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Case Report

Cortical Blindness after Contrast-Enhanced CT: Complication in a Patient with Diabetes Insipidus

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Summary: Transient cortical blindness is an uncommon but well-known complication following cerebral angiography. One possible cause of this complication is an adverse reaction to contrast agent, resulting in an osmotic disruption of the blood-brain barrier that seems to be selective for the occipital cortex. We report the case of a 16-year-old male patient with cortical blindness after intravenous application of nonionic contrast agent during CT angiography performed because of seizure that was attributed to thrombosis of the basilar artery on the basis of clinical findings. To our knowledge, the development of cortical blindness after CT angiography has not been described in the literature. The patient's symptoms were triggered by hyponatriemia and diabetes insipidus.

Transient cortical blindness is a well-known but rare complication following administration of angiographic contrast agent. The onset of transient cortical blindness occurs within minutes to as much as 12 hours after contrast agent administration (8). Cortical blindness is self-limiting and characterized by bilateral amblyopia or amaurosis, normal papillary reflexes, unaltered extraocular movements, and normal fundi. A combination with further symptoms (eg, hemiparesis, dysphasia, seizure, headache, memory loss) is possible (1). Transient cortical blindness was reported in cerebral, vertebral, brachial, aortic arch, renal, and coronary angiography, translumbal aortography, and myelography (1–5). The largest series of cortical blindness after cerebral angiography reported an incidence of 0.3–1% (6). The highest incidence was reported following vertebral angiography (7). To the best of our knowledge, there is no report of cortical blindness after contrast-enhanced CT in the literature. We report the case of a 16-year old boy with this rare condition and give possible explanations for this phenomenon.

Case Report

A 16-year-old male patient was admitted to our unit with acute symptoms following first grand mal. Because of increasing nausea, vomiting and disturbed consciousness, he was intubated and respirated. In the emergency unit, he received a native CT scan with 5-mm section thickness on a multidetector CT scanner (Light Speed, GE, Milwaukee, WI). CT was determined to be normal except for a left temporal arachnoidal cyst without mass effect or edema. Because basilar thrombosis was clinically suspected, contrast-enhanced CT angiography of the circle of Willis was performed. The contrast medium (50 mL Ultravist 300, Schering, Berlin, Germany) was administered intravenously at a flow rate of 5 mL/s. Scanning was started with a delay of 15 seconds. No occlusion or thrombosis was detected. No side effects were reported during or after the application. The patient was hospitalized for observation. The laboratory investigation revealed hyponatriemia (120 mmol/L), increased cortisol (675.3 nmol/L [normal range 119–618 nmol/L]), a normal range of further hormone concentrations, and no pathologic findings in the CSF.

The patient had a history of enuresis nocturna and diabetes insipidus treated by Mictonorm (Propiverin-HCl). The day before his seizure, he had consumed more than 2.5 liters of beer. Hyperhydration with resulting hyponatriemia was suggested as the cause of the seizure.

Three hours after CT, he developed blurred vision, which progressed to bilateral blindness. Neurologic examination was nonfocal without significant abnormalities. Pupillary light reflexes and extraocular movements were intact. The fundoscopic examination was entirely benign. Ophthalmologic investigation revealed isolated bilateral eye-field deficits. An MR imaging examination was performed on a 1.5-T MR scanner (Vision plus, Siemens, Erlangen, Germany) with standard stroke sequences (diffusion-weighted, fluid-attenuated inversion recovery [FLAIR] sequence, T2-weighted and T1-weighted spin-echo sequences and arterial and venous MR angiography). No findings of ischemia or bleeding were observed (Fig 1). There were no diffusion abnormalities as typically seen with infarction on heavily diffusion weighted images ($b = 1000$ s/mm^2). Increased values of the apparent diffusion coefficient (ADC) were observed in the cortex of the occipital lobe (0.91×10^{-3} mm^2/s) compared with the frontal lobe (0.81×10^{-3} mm^2/s), indicating an intracellular edema caused by hyponatriema and cytotoxic contrast media. Both arterial and venous angiographies were without pathologic findings. The left temporal arachnoidal cyst was verified. No enhancing effects were observed after application of an MR contrast agent (Fig 2). The patient's vision improved 7 hours after application of the nonionic contrast media and returned fully 24 hours later. No therapy was necessary. On day 3, he was discharged. A follow-up MR image 14 days later was without pathologic findings in the occipital lobe. ADC values were in the normal range in both the visual cortex (0.82×10^{-3} mm^2/s) and the frontal cortex (0.84×10^{-3}mm^2/s). The size of the reported arachnoidal cyst was unchanged. There were also no pathologic findings in the clinical investigation.

