Transient Visuospatial Disorder From Angiographic Contrast

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**Background:** The blood-brain barrier may be permeable under the clinical settings of uncontrolled hypertension, renal insufficiency, immunosuppressive drugs, and intravascular radiographic contrast. Some reversible neurological complications after angiography are caused by cortical penetration of contrast media detected on brain computed tomographic (CT) scans.

**Objectives:** To describe the first report of a transient visuospatial disorder having elements of Balint syndrome, and caused by angiographic contrast penetration of the bilateral parieto-occipital cortex; and to review cases published between 1980 and 2001 of cortical contrast penetration, documented by CT.

**Results:** Simultanagnosia, optic ataxia, and ocular apraxia occurred in a 74-year-old woman who received non-ionic contrast media during a failed renal angioplasty. Contrast noted in the bilateral parieto-occipital cortex on the initial CT scan disappeared after 4 days with clinical resolution.

**Conclusions:** Angiographic contrast tends to breach the blood-brain barrier of the vertebrobasilar circulation, penetrating the occipital cortex and leading to transient, localizable syndromes of cortical blindness or abnormal visuospatial processing.

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Catheter-related vasospasm, intimal tears, or emboli occasionally cause focal deficits during arteriography. Other patients experience confusion, delirium, seizures, or cortical blindness that are typically reversible after several hours to a few days. In these instances, intra-arterial contrast apparently penetrates the blood-brain barrier by opening tight capillary junctions or enhancing endothelial pinocytosis. It then enters the cerebral cortex and adversely affects neuronal membranes. Contrast neurotoxicity seems to be related to the chemical or ionic properties of the contrast medium and its hyperosmolarity, lipid solubility, and viscosity. The reduced clearance of intravascular contrast in renal failure may be an additional clinical factor, but it is difficult to demonstrate in an experimental model. There are several reports of angiographic contrast entering the occipital cortex and causing cortical blindness, sometimes with denial of visual deficit (Anton syndrome). The following is the first case report, to our knowledge, of angiographic contrast causing a transient visuospatial or visual processing disorder having elements of Balint syndrome.

**REPORT OF A CASE**

A hypertensive 74-year-old right-handed woman developed renal insufficiency from bilateral renal artery stenosis. There was no prior history of transient focal neurological deficit, stroke, seizure, or substance abuse. She underwent an unsuccessful percutaneous renal angioplasty, receiving a total of 415 mL of iopamidol (Isovue 370; Bracco Diagnostics, Princeton, NJ) contrast. By the next morning, her family found her to be clumsy. She would reach with her hand too far, or not far enough, when trying to grasp nearby objects. On examination that afternoon, she was afebrile with a blood pressure of 170/70 mm Hg and a pulse of 68 beats per minute. She was repeatedly using a knife and fork to “cut” on an empty dinner plate, having great difficulty focusing on, or attending to, other food items on her dinner tray, despite being verbally directed to their location. By bedside confrontation visual field testing, she was not blind and had no extinction to simultaneous bilateral visual stimuli. Ocular movements were full, either when suddenly looking toward a noise, or tracking a target; though the latter required many attempts with continuous encouragement. Her pupillary light reaction was normal.

In attempting to get more comfortably seated, she struggled to pull herself up in bed, grasping the tray table and other objects, but not the raised siderail. Multiple attempts were needed to reach and grasp her telephone, but once it was in her hands, she was able to use it properly. Af-
When finally focusing on a vegetable or fruit item on the left or right of her tray, she could not accurately spoon or stab it with her fork. Likewise, finger-nose-finger testing was clumsily performed with either hand, but no tremor was seen. Bilateral asterixis was present.

When shown a magazine advertisement of several dancers, with a wristwatch (the largest object) in the foreground, she could only identify “people.” She could only read or spell words 4 to 5 letters long, and she refused to write or draw. However, she remained alert, oriented, and cooperative, and she successfully performed 3-step commands. Verbal fluency, comprehension, and repetition were normal and without paraphasic errors. Although a few common objects were visually identified, she could not name certain food items such as a cookie, despite accurately describing their shape and color. Touching these same items, however, allowed her to correctly name them. A picture of rock star Elvis Presley was easily identified. She had finger agnosia and trouble with calculations, but was able to distinguish left from right. She appropriately followed commands to “salute the flag as if a parade marched by” and “brush your teeth as if you had a toothbrush in your hand.”

