TRABECULECTOMY:-

THE FIRST OPTION IN THE MANAGEMENT OF AFRICAN PATIENTS

WITH PRIMARY OPEN ANGLE GLAUCOMA

By

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DEDICATED TO;

- My husband; Simon, without whose untiring support this dissertation would not have been possible.

- My sweet daughters Zawadi and Kate whose innocent smiles gave me strength.

- My parents who are a source of my inspiration.

DECLARATION

I hereby declare that this dissertation is my own original work and has not been submitted for a degree or diploma in any other university.

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ABSTRACT

This study was conducted among 93 Tanzanian African eye patients with primary open angle glaucoma; at Muhimbili Medical Centre between November, 1992 and October, 1993. The main objective was to study the effectiveness of trabeculectomy as the first option in the management of primary open angle glaucoma in African patients.

The study revealed the prevalence of primary open angle glaucoma at the time of study in Muhimbili Medical Centre to be 16%.

Trabeculectomy was done in 52% of patients studied with a success rate of 84% with minimal postopeative complications. It was found out in this study that trabeculectomy significantly lowered the intraocular pressure ($P << 0.001$) and hence reducing the progression of visual field and visual acuity loss.

In conclusion, results obtained and analysed in this study confirm that

- there is a high prevalence of primary open angle glaucoma among African patients attending the Muhimbili Medical Centre Eye Clinic,

- primary open angle glaucoma is one of the common cause of blindness in Tanzania African patients,

- most patients are not aware of the onset of the disease until late when other eye imparements are manifested,

- trabeculectomy proved to be an effective treatment in lowering intraocular pressure and hence preventing further damage to the eye and consequently preserving visual field and vision; so proving the objective of the study.

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1. DEFINITION, INTRODUCTION AND CLASSIFICATION

1.1 Definition:

Primary open angle glaucoma is a chronic ocular condition characterised by a raised intraocular pressure above a level compatible with the continued health and function of the eye. It is associated with a gonioscopically open angle and a reduced facility of aqueous outflow. It is not caused by any obvious disease of the eye. If present over a sufficient period of time it causes characteristic pathological changes at the optic disc (glaucomatous optic cupping) and visual field changes. Untreated primary open angle glaucoma ultimately leads to absolute glaucoma and irrevesible blindness(4,5).

1.2 Introduction:

Primary open angle glaucoma is the commonest form of glaucoma in many countries (1). In Dar es Salaam open angle glaucoma and cataract are the major causes of blindness. Other causes include corneal opacity, trauma, optic atrophy and macular degeration (2).

In most cases the intraocular pressure is elevated above the normal range, reflecting a reduced aqueous humour outflow facilities.

The mechanism by which elevated intraocular pressure damages the optic nerve, is not clear but most clinicians believe that ischaemia of the optic disc, direct mechanical compression of axons or both are responsible. Further research is necessary to determine whether one theory or both theories in combination explain the damage to the optic nerve (4).

This disease has a hereditary component and becomes more prevalent with age and because primary open angle glaucoma is asymptomatic until late in its course, affected individuals can develop severe damage before

they have an eye examination. Many cases of primary open angle glaucoma are discovered through screening programs or on routine ocular examination (4).

1.3 Classification:

The primary glaucomas by definition are not associated with known ocular or systemic disorders which cause the increased resistance to aqueous outflow. The primary glaucomas usually affects both eyes and may be inherited.

A number of classification schemes of the glaucomas have been proposed. They are based on the age of the patient (infantile, Juvenile, adult), the site of obstruction to aqueous outflow (pre-trabecular, trabecular, post-trabecular) and on aetiology.

In open-angle glaucoma there is impaired flow of aqueous humour through the trabecular meshwork - Schlemm's canal - venous system.

Open angle glaucoma can be classified as:-

1. Primary open angle glaucoma:
   Optic nerve damage and visual field loss occur due to increased levels of intraocular pressure. The cause of trabecular obstruction is not known.
2. The glaucoma suspect:
   Elevated intraocular pressure with normal optic disc and visual fields.
3. Normal tension glaucoma:
   Optic nerve and visual field loss occur at "normal" levels of intraocular pressure.
4. Secondary open angle glaucoma:

a) Trabecular meshwork obstruction or malfunction identifiable and associated with another ocular condition e.g. pigmentary glaucoma, phacolytic glaucoma.

b) Increased episcleral venous pressure e.g.; carotid cavernous sinus fistula

2. OBJECTIVES:

The objectives of this study were to determine:-

1. The prevalence of open angle glaucoma among African patients attending Muhimbili Medical Centre eye clinic from November, 1992 to October, 1993.
2. The ocular symptoms of patients with open angle glaucoma seeking eye treatment.
3. The visual status of patients with open angle glaucoma at the time of seeking eye treatment.
4. To determine how effective Trabeculectomy is in lowering intraocular pressure and maintaining it at acceptable levels among African patients with open angle glaucoma.
5. The success of Trabeculectomy in preserving the visual fields in African patients with open angle glaucoma.
6. To find out the intraoperative and post-operative complications of Trabeculectomy in African patients with open angle glaucoma.

3. REVIEW OF LITERATURE

3.1 Epidemiology:

Prevalance and Incidence:

Most studies performed in Western Europe and in the United States show the prevalence of primary open angle glaucoma in the population above 40 years to range from 0.5% to 1% (4). Various studies have reported different prevalences. This difference is attributed to the population sample, the age of the individuals studied, technique of examination and the definition of glaucoma employed (4). An intraocular pressure of more than 21mmHg is found in 2% of the population but only 5 - 10% of these develop cupping of the optic discs and loss of visual fields (5).

A raised pressure without optic nerve changes and with normal field is defined as ocular hypertension. Typical glaucomatous optic nerve changes and field loss may be found in eyes with normal pressure a condition termed low tension glaucoma.

The difficulties in diagnosis based on the measurement of the intraocular pressure (as pointed above) are further worsened by the difficulty in accurately testing the visual field.

Primary open angle glaucoma is the second cause of blindness (first in the black population) in the United States and the cause for 8.5% of visits to the ophthalmologists (6). The incidence of primary open angle glaucoma in the general population has not been measured adequetly. Data from the Famingham Study (4); estimated the incidence of open angle glaucoma to be 0.2% at age 55 and rises to 1.1% at age 70.

A number of studies done in Europe and North America showed an incidence of 1% to 2% in the population over the age of 40 and that it increased with age (4). The prevalence of open angle glaucoma in Beaver Dam is similar to other white population that is 2.1% (6) and was found to increase with age (from 0.9% in people of 43 to 54 years of age to 4.7% in people 75 years of age or older) (6).

Age:

The prevalence of primary open angle glaucoma increases with age. However, it is not limited to middle age and older individuals only as it occurs in children and young adults as well (4). Its overall incidence increases rapidly from the age of 40 to 70 and thereafter becomes less common. The condition is rarely found under the age of 40 years (7). The effect of age on the prevalence of open angle glaucoma holds true even after compensating for the relationship between increasing age and increasing intraocular pressure (8, 9).

The occurance of the disease in young people is referred to as juvenile glaucoma. Juvenile glaucoma may represent cases of delayed forms of congenital glaucoma, early onset form of primary open angle glaucoma or other forms of glaucoma.

Sex:

Conflicting information exists on the prevalence of open angle glaucoma in the sexes. Males (10 - 14) have a higher prevalence of primary open angle glaucoma which is surprising because intraocular pressure is generally higher in females.

Other investigators found an equal prevalence of primary open angle glaucoma in the sexes (15, 16) and even a higher prevalence in females (17,

18). There is no marked sex difference in the incidence of simple open angle glaucoma but in most reported series there appears to be a slight preponderance of males (19). The preponderance in males is more marked in patients under 50 years of age (20).

Race:

Recent studies show a higher prevalence of primary open angle glaucoma in blacks than in whites (21). Further more the disease seems to develop at an earlier age and has a more rapid progression in black patients (22 - 29).
It is estimated that the incidence and prevalence of blindness from glaucoma are eight times higher in black patients than in white patients in the United States (30).
Wilson and others (228) in a study done at Boston, USA,studied race as a risk factor for progressive glaucomatous damage. The results of the study indicated that open angle glaucoma is a more severe disease in blacks than in whites. At the initial diagnosis, the disc damage and visual field loss appear to be worse in the eyes of the blacks. Also progression of the glaucomatous disease was more marked in the eyes of black patients. Significantly at initial diagnosis the black patients were younger, which suggests that glaucoma has an earlier onset in black patients than in white patients.

Data on the prevalence of primary open angle glaucoma in other ethnic and racial groups are less complete. It is stated that primary open angle glaucoma is rare in Pacific Islanders (31, 32) Asians (33, 34, 24) and certain native American tribes. For better comparison surveys utilizing standardized technique and definitions are needed.

Bilaterality:

The earlier reports in the literature suggested that open angle glaucoma sometimes affected one eye. In a series of 1,023 cases Lehrfeld and Reber (15) found that on their first appearance at hospital 50.7% of patients showed unilateral and 49.3% bilateral involvement.

Since more accurate methods of diagnosing glaucoma early have come into use, and when the eyes are observed for a longer time the condition is found to be bilateral though one eye may be affected some considerable time before its fellow eye. It must be concluded that primary open angle glaucoma is essentially a bilateral condition (5).

Refractive Error:

Myopia has been associated with primary open angle glaucoma in many studies (9, 35 - 38). It is not clear whether myopia has a direct influence on the prevalence of the disease or acts through its known association with increased intraocular pressure and large cup/disc ratio (39, 40). It is often difficult to diagnose glaucomas in myopic individuals because they have:-

- broad, shallow optic cups with less distinct margins.
- baring of the blind spot or other refractive scotomas on visual field testing and,
- low ocular rigidity which makes Schiotz reading inaccurate.

Heredity:

Primary open angle glaucoma appears to have a genetic or familial component. Over the years autosomal dominant (41, 42), autosomal recessive (43, 44), and sex linked (14) inheritance patterns have been reported. Currently most authorities believe that the genetic influence occurs through polygenic or multifactorial transmission. It is reported that

5% to 50% of cases of open angle glaucoma are hereditary with the best estimation being 20 - 25% (45). The risk of developing primary open angle glaucoma in first degree relative is 4 - 16% (41, 45 - 52).

One study, reported that a maternal history of glaucoma had a greater impact on the inheritance of primary open angle glaucoma than did a paternal history (53). This finding was not supported by a more recent and more detailed study of the inheritance of glaucoma (54).

It appears probably that, the genetically determined steroid induced ocular hypertension bears some relationship to simple glaucoma although this is indirect (55). It is also possible that the conflicting reports on this relationship and also on the transmission of the disease are indication of the genetic heterogenecity of simple glaucoma and also of its possible multifactorial inheritance. The situation is further complicated by observation that the cup disc ratio of optic nerve is probably genetically determined in multifactorial manner (56) and that the intraocular pressure and the facility of aqueous outflow in normal eyes are similarly transmitted (56).

