# Case Report

# Use of Electroconvulsive Therapy in the Presence of GLAUCOMA: A Case Report and Review of Literature

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ABSTRACT

Limited data is available with respect to use of Electroconvulsive therapy (ECT) in the presence of glaucoma. We present a case of severe depression with psychotic symptoms who did not respond to various antidepressant trials and required ECT. His medical history showed that he was diagnosed to have open angle glaucoma and cataract in both the eyes. He had undergone bilateral trabeculectomy and cataract surgery for his ophthalmological aliments. He was safely treated with bilateral ECT, with which he achieved remission.

**Key words:** *ECT, glaucoma, intraocular pressure*

## INTRODUCTION

Recent reports suggest that patients with glaucoma have higher prevalence of depression in contrast to the control group.[1,2] Taking this into consideration, it is expected that more and more patients with glaucoma require treatment for their depression and some of these patients with severe depression may require ECT. Hence it is important to understand the consequences of ECT procedure on intraocular pressure (IOP).

Studies which have evaluated the changes in the IOP during ECT in patients with normal IOP prior to ECT have reported rise in IOP during ECT, although the magnitudes of this elevation is not hazardous and usually returns to the baseline IOP levels after 90 min of seizure activity during the ECT.[3-5] One of the studies which compared the IOP changes during ECT in patients with glaucoma and non-glaucoma patients reported significantly higher elevation of IOP in glaucoma patients.[2] However a small case series involving two patients suggested that rise in IOP is of lesser magnitude in glaucomatous patients than the non-glaucomatous patients.[6]

Evidence in the form of case reports suggests that ECT is usually associated with transient elevation in the IOP[7-9] and occasional reports suggest fall in intra-ocular pressure.[10] In this report we present the case of a patient with open angle glaucoma (who had undergone trabeculectomy for the same) who was treated with ECT and review the existing literature with respect to glaucoma and ECT.

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## CASE REPORT

A 60-year-old man, who had no personal and family history of any mental disorder presented with severe depression with psychotic symptoms (as per International Classifications of Diseases - tenth revision). Exploration of the history revealed that he had been having the depressive symptoms for last 2 years. He had been treated with Tab Escitalopram

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upto 20 mg/day for a period of 6 months and Cap Venlafaxine 225 mg/day along with Tab Olanzapine 7.5-10 mg/day for 13 months without much appreciable improvement.

His medical history revealed that he had symptoms of headache and visual difficulties about 6 months back. There was no temporal relationship of the visual symptoms with increase in the dose of Venlafaxine and Olanzapine. There was no history of raised blood pressure during this period too. On evaluation by an Ophthalmologist, was diagnosed to have open angle glaucoma and cataract in both the eyes. He underwent bilateral trabeculectomy and also had undergone cataract surgery with implantation of the intraocular lens in the posterior chamber about 5 months prior to the index presentation.

On examination he was found to have sad affect, marked psychomotor retardation, depressive cognitions in the form of ideas of hopelessness, worthlessness and suicidal ideations and plans. His physical examination showed raised blood pressure (>130/84 mm of Hg) on many occasions along with raised body mass index (31.5 kg/m$^2$). Ophthalmological examination revealed normal intraocular pressure (right eye: 10 mm of Hg and left eye: 11 mm of Hg), with low filtering bleb in the right eye. Visual acuity was 6/9 in the right eye and 6/18 in the left eye. Fundus was normal in both the eyes with no hypertensive changes. The cup disc ratio was 0.6 in right eye and 0.8 in the left eye.

His investigations in the form of heamogram, blood biochemistry, liver function test, renal function tests, thyroid function test, lipid profile, electrocardiogram, X-ray chest posterior anterior view, ultrasound abdomen and pelvis and electroencephalogram did not show any abnormality. In view of non-response to two antidepressant trials, marked psychomotor retardation, besides the change of antidepressant, option of ECT was discussed with the patient and the family. Following a written informed consent, patient was re-evaluated by the Ophthalmologist. In view of the cup disc ratio of 0.8 in left eye he was prophylactically prescribed Travaprost eye drops (which usually has effect on the IOP after 6 weeks) in the left eye to avoid further damage.

Modified Bilateral ECT was given using an indigenously manufactured brief-pulse, constant-energy machine (Medicaid Systems, India). The machine has settings for adjusting the frequency between 20 Hz and 90 Hz (settings of 20 Hz, 40 Hz, 50 Hz, 60 Hz, 70 Hz, and 90 Hz), duration of current passed (0.1-6 s with increments of 0.1 s), and adjusting the pulse width from 0.1 ms to 1.5 ms. Usually the electrical dose is varied by changing the duration of current while keeping the frequency (at 70 Hz) and pulse width constant (1 ms). ECT was administered thrice a week (Monday, Wednesday, and Saturday). During each ECT procedure electroencephalographic monitoring was done. Both the cuff method and EEG seizures were used to estimate seizure duration. Patient was ventilated with 100% oxygen and all the vitals (pulse, blood pressure, oxygen saturation and electrocardiogram) were monitored closely. After each ECT the monitoring of vitals using the cardiac monitor was continued for 1 h and after that patient's vitals were monitored every 15 min without any complications. Intraocular pressure in both eyes was monitored during the ECT. This was done at about 5 PM on the day of prior to ECT or on the day of ECT. ECT was given between 10.00 A.M. to 11.00 A.M.

