Original Article

Signal changes on magnetic resonance perfusion images with arterial spin labeling after carotid endarterectomy

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Abstract

Background: Cerebral hyperperfusion after carotid endarterectomy (CEA) is defined as an increase in ipsilateral cerebral blood flow (CBF). Practically, however, prompt and precise assessment of cerebral hyperperfusion is difficult because of limitations in the methodology of CBF measurement during the perioperative period. Arterial spin labeling (ASL) is a completely noninvasive and repeatable magnetic resonance perfusion imaging technique that uses magnetically-labelled blood water as an endogenous tracer. To clarify the usefulness of ASL in the management of cerebral hyperperfusion, we investigated signal changes by ASL with a single 1.5-s post-labeling delay on visual inspection.

Methods: Thirty-two consecutive patients who underwent CEA were enrolled in this retrospective study.

Results: On postoperative day 1, 22 (68.8%) and 4 (12.5%) patients exhibited increased ASL signals bilaterally (Group A) and on the operated side (Group B), respectively. Follow-up ASL showed improvement in these findings. Six (18.8%) patients showed no change (Group C). There was no apparent correlation between ASL signals on postoperative day 1 and the preoperative hemodynamic state, including the cerebrovascular reserve ($P = 0.2062$). Three (9.4%) patients developed cerebral hyperperfusion syndrome (two in Group A and one in Group B). Coincidence in the localization of increased ASL signals and electroencephalographic abnormalities was noted in these patients.

Conclusion: Visual analysis of ASL with a single post-labeling delay overestimates CBF and cannot identify patients at risk of cerebral hyperperfusion syndrome probably because of the strong effect of the shortened arterial transit time immediately after CEA. However, ASL may be used as for screening.

Key Words: Arterial spin labeling, carotid endarterectomy, cerebral hyperperfusion, cerebral hyperperfusion syndrome

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INTRODUCTION

Cerebral hyperperfusion after carotid endarterectomy (CEA) is defined as a major increase in ipsilateral cerebral blood flow (CBF) after surgical repair of carotid stenosis that is well beyond the metabolic demands of the brain tissue.\(^{[4,25,29,31,33,37]}\) However, prompt and precise assessment of cerebral hyperperfusion is often difficult because of limitations in the methodology of CBF measurement during the perioperative period.\(^{[2,5‑8,21,32]}\) Previous reports have demonstrated cerebral hyperperfusion after CEA with transcranial Doppler, Xe-enhanced computed tomography (CT), or single-photon emission CT (SPECT) using 99mTc-ethylcysteinate dimer (ECD) or N-isopropyl-[123I] b-iodoamphetamine (IMP).\(^{[9,10,13,20,24‑26,32,37]}\) The criteria for cerebral hyperperfusion vary among these studies.\(^{[2.5,37]}\) Diagnosis of cerebral hyperperfusion syndrome (CHS) is based not only on evidence of hyperperfusion on these CBF studies but also on the presence of clinical symptoms such as seizures, deterioration of consciousness (including restlessness), and the development of focal neurological signs such as motor weakness.\(^{[25,28,29,37]}\)

Among the various CBF studies performed to date, SPECT has been widely used in Japan to assess the cerebral hemodynamic state and quantify the regional CBF and hemodynamic reserve by measuring the patient’s cerebrovascular reactivity (CVR) to acetazolamide loading.\(^{[17,22,26,34]}\) Ogasawara et al.\(^{[29]}\) reported that IMP-SPECT can be used to preoperatively measure the CVR and identify patients at risk for cerebral hyperperfusion; in addition, post-CEA monitoring of CBF, including immediately after CEA, allows for timely and reliable identification of patients at risk for CHS. However, repeating SPECT studies is practically difficult because administration of the exogenous radioisotope is required.

