Intraprocedural Detection of Cerebral Hyperperfusion by Flat Detector Computed Tomography in the Evaluation of Cerebral Blood Volume during Carotid Artery Stenting

A Case Report

YUKINORI TERADA¹, TAKETO HATANO², YASUNORI NAGAI², MAKOTO HAYASE², MASASHI ODA², TAKEHIKO NAKAMURA²

¹ Department of Neurosurgery, Tazuke Kofukai Medical Research Institute and Kitano Hospital; Osaka, Japan

² Department of Neurosurgery, Fukui Red Cross Hospital; Fukui, Japan

Key words: neuroparenchymal blood volume, cerebral blood volume, hyperperfusion, carotid artery stenting

Summary

Cerebral blood volume (CBV) can be measured using a C-arm flat detector angiographic system. The present report describes a case in which cerebral hyperperfusion was detected with the Neuro parenchymal blood volume (PBV) system (syngo Neuro PBV IR, Siemens Medical Solutions, Erlangen, Germany) during carotid artery stenting (CAS). An 89-year-old man was referred to our hospital for cerebral brain infarction and severe stenosis of the left carotid artery. CAS was performed, and Neuro PBV was used to measure CBV both during and after the procedure. Postoperative Neuro PBV revealed dramatically increased CBV, and a hyperperfusion state was suspected. The next day, subarachnoid hemorrhage along the sulcus of the left hemisphere was revealed on computed tomography. Strict management of blood pressure was instituted just after the detection of hyperperfusion, and the patient was ultimately discharged from the hospital without any new neurological deficits. Neuro PBV has the advantage that it can be performed in the angiography suite and does not require patient transfer to an alternate setting. Therefore, intracranial hemodynamic changes can be detected during the procedure. We conclude that the Neuro

PBV system is useful for monitoring intracranial hemodynamics during endovascular procedures.

Introduction

Neuroimaging plays a critical role in identifying which patients with acute ischemic stroke might benefit from early surgical intervention. While computed tomography (CT), magnetic resonance (MR) imaging, single photon emission computed tomography (SPECT), and positron emission tomography (PET) have all been used to evaluate the hemodynamic state of the brain, recent studies have described the use of a C-arm flat detector angiographic system (Neuro parenchymal blood volume [PBV]) to measure cerebral blood volume¹⁻⁴. Neuro PBV measurements can be performed in the angiography suite and do not require patient transfer to an alternate setting, which offers significant advantages in the management of patients with acute ischemic stroke. Furthermore, Neuro PBV can be used during endovascular treatment to monitor intracranial hemodynamics. The present report describes a case in which Neuro PBV was used to detect hyperperfusion immediately after carotid artery stenting (CAS).

Case Report

An 89-year-old man with a history of atrial fibrillation, cardiogenic cerebral infarction, chronic respiratory disease and stenting for thoracic aortic aneurysm was referred to our hospital for management of severe stenosis of the left carotid artery, detected by carotid ultrasound after the patient developed transient right hemiparesis and loss of consciousness. On admission, no neurological deficits were present. Diffusion-weighted images (DWI) showed a small fresh infarction in the left precentral gyrus, but the left internal carotid artery was not visualized clearly on MR angiography (Figure 1A,B). CT angiography revealed that the left internal carotid artery was nearly occluded (Figure 1C). SPECT revealed that cerebral blood flow (CBF) was slightly decreased in the left cerebral hemisphere when compared with the cerebellum, but not decreased when compared with the right hemisphere at rest (Figure 1D).

We selected endovascular treatment rather than surgical treatment (carotid endarterectomy) based on the patient's age and the presence of cardiac and respiratory dysfunction. One month after onset of the infarction, due to the presence of a stent in the thoracic aortic aneurysm, right CAS was performed by direct puncture of the left carotid artery. At first, a 4-Fr catheter was inserted into the ascending aorta by transradial catheterization, and Neuro PBV measurements were performed. Then, the catheter was guided to the origin of the left common carotid artery (CCA) to prepare for intraoperative angiography. Angiography revealed that the left internal carotid artery was nearly occluded (Figure 2A). A transverse skin incision was placed in the left neck, and the CCA was exposed. After catheterization with a 6-Fr sheath, the proximal side of the CCA was clamped using Bulldog forceps. A distal protection balloon (PercuSurge GuardWire™ system, Medtronic AVE, Santa Rosa, CA, USA) was carefully advanced across the stenosis and dilated to 5 mm. Under distal protection, predilation was performed with a 3.5×20 mm balloon (Sterling, Boston Scientific, Natick, MA, USA). After pre-dilation, a self-expandable stent (Precise 10×40 mm, Cordis, Johnson & Johnson, Miami, FL, USA) was deployed. After aspirating debris, the distal protection balloon was deflated. Angiography showed good dilatation of the left carotid artery (Figure 2B). After

removing the catheter, the hole in the carotid artery was sutured with CV-7 (W.L. Gore & Associates, Flagstaff, AZ, USA). Finally, Neuro PBV measurements were performed.

