EFFECT OF PTERYGIUM ON CORNEAL ASTIGMATISM

DISSERTATION SUBMITTED FOR M.S (FINAL) OPHTHALMOLOGY, THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY, CHENNAI,

MARCH 2008

CERTIFICATE

Certified that this dissertation entitled **"EFFECT OF PTERYGIUM ON CORNEAL ASTIGMATISM "** submitted for M.S (FINAL) Ophthalmology, The Tamilnadu Dr. M.G.R Medical University, Chennai , March 2007, is the bonafide work done by **Dr. KARTHIK SRINIVASAN.** under the direct supervision and guidance in the **Department of Cornea Services of Aravind Eye Hospital and post graduate institute of ophthalmology , Madurai** during his residency period from May 2005 to March 2008.

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ACKNOWLEDGEMENT

I would like to thank the team support of cornea para medical staff. I acknowledge with gratitude, the many people who have contributed to the completion of this dissertation without whom this thesis would not have been a success.

I take this opportunity to pay my respect to **Dr.G.Venkatasamy,** whose dynamism and vision brought about the origin of this great institution.

I sincerely thank **Dr.M.Srinivasan,** Chief, Cornea services, Aravind eye hospitals, Madurai, whose support, patience and care towards me has guided me to the undertaking of this study to a successful completion.

I am very grateful to **Dr.Venkatesh Prajna**, Chief, Department of medical education, for all his help, guidance and support that he has extended me throughout my residency programme.

I sincerely thank **Dr.P.Namperumalasamy**, Chairman, Aravind eye care system and executive director, Aravind research department: **Dr.G.Natchiar**, Director, Human research department and

Dr.M.Srinivasan, Director of Aravind Eye Care System whose untiring dedication to the prevention of needless blindness in this community has, and will continue to inspire innumerable young ophthalmologist like me.

I sincerely thank my guide **Dr.M.Srinivasan ,** Chief, Cornea services, Aravind eye hospitals, Madurai, whose support, patience and care towards me has guided me to the undertaking of this study to a successful completion.

I am indebted to **Dr.Jeena.M,** Consultant, Cornea services who reviewed my work and provided critical evaluation and support in analysing this study.

I thank the department of bio statistics for their effort in analysing and compiling the results of this study, without whose time and effort, my work wouldn't have seen the light of this world.

I would fail in my study if I did not thank my patients involved in my study.

I also would like to thank Medianett staff for their kind co operation in bringing out this dissertation in a nice and timely manner.

I thank God for making all this happen.

CONTENTS

INTRODUCTION

A pterygium is a triangular fibrovascular overgrowth or extension of connective tissue from the bulbar conjunctiva to the cornea. A pterygium that is confined to 1 to 2 mm of the peripheral cornea has little effect on vision and maybe of only cosmetic concern. As the pterygium advances, however, induced irregular astigmatism can cause decreased visual acuity. The amount of the induced astigmatism, however small is measured using corneal topography.

A significant amount of corneal astigmatism can be induced by the encroachment of the pterygia on the cornea. The pterygium usually causes with the rule astigmatism that is hemimeridional on the side of the pterygium. There is significant correlation between the extension of the pterygium onto the cornea and the amount of corneal astigmatism induced. However there is poor correlation between pterygium induced corneal astigmatism measured topographically and that measured by manifest refraction.

Using renewed optical beam scanning topography by the use of the hybrid scanning slit and placido disk we can measure the cornea with an imperfect tear film which maybe encountered

in the pterygium cases and as the measurements are carried out independently for the anterior and posterior surfaces the actual corneal index (1.376) of refraction can be used for the power calculations instead of the empirically derived keratometric index of refraction (1.3375).

AIM OF THE STUDY

OBJECTIVE : To determine the effect of pterygium on corneal astigmatism.

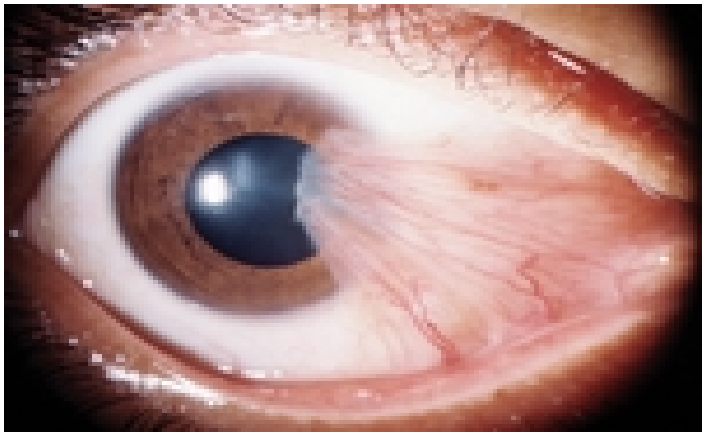
DESIGN: Prospective study.

METHODS: Fifty patients with pterygium will be selected and the extension of the pterygium will be measured using the slit lamp and the corneal astigmatism will be measured using corneal topography (Orbscan).Correlation of the data will be discussed.

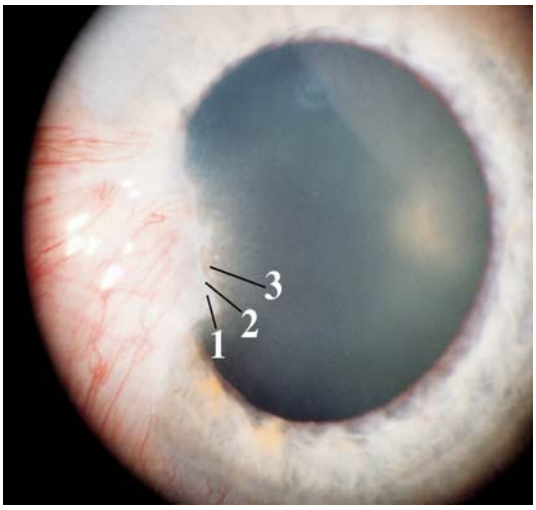
MAIN OUTCOME MEASURES: Corneal astigmatism, pterygium size.