A new magnetic resonance (MR) perfusion imaging technique with arterial spin labeling (ASL) was recently developed to assess the regional CBF without the need for contrast administration.\(^{[2,5,6,21,32]}\) Because ASL uses magnetically-labelled blood water as an endogenous tracer,\(^{[10]}\) it is a completely noninvasive and repeatable perfusion imaging technique. Previous authors demonstrated a close correlation between ASL and SPECT imaging in terms of assessment of the CBF status.\(^{[10,27,32,35,36]}\) However, ASL has some drawbacks. It has a time delay called the “post-labeling delay” (PLD) between inversion of blood spins passing through the labeling plane in the neck and collection of images in the head after the labelled blood flows into the cerebral tissue.\(^{[2]}\) An ASL sequence with no delay compensation is considerably susceptible to the arrival time of labelled blood in tissue; namely, the arterial transit time (ATT).\(^{[6‑8]}\)

Recent ASL studies have generally used a single PLD that typically ranges from 1.5 to 2.0 s as a trade-off between maintaining adequate diagnostic quality and allowing sufficient delay for visualizing tissue perfusion in clinical MR imaging machines.\(^{[11,21,39]}\) In steno-occlusive cerebrovascular disease, however, the ATT is generally prolonged due to stenosis or occlusion of the major arteries.\(^{[29]}\) Labelled blood that travels via collateral pathways of the circle of Willis or secondary collateral pathways, including those associated with leptomeningeal anastomosis, may also exhibit an increased ATT.\(^{[11,20,26]}\) In this situation, conventional ASL measurements using a single PLD may lead to an underestimation of tissue perfusion.\(^{[11]}\) Conversely, following CEA, improvement of the delayed anterograde flow in the internal carotid artery (ICA) may shorten the ATT, and ASL may lead to an overestimation of the CBF.\(^{[36]}\)

In this report, we demonstrate the chronological changes in ASL signals with a single PLD of 1.5 s immediately after CEA, and compare these findings with preoperative clinical conditions and hemodynamic states. A previous study reported signal changes on ASL with a single PLD of 1.5 s at 3 months postoperatively.\(^{[3]}\) The purpose of this study was to determine whether ASL can assess post-CEA hemodynamic changes and identify patients at risk for CHS.

PATIENTS AND METHODS

Patients

Thirty-two consecutive patients with ipsilateral ICA stenosis underwent CEA at Kyushu Rosai Hospital from November 2011 to April 2016. The inclusion criteria for CEA used in our institute are carotid stenosis of ≥70% or 50–69% with repeated ischemic cerebrovascular events, activities of daily living corresponding to a modified Rankin Scale (mRS) score of ≤2 in asymptomatic patients and 3 or 4 in symptomatic patients with repeated ischemic cerebrovascular events, small or no infarction on MR imaging, and absence of major occlusive disease (≥70% in diameter) distal to the carotid stenosis. Thirty-one of the 32 patients were men, and 1 was a woman. The mean age of the patients was 73.0 years (range, 46–85 years). We reviewed all medical records. Informed consent was obtained from all the patients.

Cerebral blood flow measurements

Arterial spin labeling measurements

Routine MR imaging and ASL perfusion imaging were performed using a 3T-MR unit (Signa HDxt 3.0T version 23; GE Healthcare, Milwaukee, WI, USA). ASL was prepared using a three-dimensional (3D) spiral fast-spin echo sequence with background suppression for perfusion imaging covering the entire brain. A pulsed continuous scheme was employed. Other acquisition parameters
were as follows: Four arms with 1004 points in each spiral arm, phase encoding in the z direction = 32, section thickness = 4 mm, Time to repeat (TR) = 4728 (AUTO) s, post-label wait = 1.525 s (1.5 s), and number of excitation (NEX) = 3. The acquisition time was 2 min 22 s.

All patients underwent preoperative ASL examination with routine MR examination. In all patients, a second ASL examination was performed on postoperative day 1 (POD1); i.e., immediately after extubation of the orotracheal tube. At that time, routine MR imaging was also performed to rule out the appearance of perioperative de novo ischemic events. Follow-up ASL was performed when needed. The timing of follow-up ASL varied in this study. Evaluation of decreased or increased ASL signals was based on visual inspection by two experienced radiologists who were blind to the clinical and imaging data. No differences in the radiologists’ interpretations were noted on independent assessments.

