Case Reports

Two Cases of Occipital Infarction Following Cardiac Catheterization

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Transient visual disturbance following cardiac catheterization has been regarded as a benign and transient complication. We describe two cases of computed-tomography-documented occipital infarction after cardiac catheterization that emphasize a complication more serious than previously thought. (Stroke 1988;19:773–775)

Transient cortical blindness is a rare but well-described complication of cardiac catheterization.\(^1^\)\(^-\)\(^5\) Reports indicate that it is a benign condition, and complete recovery of vision should be expected. We report two cases of computed-tomography-documented occipital lobe infarction following cardiac catheterization suggesting a complication more serious than previously regarded.

**Case Reports**

**Case 1**

A 71-year-old man was admitted for preoperative evaluation of a 5-cm abdominal aortic aneurysm. Medical history included diffuse peripheral vascular disease, one-block claudication, amaurosis fugax, bilateral carotid endarterectomies, and Type IV hyperlipidemia. Cardiac catheterization via the femoral approach revealed significant two-vessel coronary artery disease. Immediately after the procedure, he was noted to be somnolent but easily arousable and he could attend for brief periods. Pupils were 3 mm, symmetric, and briskly reactive to light. Fundoscopic examination was unremarkable. The patient denied any subjective visual disturbance yet could not count fingers or distinguish light from dark. He confabulated answers requiring intact vision. The remainder of his neurologic examination was unremarkable. An emergency CT of the head without contrast revealed an area of decreased density in the right occipital lobe (Figure 1).

Over the next 2 days, both mental status and vision returned to normal. A head CT with contrast performed 2 weeks after the acute event revealed an enhancing right occipital lobe infarction (Figure 2).

**Case 2**

A 69-year-old man with a history of a myocardial infarction was admitted for evaluation of worsening dyspnea on exertion. Cardiac catheterization via the femoral approach revealed a 50% right coronary artery lesion and no evidence of valvular heart disease. Immediately after catheterization, the patient complained of decreased vision bilaterally. Neurologic examination revealed an intact mental status. Visual acuity was limited to light perception only. Pupils were 4 mm and briskly reactive. Fundoscopic examination was unremarkable. The remainder of the neurologic examination was otherwise normal. An emergency noncontrast CT of the head was negative.

The next day, the patient’s vision improved to counting fingers at 2 feet, but a left homonymous hemianopsia was also present. CT of the head with contrast 2 weeks later demonstrated bilateral occipital infarctions, right greater than left (Figure 3). One month later, the patient’s best corrected visual acuity was 20/50 oculus dexter (OD) and 20/40 oculus sinister (OS) with a persistent left homonymous hemianopsia.

**Discussion**

Numerous reports describe the varied complications of cardiac catheterization\(^1^\)\(^-\)\(^6\); however, only 33 cases of transient visual disturbance have been described.\(^7\)\(^-\)\(^11\) The most comprehensive analysis, a prospective series of 2,006 cases undergoing cardiac catheterization, reported a total of 20 patients (1%) who experienced transient visual disturbances.\(^7\) The incidence was higher in women (2%, 11 of 548) than in men (0.6%, 9 of 1,458). The visual disturbance was recognized within the first hour following catheterization, and all patients had a complete recovery within 24 hours. Fifteen patients had only a partial loss of vision, and five had complete cortical blindness. An alteration of mental status was seen in seven cases, and six cases denied their blindness (Anton’s syndrome), as in Case 1. One patient had a transient visual agnosia. The
neurologic examination was otherwise unremarkable in most patients (95%). One patient had a mild right hemiparesis that resolved over 4 days. Eight of 13 electroencephalograms (EEGs) were abnormal, demonstrating bilateral temporal or temporo-occipital slowing that resolved on subsequent tracings. CT scans were not obtained.

Braunwald and Swan reported a 31-year-old man who developed the inability to detect light immediately after catheterization. Complete recovery of vision was noted the following day. In another report, a 52-year-old woman developed cortical blindness, headache, and nausea after cardiac catheterization. Her symptoms were associated with transient hypotension (140/100 to 84/60 mm Hg), and the deficit resolved spontaneously over 18 hours. An EEG performed during the acute episode demonstrated intermittent bifrontal 2-Hz delta activity and a depressed alpha rhythm over the left occipital leads. In addition, absence of an evoked response following photic stimulation was noted. Three other cases of "transient blurred vision" were also reported in a series of 5,250 cardiac catheterizations performed via the femoral route; however, details of their clinical course are lacking. In a brief report, eight of >30,000 patients undergoing cardiac catheterization experienced neuro-ophthalmologic complications; of these, four had radiographic evidence of bilateral occipital infarctions, all of whom had permanent visual deficits.

Transient visual disturbances and cortical blindness are better-known complications following vertebral angiography, with an estimated incidence of 1-4% and accounted for 12% of all cases of cortical blindness following cerebral angiography in one series. Several mechanisms have been proposed to explain the clinical findings, and similar mechanisms may explain the transient visual disturbances after cardiac catheterization. Blindness results presumably from occipital lobe ischemia. Somnolence, commonly seen in "top of the basilar" syndrome, frequently accompanies postcatheterization visual disturbances and suggests ischemia to the rostral brainstem. Presumably, both upper brainstem and bilateral occipital ischemia develop due to a lesion at the distal basilar artery. Occipital lobe ischemia may result from 1) dislodgment of atheromatous material, 2) embolization of thrombus from the catheter tip, 3) in situ thrombosis or spasm of cerebral vessels, 4) a hypotensive episode, 5) preexisting hypertensive disease or migraine headaches, or 6) selective vulnerability of occipital lobes to contrast media toxicity. Of 20 patients studied prospectively, the incidence of transient visual disturbance appeared to be significantly higher in patients with normal coronary arteries (3.7% vs. 0.8%). If presence of atheromatous coronary artery disease is taken as a marker of atherosclerosis elsewhere, dislodgment of atheromatous material causing blindness would appear to be a less likely mechanism. Although hypotension may be a contributing factor, only one patient developed a visual disturbance during transient hypotension. Another patient had transient hypotension that was treated and then developed visual loss with
documented normal blood pressure. The other mechanisms suggested may indeed play significant roles; however, none of these has ever been demonstrated radiologically or pathologically.

The largest series studying transient visual disturbance following cardiac catheterization regards it as a benign complication. The clinical course of one patient described here suggests that permanent neurological deficit can be present. Furthermore, CT findings in both of our patients imply that, at least in some cases, structural occipital lobe damage can result, even in patients who appear to recover clinically. Since most patients in previous reports were not studied with CT, it is impossible to say how often these patients will have residual occipital infarctions. Our two cases suggest that transient visual disturbance following cardiac catheterization is a much more serious complication than has been suggested previously.

References


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