REVIEW OF LITERATURE

Physicians have struggled for thousands of years with an unsightly elevated peribulbar lesion known as pterygium. It takes its name from the Greek word “pterygos” for wing and was described by Hippocrates, Galen, and others. A pterygium is a horizontally oriented triangular growth of abnormal tissue that invades the cornea from the canthal region of the bulbar conjunctiva. Its development is unrelated to antecedent injury or inflammation.



A pterygium can be divided into three recognizable parts: body, apex (head), and cap. The raised triangular portion of the pterygium with its base toward the canthus is the body, while the head forms the apex of the triangle, just posterior to the cap. A subepithelial cap or "halo" may be present just central to the apex and forms its leading edge.



Parts of a pterygium

1. The head of the pterygium has a thick white scar that is avascular
2. There is an iron line (Stocker line) in the epithelium which results from an irregular tear distribution adjacent to the raised edge of the pterygium.

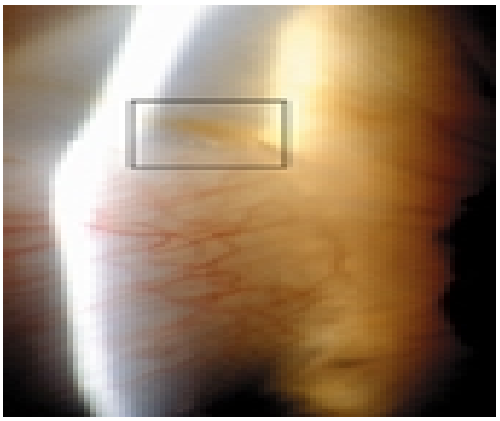
Natural History

In its earliest stages, a pterygium arises in the interpalpebral fissure as an elevated, fleshy mass on the bulbar conjunctiva near the limbus most often nasally. Engorged radial vessels appearing over the pterygium and adjacent conjunctiva usually signal a period of rapid growth. The bulbar conjunctiva may become increasingly taut as the pterygium enlarges

toward the limbus. Symptoms of burning, irritation, lacrimation, and foreign body sensation may accompany the growth of a pterygium onto the cornea. Significant astigmatism may be induced either with or against the rule as sectoral corneal steepening occurs. The astigmatism is often irregular and may lead to decreased vision. Gaze-evoked Descemet's folds and 20 diopters of astigmatic change have been observed in a patient with a pterygium containing a densely fibrotic central band[1]. As the apex approaches the visual axis, glare and decreased contrast sensitivity appear. In severe cases, symblepharon formation may limit ocular motility and result in diplopia.

For poorly understood reasons, the growth of a pterygium may stop at any stage during its evolution. Decreased elevation and vascular injection, along with a fading of the subepithelial cap, are usually seen. The lesion may remain quiescent for the remainder of the patient's life or resume growth again at a later time. Older, static lesions are often associated with an arcuate line of iron deposition in the superficial cornea immediately central to the cap (Stocker's line).

Pterygium with a Stocker line (box).



Iron lines result from pooling of tears in areas where the corneal surface is irregular. Histologically, iron is found within the basal epithelial cells and can be demonstrated using the Prussian blue stain or Perls test.

ETIOLOGY AND EPIDEMIOLOGY

There is a worldwide distribution of pterygium, but it is more common in warm, dry climates. Prevalence is as high as 22% in equatorial areas and less than 2% in latitudes above 40°. A large case control study in Australia identified a number of risk factors for the development of pterygium .There was a 44-fold greater relative risk of pterygium development for persons living in the tropics (less than 30° latitude), 11-fold for working in a sandy, outdoor environment, 9-fold for patients

without a history of wearing spectacles or sunglasses, and 2-fold for those who never wear a hat[2]. Another study demonstrated a higher prevalence among men. However, the difference between the sexes was eliminated when only indoor workers were considered.

In the northern climates, pterygium is almost exclusively confined to fishermen and rural workers. Taylor and colleagues found a statistically significant association between ultraviolet light exposure (both UV-A and UV-B) and the development of pterygium in a large group of Chesapeake Bay fishermen[3]. From these studies, the relationship between ultraviolet radiation and the formation of pterygia is obviously strong.

Ultraviolet light exposure may not be the only factor associated with the development of pterygium. Among Punjabi workers, those exposed to a dusty, indoor environment had a higher prevalence of pterygia than Punjabi workers who experienced higher levels of outdoor ultraviolet radiation[4]. One study of pterygia among welders who are exposed to increased levels of ultraviolet light showed a direct relationship between the length of employment and the incidence of pterygium. In contrast, a more recent study found pterygium to be rare (less than 0.5%) among welders[5].

Long standing nasal pterygia in elderly patients may induce deep corneal changes at the level of the endothelium and Descemet's membrane.

