
Case Report

Unilateral Contrast Neurotoxicity as a Stroke Mimic After Cerebral Angiogram

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ABSTRACT

Contrast neurotoxicity (CN) following exposure to iodinated contrast is uncommon, and usually presents as cortical blindness due to bilateral occipital lobe involvement. Unilateral cortical dysfunction due to CN could mimic an acute stroke and has been rarely described. We report the case of an 89-year-old female who developed a transient dense/complete left homonymous-hemianopsia and left-sided tactile extinction after undergoing a right internal carotid (ICA) artery rotational angiogram with a standard high-volume iodinated contrast injection for 3D visualization a 6×4-mm right posterior communicating artery aneurysm with a fetal posterior cerebral artery (PCA) incorporated in the neck. This was associated with transient fullness and loss of gray-white matter differentiation in the right occipital and parietal lobes. The potential mechanism of CN in our case was the injection of a high volume of contrast in the ICA for the rotational angiogram. The presence of a right fetal PCA possibly allowed the contrast to reach the right occipital lobe. CN manifesting as an acute focal neurologic syndrome should be considered in the setting of recent iodinated contrast exposure.

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Introduction

The occurrence of contrast neurotoxicity (CN) leading to transient neurological symptoms following exposure to iodinated contrast is uncommon but well reported, ranging between 0.3% and 2.9%.¹⁻³ Although it is most commonly seen after cerebral angiograms, it has been also described after contrast-enhanced computed-tomography (CT), cardiac and peripheral angiograms.¹⁻⁴ The vast majority of cases of CN present with cortical blindness due to bilateral occipital lobe involvement. The symptoms might develop within minutes or hours of contrast exposure (including cases where deficits are noticed immediately at the end of the procedure) and resolve over hours to days.^{2,5-8} Unilateral cortical involvement due to CN has been rarely reported.^{2,5,8} Such unilateral dysfunction could mimic an acute stroke, posing an urgent diagnostic challenge. We

report a case of unilateral occipitoparietal CN after an internal carotid artery angiogram.

Case Report

An 89-year-old female with a history of hypertension, stage-I chronic renal disease, and bradycardia status-postpacemaker insertion was admitted with worsening right eye pain, diplopia and ptosis. On exam, she was found to have a right cranial nerve III palsy. A computed-tomography angiogram (CTA) of the brain demonstrated a posterior communicating aneurysm (PcomA). Bilateral internal carotid artery (ICA) and left vertebral artery catheter cerebral angiograms were performed. A right ICA rotational angiogram with a standard high-volume iodinated contrast injection (18 cc at 2.5 cc/second) was

