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Successful treatment of cerebral hyperperfusion syndrome after secondary carotid artery reconstruction in a patient with Marfan syndrome: a case report

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INTRODUCTION

Cerebral hyperperfusion syndrome (CHS) is a rare but serious complication that can occur following carotid revascularization procedures.

AIM

To report a clinical case of the successful treatment of CHS using a combination of candesartan, carvedilol, and citicoline in a young patient with Marfan syndrome.

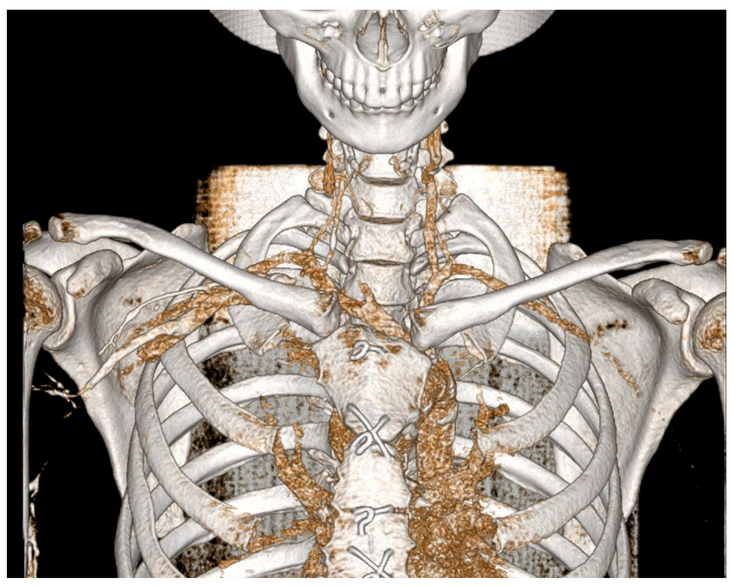

Fig.1. A 3D reconstruction of the patient's CTA scan.

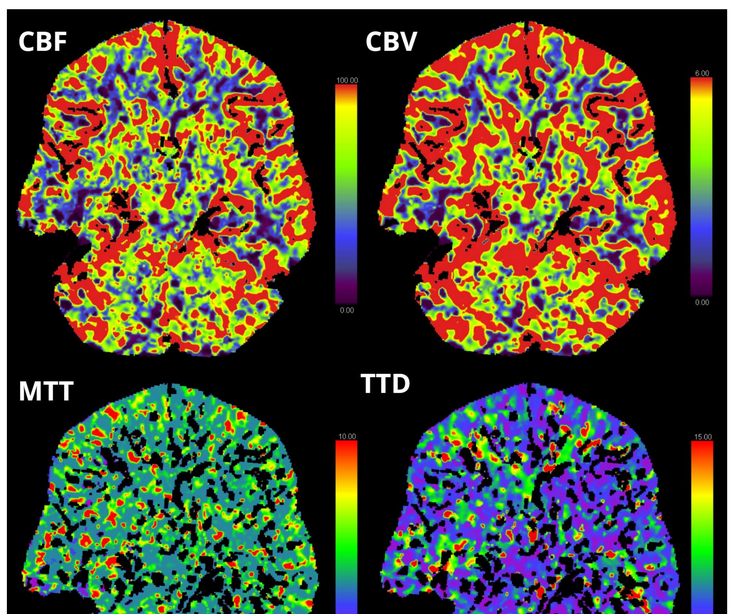

Fig.2. Preoperative brain CT perfusion data indicated a CBV of 3.5 mL/100g, CBF of 32.6 mL/100g/min, and MTT of 6.8 seconds in white matter; and a CBV of 5.7 mL/100g, CBF of 75.6 mL/100g/min, and MTT of 6.1 seconds in gray matter of the MCA territory.