Evaluation by preoperative Neuro PBV revealed an obvious decrease in CBV in the left hemisphere, and it was thus speculated that cerebral autoregulation had failed (Figure 2C,D), although preoperative SPECT revealed slightly decreased CBF in the left cerebral hemisphere. Postoperative Neuro PBV showed dramatically increased CBV in the left hemisphere, partially more than the normal right hemisphere (Figure 2E,F). The risk of hyperperfusion was thought to be high. Therefore, heparinization was immediately neutralized, and systolic arterial blood pressure was kept strictly under 120 mmHg. No changes were observed in neurological status after CAS, but the next day CT revealed subarachnoid hemorrhage (SAH) in the left hemisphere (Figure 3A,B). SPECT revealed increased CBF in the left cerebral hemisphere (Figure 3C). Strict control of blood pressure was maintained, and follow-up CT did not reveal any enlargement of SAH. SPECT performed two weeks after the operation showed resolution of hyperperfusion in the left hemisphere. The patient was ultimately discharged from the hospital without any new neurological deficits. Follow-up MR angiography clearly depicted the left internal carotid artery (Figure 3D).

Discussion

Neuro PBV measures CBV by characterizing the difference in contrast agent distribution in brain tissue³. Ahmed et al¹, and Bley et al², reported that CBV measured by Neuro PBV exhibited a good correlation with that measured by CT perfusion. Neuro PBV can measure CBV without the need to transfer patients from the angiography suite to another setting, which represents a significant advantage when managing patients with acute ischemic stroke or those in the perioperative term of the revascularization.

Neuro PBV data are acquired by injecting the contrast medium into a peripheral vein in the same manner as that used for CT perfusion studies. Itokawa et al.³ described conducting measurements with Neuro PBV after injecting the contrast medium from the ascending aorta with cerebral angiography. Their method was as

Figure 1 A,B) Diffusion-weighted image shows a small fresh infarction in the left precentral gyrus, and the left internal carotid artery is not clearly visualized on magnetic resonance angiography. C) Computed tomography angiography shows that the left internal carotid artery is nearly occluded. D) Single photon emission computed tomography shows that the cerebral blood flow in the left cerebral hemisphere is slightly decreased compared with the cerebellum, but not when compared with the right hemisphere at rest.

follows: acquisition time, eight seconds; 70 kV; 460 mA; 200° total angle; 60 frames/second; and 397 total frames. A 50% diluted contrast medium (Iomeron 300, Eisai, Tokyo, Japan) was used to reduce to the total amount of contrast agent. The distribution of contrast agent is required to be constant throughout the intracranial tissue at the time of imaging. Therefore, scan delay time was determined as the time from injection of the contrast medium to the visualized time of confluence of the sinuses. Following their method, we performed CBV measurements using biplane angiography for digital subtraction angiography. A 4-Fr catheter

was threaded into the ascending aorta. The C-arm was rotated 200° (right anterior oblique position 100°-left anterior oblique position 100°), and the images were taken at a rate of 60 frames/second for 6.6 seconds during the mask run. Then, 50% diluted contrast was administered via the catheter at the same position at a rate of 4 ml/s for 6.6 seconds, with an additional 12 seconds (scan delay time) for the second rotation of the C-arm. The PBV map was immediately available after subtraction of the two sets of source images.

In this case, cerebral blood flow at rest was slightly reduced in SPECT before surgery (Fig-

ure 1D). Therefore, we considered that the patient's preoperative state corresponded to Power's stage 0 or 1.5). However, Neuro PBV showed severely decreased CBV in the left cerebral hemisphere just before the procedure. It was speculated that cerebral autoregulation had failed, and that the patient's hemodynamic state was worse than his preoperative status (Figure 2C,D) ⁵. Therefore, we could assume the possibility of hyperperfusion just before the procedure. Furthermore, postoperative CBV measured by Neuro PBV was markedly increased in the left cerebral hemisphere, and the possibility of hyperperfusion was detected im-

mediately (Figure 2E,F). Therefore, we neutralized the heparinization and instituted strict management of blood pressure just after the diagnosis. As a result, the patient was ultimately able to be discharged from the hospital without neurological deficit.