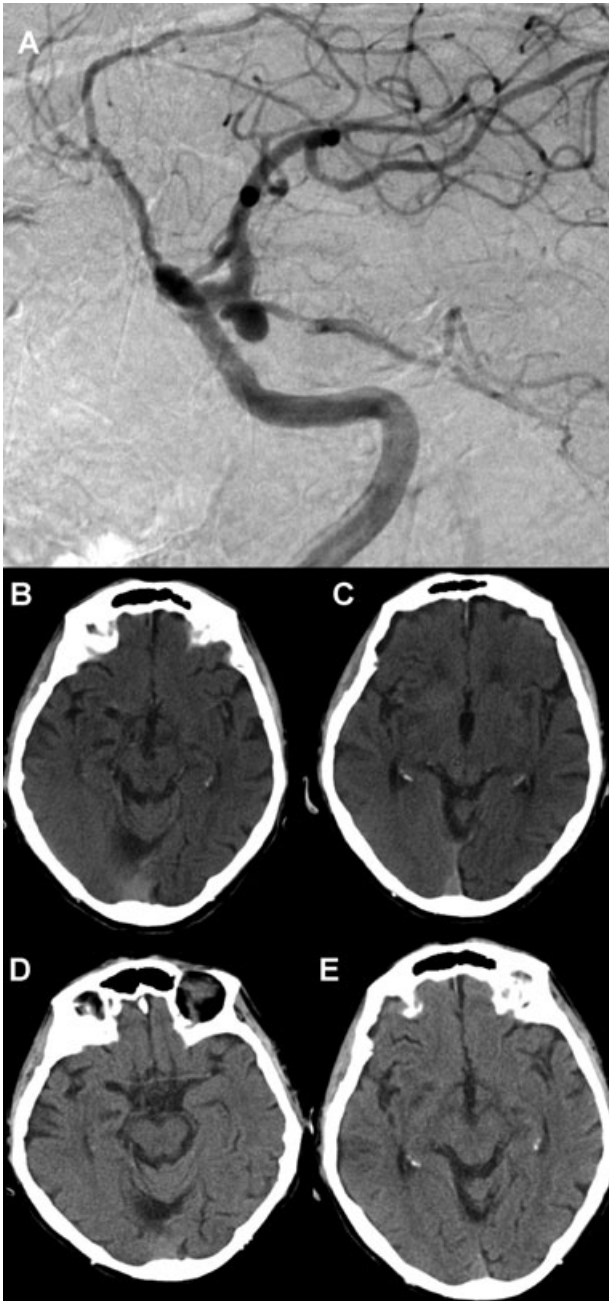


Fig 1. Conventional angiogram depicting the aneurysm and CTs demonstrating the edema and its resolution after 24 hours. (A) Conventional angiogram depicting right posterior communicating artery aneurysm (lateral view). (B and C) CT brain without contrast at 6 hours after the procedure revealing fullness of the sulci in the right occipital lobe. (D and E) Repeat CT at 24 hours demonstrating resolution of the abnormalities.

done for 3D visualization of the aneurysm. A right PcomA aneurysm (6 mm × 4 mm) with a fetal PCA incorporated in the neck was noted (Fig 1A). No thromboembolic occlusions were noted on angiography and the patient was neurologically intact after the procedure. Six hours after angiogram, although the patient remained asymptomatic, a routine neuro-ophthalmological examination showed a dense/complete left

homonymous-hemianopsia, and left-sided tactile extinction. The use of conscious sedation during the procedure might have delayed the symptom recognition. Immediate CT demonstrated fullness in the sulci over the right occipital and parietal lobes, with loss of gray-white matter differentiation (Figs 1B, C). MRI was contraindicated due to presence of a pacemaker. Vigorous hydration was started for possible CN. At 24 hours, the visual field deficit improved to a left lower homonymous quadrantanopsia, the sensory abnormality was minimal, and a follow-up brain CT demonstrated resolution of the above findings (Figs 1D, E). At 36 hours, the neurological deficits were completely resolved.

Discussion

Despite the advancement of non-invasive cerebrovascular imaging techniques, catheter angiography remains the gold-standard and a common procedure for imaging of cerebral aneurysms and other intracranial vascular disorders. In patients who develop a stroke syndrome postangiography, urgent accurate diagnosis is paramount. The non-recognition of CN presenting as a stroke mimic might lead to unnecessary repeat angiography for endovascular therapy. We report a rare case of unilateral reversible dysfunction of the occipital and parietal lobes due to CN after cerebral angiography that mimicked an acute stroke syndrome.

Although uncommon, CN is a striking clinical condition most often presenting as transient cortical blindness due to bilateral occipital cortex involvement. Postangiogram unilateral CN presenting as a stroke mimic was previously reported in one patient that developed encephalopathy and hemiparesis. However, the brain imaging in this case was not compatible with stroke as the entire right hemisphere was affected, not respecting vascular territories.⁵ Another subject was found drowsy, globally aphasic and with worsening of a prior hemiparesis. Nevertheless, this case had clear abnormal cortical high density (probably related to contrast hangout) involving the entire right hemisphere.⁸ Unilateral involvement was also described in a case where the initial presentation was cortical blindness related to bilateral occipital lobe toxicity and the hemianopsia was only noted during the recovery phase.² In another case, hemianopsia related to CN was postulated but multiple small-artery occlusions were also observed and treated with intraarterial ab-ciximab during the angiogram.²

In our patient, the clinical presentation raised a strong suspicion for cerebral thromboembolism postangiography. However, no evidence for arterial occlusion in the right parietal or occipital lobes was seen on the angiogram. CN is usually associated with imaging findings of persistent presence of contrast in the cortex (gyriform hyperattenuation) and vasogenic edema. In this case, the sole CT finding of effacement of sulci in the right parietal and occipital lobes was atypical, but felt to be consistent with CN. The complete resolution of the clinical syndrome and CT findings within 36 hours confirmed the diagnosis of CN. The potential mechanism of CN in our case was the injection of a high volume of contrast in the ICA for the rotational angiogram. The presence of a right fetal posterior cerebral artery (PCA; Fig 1A) possibly allowed the contrast to reach the right

occipital lobe. The previously noted risk factors for CN present in our case included renal insufficiency, long duration of the procedure, and high infusion rate of contrast.^{2,6,7}

In conclusion, CN manifesting as an acute focal neurologic syndrome should be considered in the setting of recent iodinated contrast exposure.

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