It is now established that simple glaucoma is genetically determined and that the appreciable familial incidence of this condition leads us to examine with particular care the relatives of affected individuals, so that if the disease develops it is diagnosed and treated at an early stage.

3.2 Pathophysiology of Open Angle Glaucoma:-

1. <u>Reduced aqueous humour outflow:-</u>

   Decreased facility of aqueous humour outflow has generally been accepted to be the cause of the increased intraocular pressure seen in in most cases of primary open angle

glaucoma. Hypersecreation of aqueous humour is a rare cause of open angle glaucoma.

Experiments by Grant (4)on enucleated human eyes have demonstrated that by incising the entire trabecular meshwork the resistance of outflow is reduced by 75%. Inference drawn from this observation is that the increased resistance to outflow seen in glaucoma must also lie between the anterior chamber and the lumen of canal of Schlemm (86). Other authorities however propose that outflow facility is reduced because the trabecular meshwork prolapses into Schlemm's canal thus occluding the lumen and this prevents circumferential flow of aqueous humour into the collector channel's (88 - 91). However no occlusion of Schlemm's canal has been proven to happen when intraocular pressure is in the range of 25 to 35 mmHg (89), which is the situation in most eyes with primary open angle glaucoma. Thus the collapsing of the canal cannot be a feature in open angle glaucoma.

The decreased outflow facility in open angle glaucoma has at times been ascribed to obstruction of intrascleral collector channels as caused by accumulation of glycossaminoglycans in the adjacent sclera (92 - 97). Karsnov (96) believes that primary open angle glaucoma is infact a disease caused by several different diseases with different sites of resistance and that obstruction of the collector channels accounts for approximately 50% of the cases of primary open angle glaucoma.

Experiments (91) have however refuted this hypothesis by demonstrating that unroofing Schlemm's canal did not reduce resistance to outflow in glaucomatous eyes until the canal was entered. Hence no sclera blockage to outflow of aqueous.

If we accept the hypothesis that the trabecular meshwork or the endothelium of Schlemm's canal is the site of the increased resistance to outflow in primary open angle glaucoma, then theories proposed to explain this could be:-

1. (a) Obstruction of the trabecular meshwork by foreign materials which accumulates in the trabecular meshwork and juxtacannalicular tissue e.g.

   Pigment, Red blood-cells, glycosaminoglycans, amorphous material, Protein.

   (b) Obstruction by a normal constituent that is catabolised insufficiently or synthesized excessively.

2. A loss of trabecular endothelial cells interfering with various important trabecular functions including phagocytosis and synthesis and degradation of macromolecules. Lack of complete endothelial covering could thus allow the trabecular beams to fuse and hence narrow the spaces between the beams (intertrabecular spaces).

3. Loss of giant vacuoles in the inner wall of Schlemm's canal thus preventing movement of fluid from the meshwork into the lumen of Schlemm's canal (98 - 101).

4. A decrease in the permeability of the trabecular meshwork.

5. A loss of normal phagocytic activity due to the endothelial cells being underactive or overwhelmed by foreign material leading to death or migration from beams.

Histology of the eyes from patients with primary open angle glaucoma has revealed abnormalities which include;

(a) Alterations in the trabecular beams, increased fragmentation of collagen, increased curly and long spacing collagen, and coiling of fibres bundles (91, 92, 102).

(b) Thickened basement membrance.

(c) Narrowed intertrabecular space (101, 103 - 105).

(d) Fused trabecular beams (106).

(e) Reduced actin fillaments (107).

(f) Accumulation of foreign materials.

(g) Decreased number of giant vacuoles.

(h) Narrowing of the collector channels.

(i) Closure of Schlemm's canal.

(j) Thickened scleral spur.

As most specimens are obtained during surgery (thus producing multitudes of artifacts) and from eyes with advanced damage, these histopathologic changes should be interpreted with caution as it is difficult to know whether the changes seen are primary phenomenon or secondary to the effects of intraocular pressure, the medical or surgical treatment.

At times it has been suggested that outflow changes of primary open angle glaucoma could be an acceleration of the normal aging process (108).

Fundamental defects of aqueous humour outflow in primary open angle glaucoma are difficult to ascertain. However, the balance of evidence favours the site of increased resistance to be in the trabecular meshwork. Various investigations have linked the increased resistance to outflow with:-

1. Altered corticosteroid metabolism.

   Following the description of topical corticosteroid induced intraocular pressure elevation by Armaly (109), Becker and Hahn (110), it was proposed that the intraocular pressure response to topical corticosteroids was inherited the same way as, or closely linked to, the inheritance of primary open angle glaucoma (104, 110). The corticosteroids hypothesis was then extended to include patients with primary open angle glaucoma. Various investigators noted that patients with primary open angle glaucoma had:-

(i) Increased plasma levels of cortisol (111).

(ii) Increased suppression of plasma cortisol with different doses of exogenous dexamethasone (112).

(iii) Continued suppression of plasma cortisol by dexamethasone despite concomitant administration of diphenylhydrantoin (phenytoin) (113, 114).

(iv) Disturbed pituitary adrenal axis function (115).

(v) Increased inhibition of nitrogen - stimulated lymphocyte transformation by glucocorticoids (116, 117).

The corticosteroid hypothesis was challenged when subsequent studies in glaucomatous patients (118 - 124) failed to confirm the increased sensitivity of glucosteroids to monocular tissues. Also intraocular pressure response to topical corticosteroids was shown to lack reproducibility (125).

2. Dysfunctional adrenergic control.

Following the corticosteroid theory it has also been proposed that diminished outflow facility in patients with primary open angle glaucoma could be explained by an increased sensitivity to adrenergic agonists.

It has been indicated that patients with primary open angle glaucoma have:-

(i) a greater response to epinephrine or theophyline in inhibiting nitrogen stimulated lymphocyte transformation.

(ii) a greater intraocular pressure reduction after the administration of topical epinephrine.

(iii) more frequent premature ventricular contraction after topical administration of epinephrine.

Ocular hypertensive subjects demonstrating intraocular pressure greater than 5 mmHg after topical epinephrine administration had a higher rate of developing visual field loss (126).

It has however not been confirmed that there is increased sensitivity to adrenergic agonists in patients with primary open angle glaucoma.

3. **Abnormal immunologic processes.**

In explaining diminished aqueous humour outflow in primary open - angle glaucoma by abnormal immune response increased levels of gamma globulin and plasma cells have been detected in the trabecular meshwork of patients with primary open angle glaucoma. In addition glaucoma patients were noted to have a high prevalence of antinuclear antibodies (17, 128). Further detailed studies have however failed to confirm these findings (129 - 134).

4. **Oxidative damage.**

No evidence has so far confirmed whether the trabecular meshwork could be damaged by oxidative insult.

In summary it could generally be accepted that the cause of trabecular dysfunction in primary open angle glaucoma is yet unclear.

Optic nerve cupping and atrophy.

Optic nerve cupping includes the loss of all three elements of the disc i.e. axons, blood vessels and glial cells (4).

Generally it is believed that the lamina cribrosa is the site of glaucomatous optic nerve damage (135). The lamina is a relatively rigid structure that surround the densely packed axons and divides the higher intraocular pressure space from the lower subarachnoid pressure space. Early in glaucoma, the lamina is compressed. In the later stage of the disease the lamina sheets become fused and the entire lamina bows backwards (135).

Increased intraocular pressure either directly or indirectly causes optic nerve cupping as it has been noted that:-

(i) Most patients with primary open angle glaucoma have increased intraocular pressure many years before the development of cupping and visual field loss.

(ii) Elevated intraocular pressure is a major factor for the development of primary open angle glaucoma in glaucoma suspects.

(iii) Elevated intraocular pressure is the only common element to a wide variety of secondary glaucomas.

(iv) In a variety of animal models elevated intraocular pressure preceeds optic nerve damage and visual loss.

The linkage between elevated intraocular pressure and optic disc cupping has been challenged. Some authors (136 - 139) observed that, some patients develop optic nerve cupping and visual field loss at normal levels of intraocular pressure. Some patients maintain normal optic nerve and visual function despite elevated intraocular pressure. Also the level of intraocular pressure does not correlate with the progression of established primary open angle glaucoma. The linkage between intraocular pressure and optic nerve damage has however not been refuted rather implications of variable resistance of optic nerve to pressure induced damage have been made.

More than 130 years ago Mueller (140) and Von Jaeger (141) introduced the following theories:-

Mueller's mechanical theory:

He proposed that elevated intraocular pressure damages and destroys optic nerve axons in the region of the lamina cribrosa. Earlier it was postulated that axons are compressed or stretched over rigid tissue of the disc (142). Recent suggestions infer that elevated intraocular pressure interferes with axoplasmic flow. Evidence to support this hypothesis include the following:-

(i) Blood vessels are not lost selectively in glaucomatous cupping. Even severely damaged discs have blood

vessels present in proportion to the number of remaining axons (148 - 151).

(ii) The lamina cribrosa in glaucomatous eyes undergoes progressive compaction and deformation. This could damage axons directly or interrupt axoplasmic flow (149, 150, 135).

(iii) In primary open angle glaucoma axons are lost in greater numbers at the superior and inferior poles of the optic disc. These are the areas of the lamina cribrosa that have the least amount of connective tissue support (135).

(iv) In a variety of animal models, orthograde and retrograde axoplasmic flow are interrupted at the lamina cribrosa (143 - 147).

(v) The optic nerve blood vessels have sufficient autoregulation so that moderate elevations of intraocular pressure should not cause ischaemia (142, 152 - 154).

(vi) Greater loss of the peripheral axons occurs in the bundles which is the opposite of what would be expected from ischaemic damage (146).

Jaeger's ischaemic theory.

The theory proposed that elevated intraocular pressure produces a relative ischaemia to the optic nerve head that damages and eventually destroys axons. Evidence supporting the theory include the following:-

(i) Marked elevation of intraocular pressure produce optic nerve ischaemia with pallor, hemorrhages and arteriolar attenuation.

(ii) Ischaemia of the optic disc from disease such as temporal arterities can produce cupping and visual loss indistinguishable from primary open angle glaucoma (155, 157).

(iii) a haemodynamic crisis may produce optic nerve cupping and visual field loss that closely resembles primary open angle glaucoma (157 - 160).

(iv) Visual field loss can progress rapidly when blood pressure is reduced too rapidly from overzelous treatment (161 - 163).

(v) The blood supply to the prelaminar optic nerve comes from the short posterior ciliary arteries, which are not autoregulated (164, 153, 165).

(vi) Diabetes mellitus and other vascular diseases are risk factors for the development of open angle glaucoma.

