% higher than that of the left ophthalmic artery and the left siphon artery. The results of the head DSA examination revealed a 50%
stenosis in the left internal carotid artery (ICA). After the application of the Carotid Artery Stenting (CAS) procedure, the patient showed improvements in clinical manifestations and diagnostic imaging as shown on the TCD and DSA examinations. Hemodynamic strokes have different characteristics from infarct and embolic ischemic stroke. The CAS procedure is proven beneficial in improving cerebral perfusion in hemodynamic stroke as demonstrated by clinical evaluations, TCD, and DSA.

**Keywords**—Hemodynamic stroke, hypoperfusion, carotid artery stenting.

**Introduction**

Hemodynamic stroke is used for ischemic stroke caused by hypoperfusion, not caused by infarction or embolism (Klijn et al., 2010). Hypoperfusion may result from decreased blood pressure and severe arterial or systemic obstruction (heart failure or systemic hypotension). The prevalence of hemodynamic stroke is estimated to reach approximately 10% of all brain infarctions (Joinlambert et al., 2012), but hemodynamic disorders as a mechanism that causes stroke have not been widely discussed (Kim et al., 2016). Patients with hemodynamic stroke generally have milder symptoms (Kim et al., 2011), than those with other stroke infarct subtypes. Hemodynamic strokes are rarely fatal and thus receive less attention. However, hemodynamic stroke may be accompanied by severe arterial stenosis (Momjian-Mayor et al., 2005).

Hypoperfusion is used to be considered the main cause of ischemic stroke, but more advanced studies revealed that thromboembolism is the main causative factor. The debate about hypoperfusion as a cause of stroke intensifies due to the absence of a “gold standard” examination for diagnosing hemodynamic stroke and the interaction between low perfusion pressure and embolism that causes ischemic stroke. In recent years, clinical symptoms of hemodynamic disorders and their effects on the prognosis of hemodynamic stroke have been reported. In addition, the recent development of diagnostic tools to determine cerebral blood flow is also very beneficial in establishing the diagnosis and management of hemodynamic stroke (Klijn et al., 2010).

We reported a case of hemodynamic stroke with hemodynamic symptoms, in which we also described the characteristics of this case in terms of epidemiology, etiology, clinical symptoms, supportive investigations and diagnosis, prognosis, and treatment management given in such a case.

**Case report**

A 49 years old male complained of weakness of the right half of the body suddenly after waking up since six hours before hospital admission. The patient also complained of a headache and communication difficulties since two hours before hospital admission. The patient did not experience nausea, vomiting, convulsions, fever, loss of consciousness, visual disturbances, and tingling sensation in half of...
the body. The patient did not also experience shortness of breath, chest pain, and palpitations. The patient had a history of weakness in the right arm and right leg one week before hospital admission, which improved within 10 hours. In addition, the patient had a history of hypertension and high cholesterol but did not take regular medication. The patient denied having a history of diabetes mellitus, heart disease, tumors, trauma, prolonged cough, tuberculosis treatment, or ear, dental, and mouth infections. The patient had a habit of smoking two to three cigarettes per day.

The results of the patient's physical examination indicated differences in right and left arm systolic blood pressure of more than 20 mmHg, right carotid bruit, severe dysarthria, central facial palsy, right central lingual palsy, right hemiparesis, and right hemihypesthesia.

The results of the CT scan of the head without contrast revealed a subacute cerebral ischemia infarct in the cortical/subcortical left parietal lobe. The patient underwent TCD examination, and the results revealed that the MFV of the right ophthalmic artery and the right siphon artery (branches of the internal carotid artery) was 50% higher than that of the left ophthalmic artery and the left siphon artery. The results also showed a peripheral index of more than 1.2 in each blood vessel (possibly due to due to generalized atherosclerosis and chronic hypertension). The results of the DSA examination indicated a 50% stenosis in the left internal carotid artery and the left common carotid artery.

After the application of the CAS procedure, the patient showed improvements in clinical manifestations and diagnostic imaging as shown on the TCD and DSA examinations. The clinical improvements included improvement in dysarthria, motor skills, and TDS differences in the two arms. Improvements in diagnostic imaging results include normal MFV in the right and left blood vessels and peripheral index in each blood vessel greater than 1.2 (possibly due to due to generalized atherosclerosis and chronic hypertension) based on TCD images and normal perfusion based on DSA images.

