Transient cortical blindness after thoracic endovascular aneurysm repair

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We report a patient who presented with transient cortical blindness 12 hours after completion of a thoracic endovascular aneurysm repair. Computed tomography of the brain demonstrated no acute findings. The patient’s symptoms resolved spontaneously after 72 hours. To our knowledge, this is the first report of transient cortical blindness after endovascular aortic aneurysm repair. This is an uncommon diagnosis that is important to recognize in a modern vascular surgery practice. (J Vasc Surg 2011;53:1405-8.)

Transient cortical blindness is a rare complication associated with angiography. We report a patient who presented with transient cortical blindness 12 hours after completion of a thoracic endovascular aneurysm repair. Computed tomography (CT) of the brain demonstrated no acute findings. The patient’s symptoms resolved spontaneously after 72 hours. To our knowledge, this is the first report of transient cortical blindness after endovascular aortic aneurysm repair.

CASE REPORT

A 65-year-old man presented with complaints of acute-onset abdominal pain. His history was significant for the open repair of an infrarenal abdominal aortic aneurysm with an aortobifemoral bypass graft placement 10 years prior. The left limb of the bypass was chronically occluded, and the patient did not have claudication. The patient was administered intravenous hydration and his pain resolved. A CT scan demonstrated an 11-cm thoracic aortic aneurysm arising just distal to the left subclavian artery (Fig 1). No other pathology was found. Thoracic endovascular aortic aneurysm repair with coverage of the left subclavian artery orifice and concomitant carotid-to-subclavian bypass (CSB) was planned.

A spinal drain was placed before induction of general anesthesia for protection from spinal cord ischemia. The patient was neurologically intact after placement of the drain and before induction of anesthesia. During the operation, the patient was systemically anticoagulated with intravenous heparin to maintain an activated clotting time of >300 seconds.

The procedure began with creation of the left CSB through a supraclavicular incision. The subclavian artery was clamped distal to the origin of the left vertebral artery. This was performed with an 8-mm polytetrafluoroethylene graft. Electroencephalographic monitoring during carotid cross-clamping did not show any changes during the case concerning for ischemic insult.

The right limb of the aortobifemoral graft was then accessed through a groin incision for a thoracic endograft deployment. Arterial access was obtained by puncturing the CSB graft with a 9F sheath directed toward the subclavian artery, and diagnostic aortography was performed, which confirmed the CT findings. A Zenith TX2 thoracic endograft (Cook Inc, Bloomington, Ind) was deployed at the edge of the left common carotid artery. Balloon angioplasty was performed, followed by arch angiography through the femoral access that demonstrated patency of the left common carotid artery as well as the CSB. Subclavian angiography was then performed through the 9F sheath in the CSB, and an occluder device (Amplatzer Vascular Plug; AGA Medical Corporation, Plymouth, Minn) was placed in the proximal subclavian artery to prevent retrograde perfusion of the aneurysm sac. Completion subclavian angiography showed the occluder device was in good position and the left vertebral artery was widely patent (Fig 2). A total of 195 mL of ioversol (Optiray; Mallinckrodt Medical, Las Vegas, NV) was used during the procedure, which lasted 5 hours and 44 minutes.

The patient awoke from anesthesia at the end of the procedure neurologically intact. His vision was normal bilaterally and he had no motor or sensory deficits. His mean blood pressure was maintained >70 mm Hg for spinal cord protection. At 12 hours after the procedure, the patient reported sudden, almost complete loss of vision bilaterally, followed by mental status changes. Neurologic examination demonstrated intact extraocular movements and normal pupillary reflexes. The fundoscopic examination was normal. A CT of the brain showed no acute hemorrhage or ischemic infarction (Fig 3). CT angiography of the head and neck showed patency of the extracranial and intracranial vertebrobasilar system, as well as a patent CSB graft. The posterior circulation was intact, with codominant vertebral arteries.

A formal neurologic consultation was obtained. With no evidence of ischemia or embolization on imaging, a diagnosis of transient cortical blindness was presumed. The patient was medically managed with intravenous hydration and maintenance of his mean arterial pressure >70 mm Hg to maintain adequate spinal and cerebral perfusion but <80 mm Hg to avoid significant
cerebral hypertension. After 72 hours, his bilateral visual symptoms completely resolved.

