CASE REPORT

Ozone Treatment in Retinitis Pigmentosa: Effect on Photo Stress and Contrast Sensitivity

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Retinitis pigmentosa is a hereditary disorder that frequently leads to blindness.[1] One of the more prominent effects f retinitis pigmentosa is decreased vision in low light.[2] This characteristic is what often leads patients to seek an eye examination and ultimately to discover the definitive diagnosis of retinitis pigmentosa or related disorder.

Although no cure exists for this disorder, much research is currently in progress, with hopes for promising results. Retinal cell transplantation has demonstrated some positive results and the identification of the loci of the genes for retinitis pigmentosa on the chromosomes is being researched intensively.[3, 4]

Because no cure has been established, treatment programs have been attempted to provide stabilization and visual function improvement to those with this disorder.[5]

The treatment utilizing the infusion of intravenous ozone has been performed at the Clinica Internacional de Retinosis Pigmentaria in Havana, Cuba, under the direction of Dr. Orfilio Pelaez Molino.[6] Although many prominent researchers in the field of retinitis pigmentosa believe that ozone has no benefit in the treatment of retinitis pigmentosa, [7, 8] documented evidence and research efforts did not include the testing performed in this study as a basis for their conclusion.

A 37-year-old white male presented to the Marmer Medical Eye Center on October 6, 1997, to undergo ozone therapy. His primary complaint was his inability to function under low light conditions. Specifically, he described that after arriving at work each morning it was necessary for him to cross through a bright sunlit courtyard before entering his office building and stepping into the elevator. Upon doing this, he was unable to see the elevator buttons to push his floor number and had to resort to the Braille designated panel portion adjacent to the elevator buttons. His best corrected visual acuity with contact lenses was reasonably good at 20/40 in the right eye, 20/30 in the left. Prior to the initiation of ozone therapy, the patient was exposed to a 10-second stimulus photo stress test using the Multivision Contrast Tester stress test unit (model #MCT 8000). The patient recorded a 3:32-second recovery time (Fig 1). Contrast sensitivity was also performed using the Multivision Contrast Tester test unit (model #MCT 8000). The patient responded with an acuity of 20/70 in each eye with day testing, but could not visualize test objects during day testing with peripheral glare (Fig 2). The patient was unable to read the test characters with night testing with and without central glare stimulation (Fig 3).

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PHOTO

STRESS TEST

10 Stimulus Seconds

3:32 Recovery seconds

Fig 1. –Before ozone therapy, patient recovery time during stress test.

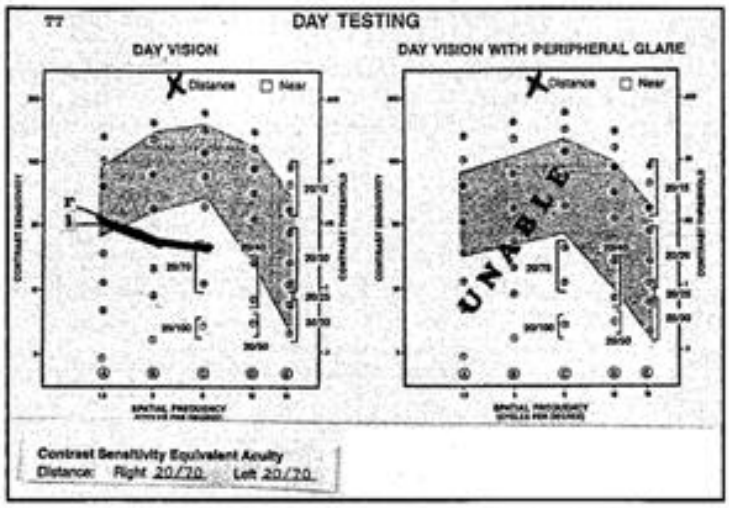

Fig 2.—Before ozone therapy, patient response to contrast sensitivity during day testing. Left, Visual acuity is 20/70 in each eye. Right, Patient is unable to visualize test objects with peripheral glare.

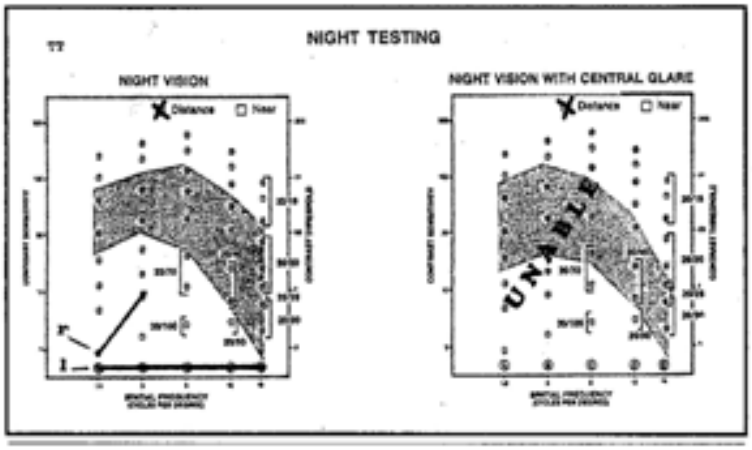

Fig 3.—Patient unable to read test characters during night testing with and without central glare stimulation.

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PHOTO

STRESS TEST

10 Stimulus Seconds

:10 Recovery seconds

Fig 4. –After ozone therapy, patient recovery time during photo stress test.