(vii) Flame shaped hemorrhages are seen on or near the optic disc in primary open angle glaucoma and are considered poor prognostic sign (166).

(viii) Fluorescein filling defects of the optic disc have been noted in primary open angle glaucomas (167, 171), and these defects correlates in location with visual field loss. Fluorescein angiography also demonstrates prolonged circulation time and leakage of fluorescein in some cases (171 - 175).

(ix) In a variety of animal experiments elevated intraocular pressure produces decreased ocular blood flow (156, 175 - 179).

In some patients mechanical forces may be of greater importance while in others ischaemia may be playing the major role. However it is possible that the two (mechanical and ischaemia) theories could be jointly responsible.

3.3 CLINICAL PRESENTATION OF OPEN ANGLE GLAUCOMA

Open angle glaucoma has very little difference in its clinical manifestation in different patients. It usually, as a rule, starts slowly, quietly and insidiously. The patients may not notice any difference in eye appearance, in vision or have any subjective symptoms e.g. pain and discomfort.

Signs of congestion are completely absent and the eye is white unless intraocular pressure is very high when the anterior ciliary vessels may be dilated.

Anterior chamber may be deep or shallow and its angle almost always open. The iris is occasionally atrophic. The optic disc appears cupped or atrophic while the intraocular pressure is always found to be above normal, (> 21 mmHg).

The central vision may remain good until late stages, but the patient will develop shrinkage of peripheral field with/or a paracental scotoma which can be annoying. Apart from a vague feeling of fullness or headache the patient may notice a slight increase in presbyopia and loss of rapid adaptation to dim illumination which may cause some inconvenience.(31)

As the disease progresses the choroid may show depigmentation especially around the disc and the excavation and atrophy of the optic nerve head increases. The shrinkage of the visual field may now cause considerable embarrassment and the patient walks hesitatingly, turning his head repeatedly from side to side, fumbling for objects and stumbling over obstacles in his path. These changes gradually progress over a period

of many years until eventually the eye becomes intensely hard, with loss of vision and a state of absolute glaucoma is reached.

There is no hope for cure when the stage of absolute glaucoma is reached. The sclera is porcelain white, the anterior ciliary vessels become; dilated and varicose show up. The cornea is insensitive, the anterior chamber is deep or shallow and its angle may be occluded by peripheral anterior synechiae. The pupil becomes widely dilated and immobile, the iris is bounded by a dark rim of pigmented epithelium and may show advance changes of atrophy, the fundus become tesselated and the white optic disc shows a deep excavation with prominent overhanging edge. At this stage the vision is permanently and completely lost. Pain and headache are usually constant and severe. Exacerbation of headache with the recurrence of congestive attacks are liable to occur.

Finally the degenerative changes occur as hyalin-like opacities on the cornea, running in a band-shape across it or spreading irregularly over its surface. Chronic oedema results in the formation of bullae which periodically rupture. Opacification of the cornea occurs with the development of a degenerative pannus (180). The lens becomes opaque and the iris extremely atrophic. Advancing degeneration in the sclera may result in its yielding before the sustained pressure with the formation of equatorial, ciliary or intercalary staphyloma. The patient may be relieved from continued pain by the bursting of the globe due to mild trauma. If the eye is allowed to remain, further intraocular degenerative changes occur with haemorrhages, anterior uveitis or panophthalmitis which will complicate the sufferings of the patient. Alternatively the cornea may disintegrate in a degenerative or hypopyon ulcer which eventually may perforate. In most cases if the eye survives high intraocular pressure for a

longtime, the tension falls and it may even become soft resulting in pthisis bulbi. The picture is tragic and worse still as primary open angle glaucoma is a bilateral disease.

Diagnosis and Investigation

A patient presenting with classical features of primary open angle glaucoma, that is, a cupped and atrophic optic disc, typical defects in the visual fields, a raised intraocular tension with a gonioscopically open angle, all in absence of other ocular disease, offers no difficulty in diagnosis. As mentioned earlier patients with primary open angle glaucoma are asymtomatic until late in the course of the disease where there is significant loss of visual field. This indicates a need for thorough examination and screening of patients with ocular problems in order to diagnose the condition early.

Ocular Examination

I. External Examination

1.1 Conjunctiva: shows little changes in Primary open angle glaucoma. High intraocular pressure may induce a visible dilatation of anterior ciliary arteries.

1.2 The cornea: with a raised intraocular pressure two things can be seen, haziness and anaesthesia. The oedema is largely responsible for the appearance of haloes and the transient periods of smoky vision that may occur in these patients. In absolute glaucoma corneal oedema and anaesthesia are commonly present.

1.3 The iris: shows some degree of atrophy. The atrophy involves the layer of pigmented epithelium.

1.4 Anterior chamber: in primary open angle glaucoma varies considerably in depth. The variations are similar to those seen in non glaucomatous individuals of the same age (182).

As a rule in the young the anterior chamber is normal or deep, while in the old it becomes more shallow. In emmetropes it tends to be normal, in myopes deep and in hypermetropes shallow.

1.5 The pupil undergoes some changes in constriction. At first it appears normal but close examination reveals in early cases a bilateral reduction in the later phase of pupillay constriction, while in advanced primary open angle glaucoma all phases of constriction may be sluggish and the latent period of constriction increased (182). In absolute glaucoma the pupil becomes widely dilated and fixed.

1.6 The lens usually has no changes related to glaucoma but with patient having pseudoexfoliation of the capsule changes can be seen. In absolute glaucoma the development of a cataract is the rule, starting at first as a general haze especially in the posterior cortex but eventually progressing to form a complicated cataract (184). The opacifications is presumably due to raised intraocular pressure and disturbance of nutrition caused by degenerative changes in the eye.

2. **Fundus Examination:-**

2.1 General considerations:

Fundoscopy is the most critical part of the primary open angle glaucoma examination. This investigation has three fold value in diagnosis, therapy and prognosis. Follow up of patients require frequent and accurate ophthalmoscopy to visualize the optic nerve head. Surgical decision for patients receiving maximum medical therapy may depend on the detection of early changes in optic nerve head. In all eyes, the diameter of the cup can be expressed as a fraction of the

diameter of the disc both in the vertical and horizontal meridian (cup/disc ratio C:D). In most normal eyes, about 66% have horizontal C:D ratio of 0.3 or less. Only 3% have a ratio of more than 0.6. A difference in ratio between the two eyes greater than 0.1 is seen in 8% of normal population, where a difference of more than 0.2 is found in less than 1% (184). Any difference should be regarded with suspicion until the possibility of glaucoma has been excluded.

2.2 **Optic disc cupping:-**

The optic nerve head is examined commonly by a direct hand held ophthalmoscope. There is no standard pattern for the development of glaucomatous cupping of the optic nerve head (186 - 194). Cupping may start as a symmetric enlargement of the physiological cup, but usually some portion of the rim erodes more rapidly than the rest. As the cup enlarges, the retinal vessels are pushed aside following receding nasal wall of the cup. Where the vessels shift from a more vertical orientation along the cup wall to a horizontal orientation on the retinal surface there is a bend in the vessel. Change in the shape or position of that bend is a sensitive indicator of disc change and can be monitored with computer analysis (194).

As the cup enlarges its colour often turns from pink to white in the terminal stages. Cupping occurs slowly unless the pressure is very high.

Reversal of disc cupping can be dramatic in young patients following surgical or medical lowering of intraocular pressure (195 - 196). Reversal is less dramatic in older patients, presumably because reduced elasticity of the sclera tissue in older patients does not allow the cup to resume its prior configuration. Also cupping that results from actual loss of nerve tissue is unlikely to reverse (197).

2.3 **Optic disc haemorrhage:-**

The reported prevalence of disc haemorrhage in open angle glaucoma varies considerably 1.8% to 8.0% of glaucoma suspect cases; 9.2% to 36.9% in Primary open angle glaucoma cases and 10.4% to 24.4% in normal tension glaucoma cases (198 - 203).

These flame shaped haemorrhages usually occur in the inferior temporal disc margin, although they may be found at any location around the disc rim. Haemorhage may be more common in patients with Normal tension glaucoma (200, 202, 204) and seen to indicate that glaucoma is or will be progressive. Eyes with disc haemorrhages frequently require more aggressive therapy, including surgery (205 - 207); than eyes without disc haemorrhages.

3. **Visual field Examination:**

The visual field examination is also an important and crutial investigation in open angle glaucoma with 3 fold value in diagnosis, prognosis and therapy.

It is thought that axonal damage is the cause of defects in glaucoma and the pattern of these defects correspond to the pattern of distribution of intraocular axons (32).

3.1 **General considerations:-**

The patient's distant correction and appropriate add for near should be available for accurate testing. Visual fields are examined prior to the instillation of drops and prior to the use of examining lenses or any other equipment that would create visual disturbance. Time and patience are needed to help the patient produce a reliable visual field. Repeated visual field studies with a technique that is constant will indicate the fate of progression of functional loss regardless of other findings that may appear to be relatively the same from one

examination to the next. Intraocular pressure may be normalized by medical or surgical means but if the eye continues to loose field the prognosis is poor.

Extreme care must be taken in assessing visual field changes in the presence of simultaneous glaucoma and cataract. The effect of clouding of the media is to reduce the stimulus illumination and to exaggerate the field defect. Continued or increased use of miotic drugs, resulting in extreme miosis, will decrease retinal illumination and simulate progress of a field defect. No matter what the other findings in glaucomatous eye; the visual field changes must always be judiciously considered in any discusion of therapy. The greater the visual field loss the worse the prognosis and the more difficult and urgent the therapy must be.

3.2 **Glaucomatous visual field defects:-**

Generalized depression can be the earliest sign of glaucoma, but it also can occur with aging, miosis or hazy media. Generalized depression can increase the clinicians suspicion that glaucomatous damage has occured, especially if it is unilateral or it is more pronounced in the eye with higher pressure or large cup/disc ratio. The nasal portion of the visual field is often affected in glaucoma. Depression may be evidenced by hesitancy in patient response when testing this area, as an inward turning of the isopter in manual perimetry or by reduced sensitivity on static testing.

Enlargement and baring of the blind spot are considered non-specific changes that can occur in normal patients. If the blind spot enlarges in an arcuate manner, it may be called Seidel scotoma and may be seen in early glaucoma.

Careful manual perimetry using combined static and kinetic technique may demonstrate small paracentral scotoma. Aulhorn

and Harms (208) found similar small defects that did not connect to the blind spot in 20% of glaucomatous visual fields. Early glaucomatous defects may have a small dense center. Over time, if the glaucoma is progressive, they enlarge, deepen, and coalese to form arcuate scotoma. Inconsistence of response in the paracentral area may be an early sign of glaucomatous change (209 - 211).