Chronic findings from an idiopathic peripheral neuropathy included distally weak (4/5), areflexic lower limbs with decreased pinprick and proprioception. There was extinction to double-simultaneous tactile stimulation on the left limbs, but graphesthesia was intact. An uninfused computed tomographic scan of the brain (Figure 1) showed persistent intravascular contrast, especially in the circle of Willis, plus bilateral gyrical enhancement of the occipital and posterior parietal lobes. Hemodialysis was begun for acute oliguric renal failure.

Three days later, her visual fields were easily tested and full to confrontation. Spoken language, reading, and repetition were normal. When viewing a picture of people seated at a table with a background of a fountain and city skyline, she could only identify “people.” She was unable to reach and grasp a glass of water. She described the colors of a pen (“gold and black”) and knew “ink comes out of it,” but could only name “pen” after touching it. However, other common items were named easily by sight. On the fourth day, she could interpret and identify all musicians and their instruments in a picture of an orchestra, and described various cosmetics illustrated in a magazine advertisement. Visual fields, visual tracking, reading, and comprehension remained normal. There was no asterixis, and finger-nose-finger tests and other limb maneuvers were performed well. A repeated uninfused computed tomography brain scan was now normal (Figure 2). She remained neurologically normal the next 3 days and was discharged home.

**COMMENT**

Simultanagnosia is a visual processing problem in which only one object from an array is “seen” at one time, with the inability of the individual to maintain visual awareness of the other objects. This occurs despite relatively in-
Table 1. CT-Confirmed Cortical Penetration of Angiographic Contrast*

