

## Comparative study of dorzolamide and latanoprost on intraocular pressure reduction in patient with age 30-50 years

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### Abstract

Anti Glaucoma agents used for reduction of intraocular pressure. The present work carried out to compare effect of dorzolamide and latanoprost on intraocular pressure reduction in patient with age 30-50 years. In present work done by me Anti Glaucoma effect of drugs namely dorzolamide and latanoprost was compared with the control and with each other. Analysis of variance and student t-Test, ANOVA were applied to compare the result. It was found that intraocular pressure varied significantly across the three groups ( $p=0.000$ ) compared to control group intraocular pressure was significantly less in both dorzolamide and latanoprost groups ( $p=0.000$ ) reduction of intraocular pressure with dorzolamide was less in comparison to that of latanoprost ( $p=0.011$ ) and the end of work. latanoprost is more efficacious than dorzolamide as far as intraocular pressure reduction is concerned.

**Keywords:** Dorzolamide, Latanoprost, Anti Glaucoma effect.

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### Introduction

Glaucoma is a slowly progressive insidious optic neuropathy that usually is associated with chronic elevation of intraocular pressure. The rise in intraocular pressure is due to the increase in aqueous formation, low rate of outflow or a raised pressure in the episcleral veins [1]. Retinal ganglion cell death is initiated when some pathologic event blocks the transport of growth factor from the brain to the retinal ganglion cell. These neurotrophins initiate a damage cascade and the cell is unable to maintain normal functions. Patient risks for glaucoma are those with affected first degree relatives, diabetes and myopes should be examined after the age of 40 years. Essentials of Diagnosis are rapid onset in older age groups, particularly hyperopes and Asians, Severe pain and profound visual loss, Red eye, steamy cornea, dilated pupil, Hard eye. In General Considerations, primary acute angle-closure glaucoma can occur only with closure of a preexisting narrow anterior chamber angle, as is found in elderly persons (owing to physiologic enlargement of the lens), hyperopes, and Asians. About 1% of people over age 35 have narrow anterior chamber angles, but many of these never develop acute glaucoma; thus, the condition is uncommon [2]. Angle closure is associated with pupillary dilation and thus might occur from sitting in a darkened movie theater, at times of stress (owing to increased circulating epinephrine), from pharmacologic mydriasis for ophthalmoscopic examination, or from systemic anticholinergic medications such as atropine (eg, preoperative medication), antidepressants, or nebulized bronchodilators.

Acute angle-closure glaucoma may also occur secondary to long-standing anterior uveitis or dislocation of the lens. Symptoms are the same as in primary acute angle-closure glaucoma, but differentiation is important because of differences in management.

In Clinical Findings patients with acute glaucoma usually seek treatment immediately because of extreme pain and blurred vision, though there are subacute cases in which presentation is delayed. The blurred vision is characteristically associated with halos around lights. Nausea and even abdominal pain may occur, and acute glaucoma must be remembered in the differential diagnosis of abdominal discomfort and vomiting [3]. The eye is red, the cornea steamy, and the pupil moderately dilated and nonreactive to light. Tonometry (or palpation of the globe) reveals elevated intraocular pressure. As far as treatment is concerned, it is divided into two parts

#### A. Primary

In primary acute angle-closure glaucoma, laser peripheral iridotomy will usually result in permanent cure. Intraocular pressure must be lowered beforehand. A single 500 mg intravenous dose of acetazolamide, followed by 250 mg orally four times a day, is usually sufficient. Osmotic diuretics, such as oral glycerol and intravenous urea or mannitol—the dosage of all three being 1-2 g/kg—can be used if necessary. Once the intraocular pressure has started to fall, topical 4% pilocarpine, 1 drop every 15 minutes for 1 hour and then four times a day, is used to treat the underlying angle closure. The fellow eye should undergo prophylactic iridectomy.

#### B. Secondary

In secondary acute angle-closure glaucoma, systemic acetazolamide is also used, with or without osmotic agents, to control intraocular pressure. Further treatment is determined by the cause.

Untreated acute glaucoma results in severe and permanent visual loss within 2-5 days after onset of symptoms. Most patients blinded by the disease were blind at the time of diagnosis. Open-Angle Glaucoma is insidious in onset in older age groups. No symptoms in early stages, Gradual loss of peripheral vision over a period of years, resulting in tunnel vision. Persistent elevation of intraocular pressure associated with pathologic cupping of the optic disks [4]. "Halos around lights" are not present unless the intraocular tension is markedly elevated.

