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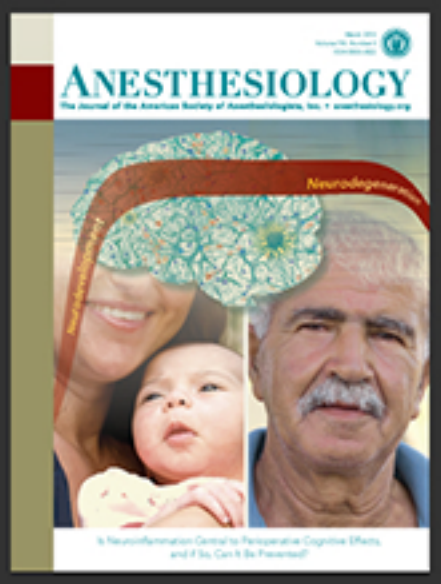

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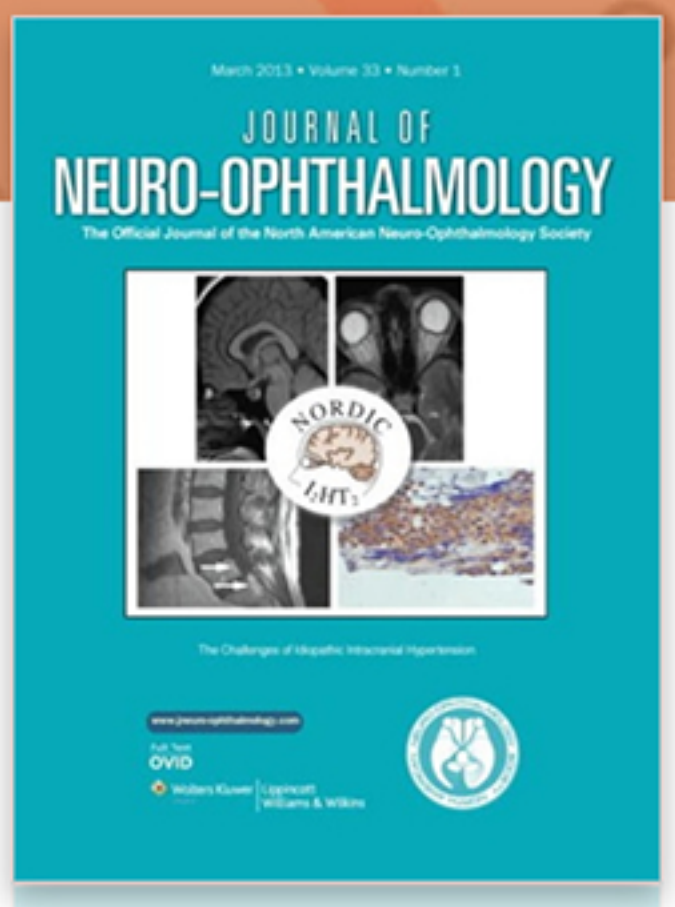

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ORIGINAL CONTRIBUTION

Idiopathic Intracranial Hypertension: Relation Between Obesity and Visual Outcomes

Aimee J. Szewka, MD; Beau B. Bruce, MD; Nancy J. Newman, MD; et al.

Abstract:
Background: Increased body mass index (BMI) has been associated with increased risk of idiopathic intracranial hypertension (IIH), but the relationship of BMI to visual outcomes in IIH is unclear.

Methods: A retrospective chart review of all adult cases of IIH satisfying the modified Dandy criteria seen at our institution between 1989 and 2010 was performed. Demographics, diagnostic evaluations, baseline visit and last follow-up examination data, treatment, and visual outcome data were collected in a standardized fashion. Groups were compared, and logistic regression was used to evaluate the relationship of BMI to severe visual loss, evaluating for interaction and controlling for potential confounders.

