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Transient Bilateral Blindness in a Patient after Cardiac Surgery

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ABSTRACT

Our report describes a case of temporary bilateral blindness in a patient after undergoing mitral valve replacement with a mechanical prosthesis and coronary artery bypass grafting.

CASE REPORT

A 63-year-old 68-kg man with a history of endocarditis 10 years prior presented with moderate regurgitation and severe prolapse of the mitral valve, as well as with coronary artery disease. Medications at admission included valsartan, furosemide, and aspirin. There was no known history of lidocaine exposure, but the patient had previously undergone an appendectomy and phacoemulsification without significant complications. His medical history was otherwise significant for hypertension. No significant carotid artery disease was present before surgery; there were no underlying vision problems. The patient was admitted the day before surgery and underwent a standard preoperative workup. The next day, he underwent mitral valve replacement with a mechanical prosthesis, and 4-vessel coronary artery bypass grafting. The procedure lasted 7 hours, including induction. No unusual amount of intravenous fluids were administered, and no intraoperative complications, including hypotensive episodes, occurred. An epiaortic ultrasound evaluation was performed to rule out the presence of significant atherosclerosis in the ascending aorta at the cross-clamping and proximal graft-anastomosis sites. Intraoperative transesophageal echocardiography was used to confirm adequate deairing of the cardiac chambers. The left atrial appendage and the left ventricle were free of thrombus.

The patient was transferred from the operating room to the cardiac surgery intensive care unit (ICU) at 6 PM with continuous infusion of norepinephrine bitartrate at a rate of 5 µg/min. The patient's blood pressure was labile, requiring administration of 1.5 L of lactated Ringer solution and 250 mL of 5% albumin during next few hours. In addition, a continuous infusion of epinephrine (2 µg/min) was started 2 hours after the patient's arrival in the ICU. At approximately the same time, the patient started to have paroxysmal ventricular trigeminy and quadrigeminy, which became increasingly irregular, contributing to a relatively unstable blood pressure. An increase in the arrhythmogenic activity was attributed to the epinephrine infusion, and its dose was slowly reduced and subsequently turned off for a brief time. Because of the unstable blood pressure, however, the infusion was restarted.

At 11 PM, 5 hours after the patient's admission to the ICU, 100 mg lidocaine was administered intravenously for the worsening arrhythmia. At that time, the patient had a high oxygen requirement (100% fraction of inspired oxygen [Fio_2]) and a low arterial oxygen pressure (PaO2, 68 torr), and we did not use amiodarone infusion as a first choice. At midnight, we started a continuous infusion of intravenous lidocaine at a rate of 1 mg/min, which was followed by 1 more intravenous bolus of 100 mg lidocaine at 1 AM. We increased the lidocaine infusion rate to 2 mg/min at 2 AM. At approximately the same time, a continuous intravenous administration of vasopressin was initiated at a rate of 0.04 U/min for blood pressure support. After that, the patient became hemodynamically stable, the heart remained in a normal sinus rhythm, and no significant arrhythmias occurred. The next morning at 11 AM, the patient was extubated without complications. Four hours later, one of the patient's first statements was that he could not see, "not even the darkness." A physical examination revealed that the patient was completely blind. In response, we almost immediately discontinued the lidocaine infusion, because we considered it a possible cause for the blindness. The patient had the first signs of vision return in 1 hour and started to see shapes of objects by 3 hours after lidocaine discontinuation. He remained color-blind, however.

Neurology and ophthalmology services were consulted on the same day (first postoperative day). Upon examination, they were able to confirm normal discs and retina, and an absence of nystagmus or diplopia. The ophthalmologic examination confirmed the absence of color vision and that the patient was able to only see hand motions. A computed tomography/computed tomographic angiography evaluation of the head revealed no signs of acute stroke or hemorrhage.

Eight hours after discontinuation of the lidocaine infusion, signs of color vision started to appear. During the following days, the patient's vision improved dramatically; however, he still complained of seeing "floaters" and seeing "like through the kaleidoscope." The patient was transferred out of the ICU on postoperative day 5. He was seen by the neuro-ophthalmology service on postoperative day 6. This

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examination revealed dramatic improvement in visual acuity, from being able to see hand motions to 20/30 vision with a pinhole at distance.

The patient was discharged home on postoperative day 10, and a follow-up examination on postoperative day 20 showed a return of his vision in both eyes to the near-normal baseline.

