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Transient Bilateral Cortical Visual Loss after Coronary Artery Bypass Grafting in a Normotensive Risk-Free Patient

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ABSTRACT

Background: Postoperative blindness has been mentioned as a rare complication of heart surgery. The majority of reported cases are caused by retinal artery occlusion or ischemic optic neuropathy. We report a case of transient visual loss due to cortical ischemia after coronary artery bypass grafting (CABG).

Case Report: A 52-year-old nondiabetic man developed complete bilateral visual loss immediately after CABG. He had been normotensive throughout the operation. An ophthalmologic exam detected no causative ocular damage. The results of a Doppler study of the carotid, vertebral, and ophthalmic arteries were completely normal, and an echocardiography examination showed no left ventricular clot. Magnetic resonance imaging of the brain, however, showed several ischemic plaques in watershed areas and a small subacute infarct in the occipital lobe. Recovery began on postoperative day 4, and the patient's vision was restored in 6 months.

Conclusion: Although most cases of visual loss after open heart surgery have been caused by injuries to the peripheral optic system, cortical blindness may occur following open heart surgery in the absence of any preexisting risk factor. Fortunately, the course of recovery is promising one, as it was for our patient.

INTRODUCTION

Postoperative visual loss is a rare but devastating complication of nonocular surgery and general anesthesia [Roth 1999]. Heart surgery or intervention has been a major type of operation that has accompanied such accidents [Roth 1996]. The majority of reported cases are caused by damages to peripheral parts of the visual structures and pathway, especially ischemic optic neuropathy and retinal artery occlusion [Moster 1998; Sha'aban 2000; Nuttal 2001; Warner 2006], and cortical visual loss has been reported to be responsible in a minority of reported cases [Taugher 1976]. Cortical blindness is distinguished clinically by visual loss along with an absence of optokinetic nystagmus and lid reflex, normal pupillary response and eye motility, and a normal retina and optic nerve. Coronary artery bypass grafting (CABG) surgery is the most common operation leading to postoperative cortical blindness [Roth 1999], and many of these operations have been considered to be associated with risk factors such as intraoperative hypotension, significant anemia, diabetes, carotid artery stenosis, and so on [Newman 2002; Williams 2002]. We report a case of transient visual loss due to cortical ischemia after CABG in a relatively risk-free patient.

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CASE REPORT

A 52-year-old man with a recent myocardial infarction (1 month previous) was referred to the cardiac surgery department for CABG surgery. No risk factor for coronary disease was found except previous acute coronary syndrome and admission to a cardiac care unit. The patient was moderately symptomatic (New York Heart Association class II) and was taking metoprolol tartrate (Metoral), isosorbide dinitrate (Isordil), and atorvastatin. An electrocardiogram showed a Q wave in leads II and III, aVF, and left anterior hemiblock. A coronary angiography examination showed a completely occluded left anterior descending coronary artery (LAD) and mildly diseased left circumflex and right coronary arteries. An echocardiography evaluation revealed an ejection fraction of 25%, a moderate restrictive left ventricular pattern (E>A), mild mitral regurgitation, mild tricuspid regurgitation (TR), no left ventricular clot (confirmed later with transesophageal echocardiography), a pulmonary artery pressure of 36 mm Hg, and an enlarged left ventricle (end-diastolic volume, 153 cm^3; end-systolic volume, 111 cm^3). A dobutamine stress echocardiography examination performed to assess the viability of the myocardium in the coronary artery disease territory revealed at least 4 viable segments in this territory. A carotid duplex study showed normal internal and external carotid arteries. The results of pulmonary function tests and arterial blood gas measurements were normal.

CABG surgery (left internal mammary artery [LIMA] to the LAD) was performed with cardiopulmonary bypass (CPB) (temperature, 32°C; CPB time, 74 min; cross-clamp time, 30 min) because of technical difficulty in finding the intramuscular LAD. The patient was in a supine position, and no hypotension occurred throughout the operation. The patient's pre-pump blood pressure was 105/75 mm Hg with a pulse rate of 60 to 70 beats/min, the on-pump perfusion pressure was kept between 80 mm Hg and 90 mm Hg, and the patient's post-pump blood pressure was 110/70 mm Hg. The on-pump blood flow rate during the operation was set at 2.6 L/m^2. On-pump hematocrits were between 25% and 27%, and arterial blood gas measurements revealed normal oxygen saturation without hypoxemia or significant acid-base disturbance in the pre-pump, on-pump, and post-pump phases. The surgeons and the perfusion team took precise precautions to prevent the introduction of air into the patient's arterial line, and the perfusion team checked the bypass route. In fact, no entrance route for air existed because we used no vein graft and did not punch or open the aorta. Weaning the patient off CPB was somewhat difficult, but he was removed from the pump without the use of an intra-aortic balloon pump. No adverse event (e.g., atrial fibrillation) occurred in the postoperative course except for the bilateral total blindness, which was apparent immediately after the patient regained consciousness. An ophthalmologic examination showed the following: bilateral complete blindness (no light perception [NLP]); normal papillary reflexes; no nystagmus; and mild bilateral lateral gaze palsy to the left, bilateral elevation limitation, and mild dry-type age-related macular degeneration that were not considered relevant to the blindness. The results of a neurologic examination were normal except for mild ocular motor impairment. A brain computed tomography scan showed normal findings for the occipital lobes but did demonstrate some hyperdensity in the basilar region (Figure 1A). A Doppler examination of the intracranial and extracranial arteries revealed no evidence of significant stenosis in the carotid and vertebral systems, and both ophthalmic arteries had normal time-velocity spectral patterns. Brain magnetic resonance imaging (MRI) revealed small hypersignal foci in watershed areas of the occipital lobes indicating ischemic plaques, along with a hypersignal area in the right occipital lobe near the posterior commissure, which was positive in the diffuse image and supportive of a subacute infarction (Figure 1B).