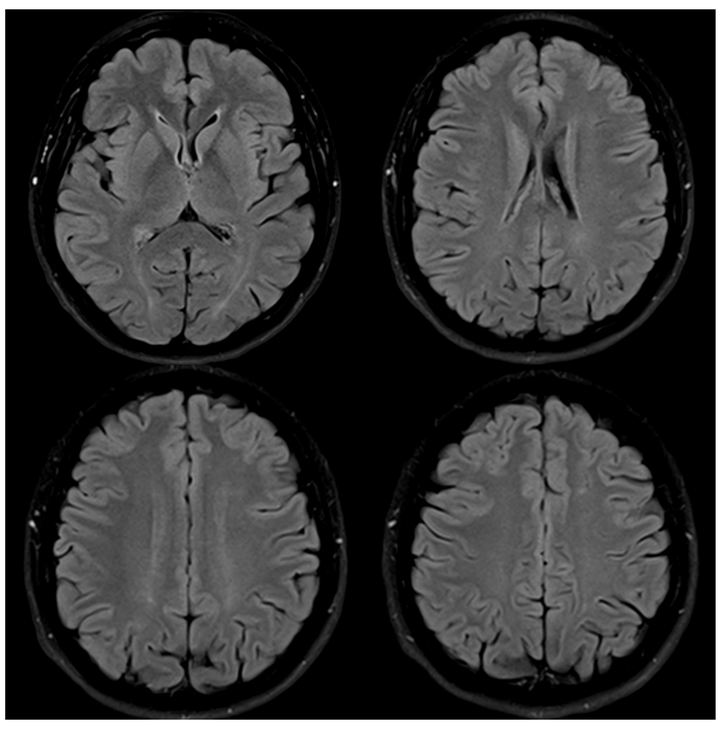

Fig.3. Results of preoperative brain MRI. The MRI showed punctate white matter hyperintensities on T2w-FLAIR images, particularly in regions corresponding to the carotid artery territory, indicating mild ischemic changes.

METHODS

A 40-year-old female with Marfan syndrome was admitted to the hospital, complaining of dizziness, fatigue, and frequent episodes of unconsciousness. She has a history of ascending aortic aneurysm resection and aorto-bicarotid graft placement in 2005. Recent imaging showed thrombosis of both graft branches, requiring reoperation on the right common carotid artery using a Vascutek Gelweave vascular graft.

Transcranial dopplerography (TCD) was performed to measure the peak systolic (PSV) and the volumetric (VBF) blood flow velocities in the middle cerebral artery (MCA). Concurrently, brain CT perfusion imaging was performed to evaluate cerebral blood flow (CBF), cerebral blood volume (CBV), mean transit time (MTT), and time to drain (TTD). MRI scans were obtained on a 1.5T MR machine, including T2, T2-weighted FLAIR (T2w-FLAIR), diffusion-weighted imaging (DWI), T1, susceptibility-weighted imaging (SWI), and 3D time-of-flight magnetic resonance angiography (3D TOF MRA). To evaluate oxidative stress, blood samples were collected from the internal jugular vein to measure total oxidative status (FORT – Free Oxygen Radical Testing) preoperatively and 24 hours post-surgery.

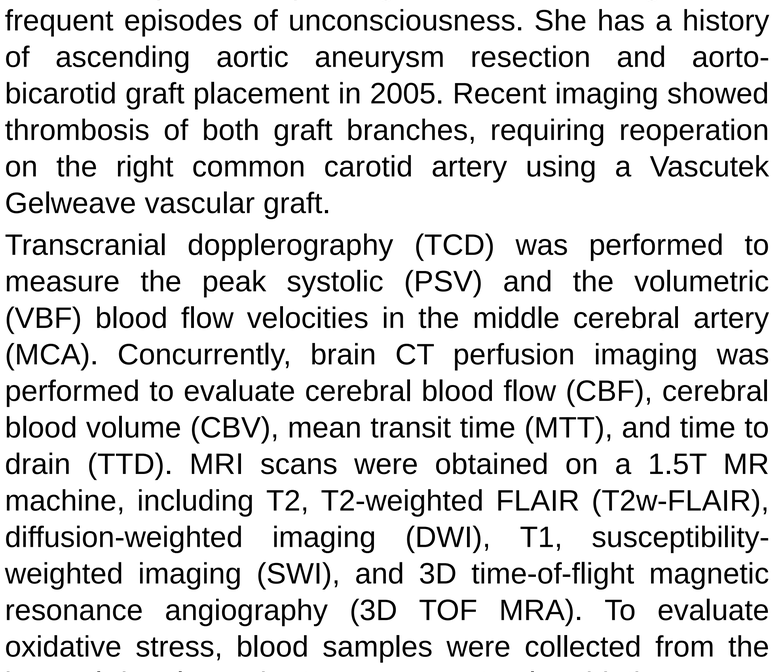

Fig.4. Results of the brain MRI on the 7th day after surgery show no signs of ischemia or hemorrhage as a cause of the neurological symptoms that developed.