The patient then underwent ozone therapy. Using the Yanco Ozone Generator (model #GE60/FM5000), oxygen was conducted through the generator and ozone was produced. The ozone was collected in four 50-cc gklass syriunges, with the concentration of ozxone being 50 µg/ml O_2. A phlebotomy was performed in the antecubital vein and 200 cc of the patient's blood was collected through standard sterile tubing into a sterile vacuum collection bottle. The 200 cc of ozone was mixed with the 200 cc of blood and the blood was then reinfused into the patient. The total time of the procedure was approximately 30 minutes. Following 15 applications of ozone, on a schedule of 1 application per day, the above tests were repeated. The best corrected

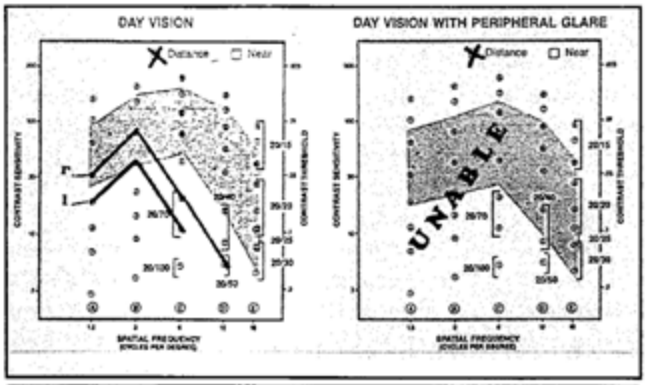

Fig 5.—After ozone therapy, patient response to contrast sensitivity during day testing. Left, Acuity has improved to 20/50 in the right eye. Right, Patient is still unable to respond to day testing with peripheral glare.

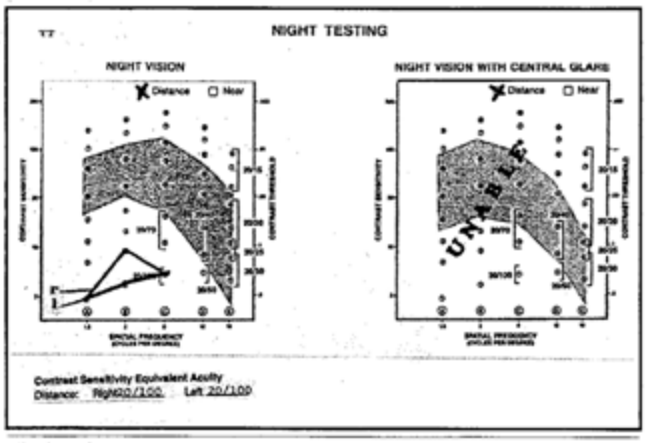

Fig 6.—After ozone therapy, patient response to night testing. Left, Acuity has improved to 20/100 in each eye. Right, Patient is still unable to respond to night testing with central glare.

contact lens visual acuity following the testing measured 20/25 -3 in the right eye, and 20/40 in the left eye. The photo stress test required only 10 seconds of recovery (Fig 4), and the contrast sensitivity also showd an improvement to 20/50 in the right eye and remained 20/70 in the left with day testing (Fig 5), but still was unable to respond to day vision testing with peripheral glare. Whereas before the administration of ozone the patient was unable to see any test characters with night testing, afterward he could discern 20/100 in each eye on night testing (Fig 6) with persistent responsiveness to night vision testing with central glare.

Conclusion

Retinitis pigmentosa is a hereditary degenerative disease that results in progressive peripheral field loss. Although there is no known cure at this time, application of ozone intravenously appears to improve the visual function of contrast sensitivity and glare recovery. The improvements in these areas appear to allow patients with retinitis pigmentosa the ability to function more effectively and more confidently in their daily activities. This includes improved confidence in performing activities in low light situations. The duration of these improvements has not been established; however, these patients have reported that it is their impression that these improvements do last at least several months. Periodic reapplication of ozone appears to be a viable method and may not only allow patients to maintain these improved visual functions but also decrease the rapidity of deterioration that is now anticipated if this type of intervention has not been administered. These results are only preliminary findings; more study is necessary in this area.

References

1. Dukes-Elder S. *Systems of Ophthalmology diseases of the Retina*. London: Henry Kumpton: 1967:577-589.
2. Tasman W, Jaeger EA. Biomedical foundations of ophthalmology. In: Yanoff M, Fine BS, eds. *Duane's Foundations of Clinical Ophthalmology.* Philadelphia, PA: JB Lippincott Co; 1970:34-35.

3. Kaplan HJ, Berger AS, Wolf MI, Del Priore LV. Human photoreceptor transplantation in retinitis pigmentosa: a safety study. *Arch Ophthalmol.* 1997; 115:1168-1172.
4. McGuire RE, Jordan SR, Branden VV, Bouffard GG, Humphries P, Daiger SP. Mapping the RP10 focus for Autosomal dominant retinitis pigmentosa on 7q: refined genetic positioning and focalization within a well-defined YAC contig. *Genome res.* 1996; 6:265-266.
5. Katznelson LA, Khoroskilova-Maslova IP, Eliseyeva RF. A new method of treatment of retinitis pigmentosa/pigmentary abiotrophy. *Ann Ophthalmol.* 1990;22:167-172.
6. Personal communication.
7. Berson EL, Remulla JFC, Rosner B, Sandberg MA, Weigel-Franco C. Evaluation of patients with retinitis pigmentosa receiving electric stimulation, ozonated blood and ocular surgery in Cuba. *Arch Ophthalmol.* 1996; 114:560-563.
8. Weleber RG. The Cuban experiment: false hope for a cure for retinitis pigmentosa. *Arch Ophthalmol.* 1996; 114:606-607.

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Acknowledgment:
This study was conducted with approval of South Fulton Medical Center of East Point, Georgia.