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Use of Oxygen and Lactate-Glucose Indices to Evaluate Neuronal and Glial Cell Bioenergetics in Patients with Occlusive-Stenotic Carotid Artery Lesions

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INTRODUCTION

Cerebral metabolism plays a critical role in neurological outcomes following carotid artery revascularization. Patients with occlusive-stenotic lesions are at risk of developing cerebral hyperperfusion syndrome (CHS), a condition linked to disrupted metabolic balance. This study explores the potential of oxygen-glucose and lactate-glucose indices as non-invasive markers to evaluate neuronal and glial bioenergetics. By assessing the interplay between aerobic and anaerobic pathways, these indices may help identify early metabolic shifts and guide clinical decision-making.

AIM

To assess the feasibility of using oxygen-glucose and lactate-glucose indices to identify cerebral metabolic changes and evaluate the balance between aerobic and anaerobic metabolism in patients with occlusive-stenotic carotid artery lesions undergoing revascularization, with a focus on detecting risk for cerebral hyperperfusion syndrome.

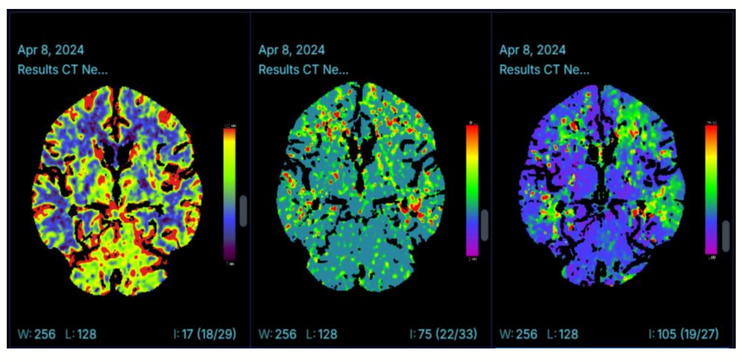

Fig.1. Preoperative brain CT perfusion data from the patient who did not develop CHS postoperatively indicated a CBF of 55.9 mL/100g/min, an MTT of 5.8 seconds, and a TTD of 6.2 seconds in the gray matter of the MCA territory. The $CMRO_2$/CBF ratio was 10.14, and the OEF value was 51.13 %.

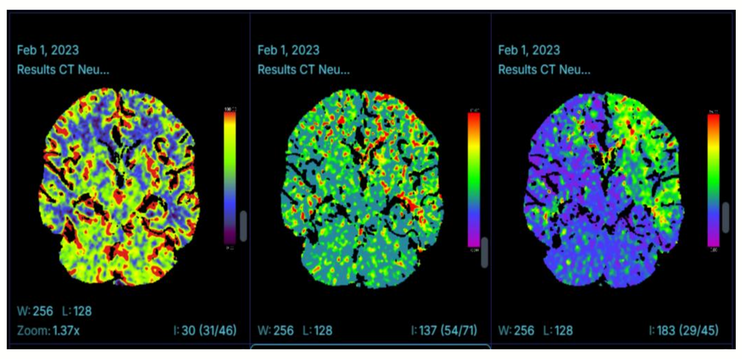

Fig.2. Preoperative brain CT perfusion data from the patient who developed CHS postoperatively indicated a CBF of 52.6 mL/100g/min, an MTT of 8.4 seconds, and a TTD of 8.7 seconds in the gray matter of the MCA territory. The $CMRO_2$/CBF ratio was 3.90, and the OEF value was 20.4 %.