Single photon emission computed tomography measurements
Using SPECT (Headtone SET-031; Shimadzu Co, Kyoto, Japan), CBF was semiquantitatively measured before and 15 min after intravenous injection of 10 mg/kg of acetazolamide (ACZ) on separate days at an interval of 2 to 3 days. Regions of interest were placed automatically in the target area (e.g. in the middle cerebral artery [MCA] territory) using commercially available software (E. CAM Signature; Toshiba Medical, Tokyo, Japan/GMS7700R). The CVR to ACZ was calculated as follows: CVR (%) =100 × (CBF_ACZ − CBF_REST)/CBF_REST, where CBF_REST and CBF_ACZ represent CBF before and after intravenous injection of ACZ, respectively. A CVR of <20% was considered to be a reduced CVR according to a previous report.28

Twenty-nine patients underwent preoperative SPECT imaging at rest. In 24 patients, CBF was assessed with an ACZ challenge. Of these 24 patients, 21 were assessed with IMP-SPECT and 3 with ECD-SPECT.

Electroencephalography
Twenty-nine of the 32 patients underwent routine EEG recording preoperatively and on POD1. Follow-up EEG was performed in 3 patients who developed CHS. Routine EEG recordings were obtained from an 18-channel digital EEG machine (Neurofax; Nihon-Kohden, Tokyo, Japan) with electrode placement according to the International EEG 10-20 system. The EEG recordings were performed for at least 30 min for each patient at rest.

Perioperative management of patients
Twenty-eight patients underwent CEA more than 1 month after the last ischemic event, and 4 patient underwent CEA urgently. All CEA procedures were performed under an operative microscope with the use of internal shunts.

After CEA, general anesthesia with propofol was continued under controlled ventilation until the next morning. Blood pressure was maintained at <130 mm Hg (systolic) and <90 mm Hg (diastolic) in all patients using intravenous nicardipine. Immediately after extubation, all patients underwent routine MR imaging examination including ASL, as described above. In patients with increased ASL signals, arterial blood pressure was more closely monitored and strict control of blood pressure using intravenous nicardipine and diltiazem and an oral angiotensin II receptor blocker was continued until POD7. Diagnosis of CHS was based on the appearance of clinical symptoms and EEG abnormalities that could explain the clinical findings in addition to evidence of increased ASL signals. In Case 5, postoperative ECD-SPECT was also performed to confirm the cerebral hyperperfusion. Functional outcomes were assessed at discharge using the mRS.28

RESULTS

Relationship between preoperative arterial spin labeling signals and preoperative single photon emission computed tomography
[Table 1]. Preoperative ASL signals mostly coincided with the CBF map on resting SPECT with visual inspection. However, ASL had a tendency to underestimate the CBF. On preoperative ASL, 19 of 32 patients exhibited decreased ASL signals on the ipsilateral side. On preoperative SPECT of these patients, an ipsilateral decrease in the CBF was noted in 12 patients, no decrease was noted in 5, and a bilateral decrease was noted in 1. Nine of the 12 patients with no decreased signal on ASL also exhibited no decreased CBF on SPECT. One other patient showed a decreased CBF of the ipsilateral side on SPECT. In a patient with decreased ASL signals on the contralateral side, no decreased CBF was noted on SPECT.

Arterial spin labeling signals on postoperative day 1 and their chronological course
On POD1, 22 (68.8%) of 32 patients exhibited increased ASL signals on both sides (Group A) [Figure 1a]. All patients except two (Case 5; detailed clinical course described below and Case 18) did not develop a de novo neurological deficit attributed to increased ASL signals on both sides. The increased ASL signals were improved in all 15 patients who underwent re-examination of their CBF by follow-up ASL. The increased ASL signals had disappeared by POD7 in six patients, by POD14 in 5, by postoperative month 2 in 1, by postoperative month 6 in 1, and by postoperative month 14 in 2.