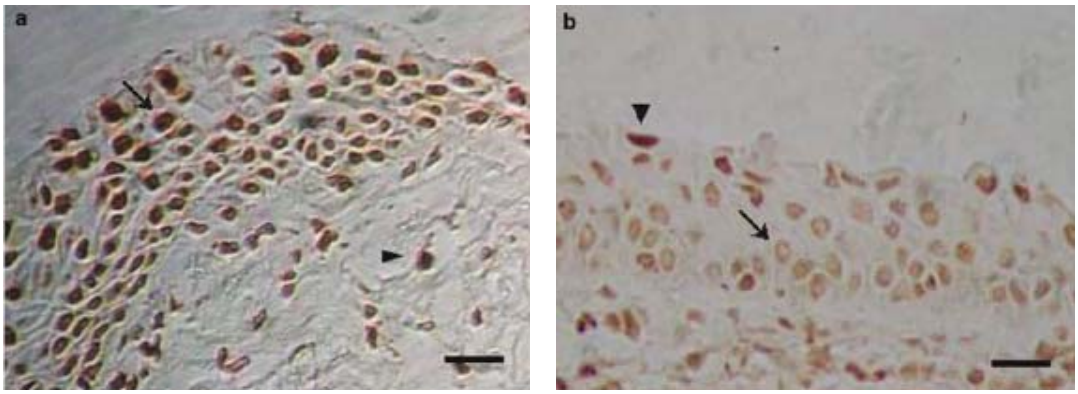
Endothelial cell density may be lower in eyes with pterygia with these deep corneal changes[24].

Local drying of the cornea and conjunctiva in the interpalpebral fissure from tear film abnormalities may lead to new fibroblastic growth according to one theory. The increased incidence of pterygium in windy, dry climates is consistent with this hypothesis.

Patients younger than the age of 15 rarely acquire a pterygium. Although the prevalence of the lesion increases with age, the highest incidence occurs between the ages of 20 and 49. Recurrences may be more frequent in young adults than older individuals. Pedigree analysis has demonstrated families with a dominant mode of inheritance, although most cases appear to be sporadic.

Histopathology

The histopathologic features of pterygia were thoroughly outlined by Fuchs in the 1890s. These include an increased number of thickened elastic fibers, hyaline degeneration of the conjunctival tissue, concretions, and epithelial changes.



(Figures illustrating the histology of pterygium)

Austin et al[6] have similarly summarized the histopathologic findings as follows:

(1)Hyalinization of the subepithelial connective tissue of the substantia propria,

(2) Diffuse or lobular collections of eosinophilic granular material with an associated increase in the number of fibroblasts and other cells,

(3) An increased number of thickened and tortuous fibers that stain strongly with elastic stains (elastotic material), and

(4) Concretions within the hyalinized and granular areas that may show either eosinophilia or basophilia.

In reference to the characteristic elastotic material within pterygia, the term “elastotic degeneration” was coined to describe the condition of tissue uptake by Weigert's and Verhoff's elastic tissue stains but the lack of

tissue degradation by pancreatic elastase. While this specific staining characteristic is not universal for pterygia, it is generally accepted that the elastic fibers within pterygia are abnormal.

Historically, Hogan and Alvarado[7]stated that the elastotic material within pterygia is formed from four sources:

(1) Degenerating collagen,

(2) Preexisting elastic fibers,

(3) Abnormal fibroblastic activity, and

(4) Abnormal ground substance.

More recently, ultra structural analysis by Austin et al[6] attributed the elastotic degeneration solely to abnormal fibroblastic activity with the production of abnormal maturational forms of elastic fibers. Moreover, collagen degeneration was only demonstrated in the subepithelial zone and accounted for the light microscopic finding of hyaline degeneration.

Histopathologic analysis of the leading edges of pterygia by Cameron[8, 9] disclosed the following:

1. Fibroblastic tissue separating the basal corneal epithelial layer from Bowman's layer,
2. Altered orientation of the basal corneal epithelial cells overlying the fibroblastic tissue,

3. Destruction of Bowman's layer and the superficial corneal stroma underlying the fibroblastic tissue, and

4. Normal corneal tissue proximal to the leading edge of the pterygium.

Immunohistochemical staining has demonstrated the presence of altered limbal basal stem cells between the dissolved edge of Bowman's layer and the fibrovascular tissue of the pterygia. Other histologic changes that have been identified in the epithelium of pterygia include squamous cell metaplasia, acanthosis, and dyskeratosis. A recurrent or secondary pterygium is defined as a pterygium recurrence after primary surgical excision. A secondary pterygium often has a more exuberant fibrovascular growth response than the original pterygium. The histologic findings of secondary pterygia differ from primary pterygia in that the typical degenerative connective tissue changes are absent. Cameron[8, 9] suggested that the surgical trauma after primary excision leads to an accelerated fibrovascular proliferative response.

The cytology of surface cells overlying pterygium is abnormal typically exhibiting squamous metaplasia with increased goblet cell

density. Abnormal cytology is also demonstrable in the inferior bulbar conjunctiva .This suggests a graded series of ocular surface changes occurring throughout the bulbar conjunctiva with the most advanced changes occurring directly over the pterygium surface confirming that pterygium is indeed a ocular surface disorder.[25]

Pathogenesis

Early work by Cameron[10] indicated that pterygia occur more commonly where ultraviolet light intensity is highest. Specifically, a high prevalence of pterygia occurs in an equatorial belt bounded by latitudes 37° north and 37° south. Confirming Cameron's observations, Mackenzie et al[11] found that those who live at latitudes less than 30° during the first 5 years of life have a 40-fold increased risk of pterygium development. Overall, it is generally accepted that ultraviolet light exposure is linked to the formation of pterygia. Additional support for this theory is the observation that pterygia are more common in those who work outdoors, especially if the activity is on or near a highly reflective surface.

Another suggested causative factor is the chronic ocular exposure to irritants such as dust. Detels and Dhir[12] reported that the age-adjusted prevalence of pterygia in factory sawmill workers (an indoor occupation) is approximately three times higher than a matched control group.

Subsequently, Coroneo [13] has questioned the possible presence of reflected or scattered ultraviolet light in these particular work environments.