Discussion

Epidemiology

In a study involving 1,000 patients with acute atherosclerotic ischemic stroke in Korea in 2012, 0.9% of cases were hemodynamic strokes. All of these cases were associated with lesions in the anterior circulation, namely the middle cerebral artery as well as proximal and distal internal carotid arteries (Kim et al., 2012). A study involving 245 patients with acute intracranial atherosclerotic ischemic stroke in Pakistan indicated that border-zone infarction pattern was quite common, accounting for 25.2% of all infarcts. Border-zone infarction was the most common infarct pattern after cortical territorial infarction. The most commonly affected vessels were the middle cerebral artery (45.7%) and the internal carotid artery (16.6%). The study also showed an increase in cases of border-zone infarction as the degree of arterial stenosis increased (P=0.002)(Khan et al., 2013). In a study in Greece, hemodynamic stroke was found in 31% of 502
atherosclerotic acute ischemic stroke patients. In these cases, stenosis was found in the left ICA.

**Etiology**

Cerebral hypoperfusion in hemodynamic stroke can occur locally or globally (Caplan et al., 2016a). Local hypoperfusion is associated with structural changes in the cerebral arteries. Global hypoperfusion can be caused by systemic disorders related to arterial pressure, blood volume, and blood hyperviscosity conditions (Klijn et al., 2010; Caplan et al., 2016a).

Cerebral atherosclerosis is a major risk factor for ischemic stroke (Bonati et al., 2016). Extracranial atherosclerosis is more common in Caucasian populations, whereas intracranial atherosclerosis is more common in Asian and African populations.

Most atherosclerotic deposits are commonly found distal to the common carotid artery, bifurcation, and the first few centimeters from the internal carotid artery (Bonati et al., 2016). Intracranial atherosclerosis is commonly found in the middle cerebral artery and the cavernous and supraclinoid internal carotid arteries (Fig. 1.1).

*Lesions in increasing frequency*

![Distribution of carotid and vertebrobasilar atherosclerotic lesions.](image)

Risk factors for atherosclerosis in carotid artery stenosis include advanced age, smoking, hyperlipidemia, hypertension, and diabetes. The prevalence is higher in male patients than females. In this case, the risk obtained factors included smoking and hyperlipidemia. Active smokers were at increased risk of developing carotid artery stenosis. The stenosis grade was associated with the number of cigarettes smoked over time.
Clinical Symptoms

Clinical signs and symptoms of a hemodynamic stroke are difficult to distinguish from other ischemic stroke subtypes/mechanisms (Klijn et al., 2010). Most of the neurologic deficits in hemodynamic stroke are nonspecific (Table 1.1 and Table 1.2). However, certain precipitating factors, signs, and symptoms can indicate hemodynamic disturbances.

Table 1.1
Clinical symptoms of patients with border-zone infarction

<table>
<thead>
<tr>
<th>Infarction Patterns</th>
<th>Neurological Symptoms</th>
</tr>
</thead>
</table>
| Contralateral weakness (proximal limb weakness is more severe than distal limb weakness, not affecting face)  
Motor transcortical aphasia (left hemisphere infarction), word-finding difficulties  
Mood disorders (right hemisphere infarction)  
Crural hemiparesis  
Hemihypesthesia |
| Sensory transcortical aphasia (left temporal-parietal border zone)  
Homonymous hemianopsia  
Quadranopia  
Hemineglect  
Anosognosia (right hemisphere infarction) |
| Brachiofacial hemiparesis with/without sensory disturbances  
Subcortical aphasia (left hemisphere infarction) |
| - Man-in-the-barrel syndrome |
| Prosopagnosia (bilateral occipital-temporal zone border)  
Balint's syndrome (bilateral occipital-parietal zone border) |
Table 1.2
Frequency of clinical features of border-zone infarction

