Intraoperative perfusion computerized tomography scanning for management of intracranial stent placement in a patient with tandem intracranial stenoses

Case Report

MATTHEW F. SANFORD, M.D., AQUILA S. TURK, D.O., DAVID B. NIEMANN, M.D., KARI A. PULFER, B.S., R.R.T., AND BEVERLY A. AGAARD-KIENITZ, M.D.

Departments of Radiology and Neurosurgery, University of Wisconsin Hospital and Clinics, Madison, Wisconsin

The authors describe the novel use of cerebral perfusion computerized tomography studies to evaluate the effectiveness of internal carotid artery stent placement in a man with symptomatic transient ischemic attacks caused by tandem stenoses of the internal carotid and middle cerebral arteries.

KEY WORDS • perfusion computerized tomography • intracranial stenosis • tandem stenoses • intracranial stent

Effective surgical management of tandem stenosis of the ICA system requires that the role of each lesion in the patient’s symptoms is accurately assessed. The degrees of stenosis in lesions affecting the extracranial ICA, as opposed to the intracranial ICA, may have a different significance because of differences in the size of the artery lumen. Treating both stenoses increases the risks of the procedure, whereas treating only one stenosis risks a suboptimal outcome. Clinical improvement cannot be assessed intraoperatively due to the use of general anesthesia. Whereas treating the most stenotic lesion seems indicated by anatomical studies, no reports have detailed a physiological approach to lesion stratification.

Cerebral perfusion CT scanning has been applied in the evaluation of acute stroke, vasospasm, and cerebral reserve. Atherosclerosis, when significant, is a systemic disease affecting the cervicocerebral circulation that is well suited to evaluation by CT angiography and cerebral perfusion CT studies. We hypothesized that intraoperative perfusion CT scanning would prove useful in tailoring treatment of tandem stenoses of the ICA. The goals of this report are to describe the intraoperative technique we used in a patient, suggest suitable criteria for performing additional revascularization, and demonstrate that a perfusion CT study was able to predict the patient’s postoperative outcome accurately.

Case Report

History. This 68-year-old right-handed man presented with innumerable daily episodes of extremity shaking, immediately preceded by an aura. These frequent spells consisted of jerking of the right extremities and were thought to represent limb-shaking TIAs. During the previous 6 months the patient had noted that these spells were becoming more frequent and that he had been experiencing progressive difficulty with walking. His gait disturbance was further complicated by a history of severe peripheral vascular disease, which had required placement of a left-to-right femoral–femoral artery bypass graft.

Examination. The patient walked with a limp, favoring his right leg. There was an asymmetrical weakness of the right extremities and pronounced muscle atrophy of the right leg. The patient exhibited memory impairment, confusion, and difficulty with concentrating. A diffusion-weighted magnetic resonance image revealed multiple small infarctions within the subcortical and border zone regions of the MCA and ACA vascular territories. The patient was referred to our facility to be evaluated for potential endovascular therapy.

At the time of the initial consultation a cerebral perfusion CT study was performed in conjunction with CT angiography (Lightspeed; General Electric Medical Systems, Milwaukee, WI). The CT angiograms revealed at least a 60 to 70% stenosis of the left supraclinoid CA and a tandem stenosis of the ipsilateral MCA, which was estimated to be at least 50%. An assessment of cerebral perfusion (Advantage Windows; General Electric Medical Systems) indicated an increased MTT and CBV with a decreased CBF, indicators of failed autoregulation of the affected vascular territories (Fig. 1).

Diagnostic Angiography, Treatment, and Intraprocedure Perfusion CT Scanning. The patient was admitted to the hospital and diagnostic angiography was performed using a standard angiographic technique combined with general anesthesia with the intent to intervene. Successive surgical
repairs of each stenosis were anticipated until a normal MTT could be achieved. The plan was to repair the proximal, more stenotic lesion first and then, if necessary, the distal stenosis. Conventional angiography confirmed the CT angiography findings of tandem lesions in vessels supplying the left hemisphere. The stenosis involving the supraclinoid CA was deemed to be approximately 80 to 90%, whereas the left M1 stenosis was estimated to be 60 to 70% (Fig. 2 left). There was no sign of the left A1 segment. A right CA angiogram demonstrated filling of both A2 segments, but with a significant delay in the filling of the left A2 across the ACoA (Fig. 2 right).

A No. 6 French Shuttle Select guide sheath (Cook Inc., Bloomington, IN) was advanced into the left cervical ICA. Five milligrams of Verapamil was injected through the guide catheter as a prophylaxis for vasospasm. A Luge wire (Boston Scientific, Freemont, CA) was used to cross the lesions and a 3 × 9-mm Driver stent (Medtronic, Inc., Minneapolis, MN) was successfully placed across the site of the ICA stenosis with the balloon inflated to 10 atm. There was no residual stenosis remaining (Fig. 2 left). The tandem MCA stenosis persisted without change. The effect of the intervention on the patient’s symptoms was unknown due to the general anesthesia. He remained intubated and a sheath placed in the groin was maintained. He was brought from the interventional radiology suite to the CT scanning suite and an immediate cerebral perfusion study was performed.