Discussion

Transient cortical blindness following angiography was first reported in 1970 (9). Cerebrovascular dis-

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From the Institute of Diagnostic and Interventional Radiology (H.-J.M., A.M., C.F., J.R.R., W.A.K.) and the Department of Pediatrics (J.B.), University of Jena, Jena, Germany

Address correspondence to Hans-Joachim Mentzel, M.D., Institute of Diagnostic and Interventional Radiology, Bachstraβe 18, 07740 Jena, Germany.

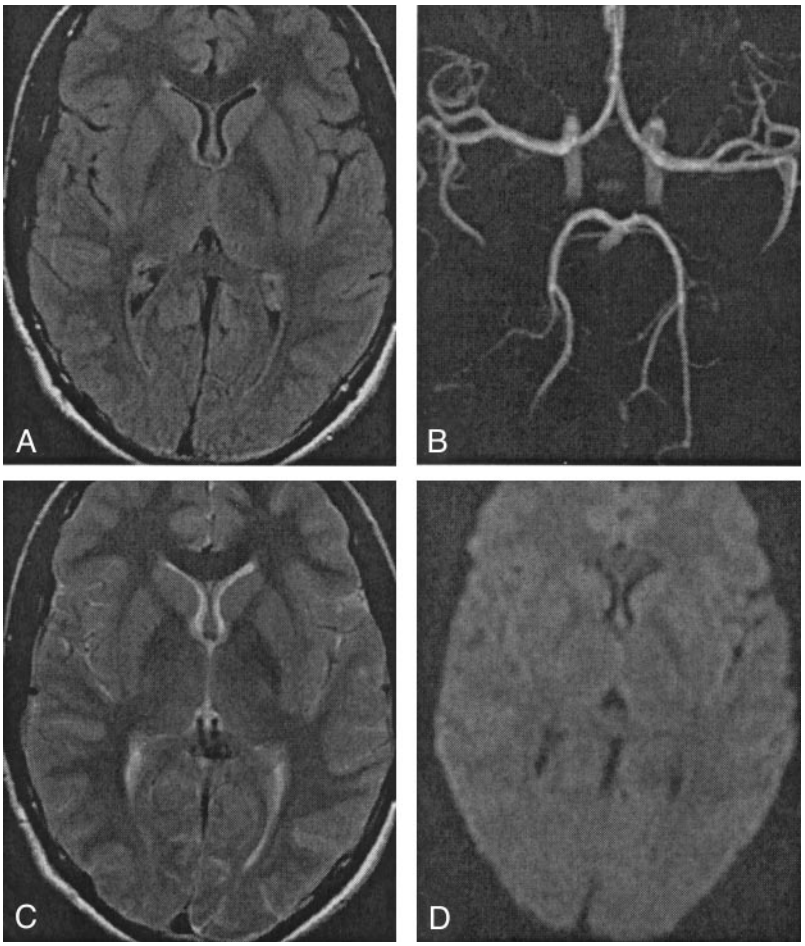

FIG 1. MR imaging revealing no abnormalities in the occipital lobe, no hyperintensities on T2-weighted or FLAIR sequences, no infarction with reduced diffusion, and no vessel occlusion.