DISCUSSION

Visual loss after cardiopulmonary bypass is not uncommon. Shen et al analyzed the data for more than 5.6 million patients who underwent various nonophthalmic surgeries between 1996 and 2005, and found that cardiac surgery had the highest rates of perioperative visual loss among nonophthalmic surgeries, specifically 8.64 cases per 10,000 patients [Shen 2009]. These cases are usually caused by emboli, hypotension, acute anemia, hypoxia, intraparenchymal hemorrhage, or a combination of these factors [Slavin 1987; Shahian 1989; Moster 1998; Chen 2000; Sha'aban 2000; Loubani 2001; Suzuki 2001; Bagheri 2008; Thurtell 2008; Shin 2010]. In the case of our patient, these causes have been carefully considered and ruled out by the workup of the neurology, ophthalmology, and neuro-ophthalmology services. Immunosuppressive therapy in heart transplant recipients also has been associated with visual loss [Schowengerdt 1993; Drachman 1996]; however, this possibility was irrelevant in our case.

We think that this event can be explained by the intravenous lidocaine administration. The onset and resolution of the blindness do correlate well with the timing of starting and discontinuing the lidocaine infusion. Although blindness is neither a known side effect of lidocaine nor a known result of interaction with other drugs, a literature search does reveal cases of visual loss after lidocaine administration [Ellis 1968; Sawyer 2002; Rishiraj 2005]. Sawyer et al described a case of temporary bilateral blindness that occurred during a regional anesthesia procedure after inadvertent deflation of the upper arm tourniquet and the subsequent release of lidocaine into the systemic circulation [Sawyer 2002]. In fact, the symptoms described are similar to the symptoms of our patient. As a possible explanation, the authors suggested occipital lobe seizure activity versus subcortical stimulation caused by lidocaine overdose. The authors did not measure the serum lidocaine level.

Unfortunately in our case, we neither measured the blood lidocaine level during the blindness episode nor did we perform further testing for lidocaine hypersensitivity. Thus, we can only provide a suggestion that requires further investigation.

REFERENCES

Bagheri J, Mandegar MH, Sarzaeem MR, Chitsaz S. 2008. Transient bilateral cortical visual loss after coronary artery bypass grafting in a normotensive risk-free patient. Heart Surg Forum 11:E248-51.

Chen FC, Chu KM, Lai CP. 2000. Delayed onset of amaurosis fugax in a patient with type A aortic dissection post surgical repair. Jpn Heart J 41:787-91.

Drachman BM, DeNofrio D, Acker MA, Galetta S, Loh E. 1996. Cortical blindness secondary to cyclosporine after orthotopic heart transplantation: a case report and review of the literature. J Heart Lung Transplant 15:1158-64.

Ellis PP. 1968. Visual loss following tonsillectomy; possible association with injections in tonsillar fossae. Arch Otolaryngol 87:436-8.

Loubani M, Galinanes M. 2001. Pituitary gland macroadenoma: a cause of transient blindness after cardiac surgery. Ann Thorac Surg 72:929-31.

Moster ML. 1998. Visual loss after coronary artery bypass surgery. Surv Ophthalmol 42:453-7.

Rishiraj B, Epstein JB, Fine D, Nabi S, Wade NK. 2005. Permanent vision loss in one eye following administration of local anesthesia for a dental extraction. Int J Oral Maxillofac Surg 34:220-3.

Sawyer RJ, von Schroeder H. 2002. Temporary bilateral blindness after acute lidocaine toxicity. Anesth Analg 95:224-6.

Schowengerdt KO Jr, Gajarski RJ, Denfield S. 1993. Progressive visual deterioration leading to blindness after pediatric heart transplantation. Tex Heart Inst J 20:299-303.

Sha'aban RI, Asfour WM. 2000. Visual loss after coronary artery bypass surgery. Saudi Med J 21:90-2.

Shahian DM, Speert PK. 1989. Symptomatic visual deficits after open heart operations. Ann Thorac Surg 48:275-9.

Shen Y, Drum M, Roth S. 2009. The prevalence of perioperative visual loss in the United States: a 10-year study from 1996 to 2005 of spinal, orthopedic, cardiac, and general surgery. Anesth Analg 109:1534-45.

Shin YD, Lim SW, Bae JH, Lee DH, Baek DH, Hong JS. 2010. Transient cortical blindness after heart surgery in a child patient: a case report. Korean J Anesthesiol 59:61-4.

Slavin ML. 1987. Acute remitting visual loss after coronary artery bypass surgery caused by a suprasellar meningioma. Am J Ophthalmol 104:434-5.

Suzuki Y, Kiyosawa M, Mochizuki M, Ishii K, Senda M. 2001. Cortical blindness following aortic arch surgery. Jpn J Ophthalmol 45:547-9.

Thurtell MJ, Besser M, Halmagyi GM. 2008. Pituitary apoplexy causing isolated blindness after cardiac bypass surgery. Arch Ophthalmol 126:576-8.