Four (12.5%) patients demonstrated increased ASL signals only on the operated side (Group B) [Figure 1b]. All patients except one (Case 23; detailed clinical course described below) did not develop a de novo neurological
Table 1: Clinical profile of 32 patients with carotid stenosis who underwent carotid endarterectomy

| Group | Case | Age/ Sex | Symptom | Side | Postoperative day 1 ASL signal | Disappearance of increase on ASL signal/ Postoperative period | CBF (rest- SPECT) | CVR | Potency of ACOM-A1/ PCOM | CHS | Outcome (mRS score)** |
|-------|------|----------|---------|------|-------------------------------|-------------------------------------------------------------|------------------|-----|------------------------|-----|------------------------|
| A     | 1    | 82/M     | Infarction | Bilateral side | L | Ipsilateral D | Ipsilateral D | Ipsilateral i | +/+ | - | 0→0 | 0
| 2     | 76/M | Infarction | L | Bilateral side | + /14 days | Ipsilateral D | Ipsilateral D | Ipsilateral i | +/+ | - | 1→1 | 1
| 3     | 68/M | TI A      | R | Bilateral side | NE | Ipsilateral D | Ipsilateral D | Ipsilateral i | +/+ | - | 0→0 | 0
| 4     | 71/M | -         | R | Bilateral side | + /12 months | Ipsilateral D | No D | Ipsilateral i | -/+ | - | 0→0 | 0
| 5     | 83/M | -         | R | Bilateral side | + /14 days | Ipsilateral D | No D | Ipsilateral i | +/+ | + | 0→0 | 0
| 6     | 80/M | TIA       | L | Bilateral side | + /7 days | No D | No D | Bilateral i | +/- | - | 0→0 | 0
| 7     | 65/M | TIA       | L | Bilateral side | NE | No D | No D | No i | -/+ | - | 0→0 | 0
| 8     | 71/M | -         | L | Bilateral side | NE | No D | No D | No i* | +/- | - | 0→0 | 0
| 9     | 72/M | -         | R | Bilateral side | + /12 months | No D | No D | Ipsilateral i | +/- | - | 0→0 | 0
| 10    | 85/M | Infarction | L | Bilateral side | NE | No D | No D | Ipsilateral i | +/- | - | 1→1 | 1
| 11    | 72/M | TIA       | L | Bilateral side | NE | No D | No D | Ipsilateral i | +/- | - | 0→0 | 0
| 12    | 66/M | Infarction | R | Bilateral side | + /14 days | Contralateral D | No D | Contralateral i | +/- | - | 1→1 | 1
| 13    | 74/M | -         | L | Bilateral side | + /7 days | Ipsilateral D | No D | No i | +/- | - | 0→0 | 0
| 14    | 74/M | Infarction | L | Bilateral side | + /7 days | Ipsilateral D | Ipsilateral D | NE | +/- | - | 1→1 | 1
| 15    | 76/M | Infarction | L | Bilateral side | + /10 days | Ipsilateral D | Ipsilateral D | NE | +/- | - | 0→0 | 0
| 16    | 75/M | TMB       | R | Bilateral side | + /2 months | Ipsilateral D | No D | Ipsilateral i | +/- | - | 1→0 | 1
| 17    | 74/M | -         | R | Bilateral side | + /10 days | No D | NE | NE | +/- | - | 0→0 | 0
| 18    | 73/M | Infarction | L | Bilateral side | + /7 days | Ipsilateral D | Ipsilateral D | Ipsilateral i | +/- | + | 1→1 | 1
| 19    | 71/M | -         | L | Bilateral side | + /7 days | Ipsilateral D | No D | No i | +/- | - | 0→0 | 0
| 20    | 72/M | Infarction | L | Bilateral side | + /7 days | No D | NE | NE | +/- | - | 1→1 | 1
| 21    | 82/M | Infarction | L | Bilateral side | NE | Ipsilateral D | NE | NE | +/- | - | 2→2 | 2
| 22    | 83/M | Infarction | L | Bilateral side | NE | Ipsilateral D | Ipsilateral D | NE | +/- | - | 1→1 | 1
| B     | 23    | 46/M    | Operated side | L | + /8 days | Ipsilateral D | Ipsilateral D* | Ipsilateral i* | +/- | + | 1→1 | 1
| 24    | 59/M | Infarction | R | Operated side | + /18 months | Ipsilateral D | Ipsilateral D | Ipsilateral i | +/- | - | 1→1 | 1
| 25    | 75/M | Infarction | L | Operated side | NE | Ipsilateral D | Ipsilateral D | Ipsilateral i | +/- | - | 3→3 | 3
| 26    | 83/M | Infarction | L | Operated side | NE | No D | No D | No i | +/- | - | 3→3 | 3
| C     | 27    | 61/M    | Infarction | R | No change | NE | No D | Ipsilateral D | +/- | - | 1→1 | 1
| 28    | 73/M | Infarction | R | No change | No change/ 24 months | No D | No D | Ipsilateral i | +/- | - | 3→3 | 3
| 29    | 80/M | Infarction | L | No change | No change/ 3 months | No D | No D | Ipsilateral i | +/- | - | 2→2 | 2
| 30    | 55/M | Infarction | R | No change | NE | Ipsilateral D | Ipsilateral D | Ipsilateral i | +/- | - | 1→1 | 1
| 31    | 81/M | Infarction | R | No change | NE | Ipsilateral D | Ipsilateral D* | Ipsilateral i* | +/- | - | 2→2 | 2
| 32    | 70/F | Infarction | L | No change | No change/ 6 months | No D | No D | Ipsilateral D | +/- | - | 4→4 | 4