The arcuate scotoma represents a complete nerve fibre bundle defect. It begins at the blind spot, arc around fixation, and ends at the horizontal nasal raphe. The defect may "break through" into the peripheral nasally and subsequently, expand further to ultimately become an altitudinal defect.

In the late stages of glaucoma most of the axons at the superior and inferior poles of the disc are destroyed, leaving only the papillomacular bundle and some nasal fibres. This destruction produces the characteristic end-stage field, with a small central island and a large temporal cresent remaining. The central island may split fixation so that only fibers from half of the papillomacular bundle remain. Kolker (212) found that patients with split fixation are more susceptible to central vision loss at surgery.

4. **Tonometry:**

Measurement of intraocular pressure can be misleading when measured with only Schiotz tonometer. Applanation tonometry when available is the suggested and more reliable method. Schiotz tonometer is useful as an inexpensive screening device.

Patients with primany open angle glaucoma frequently show a wide swing of intraocular presssure than normal. So a single pressure reading of 21 mmHg or less does not exclude the diagnosis. In order to detect such fluctuations it may be necessary to measure the intraocular pressure at different times of the day or better still at

periodic intervals around the clock (phasing). A large discrepancy in the level of intraocular pressure between the two eyes should be viewed with suspicion on the assumption that, irrespective of the absolute pressure value, the eye with the higher reading is probably diseased.

5. **Gonioscopy:**

This is employed for examination of the anterior chamber angle. Accurate evaluation of the angle of the anterior chamber is essential in the glaucoma workup.

In open - angle glaucoma gonioscopy may reveal inflamatory precipitates (213) or a fibrovascular membrane, or it may reveal a tumour that is covering or invading the trabecular meshwork. Such cases would be classified as secondary glaucoma. The early sign of impending neovascular glaucoma may be a network of blood vessels growing anteriorly on the trabecular wall.

6. Other important tests in open angle glaucoma include:-
   1. Fluorescein angiography.
   2. Fundus photography.
7. Patients with open angle glaucoma also need some systemic studies to exclude other causes of glaucoma. The investigations include:-
   1. Blood sugar.
   2. Blood pressure
   3. Blood coagulation factor
   4. Cardiovascular evaluations.
   5. Skull films.

3.4 Treatment of Open Angle Glaucoma

Treatment of simple glaucoma (open angle glaucoma) is directed at lowering the intraocular pressure to within "normal" limits. Treatment is either medical or surgical and sometimes a combination of the two.

Medical Treatment:

The management of primary open angle glaucoma is primarily medical therapy and surgery is undertaken only when medical treatment has failed, not well tolerated or when patients fail to comply with therapy. Medical therapy includes antiglaucoma agents divided into several groups based on chemical structure and pharmacological action. The common five groups are:-

1 **Beta-adrenergic antagonists:**

These lower intraocular pressure by reducing aqueous humour secreation and have little effect on outflow facility. Drugs in this group include

a) Timolol - nonselective *B1 & B2* blocker

b) Levobunolol - nonselective *B1* & *B2* blocker

c) Betaxralol - *B1* blocking agent useful in patients with history of bronchospastic disorders.

2. **Adrenergic agonists**: (sympathomimetic agents)

These increase both conventional and uveoscleral outflow.

These include, epinephrine and bipivefrin a pro-drug transformed into epinephrine by estarase enzyme in the cornea.

3. **Parasympathomimetic agents:** (Cholinergic agonists):

These lower intraocular pressure by causing contraction of the ciliary muscle. This contraction pulls on the scleral spur and trabecular meshwork, increasing the outflow of aqueous humour. In addition these agents cause the pupillary sphincter to contract hence the common name "miotics". e.g. Pilocarpine.

4. **Carbonic anhydrase inhibitors:** These are systemic agents that reduce aqueous formation by direct inhibition of carbonic anhydrase in the ciliary body and, to a lesser extent, by producing generalized acidosis. The carbonic anhydrase inhibitors are given orally or by injection. Commonly used agents are acetazolamide and methazolamide. Other drugs include Dichlorphenamide (Daranide) and Ethoxzalamide. The enzyme carbonic analydrase catalize the reaction. $CO_2 + OH^- \overset{CA}{\rightleftharpoons} HCO_3$ - (C.A - carbonic anhydrase) where the bicarbonate iron appears to be linked to the movement of sodium and water; suggesting a direct role for carbonic anhydrase in aqueous formation and a direct role for the carbonic anhydrase inhibitors in reducing aqueous formation.

5. **Hyperosmotic agents:**

When given systemically, hyperosmotic agents lower intraocular pressure by increasing the blood osmolarity. The increased blood osmolarity creates an osmotic gradient between the blood and the vitreous humour, thus drawing water from the vitreous cavity. As the gradient rises, the intraocular pressure is reduced.

A substance distributed only in extracellular water e.g. Manitol is more effective than a drug distributed in total body water e.g. urea. Commonly used hyperosmotic agents include, oral glycerine, intravenous manitol and oral isosorbide. Isosorbide is a useful oral agent for diabetics because unlike glycerine or manitol it is not metabolized into sugar.

Hyperosmotic agents are used to lower acute episodes of raised intraocular pressure. They are rarely administered for more than a few days at a time. A rebound elevation of intraocular pressure can occur if the agent penetrates the eye and reverses the osmotic

gradient. Treatment in open angle glaucoma is usually started with a single drug unless the initial intraocular pressure is found to be extremely high when a combination of drugs is used. If the use of one particular drug is not enough to bring the intraocular pressure to the estimated "safe" level another agent should be added. A betablocker with a miotic or an adrenegic agonist with a miotic is usually the best combination. Carbonic anhydrase inhibitors may be tried if the potentially less toxic agents are not effective or well tolerated. This however should be only for a short period to avoid toxic side effects. Patients should be informed that treatment is for life and that they should use the drugs as adviced and report for check up as recommended by the eye specialist.

As patients are not able to associate systemic side effects with topical drugs and consequently seldom volunter symptoms, the ophthhalmologist should conduct an active search for these symptoms by direct questioning.

Physicians in the developing world should be aware that as the drug shortage gets worse and the drug price increases compliance may become an increasing problem and they should think of an alternative safe and cheap method of managing their patients.

Surgical Treatment:

1 The decision to undertake surgical therapy is usually made when maximum medical therapy fails to control the intra-ocular pressure, not well tolerated by the patient or when for any reason compliance is poor.

Maximum medical therapy means a combination of more than one type of topical antiglaucoma used with a systemic agent e.g. a

combination of a miotic, a betablocker and a carbonic anhydrase inhibitor.

When considering surgical procedure for open angle glaucoma, it is important to note that patients with far advanced visual f ield loss or field loss that crosses fixation are at risk of total loss of central visual acuity following the procedure. The mechanism of this phenomenon is not known but possibilities include, cystoid macula oedema, early postoperative pressure spikes and optic nerve ischaemia. The surgeon must consider many other factors such as the patients general health and status of the fellow eye.
Patients should be informed that the purpose of surgery is to lower the intraocular pressure, thus arresting progressive visual loss and optic nerve damage caused by the raised intraocular pressure. They should also know that glaucoma surgery might cause further loss of visual acuity and that glaucoma medication may still be required postoperatively, as surgery might fail to control the intraocular pressure. The incidence of decreased visual acuity after filration surgery varies.
About 10 - 25 percent of eyes lose two or more Snellen's lines within 2 years after surgery (3).
Surgical treatment currently in use for open angle glaucoma include:-

a) Laser trabeculoplasty
b) Filtering procedures
    i) Partial thickness - Trabeculectomy.
    ii) Full thickness - Sclerotomy.

In ideal situations surgical treatment of open angle glaucoma should begin with a laser trabeculopasty. If laser trabeculoplasty fails to

bring the intraocular pressure under control then trabeculectomy or some other filtering procedure should be considered.

The aim of filtering surgery is to create a new path-way for the outflow of aqueous humour from the anterior chamber through the sclera into the subconjunctiva and subtenon's space.

2. **Trabeculectomy:**

Trabeculectomy is the standard drainage operation for primary open angle glaucoma (57 - 60). Its main advantage over full thickness drainage procedure is a reduced incidence of complications (61 - 64). The success and safety of this procedure has been well established following earlier operations (65 - 71).

Trabeculectomy was first described by Carin's in 1968 (72). He performed the operation at the 12 oclock position but added that it could be performed anywhere in the corneoscleral circumference (73). Since then the operation has traditionally been performed and described most frequently at the 12 oclock position (74). The reason for this is probably the surgical access is easiest superiorly. The peripheral iridectomy is unlikely to cause diplopia or a cosmetic defect at 12 oclock position and the resultant bleb is well covered by the upper eyelid.

Trabeculectomy lower intraocular pressure by fistula formation into a subconjunctival bleb (75 - 76). The success rate of trabeculectomy in primary glaucoma is reported to be between 67% and 94% (77). Surgical failure is usually apparently within the first 6 (six) postoperatively months and is generally due to increased subconjunctival fibrosis (78).

Several studies have investigated the reasons for success and failure of trabeculectomy. The type of conjunctival flap (fornix based - versus limbal based) has shown to influence the bleb morphology

and vascularity but to have no influence on the control of intraocular pressure (79). Other studies have reported that an avascular area in the bled correlates with better intraocular pressure control (80). Trabeculectomy has been performed with partial and total tenectomy. Total tenectomy was more likely to result in a cystic bleb. No difference in intraocular pressure control was found between the two groups (81). Recently, studies have shown that subconjunctival 5 - flourouracil decreases fibroblast proliferation and so reduces the risk of drainage failure (82).

The success rate of trabeculectomy has been shown to be significantly higher when it is carried out as a primary procedure without prior topical antiglaucoma medication (83). Topical medication is associated with increased number of inflamatory cells and fibroblasts in the conjunctival and episcleral tissue leading to a greater risk of filtration failure (84, 85).

Trabeculectomy is performed by removing a block of limbal tissue beneath a scleral flap. The scleral flap limits the out flow of aqueous and thereby reduces the incidence of hypotony and shallow anterior chamber which may lead to cataract, macular oedema, optic nerve swelling and peripheral anterior synechias.

Hypotony may be caused by excessive leakage through a big hole, a tear in conjunctival, a cyclodialysis cleft or a choroidal detachment. A direct leak is detected by Seidel test.

4. MATERIALS AND METHODS

4.1. Study population:

The study population consisted of all African patients with open angle glaucoma attending Muhimbili medical centre eye clinic from November, 1992 to October, 1993.

4.2. Selection of patients

Patients selected for the study were all male and female African patients who presented with raised intraocular pressure, glaucomatous optic disc cupping, glaucomatous visual field defects and open anterior chamber angle.

They were selected during the study period (from November, 1992 to October, 1993).