Along with ECT, Cap Venlafaxine was tapered off and patient was started on Cap Milnacipran 25 mg/day which was gradually built-up to 125 mg/day. Tab Olanzapine was increased to 12.5 mg/day and Tab Amlodipine 10 mg/day was started for hypertension. During ECT, he was given glycopyrrolate (0.2 mg) as premedication, induction was done with thiopental sodium and in the initial few sessions atracurium was used for muscle relaxation (Table 1 for details), but it was not sufficient to provide adequate muscle relaxation. Resultantly a combination of atracurium and succinylcholine was used for muscle relaxation and after ECT patient was administered neostigmine to reverse the effect of atracurium. Prophylactic use of esmolol was done to avoid hypertensive surge during ECT. Precautions were taken to reduce head movements during ECT. For the initial two ECTs, the response was inadequate (i.e., seizure duration was less than the minimum duration required for effective ECT), hence, thiopentone was replaced with propofol. In total he received 12 ECTs.

Prior to starting of ECT, his intraocular pressure was 10-11 mm of Hg. However, after 3$^{rd}$ ECT the IOP increased to 18 mm and 20 mm of Hg in the right and left eye respectively. However, later monitoring did not show much rise in IOP.

With ECT, Cap Milnacipran and Tab Olanzapine patient achieved remission and was discharged. Over the period of next 3 months, there was no major fluctuation in the intraocular pressure and patient had been maintaining well.

## DISCUSSION

Studies suggest that there is clinically insignificant rise in IOP during ECT in patients with no ocular pathologies.[3-5] Data in the form of case reports since early 1950s suggests that ECT leads to increase in

**Table 1: The electroconvulsive therapy treatment parameters**

| Atracurium in mg | Suxamethonium in mg | Thiopentone in mg | Propofol in mg | Duration of current in seconds | Charge in millicoulombs | Length of EEG seizure | Length of motor seizures |
|---|---|---|---|---|---|---|---|
| 30 | – | 350 | – | 0.6 | 72 | 21 | 0 |
| 45 | – | 400 | – | S1:0.8 | 96 | 0 | 0 |
| | | | | S2:1.2 | 144 | | |
| | | | | S3:1.6 | 192 | | |
| – | 100 | 250 | – | S1:1.8 | 216 | 38 | 27 |
| | | | | S2:2.4 | 287 | | |
| 20 | – | – | 170 | 2.6 | 311 | 40 | 30 |
| 20 | – | – | 170 | 2.8 | 335 | 49 | 35 |
| 20 | 20 | – | 190 | 2.8 | 335 | 56 | 40 |
| 10 | 100 | – | 120 | 2.8 | 335 | 35 | 25 |
| 10 | 100 | – | 160 | S1:2.8 | 335 | 16 | 10 |
| | | | | S2:3.2 | 383 | 24 | 15 |
| 10 | 75 | – | 140 | 3.4 | 407 | 35 | 25 |
| 10 | 100 | – | 160 | 3.4 | 407 | 36 | 25 |
| 10 | 100 | – | 140 | 3.4 | 407 | 35 | 25 |
| 10 | 100 | – | 120 | 3.6 | 431 | 38 | 25 |

S1 – First stimulus; S2 – Second stimulus; S3 – Third stimulus

IOP in patients with glaucoma.[3-5] These case reports have described varied clinical situations in the form of use of ECT in patients after bilateral iridectomy,[7] bilateral iridectomy with cataract surgery in the past,[8] and glaucoma tube implants.[11] Some of the case reports which have evaluated the changes in IOP in glaucoma patients with ECT have discussed the anaesthetic issues. In one of the case report, the authors reported that IOP increased by about 30 mm Hg during ECT.[12] However, this change in IOP almost stabilized when the patient was administered ganglionic blocking medication trimethaphan prior to ECT stimulus.[12] Two case reports described prolonged apnea as a complication of ECT in glaucoma patients receiving echothiophate eye drops.[13,14] Prolonged apnea was attributed to the increased duration of action of succinylcholine due to low levels of serum cholinesterase caused by ecothiophate.[13,14] In another case report authors described the successful use of short-acting nondepolarizing agent atracurium instead of succinylcholine for muscle relaxation.[15] From the above literature it can be concluded that whenever patient with glaucoma are recommended to receive ECT, eye drops like ecothiophate, which is a cholinesterase inhibitor should be avoided. Similarly use of agents like succnylcholine should preferably be avoided.