*CBF and CVR were assessed using 99mTc-ECD-SPECT; other cases were assessed using 123I-IMP-SPECT. **Preoperative→postoperative. M: Male, F: Female, TIA: Transient cerebral ischemic attack, TMB: Transient monocular blindness, ASL: Arterial spin labelling, I: Increase, D: Decrease, i: Impaired, CHS: Cerebral hyperperfusion syndrome, NE: Not examined. CBF: Cerebral blood flow, SPECT: Single photon emission computed tomography, CVR: Cerebral blood flow, ACOM: Anterior communicating artery, A1-A1 portion of anterior cerebral artery, PCOM: Posterior communicating artery, mRS: Modified Rankin scale.

deficit attributed to the ipsilateral increased ASL signals. Two patients underwent re-examination of their CBF by follow-up ASL; the increased ASL signals had disappeared by POD8 in one patient and by postoperative month 18 in the other.

Six (18.8%) patients showed no change in their postoperative ASL signals compared with the preoperative ASL images (Group C) [Figure 1c]. None of these patients developed de novo neurological deficits. Three patients underwent re-examination of their CBF by follow-up ASL, and the ASL signals exhibited no change in all 3 patients.

Relationship between arterial spin labeling signals on postoperative day 1 and pre and postoperative clinical conditions

Preoperatively, 13 of 22 patients in Group A were asymptomatic or had a history of transient ischemic attack or transient monocular blindness. Nine patients
had experienced small but symptomatic infarctions. The preoperative mRS score was 0 in 13 patients, 1 in 8 patients, 2 in 1 patient. Their mRS scores did not change postoperatively except one (Case 16; improved).

In contrast, all patients in Groups B and C had old infarctions. In Group B, the preoperative mRS score was 1 in 2 patients and 3 in 2 patients. In Group C, the preoperative mRS score was 1 in 2 patients, 2 in 2 patients, 3 in 1 patient, and 4 in 1 patient. The mRS scores in Groups B and C did not change postoperatively.

**Relationship between arterial spin labeling signals on postoperative day 1 and preoperative hemodynamic state**

There was no apparent correlation between the ASL signals on POD1 and the preoperative hemodynamic state, as indicated by preoperative ASL, CBF detected by SPECT at rest, and CVR. Among 22 patients in Group A, the preoperative ASL signals exhibited an ipsilateral decrease in 15 patients, no decrease in 8, and a contralateral decrease in 1 with contralateral internal carotid artery occlusion. Preoperative SPECT imaging showed an ipsilateral decrease in the CBF in seven patients and no decrease in twelve. CVR impairment was observed on the ipsilateral side in 7 of 15 patients, and 1 patient demonstrated no CVR impairment.

In 6 patients in Group C, the preoperative ASL signals exhibited an ipsilateral decrease in 3 patients and no decrease in 3. Preoperative SPECT images showed an ipsilateral decrease in the CBF in 4 patients and no decrease in 2. CVR impairment was observed on the ipsilateral side in 5 of the 6 patients.

Conversely, in 15 patients with impaired ipsilateral CVR, increased ASL signals on POD1 were seen on both sides in 7 patients and unilaterally in 3. However, in the remaining 5 patients, increased ASL signals were not seen. In 5 patients without ipsilateral CVR impairment, increased ASL signals on POD1 were seen on both sides in 4 patients and unilaterally in 1. The lack of a correlation between the ASL signals on POD1 and CVR was statistically significant ($P = 0.2062$, Chi-square test using JMP Pro 10.0.2 [SAS Institute Inc., Cary, NC, USA]).