Pterygium is strongly related to ocular sun exposure with little evidence that exposure during any particular period of life is more important than in other periods.[18]

Interestingly, neither exposure to ultraviolet light nor exposure to irritants precisely explains the observation that pterygia are predominantly found on the nasal bulbar conjunctiva. Several theories have been put forth to explain this finding:

1) The temporal surface of the eye is normally shaded from light by the longer lashes and curvature of the temporal upper eyelid,
2) The normal orbicularis contraction in bright light provides greater relative coverage of the temporal bulbar conjunctiva, and
3) Light incident from a posterolateral aspect to the eye is focused by the temporal peripheral cornea to the nasal limbus, causing focal limbal stem cell dysfunction.

Regarding the third theory, it is presumed that the normal anatomic relationships of the eyelids and nose would provide relative ocular shielding of incident light from the superior, inferior, and nasal directions.

In support of the notion that abnormal limbal stem cells are the primary abnormality in the pathogenesis of pterygia is the recent localization by immunohistochemical techniques of altered limbal epithelial stem cells at the leading edge of pterygia along the normal corneal epithelial basement membrane. It is accepted that a healthy limbal stem cell population provides a stable junctional barrier that prevents conjunctivalization of the cornea. Based on these findings, pterygium formation may ultimately represent a focal limbal stem cell dysfunctional state. This tenet is in contradistinction to other pathogenetic theories that have focused on a primary degenerative response of the conjunctiva. Specifically, Hill and Maske [14] postulated that actinic damage to the corneal or conjunctival tissue causes abnormal antigenicity and leads to a chronic inflammatory cell infiltrate with a subsequent reparative fibrovascular response.

Historically, numerous other diverse theories have been put forth to explain pterygia formation to include local tear film abnormalities, chronic ocular irritation, chronic inflammation with production of a pterygium angiogenesis factor, An immunologic mechanism probably type 1 hypersensitivity may contribute to the pathogenesis of ptreygium[22], hereditary factors, and altered elastic tissue formation by actinically

damaged fibroblasts. Vascular endothelial growth factor has been shown to be strongly increased in pterygia and is suggested to be involved directly or indirectly in the pathogenesis of pterygia.[21] The numerous different pathogenetic theories that have been proposed point to the fact that the ultimate pathogenesis of pterygia remains speculative.

Length of encroachment of the pterygium was rated the most important indicator of pterygium severity. The closer the pterygium approaches the center of the cornea and the papillary area the more likely patient will have visual consequences.[19]

MANAGEMENT

In general, conservative therapy for pterygium is warranted unless one of the following circumstances arises:

(1) Loss of visual acuity either because of induced astigmatism or encroachment onto the visual axis. An advancing pterygium can produce marked changes in the refractive state and curvature before entering the optical zone which can cause visual impairment. The change is usually characterized as the with –the –rule astigmatism resulting from the localized flattening of the cornea central to the leading apex.[20]

(2) Marked cosmetic deformity,

(3) Marked discomfort and irritation unrelieved by medical management,

(4) Limitation of ocular motility secondary to restriction, or (5) Documented progressive growth toward the visual axis so that it is reasonable to assume that visual loss will ultimately occur. In such circumstances, surgical intervention is required. Because recurrences after pterygium excision are frequent and aggressive, firm indications for surgical removal should exist before primary excision.

Preoperatively, a careful history and physical examination are mandatory to rule out the diagnosis of a pseudopterygium. A pseudopterygium is an inflammatory adherence of the conjunctiva to the cornea in response to chemical, thermal, or traumatic injury and can occur at any point around the limbus. Many corneal inflammatory disorders can also predispose to fibrovascular ingrowth that may resemble pterygia.

Clues leading to the diagnosis of a pseudopterygium include

(1) Any anatomic location other than the interpalpebral fissure,

(2) Diffuse corneal involvement in multiple locations,

(3) Historical information of a past significant ocular inflammatory event,

(4) The lack of anatomic configuration (“body” and “head,”) typical of a pterygium,

(5) A pterygium that bridges the limbus so that a probe can be passed underneath the body at the limbus, or

(6) The presence of corneal thinning underlying the pterygium head.

Depending on the ultimate etiology of the pseudopterygium, surgical excision may not be indicated. If the preoperative examination discloses corneal thinning underlying the pterygium head and surgery is to be performed, donor corneal tissue should be available intraoperatively in case a lamellar keratoplasty is required because of an inadvertent corneal perforation.

The differential diagnosis of pterygium should also include conjunctival intraepithelial neoplasia, squamous cell carcinoma, and a corneal macropannus. The characteristic features of these entities should distinguish these disorders from a pterygium. A limbal dermoid is also in the differential diagnosis but is less likely to be confused with a true pterygium.

MEDICAL APPROACHES

General recommendations for the prevention of pterygium formation should include the avoidance of exposure to ultraviolet radiation. A survey of patients in Australia disclosed that there was a doubling of risk for

pterygium formation associated with never wearing a hat outdoors between the ages of 20 and 29 years[2]. additionally; there was a nine-fold increased risk of pterygium formation if glasses were never worn in the decade before the pterygium developed. Since the development of pterygium is strongly associated with ultraviolet exposure within the first 5 years of life, parents should be advised to protect their children from ultraviolet exposure, especially if the latitude of residence is within 30° of the equator and a great deal of time is spent outdoors. Hence, in areas where exposure is high, the use of ultraviolet-absorbing protective spectacles, sunglasses, and hats is advisable. Lateral ocular exposure to incident light can be avoided with the newer wraparound sunglass designs.

Mild irritative symptoms from pterygium may be managed with topical lubricants or a mild topical antihistamine/vasoconstrictor (e.g., naphazoline QID). A mild topical corticosteroid (e.g., fluorometholone 0.1% QID or medrysone 1.0% QID) may be useful for moderate to severe vascular injection and irritative symptomatology. Secondary dellen may be managed with preservative-free lubricating ointments and temporary patching for 24 hours.

SURGICAL APPROACHES

The fact that numerous different techniques exist for the surgical treatment of pterygium underscores the point that no single approach is universally successful.