The intraprocedure perfusion CT study revealed a marked improvement in the MTT in the left MCA with only a mild persistent delay relative to the right hemisphere. The patient’s CBV remained elevated. There was a pronounced improvement in his CBF with luxury perfusion in the MCA territory, compared with that observed on the preintervention study (Fig. 1). A persistent delay in the MTT and a decrease in CBF were noted in the left ACA distribution; this is related to a flow insufficiency in the ACoA.

Postprocedure Course. Given the increased risks associated with MCA angioplasty or stent implantation, and the improvements in cerebral perfusion that now appeared to be within the range of the patient’s ability to autoregulate, the procedure was terminated. The patient was transferred to the neurosurgical intensive care unit. The patient’s postprocedure course was uneventful. At discharge 5 days after admission he exhibited a pronounced increase in strength in the right lower extremity and improvements in the TIAs that had affected his upper and lower extremities, more so in the upper extremity.

Five weeks after the intervention the patient was seen in the outpatient clinic and was noted to have a marked improvement in memory, concentration, and cognition. There was a considerable decrease in spasticity of the right lower extremity and his limp was less pronounced. The patient was able to walk without a cane. At a later examination 12 weeks posttherapy, the patient was able to walk with a normal gait. His upper-extremity limb-shaking TIAs had resolved and he experienced only two or three lower-extremity events per week after his systolic blood pressure had increased to a targeted range of 130 to 140 mm Hg.
The limited available data on the natural history of intracranial stenosis indicate that intracranial atherosclerotic stenoses are a dynamic process involving lesion progression and regression. Recurrent annual stroke rates from intracranial stenosis range from 4 to 12% and, according to one report, up to 55% per year for patients in whom antithrombotic therapy has failed. The clinical and hemodynamic effects of tandem stenoses are poorly understood and the prevalence of tandem stenoses is variable. In their retrospective review of 672 patients with greater than 70% stenosis of the CA bifurcation, Rouleau, et al., determined that 5.5% of their patients had ipsilateral stenosis of the CA siphon greater than 50%. Similarly, the same patient subset with high-grade stenoses of the CA bifurcation have been found to harbor tandem lesions of the proximal intracranial circulation (A1, M1, and P1 segments) in 2.2%. Mathematical models of tandem stenoses developed using the Poiseuille law indicate that the flow-limiting component results from the greater of the two constituents. Nevertheless, that paper and the existing literature on tandem stenoses focus on proximal extracranial and distal extracranial or intracranial lesions. There is no literature focusing on management of tandem intracranial lesions or using an intraoperative physiological approach to treatment. Given the different risk profile associated with treating intracranial stenosis as compared with extracranial stenosis, we hypothesized that intraoperative perfusion is a safer mechanism for determining an endovascular surgical endpoint.

In our case we had a patient with an atypical TIA presentation related to hypoperfusion from tandem intracranial stenoses in his dominant hemisphere. We treated the proximal supraclinoid CA stenosis first because it was a more significant lesion. The remaining question was the significance of a residual 60 to 70% MCA stenosis. We decided not to treat the MCA stenosis because of a marked improvement in CBF and a luxury perfusion in the left MCA region, indicating that our intervention had led to renewal of the patient’s capacity to autoregulate. There were persistent depressed perfusion parameters in the ACA territory, but this was believed to be related to an ACoA flow insufficiency, given the absence of the left A1 segment. The patient experienced a significant clinical improvement with symptoms only referable to the untreated ACA territory, which also improved through increased collateral flow.

In patients with tandem stenoses it is imperative that their symptoms be attributable to hypoperfusion and not thromboembolic disease before intervention is investigated. Next, the causative lesion should be defined. In pursuit perfusion CT scanning provides information about the cerebral vascular reserve and helps the clinician discern if the hypoperfusion correlates to the vascular territory of the stenosis, thereby directing surgical and endovascular therapy in patients with complex vascular lesions. As multiplatform approaches coupling magnetic resonance imaging and CT technologies to fluoroscopy become more prevalent, we will likely switch our conventional anatomical approach of stenosis to a more physiological perfusion-driven approach.

**Conclusions**

We treated a patient with ischemic symptoms caused by tandem intracranial stenoses resulting in cerebral hypoperfusion. Cerebral perfusion CT scanning was valuable for documenting perfusion changes in our patient and is proving to be a valuable tool in preoperative assessment and postintervention follow up. As cross-platform modalities in-

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**Fig. 2.** Left: Anteroposterior (A and C) and lateral (B and D) arteriograms obtained using a left common CA injection before (A and B) and after (C and D) stent placement. Arrowheads depict critical supraclinoid CA stenosis. Arrows indicate 60 to 70% MCA (M1) stenosis. Right: Anteroposterior angiogram obtained using a right common CA injection demonstrating normal filling of the right intracranial circulation and delayed cross-filling of the left ACA through a stenotic ACoA.
Incorporating CT and magnetic resonance imaging with fluoroscopy become more available, it is likely that we will adopt a more physiological than anatomical approach to cerebral vascular occlusive disease processes.

References


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Address reprint requests to: Aquilla S. Turk, D.O., Department of Radiology, University of Wisconsin Hospital and Clinics, 600 Highland Avenue, CSC E3/372, Madison, Wisconsin 53792.