
Transient Cortical Blindness With Leptomeningeal Enhancement After Attempted Peripherally Inserted Central Venous Catheter Placement

Transient cortical blindness is an infrequent complication of endovascular procedures.\(^1\)\(^-\)\(^3\) We describe a patient who developed transient cortical blindness associated with focal leptomeningeal enhancement on contrast-enhanced T1-weighted magnetic resonance imaging (MRI) following attempted placement of a peripherally inserted central venous catheter (PICC).

Report of a Case. A 57-year-old woman with metastatic carcinoma of the colon, which had spread to the liver and peritoneum, was hospitalized for partial small-bowel obstruction. Placement of a PICC for total parenteral nutrition (TPN) was attempted with inadvertent cannulation of the brachial artery and infusion of 10 mL of nonheparinized normal saline. The catheter was promptly removed; however, the patient began to complain of blurred vision in each eye approximately 15 minutes after the attempted PICC placement. During the procedure, the patient was hypertensive and tachycardic with a blood pressure of 170/120 mm Hz and a heart rate of 120 beats per minute. The patient’s blood pressure was controlled with low-dose oral metoprolol.

After the procedure, the patient complained of progressive visual loss, headache, and malaise, which progressed to complete binocular visual loss during the ensuing 4 hours. Ophthalmic examination revealed no light perception vision bilaterally and briskly reactive pupils, without a relative afferent pupillary defect. The anterior segment, extracocular motility, and dilated fundoscopy findings were normal in each eye. Findings from the remainder of the neurologic examination were otherwise normal.

Contrast-enhanced MRI obtained 4 hours after the onset of visual loss revealed focal leptomeningeal enhancement in the occipital lobes and superior cerebellum (Figure 1). Diffusion-weighted images showed no evidence of ischemia, and magnetic resonance angiography demonstrated normal cerebral vasculature.

Lumbar puncture showed an elevated protein level of 119 mg/dL and a normal glucose level of 81 mg/dL with a normal opening pressure. No atypical cells or white blood cells were seen. The results for cerebrospinal fluid cultures and blood cultures were negative. Twenty-four hours after the onset of visual loss, the patient experienced 2 generalized seizures, which were controlled with intravenous dexamethasone and phenytoin.

Figure 1. Sagittal (left) and axial (right) contrast-enhanced T1-weighted magnetic resonance images reveal focal leptomeningeal enhancement (arrows) in the occipital lobes and superior cerebellum, and an old right basal ganglia infarct.
During the following week, the patient’s symptoms gradually improved. After 72 hours from the time of onset of visual loss, she had light perception in both eyes, and after 5 days, visual acuity was 20/400 OD and 20/800 OS. Nine days after the initial insult, the patient’s visual acuity was 20/20 OD and 20/30 OS with normal visual fields by confrontation, and repeat MRI revealed resolution of the focal leptomeningeal enhancement (Figure 2). A repeat lumbar puncture revealed a protein level of 54 mg/dL, a glucose level of 126 mg/dL, and a normal opening pressure. No abnormal cells were seen on cytologic examination.

Comment. Transient cortical blindness following vascular intervention has been described primarily after contrast-enhanced coronary, cerebral, and other arterial perfusion studies. Inadvertent arterial entry during venous catheterization has resulted in cortical blindness in 2 prior reports. In both of those patients, arterial infusion of TPN resulted in transient bilateral visual loss with occipital lobe abnormalities on neuroimaging. In the first report, MRI showed leptomeningeal enhancement around the occipital cortices, similar to that seen in our patient, following TPN infusion into the right vertebral artery. The visual symptoms improved in 2 weeks with a residual right homonymous hemianopia. In the second report, a left homonymous hemianopia and visual hallucinations occurred following TPN infusion into the right subclavian artery. This patient experienced sudden bilateral visual loss after the onset of TPN infusion, which improved after several minutes, and flashes of light each time her central line was flushed with normal saline. Ophthalmic examination showed a left homonymous hemianopia that resolved after 3 days. Magnetic resonance imaging showed multifocal infarcts in the occipital and parietal cortices and in the right cerebellum.

The mechanisms causing visual loss following contrast and TPN infusion remain unclear. However, several authors have postulated that the hyperosmolarity of these agents may lead to osmotic disruption of the blood-brain barrier and subsequent cortical blindness through direct neurotoxic effects. Experimental studies have demonstrated that hyperosmolar agents induce vasodilation, shrinkage of cerebrovascular endothelial cells, and widened endothelial tight junctions.

The focal leptomeningeal enhancement noted on the contrast-enhanced MRI in our patient suggests that disruption of the blood-brain barrier occurred in the verteobasilar distribution. It is unclear whether the nonheparinized normal saline rinse disrupted the blood-brain barrier in our patient, but the timing of the visual symptoms with the catheterization of the brachial artery suggests an iatrogenic vascular cause for the patient’s symptoms. A possible contributing factor was the patient’s elevated blood pressure because hypertensive encephalopathy may cause transient cortical blindness associated with reversible cerebral edema on neuroimaging.

Leptomeningeal enhancement may occur from infection, cerebrovascular disorders, neoplasia, inflammatory conditions, and iatrogenic causes (Table). In our patient, the pattern of radiographic enhancement could have been attributed to carcinomatous meningitis. However, repeat cerebrospinal fluid examinations revealed no atypical cells. Although cerebrospinal fluid cytology is falsely negative in 10% of patients, the rapid resolution of the radiographic and clinical abnormalities makes the diagnosis of carcinomatous meningitis unlikely. Infectious meningitis was also considered in the differen-
Disseminated Fusarium Infection Presenting as Bilateral Endogenous Endophthalmitis in a Patient With Acute Myeloid Leukemia

Fusarium is a fungal pathogen that may cause local, as well as potentially fatal, systemic infection. Fusarium species are the most common cause of keratomycosis in the southeastern United States, and exogenous endophthalmitis is well documented in this patient population. Endogenous Fusarium endophthalmitis, however, is a rare condition with only a few cases reported.1-8 Almost all cases involve disseminated infection in immunocompromised patients, either owing to leukemia or other severe systemic conditions. The visual prognosis is poor and disease-related mortality is high despite local and systemic antifungal treatment. We describe a patient with a history of acute myeloid leukemia who developed bilateral visual loss secondary to endogenous Fusarium endophthalmitis. The patient was subsequently diagnosed as having disseminated fusariosis and died 5 days after she was first examined.

Report of a Case. A 70-year-old white woman was referred for evaluation for a 3-day history of visual loss. The medical history was significant for myelodysplastic syndrome and she was started on a regimen of filgrastim and cytarabine. A 2-cm skin lesion with foul-smelling discharge was noted on examination. Blood cultures, vancomycin hydrochloride, and tazobactam, ciprofloxacin hydrochloride, and amphotericin B were performed 3 days after the vitreous tap. Vancomycin, fusidic acid, and ceftazidime sodium were also positive for Fusarium species after 24 hours of incubation.

The patient was started on intravenous amphotericin B (0.5 mg/kg per day) and oral fluconazole (400 mg/daily). The following day, the patient died from multiorgan failure.

Comment. The Fusarium species are ubiquitous filamentous molds that are commonly found in soil and on plants. Fusarium infection may occur as toxicosis following ingestion; however, Fusarium infections are usually local and are associated with trauma (keratomycosis) or altered body surface (superficial burn wound infections). In the setting of severe immunosuppression and neutropenia, Fusarium species may cause potentially fatal dissemi-