Pterygium excision/avulsion

All procedures, regardless of adjunctive measures employed, begin with the surgical removal of the pterygium from the globe. There are numerous techniques that have been published extensively in the literature. Dissection may be carried out from the body to the head of the pterygium or, alternatively, from the head of the pterygium toward the body.

Advantages cited for this method include a resultant smooth corneal surface, rapid epithelialization, and minimal scarring postoperatively.

Another method described for removing the pterygium head that avoids inadvertent deep dissection dates back to the seventh century: a suture is passed underneath the body of the pterygium and, with a sawing motion toward the cornea; the head is dissected from the underlying corneal tissue.

After pterygium excision, numerous authors in the past advocated a “bare sclera” technique in which the resultant scleral and corneal defects would be left to epithelialize postoperatively. It was theorized that a pterygium recurrence would be prevented if the corneal epithelium could

heal before the conjunctival epithelium reached the limbus. Many authors claimed impressive success rates with this bare sclera technique. Unfortunately, controlled studies were not performed to validate these reports.

Transplantation of the head of the pterygium

Various techniques originated in the nineteenth century to redirect the head of the pterygium away from the cornea to prevent recurrences. The surgical procedure consisted of burying the pterygium head underneath the normal conjunctival edge inferiorly after surgical dissection of the head from the cornea.

Unfortunately, recurrence rates of 30% to 75% were reported with these techniques. Such transplantation procedures have been largely abandoned secondary to high recurrence rates and poor postoperative cosmetic results.

Conjunctival flaps and conjunctival autografts

Various surgical strategies for the treatment of pterygium have developed using the premise that close approximation of healthy conjunctival tissue at the denuded limbus after pterygium excision prevents recurrences. The three basic variations on this theme include excision with

primary conjunctival closure, excision with conjunctival flap formation, and conjunctival autografts.

The primary disadvantage of the conjunctival autograft technique is the prolonged operative time required when compared to other bare sclera or primary closure techniques. Additionally, an operating microscope is required for optimum results, which can be a problem for ophthalmologists in developing countries. However, these disadvantages are outweighed by the lack of sight-threatening complications and the relatively low recurrence rates after conjunctival autografts. There is a 97% chance for recurrence within 12 months of removal of the pterygium which happens more quickly with each subsequent removal regardless of the type of surgery involved, raises the possibility that there may be a host specific resistance to regrowth that surgical removal may destroy.[23]

Lamellar keratoplasty and penetrating keratoplasty

If significant corneal thinning is present as a consequence of previous pterygium surgery, a lamellar keratoplasty may be indicated to restore the normal ocular surface integrity.

Mucous membrane grafts and skin grafts

In cases in which sufficient conjunctiva is not available for a pedicle graft, a mucous membrane graft from the lower lip after a pterygium excision can be used.

ADJUNCTIVE THERAPY

CHEMOTHERAPY

Thiotepa

The nitrogen mustard analog thiotepa, or N, N`, N'', triethylene-thiophosphoramide, has been advocated as an adjunctive measure to reduce the postoperative recurrence of pterygium since 1962. Thiotepa is an alkylating agent that interferes with normal mitosis and cell division in all rapidly proliferating tissues.

It was postulated that thiotepa reduced the recurrence of pterygium by inhibiting vascular endothelial proliferation at the operative site.

While no systemic toxicity of topical thiotepa therapy has been reported, complications reported include early and late onset poliosis and periorbital skin depigmentation that can be permanent (especially in darkly pigmented patients), prolonged conjunctival injection, irritation, conjunctival deposition of black pigment, allergic reactions, and scleral perforation. Sun exposure during therapy was suggested as a contributing

factor in the skin and lash depigmentation. The periorbital skin depigmentation has been cited as the major reason thiotepa has not gained widespread acceptance in the postoperative treatment of pterygia.

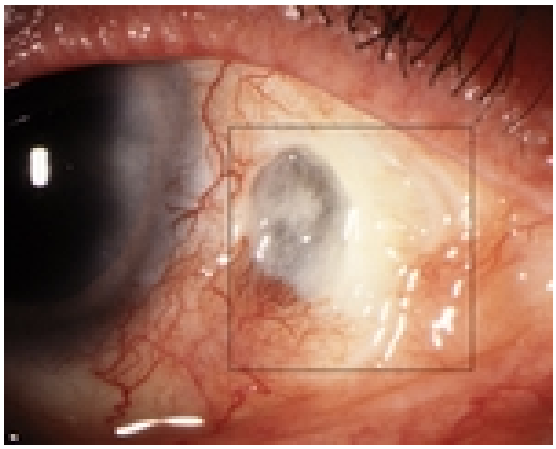
Mitomycin

Mitomycin C is an antibiotic that was first isolated from Streptomyces caespitosus by Hata in 1956. Clinical trials with mitomycin C in the United States began in the late 1960s for a variety of solid tumors to include breast, prostate, gastric, and bladder cancers. Systemic therapy with mitomycin C carries risks of myelotoxicity, hemolytic-uremic syndrome, pneumonitis, hepatic veno-occlusive disease, and rare cardiotoxicity.

The topical use of mitomycin C to prevent pterygium recurrence was first described by Kunitomo and Mori in the early 1960s in Japan.

The optimum dosage and treatment length of topical mitomycin to maximize both effectiveness and safety are not precisely known. The optimum dosage of mitomycin C may be inferred from a study on the inhibitory effects of mitomycin C on human Tenon's capsule fibroblasts in cell culture: cell colony formation was inhibited at mitomycin concentrations of 0.1 mg/ml, cell death ensued at mitomycin C

concentrations of 0.3 mg/ml, and the LD_{50} for these fibroblasts was 0.2 mg/ml.