<table>
<thead>
<tr>
<th>Clinical symptoms</th>
<th>Cortical border-zone infarction</th>
<th>Internal border-zone infarction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemiparesis</td>
<td>29%</td>
<td>96.3%</td>
</tr>
<tr>
<td>Aphasia</td>
<td>29%</td>
<td>37%</td>
</tr>
<tr>
<td>Brachiofacial weakness</td>
<td>27%</td>
<td>-</td>
</tr>
<tr>
<td>Ataxia</td>
<td>18%</td>
<td>-</td>
</tr>
<tr>
<td>Dysarthria</td>
<td>18%</td>
<td>-</td>
</tr>
<tr>
<td>Facial weakness</td>
<td>11%</td>
<td>-</td>
</tr>
<tr>
<td>Hemianopsia</td>
<td>9%</td>
<td>-</td>
</tr>
<tr>
<td>Headache</td>
<td>4%</td>
<td>-</td>
</tr>
<tr>
<td>Impaired consciousness</td>
<td>4%</td>
<td>7.4%</td>
</tr>
<tr>
<td>(confusion)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arm weakness</td>
<td>4%</td>
<td>-</td>
</tr>
<tr>
<td>Quadruponia</td>
<td>4%</td>
<td>-</td>
</tr>
<tr>
<td>Pseudo-radial weakness</td>
<td>2%</td>
<td>-</td>
</tr>
<tr>
<td>Leg weakness</td>
<td>2%</td>
<td>-</td>
</tr>
<tr>
<td>Optical ataxia</td>
<td>2%</td>
<td>-</td>
</tr>
<tr>
<td>Discretionary syndrome</td>
<td>2%</td>
<td>14.8%</td>
</tr>
<tr>
<td>cognitive impairment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemisensory</td>
<td>-</td>
<td>11.1%</td>
</tr>
<tr>
<td>Cortical dysfunction</td>
<td>48%</td>
<td>60%</td>
</tr>
<tr>
<td>Lacunar syndrome</td>
<td>17.3%</td>
<td>17.8%</td>
</tr>
</tbody>
</table>

Atherosclerotic lesions are frequently found in the arteries arising from the aortic arch. Approximately 80% of these lesions are located in the bifurcation of the common carotid artery. Auscultation of the carotid arteries is mainly performed during the examination to determine the occurrence of carotid stenosis that can predispose the patient to stroke. Carotid bruits occur in 70–89% of patients with a carotid 2 mm artery luminal narrowing. However, carotid artery luminal narrowing is not the only cause of a carotid bruit. Auscultatory sounds from heart valve murmurs radiating to the neck, cervical venous humus, and intracranial artery vascular malformations can produce vascular sounds similar to bruits. Arterial tortuosity, kinking, and high clinical output may produce bruits even in normal or non-stenotic arteries. Approximately one-third of patients with carotid bruits are found to have hemodynamically significant lesions (70–90% stenosis). Roughly 50% of patients with hemodynamically significant carotid narrowing are found to have a bruit during carotid auscultation (Lucerna et al., 2019).

In this case report, the identified symptoms include differences in right and left arm systolic blood pressure of more than 20 mmHg, right carotid bruit, severe dysarthria, right hemiparesis, and right hemihypesthesia. Differences in blood pressure between arms are a common and observable phenomenon in various populations. An increase in the difference in systolic blood pressure between arms is usually considered significant if it is greater than or equal to 10 mmHg, and its reported prevalence in the population ranges between 3.6% and 9.4%. It is
typically associated with subclavian artery stenosis, cerebrovascular disease, stroke, cardiovascular morbidity and mortality, atherosclerosis, and left ventricular hypertrophy. The difference in blood pressure in the two arms was initially suspected as a symptom of subclavian artery stenosis. However, the DSA examination results did not indicate the presence of stenosis in the subclavian artery but revealed that the cause was an atherosclerotic stenotic lesion that caused a hemodynamic stroke. A carotid bruit that occurs contralateral to a stenotic lesion is caused by arterial tortuosity.

**Diagnosis**

The diagnosis of hemodynamic stroke requires evidence of cerebral hypoperfusion with adequate imaging techniques and infarction pattern that characterizes hypoperfusion (Ringelstein et al., 2012). Currently, there is no gold standard examination for the diagnosis of hemodynamic stroke, but several supportive investigations can provide crucial clues to the presence of hemodynamic disorders. These investigations include infarction patterns on cerebral imaging, examination of collateral flow, and examination of cerebral hemodynamics (Klijn et al., 2010).

