Limitation of Near Infrared Spectroscopy Cerebral Oximetry in Predicting Neurologic Intolerance during Carotid Artery Stenting with Proximal Protection

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Proximal balloon occlusion prior to carotid artery stenting is considered a relatively safe practice during endovascular treatment of carotid artery stenosis. Transient neurological deterioration affecting the ipsilateral hemisphere is seen soon after balloon inflation, when placed proximal to the stenotic segment.[1] This occurs in cases of bilateral carotid disease due to insufficient collateral blood flow from the contralateral side.[2] Near-infrared spectroscopy cerebral oximetry (NIRS) is a valuable tool in detecting hypoperfusion-induced cerebral tissue desaturation (rSO_2) during these procedures. This helps the interventional radiologist to deflate the balloon at the earliest to reestablish the cerebral blood flow.[3]

We present the case of a 63-year-old male patient who presented with recurrent transient ischemic attacks in the form of slurred speech and right upper and lower limbs weakness. Informed consent was taken from the patient prior to publishing this report. Magnetic resonance imaging revealed 90 and 40% stenosis of the left and right carotid arteries, respectively. The patient was on telmisartan 40 mg for hypertension, which was omitted for 2 days prior to the procedure. Preprocedure electrocardiogram (ECG) was normal and echocardiography showed normal systolic function and grade I diastolic dysfunction. He was prescribed tablets aspirin 150 mg and clopidogrel 75 mg daily for 5 days prior to left carotid artery stenting, scheduled under monitored anesthesia care. Baseline rSO_2 recorded bi-frontally were 63% on the affected side (left) and 62% on the contralateral side, which remained unchanged after O_2 supplementation via facemask. ECG, pulse oximetry, end-tidal carbon dioxide, and noninvasive blood pressure were monitored throughout the procedure. Fentanyl 50 µg was administered during femoral artery puncture and frequent neurological examination was performed during the procedure. Angiography revealed almost 80% stenosis of the contralateral internal carotid artery, which was underestimated on the MRI (►**Fig. 1**). Faint opacification of the left anterior cerebral artery was seen after contrast injection via right internal carotid artery, suggestive of minimal collateral flow. Nimodipine 2 mg was prophylactically injected slowly intra-arterially prior to balloon inflation into the left common carotid artery by the radiologist. The blood pressure was maintained with boluses of mephentermine.

The patient was cooperative until the balloon was inflated proximal to the stenotic segment, when he suddenly developed restlessness and aphasia. Simultaneously, progressively increasing ST segment depression up to –2.3 mm was noted, raising the possibility of myocardial ischemia. There was a concomitant fall in mean arterial pressure from 178/69 (mean 96) mm Hg to 124/38 (mean 59) mm Hg. The balloon was deflated immediately. The patient soon developed contralateral hemiplegia. As the blood pressure did not respond to intravenous mephentermine boluses, phenylephrine infusion was started to keep the mean blood pressure of at least 65 mm Hg. In the meantime, dexmedetomidine infusion was started to control patient's restlessness as the heart rate was stable and frequent neurological examination was required. In spite of these hemodynamic and neurological changes, the rSO_2 remained stable at above 60% throughout the event. The hemiplegia and aphasia started resolving after approximately 15 minutes after the event. A distal embolic protection device was then deployed and the stent was placed directly over the stenotic segment and a balloon was inflated within the stent lumen. No significant hemodynamic changes were noted at this time. The ST segment changes gradually resolved over

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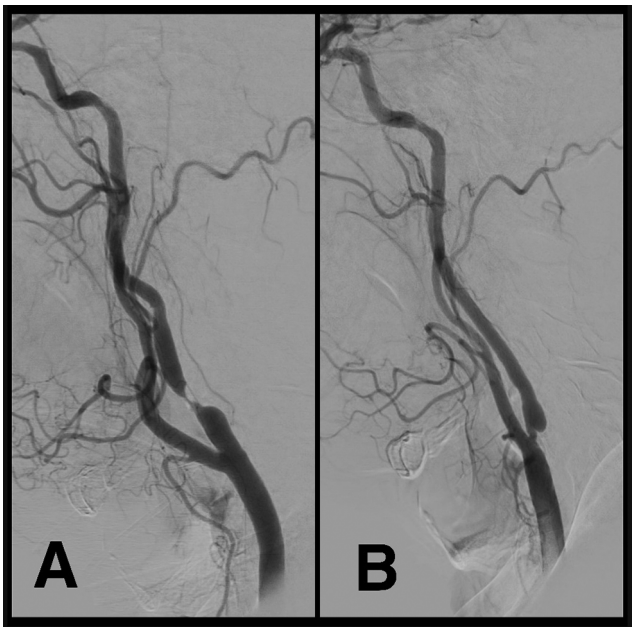

Fig.1 (**A**) Digital subtraction angiography of the left carotid artery demonstrating more than 90% stenosis of the internal carotid artery (symptomatic). (**B**) Around 80% stenosis seen in the right internal carotid artery.

the next 45 minutes and corresponded with the complete neurological recovery.

The noninvasive nature and continuous real-time interpretation make NIRS an attractive adjunct in the neuroanesthesiolgist's armamentarium for monitoring cerebral ischemia. However, significant contribution from chromophores in the extra-cerebral tissues and external carotid artery circulation can limit its sensitivity during occlusion of the internal carotid artery.[4] In our case, it did not reflect brain ischemia during hypotension and when the neurological symptoms were obvious. The ST segment depression and hypotension, probably secondary to sudden cerebral ischemia and myocardial dysfunction, confused the clinical picture.[5] The patient made neurological recovery after balloon deflation at a lower blood pressure than baseline, suggesting that the neurological symptoms were due to balloon occlusion causing total occlusion of the blood flow and not from a hemodynamic etiology. General anesthesia, if adopted, for conduct of carotid artery stenting, which the interventional radiologist requested for as this patient became restless, would have masked the occlusion intolerance and resulted in a periprocedural stroke. The sensitivity of cerebral oximetry has been reported to be modest when compared with awake testing.[6] Commonly available cerebral oximetry sensors placed over the frontal region do not cover the parietal lobe where ischemia is likely to occur during occlusion of the carotid artery.[7] In such scenarios, it has been shown that multichannel NIRS has a better sensitivity in detecting cerebral ischemia.[8]

This case report highlights the importance of frequent neurological examination during carotid stenting as rSO2 values might not always suggest cerebral ischemia.

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Conflict of Interest

None declared.

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