A, Axial FLAIR image.

B, Time-of-flight angiography (maximum intensity projection).

C, Axial T2-weighted image.

D, Diffusion-weighted image (b = 1000).

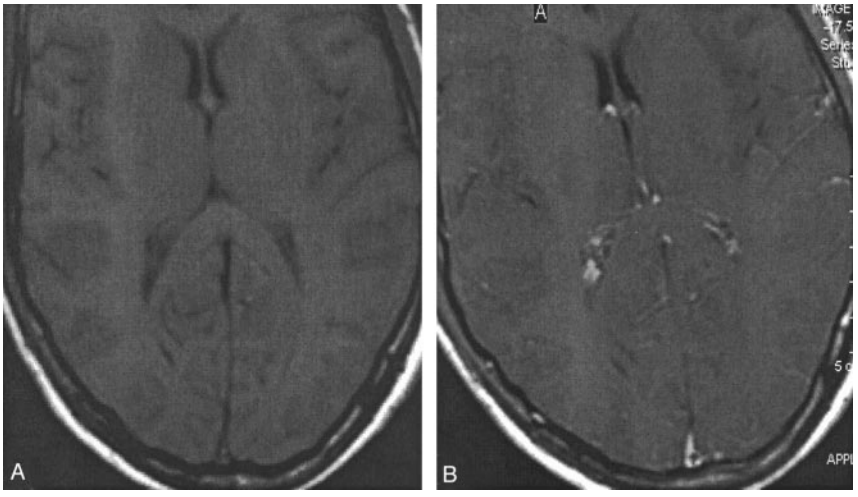

FIG 2. No contrast-enhancing effect in the occipital lobe after administration of gadopentetate dimeglumine (Gd-DTPA).

A, T1-weighted native spin-echo sequence.

B, T1-weighted spin-echo sequence after administration of 0.1 mmol/kg GdDTPA.

ease, cardiac surgery, and cerebral and coronal angiography have been recognized as major causes of cortical blindness (10). The incidence of transient cortical blindness is reported to range from 0.3% to 1% when nonionic contrast agents are used, but it can be as high as 4% when hyperosmolar iodinated contrast agents are used (11). Angiographically induced cortical blindness has to be differentiated from embolic complications (12). More than half of the patients in previous reports of transient cortical blindness revealed chronic hypertension. Hypertensive encephalopathy, cyclosporin neurotoxicity, or eclampsia may cause local vasodilatation and vasoconstriction with a breakdown of the blood-brain barrier and focal transudation of fluid with subsequent direct neurotoxiciy of the contrast media (13). Because the posterior cere-

bral circulation is known to be more sensitive to such injuries because of different sympathetic innervation, these mechanisms may lead to a contrast-enhancing effect after application of CT or MR contrast media in patients with cortical blindness (5, 14). We observed none of these changes in our patient, who developed cortical blindness 3 hours after contrast-enhanced CT was performed. There was no disruption of the blood-brain barrier discernible on the contrast-enhanced MR images, and no hyperintensities were observed in the white matter on FLAIR and T2-weighted images. No embolic lesions were seen. Sequential MR imaging showed no persistent morphologic changes indicative of embolic infarction or white matter changes after contrast media toxicity. Thus, we were not able to confirm the typical imaging findings reported with cortical blindness.

Conclusion

We believe that the symptoms of transient cortical blindness after contrast-enhanced CT were caused by neurotoxic effects of the nonionized contrast media triggered by the hyponatriemia following hypervolemia in a patient with diabetes insipidus. The cause is believed to be secondary to a direct neurotoxicity of the contrast agent itself to the occipital cerebral lobes after intracelluar edema following hyponatriemia, which could be verified in our patient by ADC measurements. This mechanism differs from other angiography-related visual events, such as embolism, changes in blood pressure, and allergy (15). The true biochemical mechanisms of cerebral injury remains speculative in our patient.

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