Hemodynamic stroke patients with internal carotid artery occlusion should undergo an assessment of collateral blood supply from the contralateral internal carotid artery, ipsilateral external carotid artery, and vertebral artery (Klijn et al., 2010). In a prospective study involving 117 patients with symptomatic internal carotid artery occlusion, the presence of leptomeningeal collateral blood supply from the pial artery branches of the posterior cerebral artery was an independent predictor of TIA/stroke recurrence (hazard ratio 4.1, 95% CI 1.3–13.1) (Klijn et al., 2010). A study involving 200 patients with internal carotid artery stenosis/occlusion indicated a worse improvement in functional outcome (P = 0.023) in patients with collateral ophthalmic arteries. A study involving 42 patients with symptomatic internal carotid artery occlusion evaluated by the performance of MRI, angiography, and PET indicated that the presence of ophthalmic or leptomeningeal collaterals was a predictor of hemodynamic disturbances in the form of increased oxygen extraction fraction (Student's t-test, P < 0.005).

Angiography, CT, MRI, and TCD are modalities that can be utilized to assess collaterals. Each modality has a method/scale of assessment of the degree of various collaterals. Conventional cerebral angiography is the gold standard for evaluating the anatomy of the collateral circulation. CTA combined with perfusion imaging showed good inter-observer reliability that correlates with clinical outcome. MRI can assess leptomeningeal collaterals but is used less frequently (Romero et al., 2009; McVerry et al., 2012). In this case report, supporting examinations were performed, including a head CT scan without contrast, TCD, and DSA to assess blood vessels.
Figure 1.2 Head CT scan without contrast showing a subacute cerebral ischemia infarction in the left cortical-subcortical parietal lobe.

Figure 1.3 TCD examination before the performance of CAS procedure showing that the MFV of the right ophthalmic artery and the right siphon artery (branches of the internal carotid artery) was 50% higher than that of the left ophthalmic artery and the left siphon artery.

Figure 1.4 TCD examination after the performance of CAS showing no differences in the MFV of the right and left ophthalmic artery and right and left siphon artery.
Figure 1.5 DSA examination showing a 50% stenosis in the left internal carotid artery and the left common carotid artery.
Therapy

Surgical techniques for hemodynamic stroke are generally aimed at increasing cerebral blood flow through collateral pathways and restoring perfusion to the ischemic cerebral area. Some of the considered surgical strategies include endarterectomy, angioplasty, stenting, and bypass surgery. However, bypass surgery is currently not recommended as a therapy in patients with intracranial atherosclerotic disease15, 16.

In patients with severe stenosis of the extracranial carotid artery (70–99%) documented by non-invasive imaging who had a TIA/ischemic stroke in the past six months, Carotid Endarterectomy (CEA) is recommended (Class I; Level A).

In patients with TIA/minor stroke (non-disabling), the revascularization procedure can be performed within two weeks (Class IIa; Level B)128. Arteries in total occlusion normally do not respond well to surgery because the clot formed can propagate distally beyond the surgical access (Caplan et al., 2016b).

CAS can be performed as an alternative to CEA for patients with symptomatic extracranial carotid artery stenosis. CAS can be considered when the internal carotid artery lumen diameter is reduced by more than 70% based on confirmed non-invasive imaging or more than 50% with catheter-based imaging (Class IIa; Level B) (Kernan et al., 2014). In elderly patients (over 70 years), CEA is preferred over CAS, especially when the arterial anatomy does not allow for endovascular intervention (Kernan et al., 2014). CAS is preferred over CEA in patients with difficult to access carotid arteries (e.g., due to obesity or high carotid bifurcation), tracheostomy, scar/fibrotic tissues in the neck due to previous surgery or radiotherapy, lesions proximal to the clavicle or distal to the C2 vertebra, coronary artery disease, congestive heart failure, chronic obstructive pulmonary disease, recurrent stenosis after CEA, and contralateral carotid occlusion. Recommendations for revascularization also take into account the risk of
complications from the procedure. CEA is recommended if perioperative morbidity and risk of surgery are estimated to be less than 6%. CAS can be considered if the complications of endovascular intervention are of moderate or low risk (Ma et al., 2015).