There was also no apparent correlation between ASL signals on POD1 and patency of the anterior communicating artery-A1 portion of the anterior cerebral artery (ACOM-A1) and the ipsilateral posterior communicating artery (PCOM) on MR angiography. The ACOM-A1 was not patent in only 2 patients in Group A. The ipsilateral PCOM was patent in 17 of 22 patients in Group A, 3 of 4 patients in Group B, and 3 of 6 patients in Group C.

**Development of cerebral hyperfusion syndrome**

Three out of 32 (9.4%) patients exhibited CHS. These 3 patients were Case 5 and Case 18 in Group A, and Case 23 in Group B. The detailed clinical courses and hemodynamic as well as EEG findings of Case 5 and Case 23 are described below as representative cases because the clinical profiles and EEG findings of Case 18 were quite similar to those of Case 5.

**Representative cases**

**Case 5 (Group A)**

An 83-year-old man presented with asymptomatic severe stenosis of the right ICA. He had a history of smoking, hypertension, hyperlipidaemia, aortic regurgitation, and chronic heart failure. His neurological examination revealed no remarkable findings. 3D-CTA revealed severe stenosis of the right ICA at the bifurcation of the common carotid artery [Figure 2a]. Preoperative ASL showed decreased signals in the right MCA territory [Figure 2b]. IMP-SPECT imaging at rest demonstrated mild CBF reduction in the right MCA territory [Figure 2c]. CVR impairment in the right anterior cerebral artery and MCA territory was noted with ACZ loading [Figure 2d].
On POD1 after CEA, the patient exhibited mildly restless confusion and a talkative state. Although no de novo ischemic events were observed on diffusion-weighted imaging [Figure 2e], ASL clearly showed increased ASL signals on both sides, especially the right [Figure 2f]. EEG on POD1 showed slow-wave activities in the bilateral frontal regions (Fp1, Fp2, F3, and F4 of International EEG 10-20 system) with poorly organized background activities. Asterisks indicate motion artefact due to restless confusion. On POD14, ASL showed disappearance of the increased signals. The preoperative decreased ASL signals in the right MCA territory were also improved. Postoperative 3D-CTA confirmed that the ICA stenosis was improved.

Figure 2: Case 5 (Group A). (a) Preoperative three-dimensional computed tomographic angiography (3D-CTA) revealed severe stenosis of the right internal carotid artery (ICA) at the bifurcation of the common carotid artery. (b) Preoperative magnetic resonance perfusion image with arterial spin labeling (ASL) showed decreased signals in the right middle cerebral artery (MCA) territory (white dotted arrows). (c) Single-photon emission computed tomography with N-isopropyl-[123I] iodoamphetamine at rest demonstrated mild reduction of cerebral blood flow in the right MCA territory (white dotted arrows). (d) With acetazolamide loading, impairment of cerebrovascular reserve in the right anterior cerebral artery (ACA) and MCA territories was noted (white dotted arrows). (e) On POD1, diffusion-weighted imaging failed to reveal any de novo ischemic lesions. (f) ASL on POD1 clearly showed increased signals in the bilateral ACA and MCA territories, especially on the right side (white arrows). (g) Electroencephalography on POD1 showed slow-wave activities in the bilateral frontal regions (Fp1, Fp2, F3, and F4 of International EEG 10-20 system, black lines) with poorly organized background activities. Asterisks indicate motion artefact due to restless confusion. (h) ASL on POD14 showed disappearance of the increased signals. The preoperative decreased ASL signals in the right MCA territory were also improved. (i) Postoperative 3D-CTA confirmed that the ICA stenosis was improved.
was fully resolved. ASL on POD14 showed disappearance of the ASL signal increase [Figure 2h]. The preoperatively decreased ASL signals in the right MCA territory were also improved. Furthermore, 3D-CTA confirmed that the ICA stenosis had improved [Figure 2i]. EEG on POD14 showed disappearance of slow waves in the bifrontal regions. The patient was discharged without neurologic deficits on POD16. His mRS score was 0 (unchanged from the preoperative state).