<table>
<thead>
<tr>
<th>Source (Year)</th>
<th>Sex/Age, y</th>
<th>Angiogram</th>
<th>Contrast</th>
<th>Clinical Symptoms</th>
<th>Region Involved</th>
<th>Clinical Resolution</th>
<th>Normalized CT Scan</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shibakiri et al† (1980)</td>
<td>M/70</td>
<td>Aortic arch</td>
<td>50 mL lothalamate meglumine</td>
<td>Partial motor SZ</td>
<td>Left (fronto)-temporo-parieto-occipital</td>
<td>. . .</td>
<td>1 d</td>
</tr>
<tr>
<td>Sage et al‡ (1981)</td>
<td>F/43</td>
<td>Aortic arch</td>
<td>130 mL Diatrizoate meglumine</td>
<td>Partial and generalized motor SZ, left central facial paresis</td>
<td>Right fronto-parietal; basal ganglia</td>
<td>. . .</td>
<td>Not done</td>
</tr>
<tr>
<td>Studdard et al‡ (1981)</td>
<td>F/59</td>
<td>Aortic arch</td>
<td>150 mL Diatrizoate meglumine</td>
<td>Cortical blindness (Anton); HA, myoclonus or SZ?</td>
<td>Bilateral (temporo)-parieto-occipital</td>
<td>3 d</td>
<td>5 d</td>
</tr>
<tr>
<td>Numaguchi et al‡ (1984)</td>
<td>F/53</td>
<td>Carotid artery</td>
<td>60 mL Diatrizoate meglumine</td>
<td>Partial motor SZ</td>
<td>Right temporo-parietal</td>
<td>1 d</td>
<td>2 wk</td>
</tr>
<tr>
<td>Utz et al‡ (1988)</td>
<td>M/72</td>
<td>Aortic arch</td>
<td>120 mL Diatrizoate meglumine; 80 mL lothalamate meglumine</td>
<td>Partial motor SZ</td>
<td>Bilateral fronto-parietal</td>
<td>1 d</td>
<td>Not done</td>
</tr>
<tr>
<td>Current report</td>
<td>F/74</td>
<td>Abdominal aorta</td>
<td>250 mL Diatrizoate meglumine</td>
<td>Cortical blindness; right central facial paralysis; left hemiparesis</td>
<td>Bilateral occipital; basal ganglia</td>
<td>4-5 d</td>
<td>Not done</td>
</tr>
<tr>
<td>Lantos‡ (1989)</td>
<td>F/49</td>
<td>Subclavian artery</td>
<td>30 mL Diatrizoate meglumine</td>
<td>Cortical blindness (Anton); nystagmus; ophthalmoplegia; general motor SZs</td>
<td>Bilateral occipital; bilateral thalamus, pons midbrain</td>
<td>1 d</td>
<td>Not done</td>
</tr>
<tr>
<td>M/64</td>
<td>Carotid artery</td>
<td>12 mL lothalamate meglumine</td>
<td>Cortical blindness; confusion; fluent aphasia</td>
<td>Left (temporo-parieto)-occipital</td>
<td>3 d</td>
<td>1 d</td>
<td></td>
</tr>
<tr>
<td>M/71</td>
<td>Carotid artery</td>
<td>38 mL Iohexol†</td>
<td>Cortical blindness; confusion; ophthalmoplegia</td>
<td>Bilateral occipital</td>
<td>10 d</td>
<td>Not done</td>
<td></td>
</tr>
<tr>
<td>F/68</td>
<td>Carotid artery</td>
<td>24 mL Iohexol</td>
<td>Cortical blindness (Anton); confusion; amnesia</td>
<td>Bilateral occipital</td>
<td>6 d</td>
<td>Not done</td>
<td></td>
</tr>
<tr>
<td>Shyn and Bell§ (1989)</td>
<td>M/70</td>
<td>Aortic arch</td>
<td>50 mL Diatrizoate meglumine; 22 mL lothalamate meglumine</td>
<td>Cortical blindness</td>
<td>Bilateral occipital</td>
<td>2 d</td>
<td>1 d</td>
</tr>
<tr>
<td>Kinn and Breishblatt‡ (1991)</td>
<td>M/55</td>
<td>Aortic arch</td>
<td>228 mL Diatrizoate meglumine</td>
<td>Cortical blindness</td>
<td>Bilateral occipital</td>
<td>1 d</td>
<td>Not done</td>
</tr>
<tr>
<td>Parry et al‡ (1991)</td>
<td>M/62</td>
<td>Aortic arch</td>
<td>270 mL Iopamidol</td>
<td>Cortical blindness; clumsy right upper limb</td>
<td>Bilateral occipital</td>
<td>3 d</td>
<td>3 d</td>
</tr>
<tr>
<td>Sticherling et al§ (1998)</td>
<td>M/55</td>
<td>Aortic arch</td>
<td>280 mL Iomeprol</td>
<td>Cortical blindness; confusion; amnesia</td>
<td>Bilateral (temporo)-occipital</td>
<td>5 d</td>
<td>1 d</td>
</tr>
<tr>
<td>Current report</td>
<td>F/74</td>
<td>Abdominal aorta</td>
<td>415 mL Iopamidol</td>
<td>Visuospatial disorder</td>
<td>Bilateral parieto-occipital</td>
<td>4 d</td>
<td>4 d</td>
</tr>
</tbody>
</table>

*CT indicates computed tomography; SZ, seizure; HA, headache; and ellipses, data not available.
†Iopamidol is a nonionized contrast media.

Our patient also had great difficulty moving her eyes, on command, to a peripheral target such as the other items on her dinner tray. This did not seem to be due to simultanagnosia since she was asked to “find the bowl of carrots on your right,” or “look next to your left hand for the plate of bread,” and would only find these items after several suggestions and directions prompting many attempts. Reflexive visual saccades, as when quickly looking in the direction of a loud noise, appeared normal. This deficit seems to be that of ocular apraxia, which was described as “psychic paralysis of gaze” and “spasm of fixation.”