4.3. Questionnaire:

A questionnaire was completed for each patient. The information required was:-

A. 1. Patients identification:-
name, age, sex and date of examination.

2. Ocular history and duration of:-
   (a) Non-visual symptoms i.e. tearing
   (b) Visual symptoms i.e. poor vision, ocular pain, haloes.
   (e) Use of topical ocular drugs i.e. steroids.
   (d) Ocular trauma.

3. Systemic vascular diseases or diabetes mellitus.

B. Ocular Examination

Ocular examination was done to determine the following:-

1. **Visual Acuity:**

Examination was carried out using Snellen chart or illiterate E-chart for literate and illiterate patients

respectively, uncorrected and best corrected visual acuity were noted.

2. **Intraocular Pressure:**

Applanation tonometer was used to measure the intraocular pressure, fluorescein 0.05% with 0.4% Novesin was instillated into the eyes to anaesthetize the cornea. Intraocular pressure more than 25 mmHg was considered raised intraocular pressure and the patient was recorded or categorized as having glaucoma.

3. **Anterior Segment Examination:**

Haag streit 900 slit lamp was used for the examination and the following structures were examined:-

(a) Eyelids:- for any abnormalities or infections.

(b) Conjunctiva:- for any infection.

(c) State of the cornea:- size, clarity, oedema (due to raised intraocular pressure) keratic precipitates (kp's).

(d) Anterior chamber depth, flare and cells.

(e) Pupil and iris were also examined:- for any iris atrophy, changes of colour, synechial peripheral anterior and posterior.

(f) Lens was examined for:- any cataractous changes or pigments on the anterior capsule, luxation and subluxation.

(g) Anterior vitreous face:-
for clarity, presence of old pigments or cells.

4. **Fundus Examination:-**

Direct ophthalmoscope was used in a darkroom to examine the fundus. The optic disc was examined for size, shape and colour.

Glaucomatous disc changes including enlargement of the physiological cup and nasal shift of vessels were recorded.

5. **Visual Fields:-**

Central visual fields were assessed by using a tangent screen or field analyser. Patients with better vision and who could cooperate on the field analyser were assessed with the analyser. Patients who had poor vision and could not understand the instructions on the field analyser were assessed using a tangent scree. Visual field defects were noted and classified as:-

(i) early detectable defects as small paracentral scotoma

(ii) arcuate scotoma

(iii) advanced visual field constriction with characteristic central and temporal island.

6. **Gonioscopy:-**

The width of the angle of anterior chamber was determined by gonioscopy using a goniolens (Goldman's) and Haagstreit 900 slit lamp. An open angle was the angle where the Schawalbe's line, trabeculae and the scleral spur were visible. Degree of pigmentation was also observed in the anterior chamber angle.

4.4 SURGERY

Patient selected for surgery were those with vision better than 6/60 (on Snellen's and illiterate E-charts). These patients (after consenting to have surgery) were admitted in the Muhimbili Medical centre eye ward for Trabeculectomy.

Patients were informed prior to the surgery as to the reasons and purpose of the surgery. The patients were advised that the procedure was not for the purpose of improving vision but rather to reduce and to control intraocular pressure which leads to progressive optic nerve damage and field loss.

Preoperative Evaluation:-

Before admission patients were examined for any signs of infection or inflammation in the eyes to be operated. Surgery was delayed if active infections of the lids or lacrimal drainage apparatus was evident to avoid postoperative endophthalmitis. These conditions were treated in the outpatient department before surgery.

The general condition of the patient was assessed including:-

(a) Blood pressure was measured to rule out hypertension.

(b) Chest examination; to rule out respiratory infection which might lead to coughing during surgery or postoperatively.

(c) any history of drug allergy was enquired for especially allergy associated with drugs used during surgery and after i.e. atropine, lignocaine.

Patients with raised intraocular pressure were treated with 2% pilocarpine eye drops in combination with Acetazolamde (Diamox) tablets.

The night before surgery all patients were given 500mg Diamox tablets before going to bed and 500mg early in the morning. In some cases where the intraocular pressure was persistently high intravenous manitol was given.

Patients were informed of this surgical procedure assured and adviced to remain quite and lie on the operating table avoiding any movements.

Anaesthesia

Trabeculectomy was done in all patients under local anaesthesia. Lignocaine 2% was used for retrobulbar anaesthesia and facial block. In addition patients were sedated by administering pethidine half an hour before surgery and topical amethocaine instilled about three times before patients being put on the operating table.

Operation Technique

Surgery in all cases was done using an operating microscope. (Magnification 7X).

The skin of the area surrounding the eye to be operated was cleaned using a weak antiseptic solution. The eye was then covered with sterile dressings allowing only the eye to be operated exposed. The eye was kept open using an eye speculum.

A superior rectus suture of 6.0 black silk was placed under the superior rectus muscle.

The conjunctiva and Tenons capsule were grasped with forceps 6mm posterior to the limbus and were incised with scissors until episcleral tissue was reached. The limbus based flap of conjunctiva and Tenon's capsule was bluntly dissected well into the corneal limbus. The conjunctival flap was handled with care to avoid making a hole (button hole) in the flap.

Heamostasis was achieved by hot cautery applied to any bleeding episcleral vessels. No cautery was applied to the conjunctival flap. The area around 12 o'clock where the scleral flap was going to be made was also lightly cauterized. Excessive cautery was avoided.

A scapel (Bard - Parker No. 15) blade was used to make a rectangular block two thirds (2/3) of scleral thickness slightly nasal at 12 o'clock area. The incision began just behind the conjunctival flap and extended posteriorly 5mm and 4mm wide at its base.

One corner of the posterior scleral block was grasped with colibri forceps and a blade was passed under the corner to begin a plane dissection. The disecting knife was moved side to side through one half of the scleral thickness while the made flap was lifted upwards.

The dissection was extended anteriorly and the colour of the bed turned from white to gray as the limbus was crossed. This indicated the location of the scleral spur which is the posterior limit of the tissue to be excised. The dissection was stopped when clear cornea tissue was reached. This superficial flap of sclera lamella was treated with care not to tear or make holes in it, since it would form a cover for the eventual window into the anterior chamber.

The sclerectomy window measuring about 1 x 4mm was then made using a surgical blade No. 11. Radial incisions into the anterior chamber were made infront of the scleral spur at both extremities of the deep corneosclera lamella bed extending 1mm posteriorly. Then a posterior circumferential incision 4mm long through the deep corneoscleral lamella just infront of the scleral spur was made. The deep corneoslera lamella was elevated with a colibri forceps, and a circumferential incision was made with a

vannas scissors through the anterior edge of the deep corneoscleral lamella parallel to the incision above. The rectangular strip of the deep corneoscleral lamella was then excised leaving a sclerectomy measuring 1 x 4mm.

An iridectomy was done and iris tissue removed from the sclerectomy by an iris repositor.

The superficial scleral flap was replaced and sutured in two positions (at the posterior edge) with 8.0 silk so that the flap was held lightly against the bed. Sponges were placed at the edge of the scleral flap to asses the flow of aqueous form the anterior chamber through the trabeculectomy area. A tight wound closure was avoided.

Silk suture (8.0 or 7.0) were used to close the conjunctiva. We used a running water tight incision closure.

A subconjunctival hydrocortisone injection was given in the 6 o'clock meridian. Topical antibiotic ointment or drops e.g. tetracycline and topical mydriatic e.g. cyclopentolate eye drops instilled into the eye. The eye was covered by a patch and a shield applied.

Postoperative Care

The eyes were examined on the first postoperative day for any signs of infection (i.e. lid oedema, discharge, conjunctival congestion, reaction in anterior chamber). The anterior chamber depth was assessed by comparing the thickness of the peripheral cornea and the space between the cornea endothelium and anterior iris. Intraocular pressure was measured by applanation tonometer.

The patch was changed daily and topical medication i.e. Dexamethasone eye drops and cyclopentolate drops applied four times a day.

Patients were allowed full activity the day after surgery and discharged home after five (5) days of observation. If the anterior chamber was not formed further in patient observation was needed.

Patients were discharged home with topical steroid i.e. Dexamethasone eye drops.

Follow up

The follow up of the operated cases was done for a period of six months postoperatively to monitor;

(a) Visual acuity

(b) Intraocular pressure

(c) Presence of filtration bleb

(d) Any probable postoperative complication

(e) Visual fields

Patients were seen one week following discharge. Examination was done to measure the visual acuity and intraocular pressure, presence of filtration bleb and signs of any intraocular inflammation i.e. presence of corneal karatic precipitates (kp's), flare and cells in the anterior chamber. The pupil was examined for any formation of posterior synechiae. The steroid instillation were tappered and stopped three weeks later.

The patients were then followed at a monthly interval check up for a period of 6 months. At each visit the above were checked. Patients with intraocular pressure less than 10mmHg to 20mmHg were followed up without medication (i.e. antiglaucoma treatment). Those patients found to have intraocular pressure of 20mmHg to 25mmHg were followed closely and instructed to report to the eye clinic immediately when in pain or experiencing blurred vision. No antiglaucoma medication was given. Patients who had intraocular pressure above 25 mmHg were maintained on pilocarpine 2% or Timolol 0.5%.

Visual field assessment was done after 6 months to look for any changes (if any) i.e. preserved fields or progression of visual field defects.

Gonioscopy was done in those cases who had persistent shallow anterior chamber and at monthly interval to rule out any formation of peripheral anterior synechiae. At the end of 6 months gonioscopy was performed on all patients to asses patency of the sclerotomy and any presence of peripheral anterior synechiae.

Complications

A number of complications can be encountered during and after trabeculectomy.

1. **Intraoperative complications**

   1.1 Button hole in the conjunctival flap.

   Accidental buttonholing of the conjunctival flap can result in a leaking wound with prolonged flattening of the anterior chamber and failure of the operation. This can be avoided by careful dissection.

   1.2 **Damage to the scleral lamelar flap**

   This can also lead to excessive leakage and flatterned anterior chamber with its consequences. This can be avoided by carefully cauterizing the episcleral vessels before making the sclera flap and avoiding cauterization after formation of the flap. Excessive handling of the flap with large toothed forceps is also avoided and a colibri forceps can be used instead.

   1.3 **Misplaced incision**

   The incision into the anterior chamber should not be more than 1mm posterior to the corneosclera limbus. If the incision is too far posterialy the ciliary body may be penetrated with resultant hemorrhage and/or loss of vitreous. Haemorrhage may also occur form

episcleral vessels. Blood leak under the flap and even into anterior chamber can be avoided by achieving a good haemostasis before formation of the scleral flap.

1.4 **Damage to the Lens**

The lens can be damaged by an improper incision or during iridectomy when the iris does not prolapse. Entering the anterior chamber with iris forceps may strike the lens and create cataract.

1.5 **Iridodialysis**

This can occur during iridectomy if the bases of the iris is torn by exessive pulling. A careful pulling of the iris tissue will avoid this.