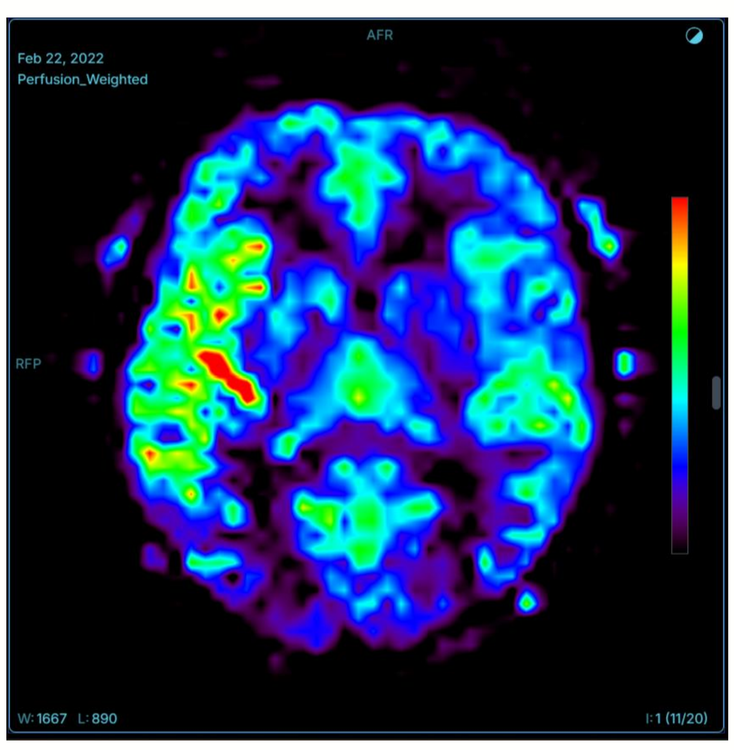

Fig.3. Postoperative pCASL MR perfusion results in a patient who developed cerebral hyperperfusion syndrome after carotid endarterectomy showed a mean CBF value of 83.7 mL/100g/min on the ipsilateral side of the operation. The $CMRO_2$/CBF ratio was 1.89, and the OEF was 12.86 %.

METHODS

A total of 125 patients (mean age: 62.3 ± 7.8 years) undergoing carotid artery revascularization were included. Among them, 9 developed cerebral hyperperfusion syndrome postoperatively. Arterial blood samples were collected from the radial artery and venous samples from the internal jugular vein. Immediately after sampling, lactate and glucose concentrations, along with blood gas parameters, were measured using the ABL800 FLEX analyzer (Radiometer Medical ApS, Denmark). Calculated indices included arterial (CaO_2) and venous (CvO_2) oxygen content, brain tissue carbon dioxide tension ($PbtCO_2$), oxygen extraction fraction (OEF), oxygen-glucose index (OGI), lactate-glucose index (LGI), and $CMRO_2$/CBF ratio. All measurements were taken prior to the main stage of revascularization. Cerebral oximetry was performed using a Nonin SenSmart® Model X-100 device (Nonin Medical, USA). Multispiral computed tomography was conducted using a dual-energy 384-slice CT scanner Siemens SOMATOM Force (Siemens Healthcare GmbH, Germany), which included native CT scans, brain CT perfusion, and CT angiography of the brachiocephalic arteries, following a standard scanning protocol. CT perfusion data were evaluated on the same slice by directly measuring quantitative perfusion parameters – CBF (Cerebral Blood Flow), CBV (Cerebral Blood Volume), MTT (Mean Transit Time), and TTD (Time to Drain) – in symmetrical regions of interest within each cerebral hemisphere. Magnetic resonance imaging (MRI) of the brain was conducted using a Siemens MAGNETOM Aera tomograph (Siemens Healthcare GmbH, Germany) with a magnetic field strength of 1.5 Tesla. Arterial spin labeling (ASL) perfusion imaging was performed using a pseudo-continuous three-dimensional (3D) ASL technique (pCASL). Post-processing of the 3D pCASL sequence data was carried out within a dedicated module of the digital intelligent ecosystem in the vascular surgery research laboratory. Statistical analysis was performed using R (version 4.4.2).

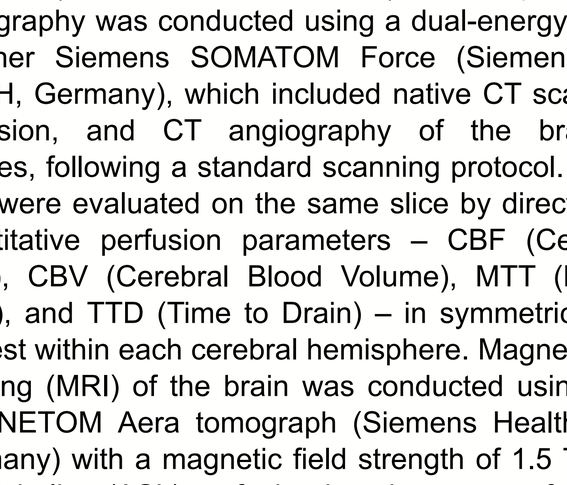

Fig.4. Box plot diagram of the $CMRO_2$/CBF ratio in CHS and non-CHS groups prior to the main stage of revascularization.

