“A Case of Visual Loss During Routine Cardiac Surgery: This Can Happen To You In Your Operating Room”

Case Presentation

A 66 year-old female patient with progressive dyspnea presented for elective mitral valve replacement (MVR) to be performed utilizing cardiopulmonary bypass (CPB). Past medical history was significant for mild congestive heart failure (for approximately 1-2 years), hyperthyroidism, and tobacco abuse. Past surgical history included several spine procedures performed in the prone position under general anesthesia without notable neurological complications. Pertinent laboratory data included an electrocardiogram with atrial fibrillation and non-specific ST- and T-wave changes; an echocardiogram noting a heavily calcified bi-leaflet mitral valve with normal opening, moderate severe MR, and moderate left ventricular hypertrophy with preserved ventricular function; and a cardiac catheterization delineating normal coronary anatomy. Additionally, she had a distant history of eye pain OS requiring several emergency room visits for urgent evaluation: presumptive diagnosis at that time was an acute ischemic optic neuropathy (ION). Further laboratory work-up revealed positive anti-nucleolar antibodies and elevations in C-reactive protein and erythrocyte sedimentation rate, suggestive of an active autoimmune process (i.e., possibly lupus or other significant autoimmune disease). An unspecified previous decrease in visual acuity OS was not documented preoperatively in the medical record at the time of her surgical procedure in question.

Intraoperatively, routine invasive monitoring included systemic and pulmonary arterial catheters (PAC, via right internal jugular vein). Balanced general anesthesia was achieved with midazolam, fentanyl, isoflurane and vecuronium. Mitral valve replacement required 76 min of CPB (clear prime), 55 min of aortic cross-clamping, and mild systemic and topical hypothermia (Tmin ~34.1 °C). Post-CPB minor inotropic support with epinephrine (≤0.03 μg/kg/min IV) was required only through the first post-operative day. Estimated blood loss was 500ml, resulting in a hemoglobin (Hgb) level of 7.7 gm/dl; after 1 unit of packed red cells, the Hgb level increased to 9.2 gm/dl. During removal of the PAC on POD 1, prolonged gentle local pressure was required to control bleeding from a laceration to the adjacent right external jugular vein: this was potentially attributed to improper placement of the PAC by the anesthesiologist during the operation.

On POD 2, she noted blurred vision in both eyes (OD>>OS) and “ziggy lines,” suggestive of a scotoma. Urgent ophthalmological evaluation noted an acuity of OS 20/40, OD no light perception, and “pale swelling” of the optic disks OU, consistent with bilateral non-arteritic ION, OD>>OS. Treatment included maintenance of blood pressure and Hgb levels at or above baseline values. Her visual deficits persisted through follow-up as an outpatient. She has inquired to her many healthcare providers, “Why did this happen to me, and what did my doctors or nurses do wrong?” She eventually filled a
lawsuit against the hospital, its agents and her doctors. A discussion of its outcome will be discussed at the session.

Key Questions

(1) What are the potential etiologies of acute post-operative visual loss following cardiac surgery? How, if at all, does the use of CPB or its management influence this risk?

(2) What is post-operative acute non-arteritic ischemic optic neuropathy and its diagnosis? How does it differ from other causes of visual loss? What are the salient ophthalmologic and clinical findings of this condition?

(3) Can a clinician identify specific risk factors perioperatively for the later development of this particular problem? If so, can any be modified to reduce the risk?

(4) Are there any preventive strategies that can be utilized to reduce the incidence or occurrence of devastating post-operative visual loss? When can these strategies and what are those to be employed?

(5) If this unfortunate and rare complication does occur, what diagnostic or therapeutic options exist for the clinicians? What do you tell your patient, if this occurs?

(6) What should clinicians do with the clinical information derived from cases such as this?

(7) What defense mechanisms does a clinician have if a patient decides to file suit for injuries and damages, alleged to be caused by medical mismanagement?

Model Discussion

- Etiologies of Acute Visual Loss Following Cardiac Surgery

Postoperative visual loss following cardiac surgery is fortunately—for both patients and clinicians—quite rare. As a general rule, it is usually unilateral and secondary to anterior retinal neuropathy, with a resultant swollen optic disk visualizable on fundoscopic examination. Potential etiologies involved in its development include: (1) thromboembolism to the ophthalmic arteries or central visual centers; (2) hypoperfusion—either regionally or globally—to the eyes or brain; or (3) vascular thrombosis. Obviously, these etiologies are not mutually exclusive, and multiple causes may be operative in enhancing the extent of visual loss. Furthermore, preexisting visual dysfunction would be expected to enhance any acquired deficits.

Source of thromboemboli when CPB is employed for cardiac procedures include clot, vascular debris, atheromatous plaque or entrained air. Global hypoperfusion can occur with hemorrhage, hypoxemia, induction and/or maintenance of anesthesia, or with use of non-pulsatile hypothermic, hemodilutional CPB. Localized organ hypoperfusion can result from direct pressure either to inflow (e.g., carotid artery) or outflow (e.g., optic veins) vessels, or by increasing intraocular pressure (e.g., direct globe compression).
Vascular thrombosis—either arterial or venous—can result from hypercoagulability, autoimmune diseases, local injury, hemoconcentration, etc. The use of CPB mandates extensive heparinization initially, followed later by its reversal with the procoagulant protamine. CPB also induces fibrinolysis, platelet dysfunction and dilutional factor deficits; treatment with antifibrinolytics, platelets and/or other factor transfusions, respectively, can induce a hypercoagulable state. Preexisting vascular disease (e.g., atherosclerosis, autoimmune processes, etc...) may further enhance thrombus formation and/or reduce organ tolerance to ischemic injury.

In the presented patient, the ultimate etiologies are likely multifactorial. Interestingly, she did not develop an acute ION following any of her previous extensive prone spine procedures—a well known risk fact. Why this is so is not clear.

**Preventive Strategies & Treatment Options**

No effective reliable treatment exists for INO: corticosteroids, osmotic diuretics, and surgical decompression have not proven useful. Therefore, prevention of possible causes is important. The Table below relates the aforementioned etiologies and risk factors with potential treatments (or risk factor modification) to reduce the incidence of postoperative ION. Avoidance of CPB—if possible—may reduce the incidence of acute ION: the incidence of visual loss is significantly less for “off-pump” coronary bypass procedures than those “on” CPB (0.00 vs. 0.11%, respectively). Thromboemboli may be reduced by meticulous valve debridement, appropriate choice of aortic cannulation site (e.g., TEE-guided), and vigorous “de-bubbling.” Hypoperfusion may be prevented by maintenance of adequate blood pressure, cardiac output, hemoglobin levels and oxygenation, limiting CPB time and use of vasoconstrictors. Hypotension, arrhythmias and anemia should be treated, as appropriate. Local pressure to the eye globe, face and neck should also be avoided, as well as any process that increases intraocular pressure.

Preexisting visual acuity and deficits (i.e., formal visual field testing) should be documented, preferably by an ophthalmologist. Patients deemed at high risk by procedure, past medical history (e.g., prior ION, glaucoma) or symptomatology should, in particular, be counseled regarding this potentially devastating complication. Clinical information from identified cases of post-cardiac surgery ION should be reported to a central databank, such as the American Society of Anesthesiologists’ Closed Claims Project. The associated web-site includes information for both patients and clinicians, including current research and understanding into the causes of this significant complication.

**References:**


(13) Gilbert TB. Acute visual loss following cardiopulmonary bypass. *J Neurosurg Anesth* 2004;16:75-6