While doing the sclerectomy window cyclodialysis may occur if the incision includes the scleral spur. This incision must always be made infront of the scleral spur.

2. **Postoperative complications**

2.1 **Problems with conjunctival bleb**

Immediately postoperatively the conjunctival incision can give way leading to leakage of aqueous and even extrusion of Tenon's capsule. These are avoided by carefully suturing the conjunctival incision.

Rupture of the filtering bleb commonly occur in thin walled blebs. This is avoided by creating a thicker wall in the procedure. If the rupture does not heal spontenously a surgical closure is done.

The bleb can be infected and endopthalmitis ensues, with loss of the eye. The thin walled blebs are

especially prone to infection. As soon as infection is recognized the patient should be hospitalized and proper antibiotic therapy instituted.

The filtering bleb may fail if the iris, lens or vitreous become incarcerated in the sclerotomy. An adequate iridectomy prevents incarceration of iris in the wound. Blood clots or vitreous may also plug the drainage site. This can be avoided by proper haemostasis, proper incisions (while making the selerotomy window) and proper technique during iridectomy.

The bleb can also dissect into the cornea and obstruct the visual axis. This must be excised.

2.2 **Hypotony**

Pressure of 10mmHg or slightly lower occur in most patients, so that some form of hypotony is the desired effect in trabeculectomy. However, if the bleb ruptures, a form of hypotony occurs which is not desired. This tear must be repaired if it does not resolve spontenously with patching.

2.3 **Corneal decompesation**

This can occur secondary to flat anterior chamber or stripping of descement's membrane.

2.4 **Flat anterior chamber**

In most patients reformation of anterior chamber occurs within the first 48 hours. Prolonged absence of the anterior chamber could lead to formation of peripheral anterior synechiae; posterior synechiae, corneal edema; cataract formation and failure to develop a filtering bleb. This is avoided by not

making too large a sclerotomy window and proper closer of the scleral and conjunctival flaps.

2.5 **Peripheral anterior and posterior synechiae**

Peripheral anterior synechiae usually develop as a result of prolonged flat anterior chamber. Extensive peripheral anterior synechiae will block the aqueous flow.

Posterior synechiae may be sever to cause pupillary block resulting into iris bombe with flat anterior chamber. This can be differentiated from the usual flat anterior chamber by presence of elevated intraocular pressure instead of hypotony.

Dilatation of the pupil will relieve this situation. If it does not, then peripheral or sector iridectomy should be performed.

2.6 **Cataract formation**

The presence of prolonged flat anterior chamber and intractable iritis may lead to cataract formation. Administration of systemic and local corticosteroids will prevent this. But if cataract was present before surgery a combination of these two complications will lead to a rapid maturation of the cataract.

2.7 **Poor vision**

Sudden loss of visual acuity may occur after Trabeculectomy due to macula oedema. A proper examination after a complaint can reveal macula oedema and systemic corticosteroids started immediately can relieve the problem.

2.8 **Malignant glaucoma (ciliary block glaucoma)**

This can happen after trabeculectomy. It is due to the misdirection of aqueous flow posteriorly in predisposed eyes; with increase in total vitreous volume leading to a flattening of the anterior chamber and closure of the angle. Treatment consist of use of longterm daily mydriatic - cycloplegic drops, systemic osmotic agents, beta blockers and carbonic anhydrase inhibitors. If this is not successful, vitreous aspiration with reconstitution of anterior chamber with air or balanced salt solution. If this fails the lens should be removed even if it is clear. If malignant glaucoma occurs in one eye, it also tends to occur in the second eye.

2.9 **Choroidal and retinal detachment**

This is not a common complication following trabeulectomy in open angle glaucoma but can occur.

5. RESULTS

5. 1. Description of the study population

A total of 1494 new cases were attended at the Muhimbili Medical Center Eye Clinic between November, 1992 and October, 1993. Out of these 887 (59.4%) were males and 607 (40.6%) were females. Primary Open Angle glaucoma was diagnosed in 93(6.2%) patients of which 57(61.3%) were males and 36(38.7%) females.

Out of these 93 cases, 34(36.6%) patients qualified for this study. The rest, 59(63.4%) patients presented with absolute glaucoma in both eyes thus did not qualify.

Sex Distribution

Of the 34 patients 23(67.6%) were men and 11(32.4%) females (Table I).

Table I: Distribution of 34 patients by sex.

Sex	No.	%
Male	23	67.6
Female	11	32.4
Total	34	100

Age Distribution

The ages of 34 patients ranged from 26 to 70 years with a mean of 53 years (Table II).

Table II: Age distribution of the 34 glaucoma patients.

Age(years)	No	%
21 - 30	1	2.9
31 - 40	2	5.9
41 - 50	12	35.3
51 - 60	9	26.5
61 - 70	10	29.4
All Ages	34	100

Distribution of presenting symptom of the 34 patients.

The main symptom at the time of presentation was poor vision in 18(52.9%) patients. None of the patient gave a history of seeing haloes.

Table III: Symptoms at presentation

Symptom	No	%
Poor vision	18	52.9
Eye Pain	5	14.7
Both Pain and Poor Vision	10	29.4
Photophobia	1	2.9
Haloes	-	-
Total	34	100

Distribution of Duration of Symptoms, before presentation

Majority of patients 21(61.8%) had been with the symptoms for 2 years or less at the time of presentation. (Table IV).

Table IV: Duration of presenting symptoms.

Duration (years)	No	%
≤ 1	12	35.3
> 1 - 2	9	26.5
> 2 - 4	3	8.8
> 4 - 5	-	-
> 5	10	29.4
Total	34	100

Treatment of glaucoma before presentation

Most patients 33(97.1%) had received no antiglaucoma treatment before attending the out patient eye clinic. Only one patient had been diagnosed as a case of open angle glaucoma and started on treatment. He discontinued the treatment as he had difficulties in finding medication.

By then he had good vision. Nevertheless this particular patient presented with poor vision when examined for this study at the out patient eye clinic.

Family history of Ocular disease

None of the 34 patients gave a history of glaucoma in their families nor occurance of any other eye disease.

Occurance of other systemic diseases

Only one patient diagnosed with open angle glaucoma was on treatment for Diabetes mellitus.

5. 2. **Findings of the ocular examination at presentation**

A total of 68 eyes were examined before surgery. Of these, 23(33.8%) eyes were blind i.e. had vision below 3m FC (three, meter finger count - according to WHO - classification), followed by 15(22%) eyes with a vision between $^{6}/_{18}$ and $^{6}/_{24}$. Only 10(14.7%) eyes had good vision of 6/6 or better. (Table V).

Table V: Distribution of Visual acuity at presentation

Visual field	No.	%
≥6/6	10	14.7
<6/6 - 6/18	9	13.2
<6/18 - 6/24	5	22.0
<6/24 - 6/60	8	11.8
<6/60 - 3mFC	3	4.4
<3mFC	23	33.8
Total	68	100

Distribution of Intraocular Pressure (IOP) at presentation

Most eyes (61.8%) presented with an intraocular pressure of above 25mmHg (Table VI).

Table VI: Intraocular pressure at presentation

IOP (mmHg)	No.	%
< 15	3	4.4
15 - 20	10	14.7
> 20 - 25	13	19.1
> 25	42	61.8
Total	68	100

Distribution of Visual field defects at time of presentation

It was not possible to asses central visual field in 22(32.4%) eyes due to both poor vision and fixation. However, 17(25%) eyes had central visual field of more than 20^0. (Table VII).

Table VII: Visual fields at presentation.

Visual field	No.	%
≥ 20^0	17	25
< 20^0 - 10^0		12 17.6
< 10^0 - 5^0	5	7.3
< 5^0	12	17.6
No fields	22	32.4
Total	68	100

5.3. **Findings after trabeculectomy procedure**

Out of 68 eyes examined, trabeculectomy was done on 36(53%) eyes only.

Out of these, 32(47%) eyes were not operated because 20(29.4%) eyes were unfit for surgery (i.e. had no visual field and vision was below 3 mere finger count), 6 patients (7 eyes), refused surgery and 3 patients (5 eyes), did not turn up for the surgery.

No intraoperative complication was encountered during the trabeculectomy procedure for the 36(8.3%) eyes. Nevertheless other postoperative complications were noted after the procedure.

Immediate complications were encountered in 3(8.3%) eyes. These eyes had shallow anterior chambers on the first postoperative day due to overfiltration.

Other complications were noted at three months and six months follow up period (Table VIII).

Distribution of complications, postoperatively (of the 36 operated eyes)

Pigment deposition on the anterior lens capsule was the commonest complication.

Table VIII: Complications after trabeculectomy.

Complications	Immediate	After 3 months	After 6 months
. Shallow (flat) anterior chamber	3(8.3%)	-	-
. Corneal KP's	-	3(8.3%)	-
. Peripheral Anterior Synechiae	-	-	2(5.2%)
. Posterior Synechiae	-	3(8.3%)	4(11.1%)
. Pigment on Anterior Lens	-	4(11.1%)	5(13.9%)
. Cataract	-	1(2.8%)	3(8.3%)
Total	3(8.3%)	11(330.5%)	14(38.9%)

5.4. **Follow up**

Patients were discharged from the hospital between day five and fourteen after surgery.

The patients were seen at the out patient clinic a week after discharge and at monthly follow up for a period of 6 months. Out of the 36 eyes operated 9 eyes were not examined 6 months after surgery as the patients did not turn up for the check up.

During the six months follow up, visual acuity was assessed, intraocular pressure measured, complications looked for and at the end of six months a central visual field assessment was repeated. 36 eyes were followed for 3 months and 25 eyes for 6 months.

. Distribution of visual acuity after surgery

None of the eyes lost vision after the procedure. However no marked change was noticed between the preoperative and postoperative visual acuity. (Table IX).

Table IX: Visual acuity after trabeculectomy

Visual acuity	After 3 months	After 6 months
$\geq 6/6$	4(11.1%)	5(20%)
$< 6/6 - 6/18$	8(22.2%)	8(32%)
$< 6/18 - 6/24$	16(44.4%)	4(16%)
$< 6/24 - 6/60$	3(8.3%)	4(16%)
$< 6/60$ - 3mFC	-	1(4%)
< 3mFC	5(13.9%)	3(12%)
Total	36(100%)	25(100%)

P - Value = 0.85

Distribution of Intraocular Pressure (IOP) after surgery

Intraocular pressure was controlled below 25 mmHg in all operated eyes within the period of 3 and 6 months follow up.

Table X: Intraocular pressure (IOP) after trabeculectomy.

IOP (mmHg)	After 3 months	After 6 months
≤ 15	26(72.2%)	29(76%)
> 15 - 20	6 (16.7%)	6 (24%)
> 20 - 25	4 (11.1%)	-
> 25	-	-
Total	36 (100%)	25 (100%)

P - Value << 0.001

Distribution of visual field defects 6 months after surgery

The central visual field assessment after 6 months follow up revealed no loss of visual field even in the severely affected eyes before surgery.