American Psychiatric Association guidelines,[16] recommend consultation liaison with an ophthalmologist, prophylactic use of medication to keep the intraocular pressure low, avoiding anti-cholinesterases as it prolongs succinylcholine induced apnea, reducing the movements of head to least during ECT and attempts to reduce the hypertensive surge. In the index case a close liaison with the ophthalmologist was maintained, esmolol was used to prevent hypertensive surge and head movements were restricted.

The index case was diagnosed with open angle glaucoma, had undergone bilateral trabeculectomy and cataract surgery prior to ECT. His intraocular pressure was normal prior to ECT. In contrast to the previous reports which have reported rise in IOP upto 30 mm of Hg, in the index case the rise of IOP was in the range of upto 10 mm of Hg. This was possibly due to bilateral iridectomy.

In contrast to previous case reports of prolonged apnea with succinylcholine,[13] we did not encounter the similar adverse effect. This could be possibly due to use of combination of atracurium and succinylcholine in lower doses and use of neostigmine after ECT to reverse the effect. Hence, the index case suggests that succinylcholine may be a safe option for ECT when used in the absence of medications like ecothiophate. Further the case demonstrates the safe use of medications like neostigmine to reverse the effect of muscle relaxants. Like the previous reports, the present case also reflects that iridectomy may have some protective effect on rise in IOP in glaucoma patients undergoing ECT. Previous reports have either have not mentioned the time of assessment of IOP along with ECT.[3] or some have just measured the effect of ECT procedure on IOP immediately after the ictal activity.[3] or after 90 min (Edwards *et al*.[4]). In contrast in the index case we were not able to measure the IOP very close to the ECT procedure, but we measured the same at a fixed time of the day prior to and after ECT and found it useful in monitoring the IOP. However, future studies should focus on the feasibility and usefulness aspect of measuring IOP in glaucoma patients during ECT.

## REFERENCES


1. Yochim BP, Mueller AE, Kane KD, Kahook MY. Prevalence of cognitive impairment, depression, and anxiety symptoms among older adults with glaucoma. J Glaucoma 2012;21:250-4.
2. Mabuchi F, Yoshimura K, Kashiwagi K, Shioe K, Yamagata Z, Kanba S, *et al*. High prevalence of anxiety and depression in patients with primary open-angle glaucoma. J Glaucoma 2008;17:552-7.
3. Epstein HM, Fagman W, Bruce DL, Abram A. Intraocular pressure changes during anesthesia for electroshock therapy. Anesth Analg 1975;54:479-81.
4. Edwards RM, Stoudemire A, Vela MA, Morris R. Intraocular pressure changes in nonglaucomatous patients undergoing electroconvulsive therapy. Convuls Ther 1990;6:209-13.
5. Van Den Berg AA, Honjol NM. Electroconvulsive therapy and intraocular pressure. Middle East J Anesthesiol 1998;14:249-58.
6. Lincoff HA, Ellis CH, Devoe AG, Debeer EJ, Impastato DJ, Berg S, *et al*. The effect of succinylcholine on intraocular pressure. Am J Ophthalmol 1955;40:501-10.
7. Manning EL, Hollander WM. Glaucoma and electroshock therapy. Am J Ophthalmol 1954;37:857-9.
8. Nathan RS, Dowling R, Peters JL, Kreps A. ECT and Glaucoma. Convuls Ther 1986;2:132-3.
9. Ottosson JO, Rendahl I. Intraocular pressure in electroconvulsive therapy. Arch Ophthalmol 1963;70:462-5.
10. Mather RW, Shuman CR, Harrison SI. Influences of curare and eserine on ocular tension following electroshock. Am J Ophthalmol 1954;37:859-66.
11. Song J, Lee PP, Weiner R, Challa P. The effect of surgery on intraocular pressure fluctuations with electroconvulsive therapy in a patient with severe glaucoma. J ECT 2004;20:264-6.
12. Ottosson JO, Rendahl I. Effect of trimethaphan on intraocular pressure in electroconvulsive therapy. Arch Ophthalmol 1963;70:466-70.
13. Packman PM, Meyer DA, Verdun RM. Hazards of succinylcholine administration during electrotherapy. Arch Gen Psychiatry 1978;35:1137-41.
14. Chessen DH, Geha DG, Salzman C. ECT, glaucoma, and prolonged apnea. Dis Nerv Syst 1974;35:152-3.
15. Messer GJ, Stoudemire A, Knos G, Johnson GC. Electroconvulsive therapy and the chronic use of pseudocholinesterase-inhibitor (echothiophate iodide) eye drops for glaucoma. A case report. Gen Hosp Psychiatry 1992;14:56-60.
16. American Psychiatric Association Committee on ECT. The Practice of Electroconvulsive Therapy: Recommendations for Treatment, Training, and Privileging. 2nd ed. Washington, DC: American Psychiatric Association; 2001. p. 41.


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