On postoperative day 4, the patient recovered some vision (light perception bilaterally). A postoperative echocardiography examination showed no new abnormal changes, including a left ventricular clot. A follow-up MRI examination 1 week after the operation showed the previous signals with some recovery (Figure 2). By postoperative day 13, the patient's vision had improved (finger counting at 10 cm) and gradually recovered to acceptable vision until postoperative day 21 (visual acuity equal to 3-4 of 10 bilaterally), when he was discharged. Because the patient had become somewhat depressed during the postoperative course, a psychiatric consultation performed before discharge resulted in the addition of a mood-enhancer drug (fluoxetine) to his regimen. Fortunately, the patient continued to recover steadily in follow-up visits, and he regained virtually normal vision by 6 months after surgery.

DISCUSSION

Postoperative blindness after nonocular operations such as cardiac surgery has been reported previously [Roth 1999]. Thus far, the major causes of this complication that have been

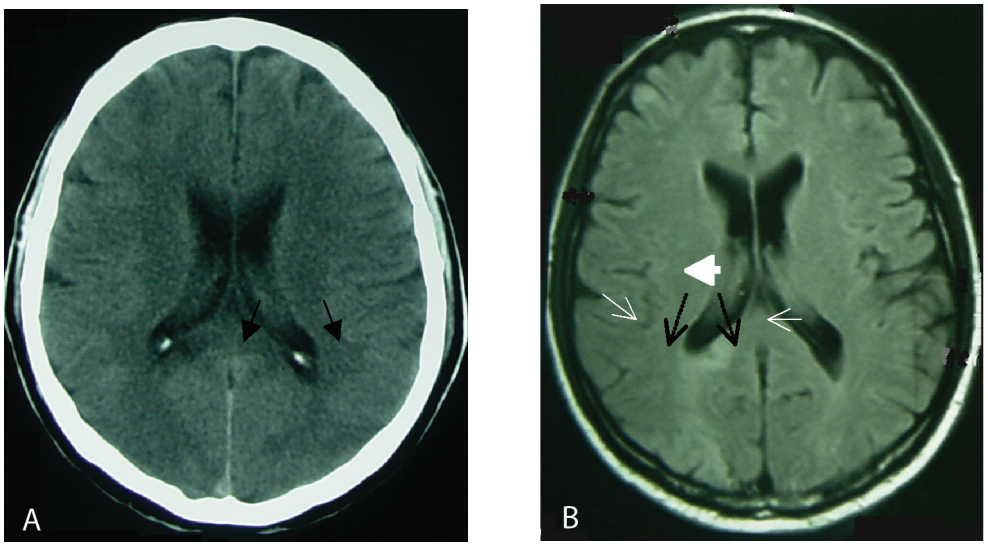

Figure 1. A, Brain computed tomography scan showing generally normal tissue with a relatively hypodense occipital cortex (black arrows). B, Brain fluid-attenuated inversion recovery (FLAIR) magnetic resonance imaging indicating ischemic plaques in occipital lobes (white thin arrows) with a hypersignal area in the right occipital lobe in favor of subacute infarction (white thick arrow). Bilateral edematous cortex and decreased cortico-subcortical discrimination (dashed black arrows) are also seen.

