Editorial

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Keep an eye on acute optic neuropathy

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Getting blind after recovery from general anesthesia in patients who undergo spinal fusion is a catastrophic event. Perioperative visual loss (POVL) is most commonly due to ischemic optic neuropathy (ION), followed by retinal arterial occlusion.[1] The first description of POVL in Medline, named "Postoperative amaurosis," was published in 1950.[2] Along with the rapid growth in spine procedures, the yearly number of POVL publications accelerated dramatically.[3] However, the number of cases reported POVL registry yearly has been decreasing from a peak in 2000 and has been dropping since.[4] Peri-operative ION (PO-ION) in spine fusion decreased statistically significantly (P = 0.002) from 1.63/10,000 in 1998 to 0.60/10,000 in 2010.[1] This seems a great news to us, but why is the incidence of PO-ION falling? It is probably due to a reduction in operative times along with intraoperative blood transfusion.[4]

One invited article in this issue of TJO highlights the need for all eye doctors to be aware of blind intraoperatively (article by MY Wang and AA Sadun *et al*.). Two articles highlight the optical coherence tomography (OCT) in neuro-ophthalmology: one shares their findings of no significantly thinner macular and peripapillary choroidal thickness (CT) in typical unilateral retrobulbar optic neuritis (ON) eye (clinical diagnosis without magnetic resonance imaging proven) and non-ON eye compared with the healthy control group (article by A Dehghani and MH Alemzadeh-Ansari *et al*.). The other one reveals that non-ON eye of neuromyelitis optica (NMO) patients had a significant reduction in retinal nerve fiber layer thickness along with changes of contrast sensitivity as compared to the control group (article by WOC Feng and WHW Hitam). A case report raises the question in differentiating between acute zonal occult outer retinopathy and ON (article by PF Peng and WC Chan). In one cross-sectional study of 122 patients referred for ON, ON was overdiagnosed in 59.8% of referral cases. The most common errors result from overreliance on a simple clinical history or not considering alternative diagnoses.[5]

The article by Wang and Sadun *et al*. in this issue describes the risks of PO-ION and summarizes PO-ION succinctly: *We need to control modifiable vascular risk factors to prevent irreversible neurologic sequelae in PO-ION*. The causation in PO-ION remains elusive, however, it is most frequent in spine fusion or cardiac surgery.[6] In ION, only the following six factors were identified as independent risk factors in a multivariate model: male sex, obesity, operation duration, the use of a Wilson frame for positioning intraoperatively, estimated blood loss, and nonblood fluid management.[1,3] Besides, they found that the average blood loss was 2.3 L (ranging from 0.8 to 7.6 L) in PO-ION cases. On an average, the lowest hematocrit was 26.1% (ranging from 20% to 36.6%), with the hematocrit percentage decreasing from baseline of about 65%. The addition of colloids will reduce the cell's third space and the resultant increase in tissue pressure. The overall visual outcome appears to be inversely related to the length of the surgery, therefore, staged surgery needed to be considered for high-risk patients. Focusing on minimally invasive surgery, staging of complex procedures, and improved anesthesia practices will probably reduce the length of surgery and blood loss. Furthermore, even though anemia and hypotension are common after major

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surgery, very few patients develop perioperative PO-ION. This suggests the multifactorial etiology and individual susceptibility in PO-ION.

There were some animal models of PO-ION (pig and rat) which represented that optic nerve injury partly resembling human ION can be produced by combining hemodilution and head-down tilt.[7,8] In those studies, the optic nerve blood flow did not have a compensatory increase in the condition of anemia with or without hypotension comorbidity, thereby resulting in significant reductions on optic nerve oxygen delivery in the model of procaine.[8] The nerves in the experimental group, after an ischemia insult, all demonstrated typical optic atrophy with gliosis. These ischemia changes of axonal degeneration were observed mainly in the intracanalicular portion of the optic nerve.[9]

