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Full Text

Postoperative visual loss is a devastating perioperative complication that has received increased attention by anesthesiologists, spine surgeons and ophthalmologists over the last five to 10 years. Despite this increased awareness, physicians remain helpless in preventing its occurrence because most of the cases have no proven etiology.

The ASA Committee on Professional Liability established the ASA Postoperative Visual Loss (POVL) Registry in July 1999 to collect detailed information on these cases obtained through anonymous submissions. The goal of the ASA POVL Registry is to collect 100 cases of postoperative visual loss and search for common patient characteristics and/or perioperative events that may be associated with the development of this complication. As of this writing, 79 cases of postoperative visual deficits after nonophthalmologic surgery have been submitted to the ASA POVL Registry.

Preliminary analysis of the database indicates that the majority of cases are associated with spine operations (67 percent) followed distantly by cardiac bypass procedures (10 percent). The remaining 23 percent of cases are composed of liver transplants, thoracoabdominal aneurysm resections, peripheral vascular procedures, head and neck operations, prostatectomies and miscellaneous cases. Because spine operations comprised such a large percentage of the ASA POVL Registry, these cases were analyzed separately.

Of the 53 cases of postoperative visual loss associated with spine surgery in the registry, ophthalmologic diagnoses included ischemic optic neuropathy (n = 43, 81 percent), central retinal artery occlusion (n = 7, 13 percent) and unknown diagnosis (n = 3, 6 percent). Potential associated factors for spine operations with ischemic optic neuropathy were compared to those for spine operations with central retinal artery occlusion [Table 1]. Patients were similar in age but had striking differences between groups for other factors.

Table 1

Preliminary Data From ASA Postoperative Visual Loss Registry: Associated Factors From Spine Cases*

	Ischemic Optic Neuropathy (n=43)	Central Retinal Artery Occlusion (n=7)
Age–years, median (range)	49 (19-73)	49 (35-71)
Headrest:		
Mayfield tongs	8 (18%)	0
Horseshoe	0	2 (29%)
Foam	33 (77%)	3 (43%)
Unknown	2 (5%)	2 (29%)
Prone time–hours, median (range)	8 (3-24)	5.5 (3.4-9)
EBL† liters, median (range)	2.3 (0.2-20.0)	0.7 (0.5-1.3)
Lowest Hct percentage, median (range)	25.5 (19-40)	33 (29-38)
Bilateral disease	25 (58%)	0
No vision recovery	24 (56%)	7 (100%)

*Three cases with unknown diagnosis for vision loss not shown. †EBL = estimated blood loss

Eight of 43 patients who developed ischemic optic neuropathy had their heads positioned in Mayfield tongs with their faces free from external pressure, whereas none of the patients who developed central retinal artery occlusion was positioned in Mayfield tongs. Two patients from the central retinal artery occlusion group were positioned in a horseshoe headrest. Patients in the ischemic optic neuropathy group had longer periods in the prone position (eight hours) with larger estimated blood loss (2.3 liters) compared to the central retinal artery occlusion group (5.5 hours and 0.7 liters). Consistent with the estimated blood loss, the median lowest hematocrit was lower in the ischemic optic neuropathy group (25.5 percent) compared to the central retinal artery occlusion group (33 percent).

More than half of the ischemic optic neuropathy group had both eyes affected, whereas none in the central retinal artery occlusion group demonstrated bilateral disease. Recovery of vision occurred in 44 percent of the ischemic optic neuropathy group compared to 0 percent recovery in the central retinal artery occlusion group.

This preliminary analysis of potential associated factors in ischemic optic neuropathy spine patients compared to central retinal artery occlusion patients from the ASA POVL Registry supports previously published literature reviews and case reports. The etiology of central retinal artery occlusion is thought to be caused by direct pressure on the globe from face masks, or cushions in the prone position, by emboli or by low perfusion pressure in the retina.[1] The findings of low estimated blood loss, lack of anemia, shorter duration of prone position, unilateral disease and no vision recovery are all consistent with these proposed etiologies. Unilateral periorbital bruising, proptosis, paresis of extraocular eye muscles and/or supraorbital paresthesias may be found in association with central retinal artery occlusion when it is caused by direct pressure on the globe.

In contrast, the etiology for ischemic optic neuropathy is unknown and possibly multifactorial. It has been associated with large blood loss, hypotension, anemia, the prone position and/or vaso-occlusive disease, though specific etiologies for anterior and posterior ischemic optic neuropathy may differ.[1,2] The preliminary data in Table 1 demonstrate a relatively large blood loss, presence of a moderate anemia and long duration in the prone position in the ischemic optic neuropathy group. The occurrence of this disease in eight spine surgery patients whose heads are suspended in Mayfield tongs strongly supports the theory that ischemic optic neuropathy is not caused by direct pressure on the globe. Moreover, the high percentage of patients with bilateral disease makes direct globe pressure an unlikely etiology.

Although the preliminary data suggest unique etiologies for different types of ophthalmologic lesions causing postoperative visual loss, larger numbers of cases will be required before a meaningful statistical analysis can be performed. Collection and analysis of these cases will provide insight into the perioperative events surrounding the development of this complication. For example, as discussed above, the ASA POVL Registry now contains strong evidence that the most commonly reported form of postoperative visual loss, i.e., ischemic optic neuropathy, occurs in the absence of direct pressure on the globe. The data refute a misperception commonly held by surgeons, patients and even many anesthesiologists, and it broadens the potential for research into this perplexing perioperative complication. More definitive data on postoperative visual loss will be gained by obtaining the goal of 100 patients in the ASA POVL Registry. For more information, please visit our ASA POVL Registry Web site at <www.asaclosedclaims.org>.

References

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