(Figure showing scleral necrosis due to use of Mitomycin C)

Radiation therapy

Until the 1950s, radon bulbs, radium plaques, Grenz rays, and x-rays were employed in the treatment of pterygia with variable success. In 1952 strontium 90 was introduced for the treatment of neoplastic disease and has been used extensively for the treatment of pterygia since that time.

Strontium 90 is produced in the fission of uranium 235 and has a half-life of 28 years. Beta rays expend their energy maximally within the superficial 2 mm of tissue as the dose drops to 41% at 1 mm, 19% at 2 mm, 9% at 3 mm, and 1% at 5 mm. This low penetration profile for strontium

90 is important, since cataracts may develop should the dose to the crystalline lens approach 1500 to 2500 rep (1 rep = 1.08 rad).

Recurrence rates after pterygium excision with beta irradiation have varied widely, with a low of 0% to a high of 80% reported in the literature. While beta irradiation lowers the recurrence rate of pterygia, significant long-term complications have been reported, including cataract formation, endophthalmitis and scleral necrosis. Other less frequently encountered complications included corneal ulcers, symblepharon, iris atrophy, ptosis, and thinned conjunctival tissue.

SUMMARY OF MANAGEMENT

Because conjunctival autografting offers a low rate of pterygium recurrence and is free from long-term sight-threatening complications, it appears that autografting offers patients a safer alternative when compared to beta irradiation. Because scleral necrosis and possible late infectious complications occur years after the original surgery, it is not surprising that numerous short- and intermediate-term studies deemed beta irradiation safe. While it is debatable whether the reported complications from beta irradiation or mitomycin therapy are at an acceptably low rate, the serious

nature of these untoward late effects make conjunctival autografting a viable alternative in the treatment of both primary and secondary pterygia.

ASTIGMATISM

For rotationally symmetrical optical systems, paraxial rays focus stigmatically. Regular astigmatism results when paraxial rays are focused by toric optical systems. However, most of the rays passing through a lens are not paraxial. Whether or not the optical system is symmetrical, rays outside the paraxial region never focus stigmatically and produce irregular astigmatism.

The most effective method developed to date for describing irregular astigmatism is wavefront analysis.

To understand irregular astigmatism and wavefront analysis, it is best to begin with stigmatism. A stigmatic imaging system brings all rays from a single object point to a perfect point focus. According to Fermat's principle, a stigmatic focus is possible only when the amount of time required for light to travel from the object point to the image point is identical for all possible paths the light might take.

Fermat's principle explains how a lens works. Rays going through the center of a lens travel a short distance in air, but they are slowed down

by moving through the thickest part of the glass. Rays going through the edge of the lens travel a longer distance in air, but slow down only briefly when they traverse a thin section of glass. The shape of the ideal lens precisely balances each path so that no matter what path the light travels, it reaches the image point at the same time. If the lens is not ideal, some rays miss the image point, and the focus is astigmatic.

Wavefront analysis is based on Fermat's principle. Construct a circular arc centered on the image point with a radius approximately equal to the image distance. This arc is called the reference sphere which is the image point. If the image is stigmatic, all rays (from point A) will cross the reference sphere simultaneously. If the image is astigmatic, the rays will cross the reference sphere at slightly different times. The wavefront aberration is the time each ray finishes minus the time of the fastest ray. In other words, it's the difference between the reference sphere and the wavefront. When the focus is stigmatic, the reference sphere and the wavefront coincide so the wavefront aberration is zero.

Another common aberration is called coma. In this case, rays at one edge of the pupil cross the finish line first; rays at the opposite edge of the pupil cross the finish line last. The effect is that the image of each object point resembles a comet with a tail. The word coma means “comet.” Coma

is common in patients with decentered corneal grafts. Coma is commonly seen in the aiming beam when performing retinal laser photocoagulation with a contact lens that produces an inverted image of the retina. If you tilt the lens too far off axis, the aiming beam spot becomes coma-shaped.

There are different ways to represent wavefront aberrations. One approach is to represent the wavefront aberrations as three-dimensional shapes. This is the approach adopted in the preceding illustrations. Some think that two-dimensional contour plots will be more popular. However, irregular astigmatism is a combination of a few basic shapes, just as conventional refractive error is a combination of sphere and cylinder.

Irregular astigmatism

If the orientation of the principal meridians changes from point to point across the pupil, or if the amount of astigmatism changes from one point to another, the condition is known as irregular astigmatism. Although the principal meridians are 90° apart at every point, it may sometimes appear by retinoscopy or keratometry that the principal meridians of the cornea, as a whole, are not perpendicular to one another. All eyes have at least a small amount of irregular astigmatism, giving rise to irregular reflexes in retinoscopy, but the term is used clinically only for gross

irregularities such as those occurring with keratoconus or traumatic corneal scars. Cylindrical lenses can do little to improve vision in these cases, although rigid contact lenses may be useful.

Contribution of the Corneal Layers and Shape to the Optics of the Eye

The air–tear film interface provides the major optical power of the eye. The tear film itself has a relatively small optical effect unless an abnormality is present. For instance, in patients with epiphora, the tear meniscus may partially cover the pupil and cause blurred vision. In addition, an uneven tear meniscus may result in deterioration of the quality of vision.

The optical power of the eye derives primarily from the anterior corneal curvature, which produces approximately two thirds of the eye's refractive power, accounting for approximately +48.0 diopters (D). The overall corneal power is less (approximately +43.0 D) as a result of the negative power (-5.8 D) of the posterior corneal surface. Standard keratometers and corneal topography instruments measure the anterior corneal radius of curvature. Because the back corneal surface curvature and the exact refractive index are not measured, these instruments estimate total corneal power from front surface measurements.