Results: Among 414 consecutive IIH patients, 158 had BMI ≥40 (World Health Organization Obese Class III) and 172 had BMI 30–39.9. Patients with BMI ≥40 were more likely to have severe papilledema at first neuro-ophthalmology encounter than those with a lower BMI (P = 0.02). There was a trend toward more severe visual loss in 1 or both eyes at last follow-up among those patients with BMI ≥40 (18% vs 11%, P = 0.067). Logistic regression modeling found that 10-unit (kilogram per square meter) increases in BMI increased the odds of severe visual loss by 1.4 times (95% confidence interval, 1.03–1.91, P = 0.03) after controlling for sex, race, diagnosed hypertension, and diagnosed sleep apnea. Conclusion: Our finding of a trend for severe papilledema and visual loss associated with increasing BMI suggests that very obese IIH patients should be closely monitored for progression of visual field loss.

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Abstract

Background: The authors investigated the correlation of protan and tritan color vision with disease characteristics in Leber hereditary optic neuropathy (LHON). The authors also characterized the therapeutic potential of idebenone in protecting patients from developing dyschromatopsia in LHON.

Methods: Color contrast data of 39 LHON patients participating in a randomized, double-blind placebo-controlled intervention study were evaluated. Patients reported disease onset <5 years before enrolment and were genetically confirmed. Protan and tritan color contrast sensitivity was measured using a computer graphics method in patients receiving idebenone (Catena; 900 mg/d; N = 28) or placebo (N = 11) for 6 months.

Results: Mean age of patients was 28.1 years, 87.2% were men, 76.9% carried the m11778G>A mutation, and mean duration since onset was 2 years. Assessing protan and tritan color vision at baseline revealed a high degree of color confusion even in young patients (<25 years) and with a short history of disease (<1 year). Treatment with idebenone improved tritan color vision compared with placebo ($P = 0.008$ at week 24); a similar trend was seen for protan. The effect of idebenone was most prominent in patients with discordant visual acuity (interocular difference of logMAR >0.2). In this subgroup, the treatment effect at week 24 was 20.4% ($P = 0.005$) in favor of idebenone for the tritan color domain and 13.5% ($P = 0.067$) for the protan domain.

Conclusion: This study confirms that protan and tritan color confusion is an early symptom in LHON. Treatment with idebenone can protect from loss of color vision, particularly in patients who are at imminent risk of further vision loss.

Leber hereditary optic neuropathy (LHON; MIM 535000) causes progressive and mostly irreversible loss of central vision in one eye, followed by a similar loss of vision in the fellow eye within days to months ([1–3]). The painless loss in central visual acuity (VA) is characterized by an enlarging centrocecal scotoma and loss of color vision.

Dyschromatopsia in LHON has been described predominantly as red–green (protan) defect with concomitant loss of blue–yellow (tritan) color contrast sensitivity. Dyschromatopsia results from function loss primarily in small-caliber retinal ganglion cells constituting the papillomacular bundle of the retinal nerve fiber layer (RNFL) ([4,5]). The smallest fibers in the retina belonging to th
highest risk of functi
carried by the koniod
disease ([7]), explaining the subtle differences in the development of protan and tritan color confusion in LHON patients.

4. Carelli V, Ross-Cisneros FN, Sadun AA. Mitochondrial dysfunction as a cause of optic neuropathies. Prog Retin Eye Res. 2004;23:53–89.

LHON is caused in most patients by 1 of 3 primary pathogenic mutations of the mitochondrial DNA (mtDNA: m.11778G>A, m.14484T>C, m.3460G>A), all of which affect complex I (NADH–ubiquinone–oxidoreductase) of the mitochondrial respiratory chain ([1,8,9]). These mutations lead to a defect of ATP synthesis accompanied by increased oxidative stress causing retinal ganglion cell dysfunction and eventually loss ([10,11]). Patients with the m.14484T>C mutation generally tend to have milder disease progression with a 37%–71% chance of some degree of visual improvement, whereas patients with the m.11778G>A and m.3460G>A mutations have a worse prognosis with a much lower (approximately 4%) chance of spontaneous recovery ([2,12,13]).