Progressive optic neuropathy in congenital glaucoma associated with the Sirsasana yoga posture

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The authors describe a case report of progressive glaucomatous neuropathy in a patient with congenital glaucoma who routinely practiced the Sirsasana (Headstand) yoga position for several years. The ophthalmic examination included best-corrected visual acuity, anterior segment examination, indirect ophthalmoscopy, ultrasound pachymetry for central cornea thickness, and intraocular pressure (IOP) before, during and after 5 minutes of headstand yoga posture. The IOP increased significantly during the head-down position. Transient elevation in IOP during yoga exercises may lead to progressive glaucomatous optic neuropathy especially in susceptible patients with congenital glaucoma.

INTRODUCTION

Yoga exercises have increased in popularity as a part of an active lifestyle. In addition to physical fitness, yoga has been promoted as an alternative form of therapy for chronic
illnesses. With the increasing utilization of these exercises, physicians need to be familiar with the potential side effects in patients with certain medical conditions.

Recent studies have described an elevation in intraocular pressure following Sirsasana (headstand) yoga posture, particularly in patients with glaucomatous optic neuropathy. However, to the best of our knowledge there are no publications describing progressive glaucomatous neuropathy in congenital glaucoma associated with yoga exercises. We report a case of progressive glaucomatous optic neuropathy in a patient with a history of congenital glaucoma who has been performing the Sirsasana headstand posture during yoga exercises for the past 5 years.

**CASE REPORT**

A 47-year-old Caucasian female with a history of congenital glaucoma, presented with progressive optic neuropathy and decreased visual acuity in the left eye. She had undergone goniotomy in both eyes in the first year of life and filtration surgery on the left eye 17 years prior to presentation. She recently had cataract extraction on her left eye. Her best corrected visual acuity was 20/20 in the right and 20/80 in the left eye, with a refractive error of -1.75 + 2.25 x 160 and -2.00 + 2.75 x 018, respectively. By Goldmann applanation tonometry, the intraocular pressure was 13mmHg in the right and 24mmHg in the left eye. At presentation she was using Alphagan, Betimol, and Travatan in the left eye only. The examination was significant for mild corectopia of the left pupil with a trace afferent pupillary defect and buphthalmos of both eyes. The anterior segment examination was significant for Haab’s striae, left greater right and a superior moderately
elevated avascular bleb in the left eye. The central corneal thickness was 486µ in the right eye and 539µ in the left eye. The IOP was unchanged after dilation in both eyes. The optic nerve showed a small healthy cup in the right eye and larger shallow cupping with a full healthy rim in the left eye.

There was evidence of progressive optic neuropathy and visual field defects in the left eye. The IOP was measured with the Tonopen XL before, during and after the headstand position. The patient’s IOP rose significant from 13 mmHg before the headstand position to 35mmHg in the right eye and from 24 mmHg to 50mmHg in the left eye, during the headstand position. The intraocular pressure after the headstand position decreased to 18 mmHg. The patient would routinely continue this position for ten minutes approximately three times a week.

DISCUSSION

Yoga exercises have being advocated as a complementary and alternative medicine technique for various diseases in many countries. While these exercises are routinely safe and promote good health, there are risks involved for certain patients. Margo et al reported that maintaining a headstand position may predispose patients to vascular thrombosis by intermittently increasing conjunctival venous pressure and decreasing venous outflow. Other more recent reports have been described an increase in intraocular pressure associated with yoga exercises.
In a prospective case observational series, Baskaran et al demonstrated a uniform 2-fold increase in the IOP during Sirsasana, which was maintained during the posture in all age groups irrespective of the ocular biometry and ultrasound pachymetry. Of significance, all seventy five subjects that were observed did not have a prior diagnosis of glaucoma or any glaucomatous optic neuropathy. Additionally, they concluded that normal patients performing the headstand posture do not need routine ocular examinations to monitor for glaucomatous damage. This study did not address how the elevation and fluctuation of IOP may affect patients with pre-existing glaucomatous optic neuropathy. In a similar presentation as our patient, Gallardo et al reported a case of a patient with primary open angle glaucoma, splinter disc hemorrhage and progression of glaucomatous optic neuropathy in association with the Sirsasana headstand posture.

It is difficult to make a direct causal relationship between the headstand posture and glaucomatous progression, but in patients with pre-existing glaucomatous optic neuropathy, the dramatic change in intraocular pressure is potentially harmful. The sudden increase in pressure may cause ischemic as well as mechanical pressure related damage to the optic nerve fibers.

The rise in intraocular pressure that occurs during Sirsasana headstand posture has been documented in multiple studies, but further controlled studies in glaucoma patients are needed to make a direct correlation with progression of glaucomatous optic neuropathy. Regardless, it is an important consideration in patients with seemingly controlled IOP, but with worsening optic neuropathy. Additionally, the Sirsasana headstand posture
should be incorporated into the standard history questionnaire for all patients at risk for glaucoma.

References


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