Another factor in corneal shape is that the central cornea is not spherical. The aspheric shape of the cornea generally reduces spherical aberration, minimizing refractive error fluctuations as the pupil changes size. When the central cornea is steeper than its periphery, the corneal shape is prolate. When the central cornea is flatter than its periphery, the corneal shape is oblate. Prolate corneas reduce spherical aberrations, while oblate corneas increase spherical aberrations.

KERATOMETRY AND PHOTOKERATOSCOPY

The keratometer is a useful instrument for measuring the curvature in the central region of the cornea. It is relatively accurate when the cornea closely approximates a spherocylindrical lens. In many circumstances, such as after keratorefractive surgery, in keratoconus, and in pellucid marginal degeneration, the keratometer is not an accurate method of corneal curvature analysis. However, additional information can be obtained by analyzing the quality of the reflected mires on the cornea. If the keratometry mires are irregular, irregular astigmatism is present. Two different patterns of irregular astigmatism are noted on keratometry. The first is seen when the examiner cannot superimpose the central keratometry

mires the second pattern is noted when the reflected mires are not crisp and sharp but have an irregular quality.

The keratometer can be used to measure inferior corneal steepening as well. This technique is very useful in evaluating patients with suspected keratoconus. First, central keratometry is performed; the patient is then instructed to look up and the measurements are repeated. The mires are now focused on the cornea, inferior to the visual axis. Attention is directed to the vertical meridian. In normal corneas the periphery is flatter than the central cornea. In keratoconus, the inferior cornea is steeper; thus the reading taken on upgaze will be steeper than the central value. Values of inferior corneal steepening greater than 1D are highly suggestive of keratoconus.

COMPUTERIZED VIDEOKERATOGRAPHY

Computerized videokeratography may also be useful in evaluating patients with minimal visual loss. Most of the currently available systems work by projecting a Placido disc onto the cornea, recording the reflected image, analyzing the image with a computer, and then displaying a color-coded curvature map of the corneal surface. These instruments generate thousands of data points from the anterior corneal surface and are

extremely sensitive in detecting subtle changes in curvature. One of the current systems, the TMS made by Tomey, calculates surface asymmetry index (SAI) with each topographic evaluation. The SAI correlates with central corneal asymmetry and is useful in monitoring changes in corneal topography. The surface regularity index (SRI) correlates with localized surface regularity of the cornea within the central area over the pupil in standard lighting conditions. The SRI is a useful prediction of the optical performance of the anterior corneal surface and has been shown to provide a good correlation with best spectacle-corrected visual acuity. There are other types of computerized videokeratography systems that use different imaging methods to examine the corneal surface, such as laser holography, raster photogrammetry, and projected-fringe contouring.

Corneal Topographer

Conventional keratometry measures the curvature of only the central 3 mm of the cornea. However, this is not representative of the entire corneal surface, since the curvature generally flattens from apex to limbus. A “map” of the corneal curvature can be useful in contact lens fitting as well as corneal refractive procedures.

Methods for measuring the corneal topography are commonly based on either a mire arrangement similar to a Placido disc which consists of many concentric lighted rings, or a standard keratometer directed to different, off-center areas of the cornea. One may consider a series of concentric lighted rings as a series of many different-sized mires, all in the same plane. Thus, the central ring would function very much like the standard mire on a keratometer and act as a target for the central 3 mm of cornea. The next ring can be considered to subtend the zone surrounding the center and produce a reflected ring representative of the curvature of that zone, and so on.

A flat series of illuminated rings held at the usual distance from the cornea can accurately measure only the central 7 mm of the cornea. To measure corneal curvature closer to the limbus, the concentric rings must be presented in the shape of a concave surface (i.e., open bowl) so that the distances from rings to cornea remain similar over the whole cornea. If the series of lighted ring targets is placed in front of a camera (the camera lens placed at an opening in the center of the ring pattern), the device is called a photographic keratoscope, and the picture of the reflected rings may be quantitatively analyzed. With irregular astigmatism (scars, keratoconus),

the irregular pattern of reflected rings can be used as a qualitative representation of the corneal map.

The use of computerized videokeratoscopes has grown rapidly in recent years. These devices provide computer analyses of multiple rings (often 16 or 32), producing color-coded dioptric maps of the corneal surface. Some of these instruments also calculate the SIM K (simulated keratometry) value, providing the power and location of the steepest and flattest meridians for the 3 mm optical zone. Other parameters include the surface asymmetry index (SAI) and the surface regularity index (SRI). The SAI is a centrally weighted summation of differences in corneal power between corresponding points 180° apart on 128 meridians that cross the four central mires. The SAI can be used to monitor changes caused by contact lens warpage or keratoplasty or by such progressive alterations as keratoconus, keratoglobus, Terrien marginal degeneration, and pellucid degeneration. The SRI is determined from a summation of local fluctuations in power among 256 hemimeridians on the 10 central mires.

Retinoscopy

Retinoscopy can detect irregular astigmatism by showing nonlinear or multiple reflexes that cannot be completely neutralized with a

spherocylindrical lens. With a multifocal cornea, retinoscopy reveals multiple regular reflexes that move in different directions. Irregular astigmatism and multifocal cornea can occur in keratoconus and after keratorefractive surgery. Abnormalities found with retinoscopy can help explain why a patient with a clear cornea cannot see well. Additionally, retinoscopy can disclose disrupted light reflexes caused by disturbances of the corneal surface. In cases where retinoscopic findings exceed the corresponding slit-lamp findings, retinoscopy can help gauge the relative effect of corneal surface changes on vision.[15]

Aberrations of the Retinoscopic Reflex

With irregular astigmatism, almost any type of aberration may appear in the reflex. Spherical aberrations tend to increase the brightness at the center or periphery of the pupil, depending on whether the aberrations are positive or negative.

As the point of neutrality is approached, one part of the reflex may be myopic while the other is hyperopic relative to the position of the retinoscope. This will produce the so-called scissors reflex.