Table XI: Visual field defects after trabeculectomy.

Visual field	No.	%	
≥ 20^0	8	32	
< 20^0 - 10^0		10	40
< 10^0 - 50^0		2	2
< 5^0	5	5	
No fields	-	-	
Total	25	100	

The occurance of filtration blebs after surgery

No eye was found to have any complication on the filtration bleb. Most of the eyes 32(88.9%) at 3 months and 21 (84%) at 6 months had good filtration blebs.

It was noted that even those with no filtration blebs maintained an intraocular pressure below 25 mmHg.

6. DISCUSSION

6.1. Age and Sex Distribution

No previous studies on African patients with primary open angle glaucoma showing the prevalence and distribution of the occurrence of the disease by age, sex have been conducted. However, this study confirms studies made by others (5) in other parts of the world which also indicate that prevalence of primary open angle glaucoma increases with age. These studies have been entirely hospital based. No studies so far have been conducted on the communities.

It is noteworthy however that primary open angle glaucoma overall incidence increases rapidly from the age of 40 years to 70 years but becomes less common thereafter. Studies by L"o"hlein, Keel and Camill (7) found the condition to be rare under the age of 40 years. Primary open angle glaucoma in this study was recorded from the age as young as 26 years. This early presentation has been confirmed in previous studies (22 - 29, 228) and that the disease has a more rapid progression in the black patients. Findings in this study suggest however that very few patients are diagnosed to have primary open angle glaucoma below the age of 40 years. The peak is between 41 to 50 years. Though with lesser occurrence patients over 50 years also showed a marked high incidence of primary open angle glaucoma. No case of primary open angle glaucoma was seen above 70 years. This is probable due to the life expectancy in Tanzania which is about 50 years. Generally we see few patients of 70 years of age and above in our out patient clinic.

Studies (10 - 20) have shown conflicting information existing on the prevalence of primary open angle glaucoma in the sexes. Some showed a high prevalence in males; others equal prevalence in both sexes and even a higher prevalence in women.

In this study 61.3% of the cases were males and 38.7% females. This is a male: female ratio of 1.6:1. Considering the female preponderance over males in the Tanzanian population, the possibility of having more males than females with primary open angle glaucoma in the population cannot be overlooked. Community based studies are required to prove this observation.

6.2. Presenting Symptoms of Patients with Primary Open Angle Glaucoma

Most of the patients (52.9%) studied came to the eye clinic due to poor vision and pain. As mentioned earlier the presentation of primary open angle glaucoma is usually quiet and insidious. Patients have no subjective symptoms and the central vision may remain good until in late stages.

Symptoms were claimed to have been present for two (2) years in many of the patients (61.8%) and over 5 years in 29.4% of patients before coming to hospital. These findings confirm that our patients came to hospital very late. This statement is supported by the fact that 63.4% of patients presented at our out patient clinic with absolute glaucoma which was already late for any treatment.

6.3. Heredity

Studies (41 - 44) have reported primary open angle glaucoma to have a genetic or familial component. The patients in our study did not show or give any family history of the same illness. This could only be established by examining members of the families, which was not done in the present study.

6.4. Systemic Diseases

Katz and Sommer (216); studied the risk factors in primary open angle glaucoma among 94 individuals with well documented glaucomatous visual field loss. They found that diabetes showed the closest association with glaucoma. MG Leska (8) also found a high incidence of primary open angle glaucoma in diabetic patients but the relationship to systemic hypertension was uncertain. Separate analysis for white and black showed diabetes to be a risk factor for both groups.

In this study only one patient was found to have primary open angle glaucoma while on treatment for diabetes mellitus. This is too small a number to draw any conclusions from. Possibly the study of diabetic patients could be more relevant.

6.5. Findings of Ocular Examinations

Visual Field

Reduction of visual field is the functional manifestation of the loss of optic nerve fibres which occur in primary open angle glaucoma. Visual field measurement is the commonest test of visual function used to follow the progression of the disease and to assess the need to introduce or change treatment.

This study revealed that 32.4% of the eyes examined had no fields due to the advanced stage. Only 25% of the eyes presented with visual field of 20^0 or better, and 17.66% had visual fied less than 5^0 of fixation.

Manual rather than automated visual field tests were used in this study. It is unlikely that automated tests would yield different results. Studies (214) have shown that the pressure rises gradually during the course of the disease and so the visual field deterioration may accelerate as the pressure rises. Similarly, as optic disc cupping increases, the remaining nerve fibres may be more vulnerable to damage.

The rapid rate of visual field loss in untreated disease, especially with pressure over 25 mmHg, and the relatively slow deterioration after optimum treatment, emphasises the importance of vigorous treatment.

Intraocular Pressure

Intraocular pressure alone as a single entity (high or low), can not lead to the diagnosis of open angle glaucoma. A high intraocular pressure need to be corollated with other findings of the optic disc and/or visual field defects. MG Leske (8) found intraocular pressure above 21 mmHg in 2% of the population but only 5 - 10% of these developed cupping of the optic disc and loss of visual field. It has been observed by others (136 - 139), that patients do develop optic nerve cupping and visual field loss at normal levels of intraocular pressure. Some patients maintain normal optic nerve and visual function despite elevated pressure.

Among the 68 eyes examined 42 (61.8%) presented with intraocular pressure above 25 mmHg. Intraocular pressure was found to be less than 20 mmHg in 18.7% of patients. All patients found to have low or raised intraocular pressure in this study had disc changes and visual field changes, hence the straight forward diagnosis.

The intraocular pressure variation in primary open angle glaucoma call for a need to screen the community not only by checking the intraocular pressure but it should be coupled with at least a fundus examination for the optic disc changes.

Visual Acuity

It was observed in this study that most of the patients examined presented with poor vision. Most of the patients presented with one eye being more affected than the other. A high percentage (63.4%) presented with absolute glaucoma in both eyes. Accordingly such cases could not be treated.

Diagnosis of primary open angle glaucoma in Tanzania Africans is late as most of the patients are blind by the time they are seen (2). Efforts should be made to identify them early by screening the community.

6.6. Trabeculectomy

Trabeculectomy Versus Medical Therapy

Medical treatment has always been the mainstay in the management of primary open angle glaucoma. Surgical intervation has been reserved for progression of visual field loss. Following the introduction of trabeculectomy, there was a move towards earlier surgery in primary open angle glaucoma with some advocating it as a primary procedure. However, others have emphasised the complications and

unpredictability of trabeculectomy and used it only as a last resort.

Thomas and Billson (227), have reviewed the literature and the factors that prevented complications and helped ensure a successful result in trabeculectomy. While the role of trabeculectomy as a primary procedure in primary open angle glaucoma is debatable, these authors favour earlier surgical intervention rather than using surgery as a last resort.

Smith (68, 217), has compared the longterm results of medical versus surgical therapy at up to 18 years. His results indicated that drainage surgery is superior to medical therapy in lowering intraocular pressure and that early randomised operation was not detrimental to visual acuity in comparison with cases initially allocated to medical therapy. Changes in visual field were less conclusive but suggested that early operation might have a beneficial effect.

Jay and Murray (70) showed that in many countries prolonged medical therapy and close supervision were impossible, and the surgeons practised early trabeculectomy. It now seems evident that this may also be the better policy even when close supervision of medically treated patient is possible. Their results provided a basis for the assessment of risk and benefits of trabeculectomy as primary treatment with secondary support from medical therapy compared with the hitherto more conventional reverse sequence. Only cases with initial intraocular pressure of 26 mmHg or more were studied. For the severity of glaucoma studied it is clear, however, that the initial attempt at medical control merely delays trabeculectomy in many cases. This delay has been identified

by others (59, 60, 65, 68, 69) and it is a criticism which also applies to argon laser trabeculoplasty (62). Where the initial intraocular pressure is over 30 mmHg with dense, though not necessarily extensive, field defects over 75% of cases treated conventionally required trabeculectomy within 3 years. This delayed operation was just as effective as primary surgery in reducing intraocular pressure and in both cases operation was more effective at reducing pressure than modern medical therapy. Although delay does not compromise the pressure reducing effect of the operation, it does jeopardise preservation of visual field.

At the Muhimbili Medical Centre eye clinic patients are started on medical therapy as soon as they are diagnosed as having primary open angle glaucoma. The poor accessibility, non compliance and high prices of relevant drugs make the patients miss required treatment for a longtime. This causes deterioration of both vision and visual field.

In this study it has been observed that trabeculectomy is effective hence the need for more emphasis and education on its early use as a surgical management for primary open angle glaucoma to both surgeon and patients. This is of importance especially at Muhimbili Medical Centre Eye Clinic where patients seen come from very far. The distance coupled with poor communication in this country makes proper follow up impossible.

Effect of Trabeculectomy on Intraocular Pressure

Since its first description, trabeculectomy has been very successful in lowering intraocular pressure in glaucoma (220 -

226). This lowering of intraocular pressure has been shown to both improve and preserve visual field (218, 219).

This study has shown a significant lowering of intraocular pressure in all patients studied (P. Value <<0.001). In all 36 eyes operated, the intraocular pressure was below 25 mmHg after 3 months and 6 months follow up.

These findings are reinforced by recent analysis of data form Moorfields primary therapy trial. This randomised prospective study which compared medical, surgery and laser treatment in primary open angle glaucoma showed statistically lower intraocular pressure in the surgery group compared with the laser and medical groups.

In some eyes with a seemingly, successful trabeculectomy and good postoperative intraocular pressure, there is progressive visual field loss postoperatively. This may be due to fluctuations in the intraocular pressure that are characteristic of glaucoma and not entirely eliminated by filtering surgery. Therefore, patients with normal postoperative intraocular pressure still

require periodic evaluation of optic disc and visual field testing.

Effect of Trabeculectomy on Visual Field

Jay and Murray (70) compared their findings with those of Kidd and O'connor (231) on the frequency of postoperative field loss with normal pressure after early surgery (231); and noted that, even surgery successfully reducing intraocular pressure, there is further loss of field in some cases (18%) despite the post operative pressure being in the normal range.

Jay and Murdoch (125), showed that some patients had a rapid rate of visual field loss during an initial trial of medical treatment and trabeculectomy halted the deterioration.

In this study none of the eyes showed a marked deterioration in the visual field for the six month follow up, even the severely affected cases with visual field less than 5^0. Aggarwal and Hendeles (241) showed a considerable risk of sudden loss of visual field after operation especially with patients who had small residual visual fields. This did not happen in our cases.

All the above studies were conducted with a follow up of over one year. This shows a need of a longer follow up after trabeculectomy as even after trabeculectomy there is still visual field deterioration. A regular postoperative perimetric assessment is essential whatever the pressure measurements recorded.

