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Loss of labelling efficiency caused by carotid stent in pseudocontinuous arterial spin labelling perfusion study

D.Y.-T. Chen [a], Y.-S. Kuo [a], H.-L. Hsu [a], F.-X. Yan [a], H.-L. Liu [b], C.-J. Chen [a], Y.-C. Tseng [a,*]

[a] Department of Radiology and Brain and Consciousness Research Center, Taipei Medical University Shuang-Ho Hospital, New Taipei City, Taiwan
[b] Department of Imaging Physics, The University of Texas M. D. Anderson Cancer Center, Houston, TX, USA

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AIM: To elucidate the cause of cerebral hypoperfusion on the stent placement side after carotid artery stent placement (CAS) measured by pseudocontinuous arterial spin labelling (PCASL) perfusion imaging.

MATERIALS AND METHODS: Consecutive patients with symptomatic internal carotid artery stenosis receiving CAS were included in the study. Cerebral blood flow (CBF) was measured by PCASL perfusion imaging at 3 T magnetic resonance imaging (MRI) the day before and 3 days after the procedure. Changes in cerebral haemodynamics after CAS were assessed.

RESULTS: Twenty-two patients were included; 17 patients had increased or stationary CBF after CAS and five patients had significantly reduced CBF on the stenting side after CAS whereas CBF increased on the contralateral side. High stent position was noticed in the five patients. After labelling plane adjustment to avoid labelling on the stent, no more cerebral hypoperfusion was noticed.

CONCLUSION: When using PCASL perfusion imaging to monitor post-stenting CBF, the stent may cause an artefact that leads to a low CBF in the territory of the stented vessel. Routinely adding a fast T2 star gradient-echo echo-planar-imaging covering the upper neck region before PCASL perfusion imaging to identify the stent position and avoid the stent-related artefact is recommended.

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Introduction

Carotid artery stenting (CAS) is being increasingly used as an alternative treatment to carotid endarterectomy for carotid artery stenosis.[1] Alteration of cerebral perfusion after CAS has been investigated in many perfusion studies.[2–7] Most of them reported improvement in cerebral perfusion on the stenting side after CAS, especially in patients with high-grade stenosis, with greater perfusion deficit prior to CAS, or with symptoms.[4–6]

Pseudocontinuous arterial spin labelling (PCASL) is a non-invasive technique that detects absolute cerebral blood flow (CBF) without the use of an exogenous contrast agent.[8,9] These properties permit repeated measurement of

* Guarantor and correspondent: Y.-C. Tseng, Department of Radiology, Shuang-Ho Hospital, No. 291, Zhongzheng Road, Zhonghe District, New Taipei City 23561, Taiwan. Tel.: +886 2 22490088 1320; fax: +886 2 22490088 1322.
E-mail address: x2mickey@yahoo.com.tw (Y.-C. Tseng).

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CBF in a short interval, which is useful in patients receiving CAS.[3,7] In Shuang-Ho Hospital PCASL perfusion imaging was used to monitor the cerebral haemodynamic changes before and after CAS. Most patients had increased or stationary CBF after CAS; however, occasionally, patients had obviously decreased CBF on the stenting side after CAS. The aim of the present study was to elucidate the causes of the post-stenting hypoperfusion measured by ASL.

Materials and methods

Patients

This study was approved by the local ethics committee. From July 2012 to October 2013, consecutive patients with symptomatic internal carotid artery (ICA) stenosis (ICA stenosis >70% by North American Symptomatic Carotid Endarterectomy Trial method on conventional cerebral angiogram[10]) receiving CAS in Shuang-Ho Hospital were included in the study. To evaluate the haemodynamic changes after CAS, whole-brain CBF was measured using the PCASL technique before and after the procedure. Patients were excluded when severe dental metal artefact was noted on the PCASL perfusion imaging.

CAS

Patients were pretreated with aspirin (325 mg/d) and clopidogrel (75 mg/d, Plavix; Bristol-Myers Squibb/Sanofi Pharmaceuticals, New York, NY, USA) for at least 3 days before CAS. Systemic anticoagulation was then given by intravenous administration of a bolus of heparin (70 U/kg) and then continuous heparin infusion (15 U/kg/h) to maintain an activated clotting time of more than 250 seconds. The self-expanding stent (Precise; Cordis, Miami Lakes, FL, USA) was deployed across the lesion, and a semi-compliant angioplasty balloon was used to post-dilate the stent to achieve >90% luminal diameter. The post-procedural angiography was performed 20 minutes after stent deployment to evaluate both the stented site and the distal cerebral vasculature. After the procedure, antihypertensive agents were used to maintain systolic pressures of <130 mmHg.