Case 23 (Group B)
A 46-year-old man presented with transient weakness of his right limbs. He had a history of smoking, hypertension, hyperlipidemia, diabetes mellitus, atrial fibrillation, and chronic heart failure. He had no abnormal neurological findings, although he had an old infarction in the white matter of the left frontal lobe. 3D-CTA revealed severe stenosis of the left ICA [Figure 3a]. Preoperative ASL showed decreased signals in the left MCA territory.
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In the present study, we measured ASL signals immediately after extubation on POD1 because general anesthesia using propofol was continued until the morning of POD1. Twenty-two (68.8%) and 4 (12.5%) of the 32 patients exhibited increased ASL signals bilaterally and on the operated side, respectively. Follow-up ASL showed improvement in these findings. The higher incidence of transiently increased ASL signals in our study is apparently attributed to the shortening of the ATT not only to the increased CBF. Immediately after CEA, improvement in anterograde ICA perfusion on the operated side and no need for slow streaming collaterals resulted in shortening of the ATT not only in the ipsilateral hemisphere but also in the contralateral hemisphere.

Previous authors have clearly demonstrated that patients with poor preoperative CVR have a potentially increased risk of post-CEA hyperperfusion. This is because the cerebral vessels are expanded and the cerebral blood volume is elevated in conditions of poor cerebrovascular reactivity, in which the cerebral vessels cannot constrict if the perfusion pressure is normalized by revascularization. Cerebral perfusion will rise as a result. In the present study, the absence of a correlation between preoperative CVR impairment and cerebral hyperperfusion (Groups A and B) may support the idea that ASL with a single PLD does not accurately reflect the CBF.

To overcome these shortcomings of ASL with a single PLD and to achieve quantitative measurements of CBF and ATT, a multiple-PLD ASL approach has been used. Wang et al. presented a multi-PLD ASL protocol using four PLDs of 1.5, 2.0, 2.5, and 3.0 s to achieve simultaneous measurements of CBF and ATT in a total scan time of 11.2 min in patients with moyamoya disease. In addition to the longer scan time, calculation of CBF and ATT requires special software and a workstation; it is not practical for clinical use. Uchihashi et al. demonstrated the usefulness of another ASL quantification approach named quantitative star labeling of arterial region (QUASAR) in the assessment of cerebral hyperperfusion. However, it also requires offline data processing with special software and a workstation. In contrast, the acquisition time of our single-PLD method was as short as 2 min 22 s, and a special calculation was not required. Although visual inspection was subjective, no assessment difference was noted with independent interpretations by two radiologists in the present study. Thus, post-CEA hemodynamic changes, including an increased CBF and shortening of the ATT, could be promptly estimated immediately after routine MR examination.

In the present study, 3 patients (Case 5 and Case 18 in Group A, Case 23 in Group B) developed CHS. In Case

DISCUSSION

Preoperative ASL signals with a single PLD of 1.5 s mostly coincided with the CBF map on resting SPECT, supporting the findings of previous reports. However, ASL had a tendency to underestimate the CBF probably because of the prolonged ATT in patients with carotid stenosis.

The incidence of post-CEA hyperperfusion detected with SPECT reportedly ranges from 0.5 to 40.0%. These contradictory results are probably attributed to different timing of post-CEA CBF measurements and the various quantitative criterion of hyperperfusion. Ogasawara et al. strictly defined post-CEA hyperperfusion as a ≥100% increase in CBF on IMP-SPECT compared with preoperative values and observed ipsilateral hyperperfusion immediately after CEA in 8 (15.6%) out of 51 patients.
CONCLUSION

In conclusion, owing to the effect of the ATT, ASL with a single PLD does not accurately reflect the CBF value and cannot identify patients at risk of CHS. However, ASL is completely noninvasive and can be performed as a part of routine MR imaging examination. Furthermore, ASL can promptly evaluate post-CEA hemodynamic changes, including both an increased CBF and shortening of the ATT. Although a more sophisticated ASL method should be explored, ASL with a single PLD may be used as screening test for cerebral hyperperfusion at present.

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Conflicts of interest

There are no conflicts of interest.

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