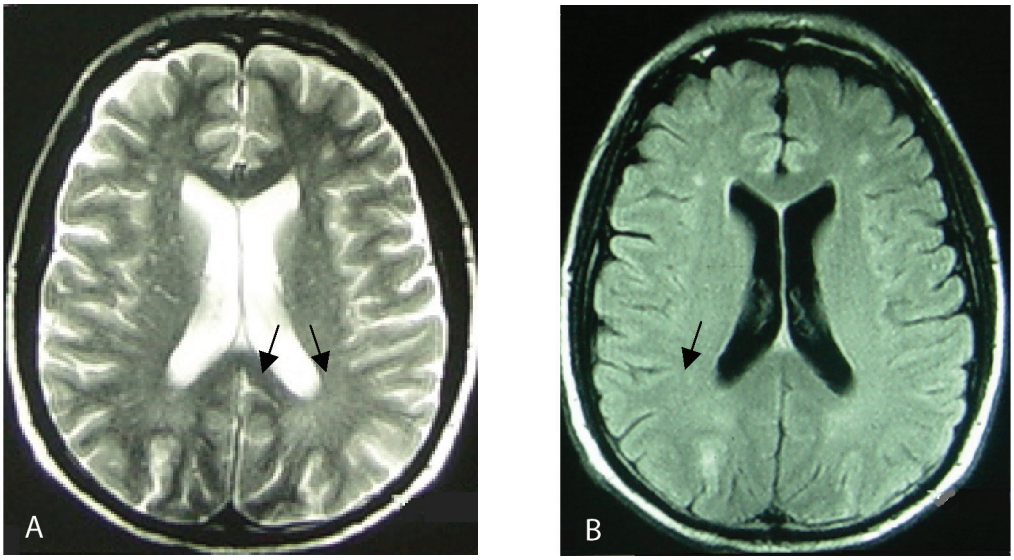

Figure 2. Brain magnetic resonance images (MRI) showing some recovery after 1 week. Arrows indicate bilateral thinned occipital cortices. T_2-weighted MRI (A); fluid-attenuated inversion recovery (FLAIR) MRI (B).

detected have been related to retinal and ophthalmic nerve damage, especially after surgeries in which the patient was in a prone position, such as in spine surgery [Moster 1998; Warner 2006]. In the present case, an ophthalmologic examination and consultation revealed no evidence of injury to these structures, which prompted our clinical presumption of cortical events as a cause. This hypothesis was later confirmed in brain-imaging studies.

The primary causes for post-CPB cortical blindness that have been proposed are emboli, profound hypotension, significant anemia or hypoxemia, or a combination of these factors [Williams 2002]. Transthoracic and transesophageal echocardiography evaluations revealed no left ventricular clot as a source of an embolism in our patient. There was no sign of atheroma in the ascending aorta by angiography, transesophageal echocardiography, or direct palpation. The results of Doppler studies of the carotid arteries before and after the surgery were completely normal, and a transcranial Doppler examination revealed no stenosis or occlusion in the major cerebral and vertebral arteries. Consequently, we found no clear source for macroembolization. On the other side, because the patient's blindness was bilateral and because we found bilateral ischemic plaques in the occipital cortices (Figure 1B), embolic events seem not to have played a great role in our patient's presentations. There was just a single unilateral hypersignal area near the right posterior commissure, which was compatible with an embolic infarction; however, this finding cannot explain the patient's visual and neurologic symptoms.

We performed a LIMA-to-LAD procedure and did not use a venous graft (which involves anastomosis to the ascending aorta); therefore, there was no route for the introduction of air into the arterial system and systemic circulation. Moreover, because air showering tends to involve anterior parts of the brain tissue, it is not probable that it would interfere with the occipital blood supply in a supine patient. However, the patient experienced no hypotensive or hypoxic episode at any time during the surgery and had no significant anemia, which can lead to such a cerebral accident.

There is increasing evidence in the literature that patients with coronary artery disease have more preexisting cerebrovascular disease that has the potential to compromise the cerebral circulation, even before surgery [McKhann 2006]. This disorder not only causes autoregulatory dysfunction of the brain vascular system but also makes the patient susceptible to CPB complications, such as microemboli, inflammatory components, and inhomogeneous perfusion. Despite a decreasing incidence of macroembolization because of the advent of modern surgical techniques, microembolization following the use of an extracorporeal circulatory pump may adversely affect the blood supply to small territories of the brain, especially in watershed areas [McKhann 2006]. In conclusion, we suppose that disturbance of the occipital circulation caused by autoregulatory compromise in the watershed areas along with adverse inflammatory and microembolic effects of CPB led to an inhomogeneous perfusion to visual parts of the cortex. This process did not produce a true ischemic infarction but rather a relatively transient ischemia in the visual cortex that gradually faded away during the subsequent months.

Although the restoration of vision may be somewhat prolonged, the prognosis is promising for cortical blindness, particularly in previously healthy patients. In this case, the recovery process began in the first postoperative week, but normal visual acuity was not regained for 6 months. Minimizing the risk of such complications requires taking preoperative and intraoperative precautions. Screening of emboli sources such as the aorta, the carotid arteries, and the heart chambers, as well as abstaining from intraoperative hypotension and hypoxemia, seems to be a good

strategy in this regard. Moreover, our experience has shown that these precautions may not be sufficient to eliminate the risk of cortical ischemia and blindness.

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