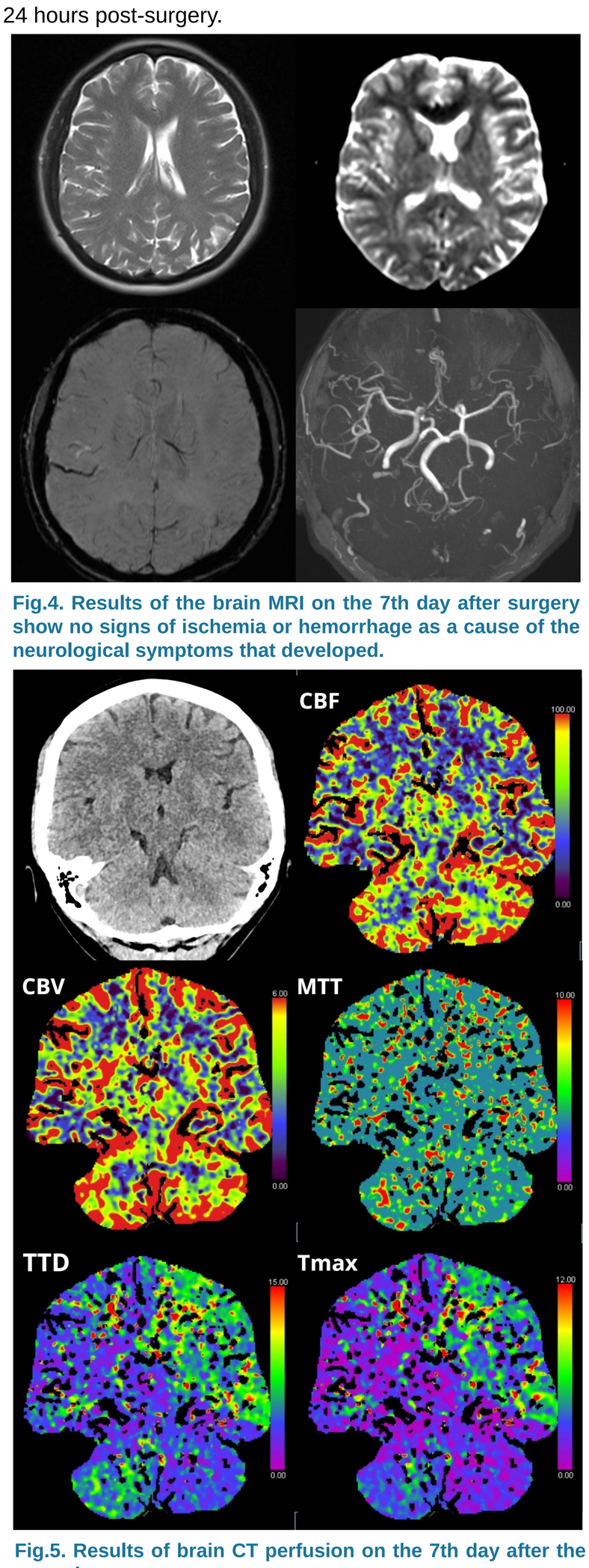

Fig.5. Results of brain CT perfusion on the 7th day after the operation.

RESULTS

Preoperative brain CT perfusion data indicated a CBV of 3.5 mL/100g, CBF of 32.6 mL/100g/min, and MTT of 6.8 seconds in white matter; and a CBV of 5.7 mL/100g, CBF of 75.6 mL/100g/min, and MTT of 6.1 seconds in gray matter of the MCA territory.

The minimum values of cerebral oximetry during surgery were 51% on the right and 63% on the left. After restoration of blood flow through the prosthesis and carotid arteries, there was an increase in cerebral oximetry values: on the right to 90% and on the left to 68%. Additionally, there was an increase in blood pressure to 180/100 mmHg, which required partial compression of the prosthesis and controlled hypotension using urapidil.

Postoperatively, on 5th day, a patient presented with neurological symptoms including severe headaches, altered mental status, and left-sided weakness. At the same time, the blood pressure was 190/110 mmHg. The patient was prescribed candesartan, carvedilol, and citicoline.

The patient demonstrated an increase in total oxidative status, with FORT values rising from 380 U to 452 U within 24 hours post-surgery. The follow-up brain CT perfusion study, performed 24 hours after surgery, demonstrated an increase in CBF to 64.7 mL/100g/min, an increase in CBV to 4.5 mL/100g, and a decrease in MTT to 3.4 seconds in the white matter of the MCA territory. Similar values of perfusion parameters were also observed on the 7th day after surgery.

TCD data revealed a post-surgery PSV of 193 cm/s (compared to 47 cm/s before surgery) and a VBF of 1690 mL/min (compared to 342 mL/min before surgery) in the right MCA, supporting the diagnosis of cerebral hyperperfusion syndrome.

Despite the high values of blood flow velocity and volume according to transcranial dopplerography, as well as obvious signs of CHS demonstrated by CT perfusion and MRI of the brain, there were no signs of subarachnoid or intracerebral hemorrhages, which are classic manifestations of cerebral hyperperfusion syndrome. There were no signs of microbleeds on the SWI sequence or ischemia on the DWI. There was an expansion of the cortical veins on SWI, which is also an indirect sign of cerebral hyperperfusion syndrome.

Following the diagnosis, the patient was treated with blood pressure management and neuroprotection, and was closely monitored. This resulted in a gradual resolution of symptoms and normalization of both TCD and imaging parameters on follow-up evaluations.

CONCLUSIONS

This case emphasizes the importance of transcranial dopplerography as a valuable tool for the early diagnosis of cerebral hyperperfusion syndrome after carotid revascularization. The integration of brain CT perfusion, along with MRI and TCD blood flow measurements, alongside oxidative stress assessments, provides a comprehensive understanding of the evolving cerebral hemodynamics in these patients.

BIBLIOGRAPHY

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