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In open-angle glaucoma, the intraocular pressure is consistently elevated due to abnormal drainage of aqueous through the trabecular meshwork. Over a period of months or years, this results in excavation ("cupping") and pallor of the optic disk with loss of vision varying from slight constriction of the peripheral fields to complete blindness. The cause of the decreased rate of aqueous outflow in primary open-angle glaucoma has not been clearly established. However a number of mutations, such as in the myocilin gene on chromosome 1, have been identified in a small proportion of cases. The disease is bilateral, and there is an increased prevalence in first-degree relatives of affected individuals and in diabetics. Primary open-angle glaucoma occurs at an earlier age, is more frequent in blacks, and may result in more severe optic nerve damage. There is increasing evidence that factors other than the level of intraocular pressure—particularly vascular abnormalities—may play a role in the pathogenesis of glaucomatous optic nerve damage. Open-angle glaucoma may also develop secondary to other eye disease, such as uveitis or the effects of trauma. Elevation of intraocular pressure is also a complication of steroid therapy, whether it be topical, systemic, inhaled, or administered by nasal spray[5]. In the USA, it is estimated that 1-2% of people over 40 have glaucoma; about 25% of these cases are undetected. Over 90% of all cases of glaucoma are of the open-angle type. Patients with open-angle glaucoma have no symptoms initially. On examination, there may be slight cupping of the optic disk observed as an absolute increase—or an asymmetry between the two eyes—of the ratio of the diameter of the optic cup to the diameter of the whole optic disk (cup-disk ratio). (Cup-disk ratio of greater than 0.3 or asymmetry of cup-disk ratio of 0.2 or more is suggestive of glaucoma.) Changes in the retinal nerve fiber layer may be observed as an earlier finding in some patients. The visual fields gradually constrict, but central vision remains good until late in the disease. Tonometry, ophthalmoscopic visualization of the optic nerve, and central visual field testing are the best studies for the diagnosis and follow-up. The diagnosis of glaucoma generally depends upon identification of consistent abnormalities in at least two of these parameters. The normal range of intraocular pressure is 10-24 mm Hg. Except in acute cases, the diagnosis of glaucoma is not made on the basis of one tonometric measurement. Intraocular pressure is influenced by various factors, including posture and diurnal variation. In many individuals, elevated intraocular pressure is not associated with optic disk or visual field abnormalities. These ocular hypertensives are at increased risk of developing glaucomatous damage but may only need regular observation. Conversely, a significant proportion of patients with glaucoma have normal intraocular pressure when it is first measured, and it is only repeated measurement which identifies the abnormally high pressure. Furthermore, there are patients with normal tension glaucoma, in which the intraocular pressure is always within the normal range despite repeated measurement even though they have glaucomatous optic disk and visual field abnormalities[6]. There are many other causes of optic disk abnormalities or visual field changes that mimic glaucomatous damage, and visual field testing may prove unreliable in some patients, particularly the elderly. Taken together, these factors mean that the diagnosis of glaucoma is not always straightforward, which greatly hampers the effectiveness of screening programs. All persons over age 40, particularly blacks, should have tonometric and ophthalmoscopic examinations every 3-5 years. In diabetics and in individuals with a family history of glaucoma, annual examination is indicated. As far as Treatment is concerned topical  $\beta$  adrenergic blocking agents, such as timolol 0.25% or 0.5%, carteolol 1%, levobunolol 0.5%, and metipranolol 0.3% solutions twice daily, or timolol 0.5% gel once daily, are still the most commonly used antiglaucoma agents. They are contraindicated in patients with reactive airway disease or heart failure.  $\beta_1$  receptor selective blocking agent, is theoretically safer in patients with reactive airway disease but is less effective at reducing intraocular pressure. Brimonidine 0.2%, a selective  $\alpha_2$  agonist, dorzolamide 2%, a topical carbonic anhydrase inhibitor, and latanoprost 0.005%, a prostaglandin analog, can all be used in addition to a  $\beta$ -

blocker (brimonidine and dorzolamide twice daily and latanoprost once daily) or as initial therapy when  $\beta$ -blockers are contraindicated (brimonidine twice daily, dorzolamide three times daily and latanoprost once daily). Brimonidine and dorzolamide are associated with allergic reactions, and latanoprost produces permanent darkening of iris and eyebrow color, but its efficacy and once-daily application have made it increasingly popular. Travaprost, a similar agent approved in mid 2001, has the potential advantage of being more heat stable. Retrospective review of pre-release data suggests that it may be more effective in blacks. Bimatoprost, also FDA-approved in 2001, has molecular similarity to prostaglandins but does not activate receptors for these compounds[7]. It also is thermally stable and appears to increase drainage of intraocular fluid by two mechanisms—a possible benefit therapeutically. Apraclonidine, 0.5-1%, another  $\alpha_2$  agonist, can be used three times a day to postpone the need for surgery in patients receiving maximal medical therapy, but long-term use is limited by a high incidence of allergic reactions. It is more commonly used to control acute rises in intraocular pressure, such as after laser therapy. Epinephrine, 0.5-1%, and the prodrug dipivefrin, 0.1%, are being used much less frequently, particularly because of allergic reactions and their adverse effects on the outcome of subsequent glaucoma surgery. Pilocarpine 1-4% (and sometimes higher concentrations in patients with dark irides) four times a day is little used because of the induced myopia in younger patients and the pupillary constriction that compromises vision in patients with cataract. Oral carbonic anhydrase inhibitors (eg, acetazolamide) may still be used on a long-term basis if topical therapy is inadequate and surgical or laser therapy is inappropriate. Laser trabeculoplasty is used as an adjunct to topical therapy to defer surgery and is also advocated as primary treatment. Surgery is generally undertaken when intraocular pressure is inadequately controlled by medical and laser therapy, but it may also be used as primary treatment. Trabeculectomy is the standard procedure. Adjunctive treatment with subconjunctival fluorouracil or mitomycin is used peri- or postoperatively in difficult cases. Viscocanalostomy and deep sclerectomy, two alternative procedures under investigation, have the advantage of avoiding a full-thickness incision into the eye[8]. Untreated chronic glaucoma that begins at age 40-45 will probably cause complete blindness by age 60-65. Early diagnosis and treatment will preserve useful vision throughout life in most cases. The glaucoma is seen in two principal clinical forms (a) Open angle (b) Angle closure. Factors modifying intraocular pressure are