NMO is an autoimmune demyelinating disease with pathogenic aquaporin-4 autoantibodies that act against the astrocyte water channel protein. NMO is associated with recurrent episodes of ON, transverse myelitis, and other brain stem symptoms, often resulting in severe disability.[10] Inflammatory diseases usually alter the vascular structure, and there is no exception in the retina. In the eye, the choroid is the vascular layer and thus can be possibly changed along with inflammatory optic neuropathy. A previous article described patients with multiple sclerosis who after affected by ON had decreased subfoveal CT.[11] Multiple sclerosis produces retinal structural loss, decreased optic nerve head perfusion, and macular vessel density in ON and non-ON eyes in OCT angiography.[12,13] However, A Dehghani *et al*. demonstrated the utilization of OCT in the evaluation of macular and peripapillary CT in patients with acute typical unilateral retrobulbar ON, which showed no significant decrease compared with that of control groups.

In conclusion, different ethnicity and comorbidity will decide the prognosis of visual function and structural loss in ION and ON. Publications of PO-ION in the neuro-ophthalmological literature are still sparse. OCT is an essential imaging technique able to assess structural changes associated with nonglaucomatous optic neuropathy. This issue has the most important theme in acute optic neuropathy which will be an active research area of neuro-ophthalmology in future, in both the basic mechanism and potential medication as well as clinical pathophysiological insight of optic nerve disorders.

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References

1. Rubin DS, Parakati I, Lee LA, Moss HE, Joslin CE, Roth S. Perioperative visual loss in spine fusion surgery: Ischemic optic neuropathy in the United States from 1998 to 2012 in the nationwide inpatient sample. Anesthesiology 2016;125:457-64.
2. Curtillet E. Postoperative amaurosis. Afr Fr Chir 1950;1:51-3.
3. Apfelbaum JL, Roth S, Connis RT, Domino KB, Lee LA, Nickinovich DG, *et al*. American Society of Anesthesiologists Task Force on Perioperative Visual Loss. Practice advisory for perioperative visual loss associated with spine surgery: An updated report by the American Society of Anesthesiologists Task Force on Perioperative Visual Loss. Anesthesiology 2012;116:274-85.
4. Todd MM. Good news: But why is the incidence of postoperative ischemic optic neuropathy falling? Anesthesiol J Am Soc Anesthesiol 2016;125:445-8.
5. Stunkel L, Kung NH, Wilson B, McClelland CM, Van Stavern GP. Incidence and Causes of Overdiagnosis of Optic Neuritis. JAMA Ophthalmol 2018;136:76-81.
6. van Wicklin SA. Systematic review and meta-analysis of prone position on intraocular pressure in adults undergoing surgery. Int J Spine Surg 2020;14:195-208.
7. Roth S, Dreixler J, Newman NJ. Haemodilution and head-down tilting induce functional injury in the rat optic nerve: A model for peri-operative ischemic optic neuropathy. Eur J Anaesthesiol 2018;35:840-7.
8. Lee LA, Deem S, Glenny RW, Townsend I, Moulding J, An D, *et al*. Effects of anemia and hypotension on porcine optic nerve blood flow and oxygen delivery. Anesthesiology 2008;108:864-72.
9. Ross-Cisneros FN, Sultan WC, Asanad S, Sadun AA. Rat model of posterior ischemic optic neuropathy. Invest Ophthalmol Vis Sci 2019;60:2265.
10. Huang TL, Lin KH, Wang JK, Tsai RK. Treatment strategies for neuromyelitis optica. Tzu Chi Med J 2018;30:204-8.
11. Esen E, Sizmaz S, Demir T, Demirkiran M, Unal I, Demircan N. Evaluation of choroidal vascular changes in patients with multiple sclerosis using enhanced depth imaging optical coherence tomography. Ophthalmologica 2016;235:65-71.
12. Spain RI, Liu L, Zhang X, Jia Y, Tan O, Bourdette D, *et al*. Optical coherence tomography angiography enhances the detection of optic nerve damage in multiple sclerosis. Br J Ophthalmol 2018;102:520-4.
13. Higashiyama T, Nishida Y, Ohji M. Optical coherence tomography angiography in eyes with good visual acuity recovery after treatment for optic neuritis. PLoS One 2017;12:e0172168.