Effect of Trabeculectomy on Visual Acuity

Many surgeons are hesitant to operate early because they believe postoperative cataract may eventually jeopardise visual acuity (70). A study done by Jay and Murray (70) showed no difference in visual acuity between patients treated with medical therapy and those subjected to surgical therapy.

This study showed no significant change in visual acuity (P - value = 0.85) of the eyes at presentation and after trabeculectomy. However, no eye lost vision after trabeculectomy even those eye with reduced visual acuity. A few eyes had a loss of one line on the Snellen's chart. This correlates with the findings by Watson and Gierson's (65) where the mean reduction of visual acuity in the surgical group was less than half a line on the Snellen's chart.

Complications of Trabeculectomy Procedure

Trabeculectomy performed early give excellent intraocular pressure control with minimal complications.

No intraoperative complication was encountered in this study. The most frequent complications encountered during the immediate postoperative period include shallow or flat anterior chamber, choroidal detachment and anterior chamber heamorrhage (232). Flat anterior chamber may be associated with the onset or accelerated progression of lens opacities corneal oedema,and the loss of the filtering bleb (233).

In this study immediate postoperative complication was encountered in 8.3% of the eyes where all had shallow anterior chamber on the first postoperative day due to over filtration (Seidel's test was negative). The intraocular pressure in these eyes were below 10 mmHg and the anterior chamber reformed spontenously during the first postoperative week. None of these eyes developed choroidal detachment.

Acceleration of cataract maturity was a complication noted in 8.3% of eyes after trabeculectomy, but non of these had a flat anterior chamber after surgery.

Pigment deposition on the anterior lens capsule was a common complication encountered. This was seen in 11.1% of eyes after 3 months follow up and in 13.9% after 6 months follow up. None of these eyes developed raised intraocular pressure due to pigment blockage of the sclerotomy or angle of anterior chamber. These findings were probably due to the excessive pigment of the African iris. In the literature,

pigment deposition on the lens was not mentioned as a frequent complication seen in the white population.

Posterior synechiae was the second complication seen in this study. This was found in 8.3% of eyes after 3 months and 11.1% of eyes after 6 months. These findings have emphased the importance of using cycloplegic mydriatric therapy after trabeculectomy due to the mild inflammation in the anterior chamber seen postoperatively; to prevent posterior synechiae.

Mydriatics have also proven to be of benefit in maintaining the depth of anterior chamber and decreasing the possibility of malignant glaucoma (242). By preventing the forward movement of the lens iris diaphragm, mydriatics may help maintain the depth of anterior chamber and protect the posterior segment from severe decompression. Mydriatic - cycloplegic effect produce relaxation of the ciliary muscle with dilatation of the ciliary muscle with dilatation of ciliary ring. The consequent increased tension of the lens zonules (which hold the lens back towards its normal position) tend to counteract forces that try to push the lens and iris diaphragm forward (234). The distance between the ciliary processes and lens equator is also increased (235). All these effects help prevent the development of shallow or flat anterior chamber and may lessen the incidence of choroidal detachment, the result of the combination of hypotony and post surgical trauma (236). Ocular hypotony may also increase the likely hood of anterior chamber haemorrhage.

Mydiatric cycloplegic therapy in addition, reduces iris lens contact, allowing access of aqeous to angle drainage and

prevent back up efflux into vitreous and hence decrease the likely hood of malignant glaucoma.

Success and Failures Trabeculectomy

Trabeculatomy lowers intraocular pressure by fistula formation into a subconjuctival bleb (75, 76). The success rate of trabeculectomy in primary open angle glaucoma is reported to be between 67% and 94% (77). Surgical failure is usually apparent in the first six postoperative months. The operation site traditionally has been at the superior corneoscleral junction. However, this site has a failure rate of 10 - 30% (78). The cause of trabeculectomy failure can be broadly classified into intraocular, scleral and extraocular (237). Extraocular factors account for the majority of filtration failures (238), the main cause being increased subconjuctival fibrosis. The main reason for this is scarring at the conjunctival - Tenon's capsule - episcleral interface, external to the scleral flap. Other observed factors are synthesis of extraocular matrix such as collagen and glycosaminoglycans (77), and an increased inflamatory reaction in subconjunctival tissue with the presence of macrophages and lymphocytes (238).

More recently studies have shown that subconjunctival 5 - flurouracil decreases fibroblast proliferation and so reduces the risk of drainage failure (82). The use of postoperative injections of antimetabolite 5 - flurouracil has been shown to increase the success rate of filtering surgery in high risk patients (239) including previous filtering surgery failure. There are indications that 5 - flurouracil is likely to be replaced by mitomycin C which is a powerful antimetabolite requiring only one 5 minute application at the time of surgery to

produce a prolonged effect on fibroblast function. There is much which is still not known about the use of antimetabolites in filtering surgery. The best drug, dose and delivery system may vary between cases. Factors such as the individual "scar-forming potential" (with age, race, glaucoma diagnosis, and previous surgery modify this tissue response) and the intraocular pressure "goal" of the surgery influence the surgeons choice of adjunctive therapy.

The success rate of trabeculectomy has been shown to be significantly higher when it is carried out as a primary procedure without prior topical antiglaucoma medication (71). Topical medication is associated with increased number of inflamatory cells and fibroblasts in the conjunctival and episcleral tissue leading to a greater risk of filtration failure (84, 85). Laving (71) suggests that administration of topical antiglaucoma medication for even a few months before trabeculectomy has an effect on subsequent bleb fibrosis and intraocular pressure control (71).

This study showed 84% success rate in the filtering procedure with presence of filtering blebs. It was noted that 16% of eyes had no filtering blebs. It was noted that 16% of eyes had no filtering blebs after 6 months follow up but still maintained intraocular pressure below 25 mmHg. Non of these cases were found to have a closed sclerotomy window. Hence the

flat bled would be attributed to the fibrosis of the conjunctival and/or Tenon's capsule.

Adala and Klauss (240) studied the cause of failure of trabeculectomy among Kenyan Africans. They found that the

commonest cause of failure was external and internal fibrosis. Internal fibrosis was strongly associated with hyphema and hence advised maximum haemostasis during surgery to avoid hyphema. In this study hyphema was not a complication encountered hence the filtration bleb failure was more of external fibrosis.

Follow up of patients after Trabeculectomy

In this study out of the 36 eyes operated, only 25 eyes were examined on the final check up at 6 months. These findings emphasise the need of early decision to perform surgery as soon as the patient with primary open angle glaucoma is seen. In view of the fact that the disease is symptomless a delay in surgical treatment through trial medical therapy would lead to loss of patient as some of them will not turn up for followup. The study has shown a high success rate of Trabeculectomy in the lowering of intraocular pressure. Advantage of the surgical process over the use of medication in treatment of primary open angle glaucoma is that a patient would have received treatment even if he/she does not turn up for follow up.

7. CONCLUSION AND RECOMMENDATIONS

Results obtained and analysed under this research have indicated a high prevalence of primary open angle glaucoma in African patients attending Muhimbili Medical Centre Eye Clinic. As the results have been obtained among patients who neither had prior diagnosis of primary open angle glaucoma nor any prior awareness that they were suffering from such a disease the findings are very significant. Inference drawn from these findings are very significant. Inference drawn from these findings also indicates the possibility of high prevalence of primary open angle glaucoma among the African population than any other population where similar studies have been conducted.

It was also observed that there is an early onset of the disease among Africans (before the age of 40) while in other studies such occurrence have been seen to start after the age of 40 years and not found after 70 years of age. Most patients were not aware of the onset of the disease until late when other eye impairements were manifested. Patients reported such impairement late to the hospital seeking cure and hoping that such cure was available.

However, in this study trabeculectomy proved to be effective in lowering intraocular pressure and hence preventing further damage to the eye and so preserving visual field and vision.

In view of these findings efforts should be made to initiate public awareness of the onset of primary open angle glaucoma through the use of media and open public health campaings. Health and medical workers in the peripheral health centres or units should be educated in diagnosing the disease early so that patients are referred early to the eye clinic for specialist attention. At the referral hospitals surgical therapy should be the first option in Tanzania African patients with primary open angle

glaucoma. This surgery could be done as early as possible so as to prevent further damage to the afflicted eye.

It is nevertheless note worthy that before individual surgeons adopt the policy of early surgery it is essential that their own recorded surgical results are seen to be as safe and effective as documented in published literature. However, the management of patients with established primary open angle glaucoma still requires individual surgeons clinical evaluation, judgment and assessment of each patient.

In conclusion, this study has shown that early surgery gives excellent intraocular control with minimal complications.

Early surgery should be the first option for Tanzania African patients due to the unavailability of drugs, the high price of the drugs, poor patient compliance and also poor turn up of patient for follow up.

8. SUMMARY

A population of ninety three African patients attending the Eye Clinic at the Muhimbili Medical Centre were studied. Males (61.3%) predominated over female (38.7%). The age range of the patients was between 26 and 70 years; only 8.8% were below 40 years.

The prevalence of primary open angle glaucoma among the patients was recorded to be approximately 16% . Out of these 63.4% patients were diagnosed as suffering from absolute glaucoma. Accordingly these were not included in the study. The rest (36.6%) of the primary open angle glaucoma patients were the subject of this study.

Examination revealed that for all patients one eye was more affected than the other. About 52.9% of the patients presented with complaint of poor vision for a duration of more than one year. The rest had symptoms for more than two years indicating late presentation to hospital even with the symptoms.

Only one patient had documented antiglaucoma treatment before attending the Eye Clinic. The rest (97%) of the patients had no glaucoma treatment before attending the clinic. None of the patients gave history of glaucoma or any other eye diseases in the family. Diabetes mellitus was however found in one of the patients with primary open angle glaucoma.

At presentation intraocular pressure was found to be more than 25 mmHg in 61.8% of the eyes examined. Blindness (inability to count fingers at 3 meters) due to severely affected vision and visual field was found in 33.8% of the eyes. Visual field assessment was however impossible in about 32.4% of the eyes examined due to poor fixation and vision.

Trabeculectomy was conducted successfully in 52% of the eye examined. It was noted that no intraoperative complications were encountered during the surgical procedure. The only immediate post operative complication was the occurrence of a shallow anterior chamber due to overfiltration.

Six months postoperative follow up did not reveal any serious complications and intraocular pressure was well controlled in all operated eyes. None of the eyes lost vision or visual field at this point. Functional filtration blebs were present in 84% of the operted eyes and 16% had no filtration blebs but had controlled intraocular pressure.

These successful results obtained after the surgical procedure indicate the effectiveness of trabeculectomy as the first option in the treatment of primary open angle glaucoma.

This study highly recommends that trabeculectomy be considered by eye specialists as the first option in treatment of African patients with primary open angle glaucoma in Tanzania.

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