Cerebral hyperperfusion is defined as a major increase in ipsilateral CBF above the metabolic demands of the brain tissue following the repair of carotid artery stenosis ⁶⁻⁸. Cerebral hyperperfusion syndrome (CHS) is characterized by unilateral headache, face and eye pain, seizure and focal symptoms due to intracranial hemorrhage (ICH), SAH, or cerebral edema

Figure 2 A) Preoperative angiography shows that the left internal carotid artery is nearly occluded. B) After carotid artery stenting, angiography shows good patency of the left carotid artery. C,D) Evaluation by preoperative Neuro parenchymal blood volume (PBV) reveals an obvious decrease in cerebral blood volume (CBV) in the left hemisphere. E,F) Postoperative Neuro PBV showed dramatically increased CBV in the left hemisphere, partially more than normal right hemisphere.

related to hyperperfusion⁷. CHS was originally reported by Sundt⁸ in connection with carotid endarterectomy, and has also been noted to occur after CAS⁹⁻¹⁹. ICH caused by hyperperfusion is associated with high mortality and morbidity rates^{6,7,9,11,16,18,19}. Ogasawara et al.⁶ reported that the incidence of hyperperfusion syndrome and ICH were 0.5-2.2% and 0.6%, respectively after CEA, and 1.1% and 0.7%, respectively after CAS. Shindo et al.⁷ also demonstrated incidences of hyperperfusion syndrome and ICH of 0.6-3.1% and 0.3-1.2%, respectively following CEA, and 1.1%-6.8% and 0.67-4.5%, respectively following CAS. Ogasawara et al.⁶ reported that CHS occurred significantly sooner after CAS than after CEA. The onset of CHS peaked on the sixth postoperative day in patients who had undergone CEA and within 12 hours after surgery in those who had undergone CAS. Therefore, it is important to detect the hyperperfusion state as soon as possible after CAS in order to prevent hyperperfusion syndrome. Evaluation by SPECT or PET is the gold standard for assessment of CBF. However, these modalities are expensive and require special equipment and facilities. Neuro PBV is considered a useful method to detect hemodynamic changes in the angiogra-

phy suite, thereby providing valuable information to guide the management of hyperperfusion states during endovascular treatment.

As mentioned above, CHS is detected via measurement of CBF. Although, the currently available flat detector CT lacks the temporal resolution to assess CBF or mean transit time, it is able to provide spatial resolution and contrast sensitivity that should be adequate for measuring levels of blood in the brain parenchyma¹. Therefore, CHS cannot be precisely defined via Neuro PBV. However, in this case, a marked change in CBV was seen when comparing preoperative and postoperative Neuro PBV values. Therefore, we could speculate that CHS had developed just after CAS, and we quickly instituted management for CHS.

Conclusions

Measurements of CBV by Neuro PBV during CAS can be helpful in predicting and preventing CHS. While we conclude that Neuro PBV is useful for the monitoring of intracranial hemodynamics during endovascular procedures, further investigation is required to establish the utility of Neuro PBV.