(1) physiological variation (b) local mechanical factors and (c) pharmacological factors. The normal intra ocular pressure of an individual ranges from 10-20 mmHg and can rise up to 60 mmHg in glaucoma patients. The management aspect includes lowering of intra ocular pressure by reducing secretion of aqueous humor or by promoting its drainage. Five general groups of drugs cholinomimetics. Alpha agonists, beta-blocker. Prostaglandin F2 alpha analogue and diuretics-have been found to be useful in reducing intraocular pressure. The latanoprost works by increasing uveoscleral outflow of aqueous. It can be used in normal pressure glaucoma also where as dorzolamide acts by decreasing aqueous secretion.

#### Material and methods

- 1) Patients of either sex with age between (30-50) years were taken for study.
- 2) For measurement of intraocular pressure:- Goldmann Applanation tonometer was used. Drug dose which was taken were namely latanoprost (.0025%), Dorzolamide (1%) .

This work was done during the period from June to July 2021 in the department of pharmacology, D.M.CH, LAHERIASARAI.

#### Statistical analysis

Data were presented in mean  $\pm$  SEM and were analysed using statistical package for social scientists 10 (SPSS). Student's t-test and ANOVA were applied to compare significance between different groups ( $p < 0.05$ ).

#### Results and discussion

Intraocular pressure with control, latanoprost and dorzolamide were ( $19.58 \pm .62$ ), ( $13.2 \pm .56$ ) and ( $14.80 \pm .40$ ) respectively, one hour

after drug administration. The mean intraocular pressure in three groups varied significantly [ $F(2,26) = 140.048$   $p = .000$ ]. The mean intraocular pressure of latanoprost group was significantly less than control group [ $t(18) = 8.04$   $p = .000$ ] It was also significantly less in the dorzolamide group in comparison to control group [ $t(18) = 6.44$   $p = .000$ ]. However the mean intraocular pressure in latanoprost was found to be significantly less in comparison to dorzolamide group [ $t(18) = 2.60$   $p = .014$ ]. Similarly at half an hour during experiment intraocular pressure with control, Latanoprost and dorzolamide were ( $22.0 \pm .62$ ), ( $15.44 \pm .62$ ) and ( $17.30 \pm .11$ ). The mean intraocular pressure in three groups varied significantly [ $F(2, 27) = 43.456$   $p = .000$ ]. The mean intraocular pressure of latanoprost group was significantly less than control group [ $t(18) = 7.42$   $p = .000$ ]. It was also

significantly less in the dorzolamide group in comparison to control group [ $t(18) = 7.44$   $p = .000$ ]. Also mean intraocular pressure in latanoprost was found to be significantly less in comparison to dorzolamide group [ $t(18) = 2.80$   $p = .011$ ]. Niazi MK et al. in year 2004 did work on intraocular pressure reduction. After three months latanoprost reduce mean base line intraocular pressure from  $27.0 \pm 2$  mmHg by  $8.48 \pm 3.26$  mmHg. The similar figures for dorzolamide were  $27.4 \pm 3.4$  and  $5.6 \pm 2.6$ . The difference of 2.8mmHg (95% CI : 2.4 - 3.6) was highly significant  $p < .001$ [9]. It was evident that latanoprost was superior to dorzolamide as far as intraocular pressure reduction was concerned.

**Table 1 : Effect on Intraocular Pressure after Instillation of Different Drugs on Day 30**

No. of Patient in each group	Drugs Used	IOP before drug administration	IOP 1/2 Hr. after Drug administration	IOP 1 Hr. After Drug administration
10	Control	24.78 ± .80	22.0 ± .62	19.58 ± .62
10	Latanoprost (.0025)%	24.62 ± .72	15.44 ± .62	13.2 ± .56
10	Dorzolamide (1%)	24.58 ± .10	17.30 ± .11	14.80 ± .40

### Conclusion

From above observations it was evident that latanoprost was more efficacious than dorzolamide as far as intraocular pressure is concerned.

### Ethical considerations

Ethical issues (including plagiarism, consent misconduct, data fabrication and /or falsification, double publication and / or submission, redundancy etc.) have been completely observe by the other. Ethical clearance to conduct the study was obtained from ethical committee of Darbhanga Medical Collage and Hospital Laheria Sarai, Bihar, India.

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