Angioplasty/stenting is not recommended as initial therapy in patients with stroke/TIA associated with major intracranial artery stenosis (Class III; Level B). However, angioplasty/stenting may be considered in patients with TIA/stroke due to recurrent severe stenosis (70-99%) despite optimal medical therapy (Class IIb; Level C) (Kernan et al., 2014).

In recent years, CAS has been used as a less invasive alternative for stroke prevention in carotid artery stenosis compared to CEA. However, there is still debate about the effectiveness of CEA and CAS in the treatment of carotid artery stenosis.

Several International Carotid Stenting Study (ICCS) and Endarterectomy Versus Angioplasty Symptomatic Severe Carotid Stenosis (EVA-3S) studies reported increased periprocedural mortality and stroke rates with the application of CAS compared to that of CEA. The Carotid and Vertebral Artery Transluminal Angioplasty Study (CAVATAS I) involved 24 centers from 3 continents (Europe, America, Australia) and ended in 2001 (followed by CAVATAS II). The CAVATAS I results indicated that carotid stenting (endovascular therapy) had the advantage of avoiding minor complications (White et al., 2016).

The Stenting and Angioplasty with Protection in Patient High Risk For Endarterectomy (SAPPHIRE) study showed that carotid stenting was superior to other management of carotid artery stenosis. The Acculink for Revascularization of Carotid in High Risk (ARCHER) study conducted from 2000 to 2002 revealed that carotid stenting performed with a protective mechanism against embolism was safe (White et al., 2016).

The Guidelines for Prevention of Stroke in Patients With Ischemic Stroke of Transient Ischemic Attack of 2006 recommended that patients who currently had a TIA or ischemic stroke in the last 6 months accompanied by severe stenosis (70%-99%) should undergo carotid endarterectomy. In patients who do not allow endarterectomy, carotid stenting is not inferior to endarterectomy. The Carotid Revascularization Endarterectomy versus Stent Trial (CRESS) showed no significant difference between CAS and CEA at the combined endpoint of periprocedural stroke, myocardial infarction, death, or postprocedural ipsilateral stroke (White et al., 2016).

Primary Prevention of Ischemic Stroke in 2006 stated that patients with asymptomatic carotid stenosis should receive intensive treatment, examination, and therapy for modifiable risk factors. Several studies and consensus suggested that carotid stenting had a unique place in managing carotid artery stenosis to prevent the occurrence or recurrence of ischemic stroke because high-risk patients could not undergo endarterectomy. In addition, it had a short recovery and treatment time and a relatively low complication rate.
In this case report, the patient underwent a stenting procedure and showed improvements in clinical manifestations and diagnostic imaging as shown on the TCD and DSA examinations. The clinical improvements included improvement in dysarthria, motor skills, and TDS differences in the two arms. The results of the TCD examination indicated that the MFV of the right and left blood vessels were within normal limits, and the results of the TCD examination indicated normal perfusion.

**Conclusion**

The prevalence of hemodynamic stroke is estimated to reach approximately 10% of all brain infarctions. The term hemodynamic stroke is used for ischemic stroke caused by hypoperfusion. Hemodynamic stroke is basically the result of decreased cerebral blood flow and the inability of the cerebral circulatory system to compensate or increase blood flow when necessary. Cerebral hypoperfusion in hemodynamic stroke can be caused by structural changes in the cerebral arteries (e.g., atherosclerosis) or systemic disorders (hypotension, hypovolemia, and hyperviscosity of the blood). Most of the neurologic deficits in hemodynamic stroke are nonspecific. However, certain precipitating factors, signs, and symptoms can indicate hemodynamic disturbances. Currently, there is no gold standard examination for the diagnosis of hemodynamic stroke, but several supportive investigations can provide crucial clues to the presence of hemodynamic disorders. The management of hemodynamic stroke includes nonsurgical therapy and surgical therapy. The considered surgical procedures for patients with hemodynamic stroke include endarterectomy and stenting/angioplasty. The patient underwent a stenting procedure and showed improvements in clinical manifestations and diagnostic imaging as shown on the TCD and DSA examinations. The clinical improvements included improvement in dysarthria, motor skills, and TDS differences in the two arms.

**References**