Imaging acquisition

All patients received brain magnetic resonance imaging (MRI) using a 3 T MRI system (Discovery MR750, General Electric Medical Systems, Milwaukee, WI, USA) with diffusion-weighted imaging (DWI), time-of-flight (TOF)-magnetic resonance angiography (MRA) of the brain, and PCASL perfusion imaging on the day before and 3 days after CAS. The protocol applied on each scan was as follows[1]: DWI: single-shot spin-echo echo-planar imaging with repetition time (TR)/echo time (TE)=8000 ms/68 ms, flip angle=90°, b=1000 s/mm^2, diffusion direction=all, field of view (FOV)=230×230 mm^2, in-plane matrix=512×512, section thickness=5 mm, intersection gap=2 mm.[2] TOF-MRA: three-slab three-dimensional (3D) sequence with TR/TE=30 ms/2.9 ms, flip angle=20°, FOV=200×200 mm^2, in-plane matrix=416×256, section thickness=1.4 mm, superior to inferior acquisition.[3] PCASL perfusion imaging: 3D background suppressed fast-spin-echo stack-of-spiral readout module with eight in-plane spiral interleaves, TR/TE=5327 ms/10.5 ms, labelling duration=1.5 seconds, post-labelling delay=2525 ms, no flow-crushing gradients, in-plane matrix=128×128, number of excitations (NEX)=4, section thickness=4 mm, echo train length=36 to obtain 36 consecutive axial sections, labelling plane=10-mm thick and placed 2 cm inferior to the lower edge of the cerebellum, total scan time=336 seconds.

Data analysis

The CBF maps were generated on an Advantage Windows workstation using Functool software (version 9.4, GE Medical Systems). Quantification of CBF was calculated with the following equation[9]:

$$CBF = 6000 \cdot \lambda \frac{\left(1 - e^{-\frac{ST}{T_{1t}}}\right) e^{\frac{PLD}{T_{1b}}}}{2\varepsilon T_{1b}\left(1 - e^{-\frac{LT}{T_{1b}}}\right)} \left(\frac{PW}{SF_{PW} PD}\right)$$

where T1 of the blood (T_{1b}) was assumed to be 1.6 seconds at 3 T, T1 of the tissue (T_{1t}) 1.2 seconds, partition coefficient (λ) 0.9, labelling efficiency (ε) 0.6, saturation time of PD (ST) 2 seconds, labelling duration (LT) 1.5 seconds, and post-labelling delay (PLD) 2525 ms. PW was the perfusion-weighted or the raw-difference image; PD was the signal intensity of the proton density image and SF_{PW} was an empirical scaling factor (= 32) used to increase the dynamic range of the PW.

To compare changes in CBF before and after CAS, two radiologists with 4 and 15 years of experience chose the regions of interest (ROIs) on two sections, the level of the basal ganglia and the level of the body of the lateral ventricle. On each section, the raters manually outlined the ROIs corresponding to the cortical flow territory of the middle cerebral artery (MCA) of both hemispheres according to the maps by Damasio,[11] taking care to exclude regions of prior cerebral infarction. The absolute CBF (expressed in millilitres per 100 g of tissue per minute) of the MCA territories in each hemisphere were obtained by averaging the mean values within the ROIs of the two sections. Relative CBF was defined as stenting side CBF/contralateral side CBF.

Callback study

In patients showing post-stenting cerebral hypoperfusion, callback studies were performed to confirm whether cerebral hypoperfusion persisted in PCASL perfusion imaging. In the callback study, the CBF was measured twice using two different labelling planes. The CBF was first measured by PCASL perfusion imaging using the routine labelling plane. On the second measurement, T2* echo-planar imaging (gradient-echo echo-planar-imaging sequence, TR/TE=925 ms/20 ms, flip angle=70°, FOV=250×250 mm^2, in-plane matrix=132×128, section thickness=5 mm, intersection gap=1 mm, NEX=4, scanning

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