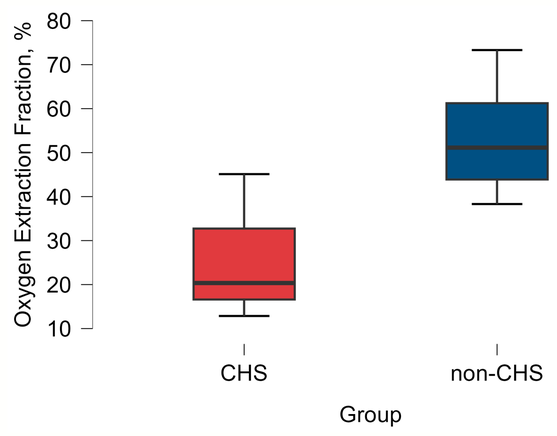

Fig.5. Box plot diagram of the OEF value in CHS and non-CHS groups prior to the main stage of revascularization.

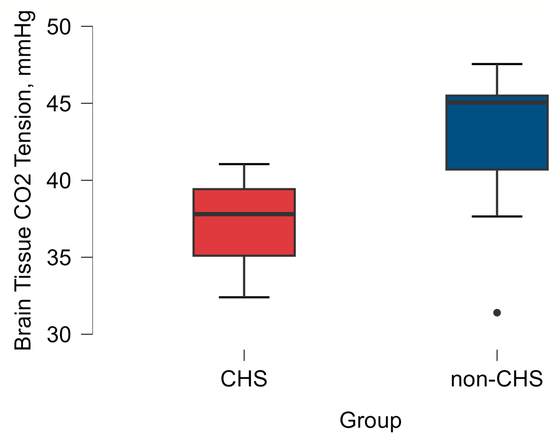

Fig.6. Box plot diagram of brain tissue carbon dioxide tension in CHS and non-CHS groups prior to the main stage of revascularization.

RESULTS

CHS group had lower $CMRO_2$/CBF values (Me 3.91 [2.90–5.20]) compared to non-CHS (Me 9.11 [8.31–10.14], p = 0.004), indicating impaired oxygen metabolism. Reduced OEF in CHS patients (Me 20.35% [14.73–38.92]) vs. non-CHS (Me 51.13% [43.89–61.23], p = 0.006), suggesting diminished oxygen extraction. Markedly lower OGI in CHS (Me 1.22 [–1.76 to –1.42]) vs. non-CHS (Me 20.27 [10.12–33.58], p < 0.001), consistent with a shift toward anaerobic glycolysis. Elevated LGI in CHS (Me 0.31 [0.07–0.41]) vs. non-CHS (Me –0.25 [–0.67 to 0.33], p = 0.088), indicating increased lactate uptake.

Other parameters such as CaO_2, CvO_2, and $PbtCO_2$ showed trends but did not reach statistical significance.

Cerebral oximetry values in both the ipsilateral (ΔrSO_2 = 6 %, p = 0.032) and contralateral (ΔrSO_2 = 7 %, p = 0.011) hemispheres of the brain were statistically significantly lower in patients with cerebral hyperperfusion syndrome compared to the control group.

Analysis of brain CT perfusion data revealed that, prior to revascularization, patients with cerebral hyperperfusion syndrome exhibited significant differences in cerebral circulation parameters compared to the control group. The most pronounced changes were observed in the temporal lobe of the ipsilateral hemisphere, where increases in cerebral blood volume and average blood transit time were noted, along with alterations in cerebral blood flow.

Following revascularization, these differences became even more marked. Patients with cerebral hyperperfusion syndrome demonstrated a further rise in cerebral blood volume and cerebral blood flow, accompanied by a significant reduction in average blood transit time, indicating the progression of severe hyperperfusion. No statistically significant differences were found between the groups in other brain regions.

At this stage of the study, no statistically significant correlation was found between the perfusion parameter values and the examined cerebral metabolism parameters.

CONCLUSIONS

The oxygen-glucose index and lactate-glucose index are promising markers for evaluating cerebral metabolic status in patients with carotid artery stenosis. OGI values below 6 suggest a predominance of anaerobic glycolysis, while positive LGI values reflect active lactate uptake by brain tissue. These indices may aid in identifying patients at risk for CHS and other metabolic disturbances following revascularization. Further research is warranted to validate these findings and explore their prognostic and therapeutic implications.

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