His postoperative course was additionally significant for transient lower extremity weakness that resolved with continued spinal drainage and raising the mean blood pressure to 80 to 90 mm Hg. He was discharged home on postoperative day 7 with a normal neurologic examination. At the 1-month follow-up, he continued to have no visual or other neurologic problems and a CT scan of the chest demonstrated no endoleak, graft migration, or aneurysm enlargement.

DISCUSSION

The development of transient cortical blindness (TCB) is a rare complication after angiography. It has been reported in several types of diagnostic procedures using contrast, including aortography, coronary angiography, and imaging of all of supraaortic vessels.1-4 In cerebral and vertebral angiography, TCB has been demonstrated to occur at a rate of 0.3% to 1.0%.2,5-7 This rate is much lower for other types of angiography.8,9 Diagnosis of TCB is made by clinical examination and imaging evaluation to rule out other pathologies. TCB is characterized by unilateral or bilateral vision loss despite a normal ophthalmic examination (with normal fundi, pupillary reflexes, and extraocular movements). The onset of TCB from angiography occurs from minutes to hours after the procedure and can be accompanied by other symptoms such as seizures, headaches, mental status change, memory loss, and blindness denial (an apparent lack of concern by the patient for their complete loss of vision).

CT of the brain can demonstrate extravasation of contrast in the posterior and occipital areas that may resolve in serial imaging. However, TCB can also result in a normal CT scan. CT angiography may be helpful in ruling out evidence of embolization or stroke as well as ensuring that the cerebral circulation is intact. Magnetic resonance imaging (MRI) may show T2 and fluid attenuation
The etiology of TCB remains unclear, but several hypotheses have been proposed. The most common postulation is that TCB is caused by an adverse reaction to the penetration of contrast material across the blood–brain barrier. Some researchers have demonstrated that hyperosmolar agents may induce vasodilation, shrinkage of cerebrovascular endothelial cells, and widened endothelial tight junctions. The development of TCB has been associated with both ionic and nonionic contrast and is not dose-related. Chronic hypertension has been linked to osmotic disruption of the blood–brain barrier and may be a contributing factor.

The optimal management of TCB also remains unclear. Although there have been reports of steroid use, anticoagulation, and intravenous hydration for treatment of these patients, there is no evidence that there is any quicker resolution of symptoms with these modalities compared to watchful waiting. This is typically a self-limiting entity and resolves within 4 to 5 days.

TCB may also be related to posterior reversible leukoencephalopathy (PRLE) syndrome, which presents with neurologic deficits, including visual loss, and is associated with reversible edema in the posterior circulation upon imaging. PRLE is usually related to chronic hypertension and transplant immunosuppression and has MRI changes in the posterior circulation similar to that of contrast-induced TCB.

The number of endoluminal procedures performed by vascular surgeons has grown dramatically during the past 2 decades. TCB has not been a widely acknowledged complication in the vascular surgery literature. To our knowledge, this is the first reported case of transient cortical blindness associated with endovascular aortic aneurysm repair. Our patient had both aortic and subclavian angiography and induced hypertension. The CT angiographic images obtained after the presentation of blindness did not show any contrast pooling in the occipital visual areas, as has been described in other reports. Our patient did have full return of his vision and his mental faculties without any sequelae.

The point of our procedure that led to the TCB in our patient is unclear, but the likely cause was contrast from the angiography entering the posterior circulation. Other potential causes of this patient’s cortical blindness may have been embolization to the posterior circulation or placement of the spinal drain. CT angiography showed an intact posterior circulation with no obvious infarct. A delayed CT scan might have shown some posterior brain changes; however, because the patient’s symptoms improved, it did not seem warranted. MRI might have been more sensitive for ischemic events but was not thought to be necessary given the patient’s presentation. Lumbar procedures have been associated with TCB, but these are most commonly associated with injection procedures, not placement of a spinal drain. They also have retinal hemorrhage as a clinical sign. TCB in lumbar procedures tend to present immediately after injection and not in a delayed fashion.

CONCLUSION

The dramatic clinical presentation of contrast-induced TCB is very unnerving, and it is important that it be entertained early on in the differential diagnosis by the endovascular surgeon. Rapid imaging to rule out an embolic event or hemorrhage is important to diagnose and treat other etiologies of these symptoms.

REFERENCES

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