References

- 1 Ahmed AS, Zellerhoff M, Strother CM, et al. C-arm CT measurement of cerebral blood volume: an experimental study in canines. *Am J Neuroradiol.* 2009; 30 (5): 917-922. doi: 10.3174/ajnr.A1513.
- 2 Bley T, Strother CM, Pulfer K, et al. C-arm CT measurement of cerebral blood volume in ischemic stroke: an experimental study in canines. *Am J Neuroradiol.* 2010; 31 (3): 536-540. doi: 10.3174/ajnr.A1851.
- 3 Itokawa H, Suzuki R, Moriya M, et al. Evaluation of cerebral blood perfusion with IVR-CT/angio system during interventional procedures. *No Shinkei Geka.* 2006; 34 (3): 281-286. [Article in Japanese].
- 4 Struffert T, Deuerling-Zheng Y, Kloska S, et al. Flat detector CT in the evaluation of brain parenchyma, intracranial vasculature, and cerebral blood volume: a pilot study in patients with acute symptoms of cerebral ischemia. *Am J Neuroradiol.* 2010; 31 (8): 1462-1469. doi: 10.3174/ajnr.A2083.
- 5 Powers WJ, Press GA, Grubb RL Jr, et al. The effect of hemodynamically significant carotid artery disease on the hemodynamic status of the cerebral circulation. *Ann Intern Med.* 1987; 106 (1): 27-34. doi: 10.7326/0003-4819-106-1-27.
- 6 Ogasawara K, Sakai N, Kuroiwa T, et al. Intracranial hemorrhage associated with cerebral hyperperfusion syndrome following carotid endarterectomy and carotid artery stenting: retrospective review of 4494 patients. *J Neurosurg.* 2007; 107 (6): 1130-1136. doi: 10.3171/JNS-07/12/1130.
- 7 Shindo A, Kawai N, Kawakita K, et al. Intracerebral hemorrhage after carotid artery stenting without evidence of hyperperfusion in positron emission tomography. *Interv Neuroradiol.* 2007; 13 (2): 191-199.
- 8 Sundt TM Jr, Sharbrough FW, Piepgras DG, et al. Correlation of cerebral blood flow and electroencephalographic changes during carotid endarterectomy. *Mayo Clin Proc.* 1981; 56 (9): 533-543.
- 9 Abou-Chebl A, Yadav JS, Reginelli JP, et al. Intracranial hemorrhage and hyperperfusion syndrome following carotid artery stenting: risk factors, prevention, and treatment. *J Am Coll Cardiol.* 2004; 43 (9): 1596-1601. doi: 10.1016/j.jacc.2003.12.039.
- 10 Al-Mubarak N, Roubin GS, Vitek JJ, et al. Subarachnoidal hemorrhage following carotid stenting with the distal-balloon protection. *Catheter Cardiovasc Interv.* 2001; 54 (4): 521-523. doi: 10.1002/ccd.1324.
- 11 Coutts SB, Hill MD, Hu WY. Hyperperfusion syndrome: toward a stricter definition. *Neurosurgery.* 2003; 53 (5): 1053-1058; discussion 1058-1060. doi: 10.1227/01.NEU.0000088738.80838.74.
- 12 Grunwald IQ, Politi M, Reith W, et al. Hyperperfusion syndrome after carotid stent angioplasty. *Neuroradiology.* 2009; 51 (3): 169-174. doi: 10.1007/s00234-008-0483-6.
- 13 Gurm HS, Nallamothu BK, Yadav J. Safety of carotid artery stenting for symptomatic carotid artery disease: a metaanalysis. *Eur Heart J.* 2008; 29 (1): 113-119. doi: 10.1093/eurheartj/ehm362.
- 14 Krajickova D, Krajina A, Nova M, et al. Fatal intraventricular hemorrhage after the extracranial carotid artery angioplasty and stent placement. *Cardiovasc Intervent Radiol.* 2005; 28 (4): 502-505. doi: 10.1007/s00270-004-0155-9.
- 15 McCabe DJ, Brown MM, Clifton A. Fatal cerebral reperfusion hemorrhage after carotid stenting. *Stroke.* 1999; 30 (11): 2483-2486. doi: 10.1161/01.STR.30.11.2483.
- 16 Meyers PM, Higashida RT, Phatouros CC, et al. Cerebral hyperperfusion syndrome after percutaneous transluminal stenting of the craniocervical arteries. *Neurosurgery.* 2000; 47 (2): 335-345; discussion 343-345. doi: 10.1097/00006123-200008000-00013.
- 17 Mori T, Fukuoka M, Kazita K, et al. Intraventricular hemorrhage after carotid stenting. *J Endovasc Surg.* 1999; 6 (4): 337-341. doi: 10.1583/1074-6218(1999)006<0337:IHACS>2.0.CO;2.
- 18 Morrish W, Grahovac S, Douen A, et al. Intracranial hemorrhage after stenting and angioplasty of extracranial carotid stenosis. *Am J Neuroradiol.* 2000; 21 (10): 1911-1916.
- 19 Wu TY, Anderson NE, Barber PA. Neurological complications of carotid revascularization. *J Neurol Neurosurg Psychiatry.* 2012; 83 (5): 543-550. doi: 10.1136/jnnp-2011-301162.

Yukinori Terada, MD
 Department of Neurosurgery
 Tazuke Kofukai Medical Research Institute
 and Kitano Hospital
 2-4-20 Ohgimachi, Kita-ku
 Osaka 530-8480, Japan
 Tel.: +81-6-6312-1221
 Fax: +81-6-6361-0588
 E-mail: yterada@kuhp.kyoto-u.ac.jp