The first deficit noted in our patient was her impaired ability to reach and grasp objects that she wanted. Despite seeing the telephone, she had trouble getting her hand on it, reaching beyond its location or not reaching far enough. After finally visually locating, on command, a specific food item on her tray, she could not spoon it up or pierce it with a fork. Such erratic reaching under visual guidance represents optic ataxia.

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Balint syndrome, consisting of simultanagnosia, optic ataxia, and ocular apraxia, is typically associated with bilateral lesions in the superior parieto-occipital lobes.\(^{12,13}\) Some have argued that these deficits may be produced by lesions elsewhere, and that this clinical triad may not exist as cohesively as previously assumed.\(^{11}\) Nevertheless, we feel our case has great similarity, both clinically and neuroanatomically, to the historical syndrome of Balint. Our patient exhibited simultanagnosia and optic ataxia, and probably ocular apraxia. These deficits often coexist as a result of bilateral lesions in the parieto-occipital, dorsal visual association areas, which in general comprise the “where” visual system, dealing with the accurate location of objects. The temporo-occipital, ventral visual association areas constitute the “what” system, or the identification and recognition of objects.\(^{14}\) Although she could name several items, our patient could not name a “cookie,” or “pen,” unless she touched them. Her partial visual agnosia shows that there was some involvement of these temporo-occipital areas as well. The presence of some elements of the Gerstmann syndrome (finger agnosia and impaired calculations) also suggests lesion extension into the dominant angular gyrus.\(^{15}\)

Whether or not our patient manifested Balint syndrome, if indeed it specifically or uniquely exists, she did have a reversible syndrome of abnormal visual processing due to penetration of angiographic contrast media into the bilateral parieto-occipital cortex. Angiographic contrast is neurotoxic, and its presence within brain parenchyma makes it unlikely that other mechanisms, such as arterial emboli or vasospasm induced by the angiographic procedure, were causative. Contrast may even appear in the occipital lobes following intra-arterial injection as remote as the abdominal aorta or renal arteries as noted in our case and in one other report.\(^{1}\) In addition to our report, there are 14 other cases of contrast penetration in the cerebral cortex documented by computed tomography scan. Twelve of these 15 cases involved the occipital cortex, with transient cortical blindness in 10 cases (Table). Some of these cases were exposed to relatively less toxic nonionic contrast media, and the amounts of contrast injected varied greatly. Renal insufficiency, delaying clearance of intravascular contrast and prolonging its exposure to the blood-brain barrier, was an additional factor in our case and in 2 others.\(^{3,4}\) Cortical blindness has occurred after contrast injection into the aorta, or the renal, subclavian, coronary, carotid, or vertebral arteries. This apparently increased frequency of contrast permeating the occipital cortex suggests that the blood-brain barrier is more vulnerable in the posterior circulation.

The sympathetic innervation of the vertebrobasilar arterial system is not as extensive or complete as that of the carotid arterial system.\(^{19}\) Thus, a relative lack of protective, sympathetically mediated arteriolar vasocostriction during severe hypertension may account for the predominance of posterior hemisphere lesions in eclampsia or hypertensive encephalopathy.\(^{20,21}\) A reversible posterior leukoencephalopathy syndrome seems to be related to transplant immunosuppressive treatment and renal insufficiency, as well as hypertension.\(^{22,23}\) Early on, vascular permeability presumably creates edematous lesions, which often resolve upon control of blood pressure or reduction of immunosuppressive drugs. Clinically, cortical blindness is common in this syndrome, and is often accompanied by agitation, delirium or seizures, making the bedside examination difficult. If the visual system could be examined thoroughly, perhaps more subtle deficits in visual processing would be detected.

The blood-brain barrier of the vertebrobasilar system thus seems breachable in the setting of several conditions that may coexist, including hypertension, renal insufficiency, immunosuppressive drugs, and angiographic contrast. Ionic or nonionic contrast tends to selectively penetrate the occipital cortex, causing transient syndromes from dramatic cortical blindness to more subtle visuospatial processing disorders.

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Author contributions: Both authors had equal responsibility for the work.

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REFERENCES