Sometimes a marked irregular astigmatism or optical opacity produces confusing, distorted shadows that can markedly reduce the

precision of the retinoscopic result. In such cases, other techniques such as subjective refraction should be used.

All of these aberrant reflexes become more noticeable with larger pupillary diameters. In these cases, considering the central portion of the light reflex yields the best approximation.

MATERIALS AND METHODS

PATIENTS AND METHODS:

Inclusion Criteria

1. Age more than 18 years.
2. Any type of pterygium with the pterygium classified into either Primary, atrophic or recurrent pterygia.
3. Growing pterygium, which invaded more than 1mm into the cornea.

Exclusion criteria:

1. Recurrent pterygium
2. Eyes with any corneal pathology
3. Double pterygium

This study included a series of 50 patients selected within the constraints of the inclusion criteria. The patients' history of presenting illness is noted and history of hours spent outdoors and history of the use of sun protection were enquired. Then the patient was examined under diffuse illumination and by using the slit lamp. The size of the pterygium its extent were noted and the

pterygium was graded accordingly by using the horizontal beam of the slit lamp. The orbital anatomy was noted and also the normalcy of the lid closure was noted. After this is done the patient then underwent refraction by a refractionist, manual keratometry readings were obtained. Then the patient was screened using a Orbscan machine. Results were depicted as scatter plots and bar graphs and analysed by linear regression to determine the relationship between diopters of induced astigmatism and the size of extension of the pterygium.

The following details are to be collected from the patient:

PROFORMA

CASE NO ☐

NAME ☐ AGE ☐

MR.NO ☐

MALE ☐

FEMALE ☐

RIGHT EYE ☐

LEFT EYE ☐

OCCUPATION ☐

TOTAL NO OF HOURS SPENT OUTDOORS ☐

TOTAL NO OF HOURS SPENT IN DAYLIGHT ☐

PERSON USING SUNGLASSES Y ☐

(U-V protective) N ☐

PERSON USING UMBRELLA	Y	☐
	N	☐
CAP	Y	☐
	N	☐
VISOR	Y	☐
	N	☐
CLOTH	Y	☐
	N	☐

GRADE OF PTERYGIUM

T1	☐
T2	☐
T3	☐

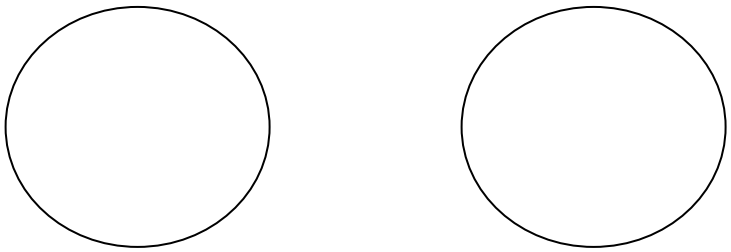
TYPE OF PTERYGIUM	Primary	☐
	Atrophic	☐
	Recurrent	☐

LOCATION OF PTERYGIUM	Nasal	☐
	Temporal	☐

NATURE OF THE PTERYGIUM:

Extension:

RE LE



Size in mm RE ☐ LE ☐

LID CLOSURE Complete ☐

Incomplete ☐

ORBIT Normal ☐

Abnormal ☐

DILATED REFRACTION

RE

LE

MANUAL KERATOMETRY

ORBSCAN

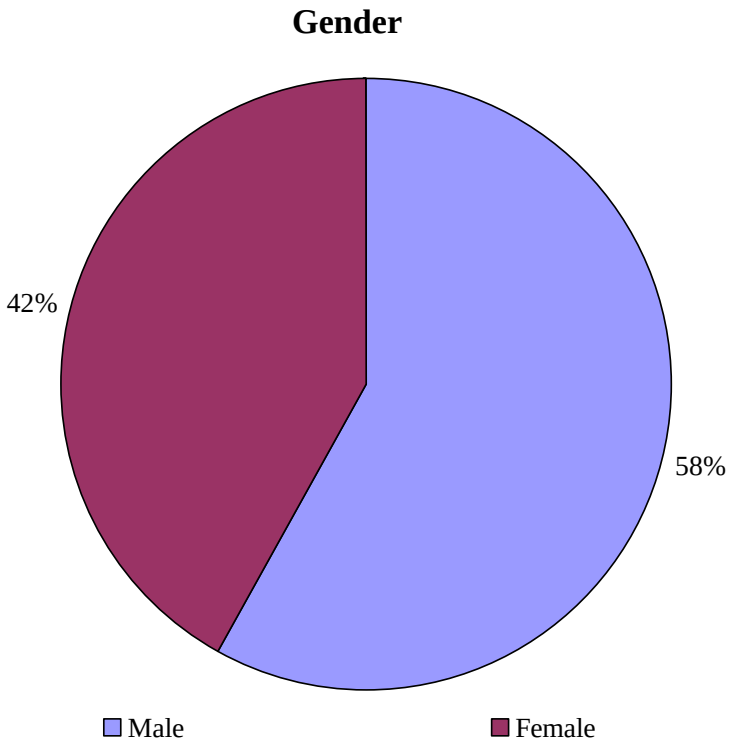
The grade of the pterygium was classified based on relative translucency of the body of the pterygium on slit lamp examination. In this grading Grade T1 denotes a pterygium in which episcleral vessels underlying the body of the pterygium were unobscured and clearly distinguished. Grade T3 denotes a thick pterygium in which the episcleral vessels underlying the body of the pterygium are totally obscured by the fibrovascular tissue. All other pterygia that donot fall into these two categories fall into Grade T2.[17]

OBSERVATIONS

SEX INCIDENCE:

SEX	
MALE	**FEMALE**
29	21

Of the 50 patients in the study 29(52%) were male and 21(48%) were female.



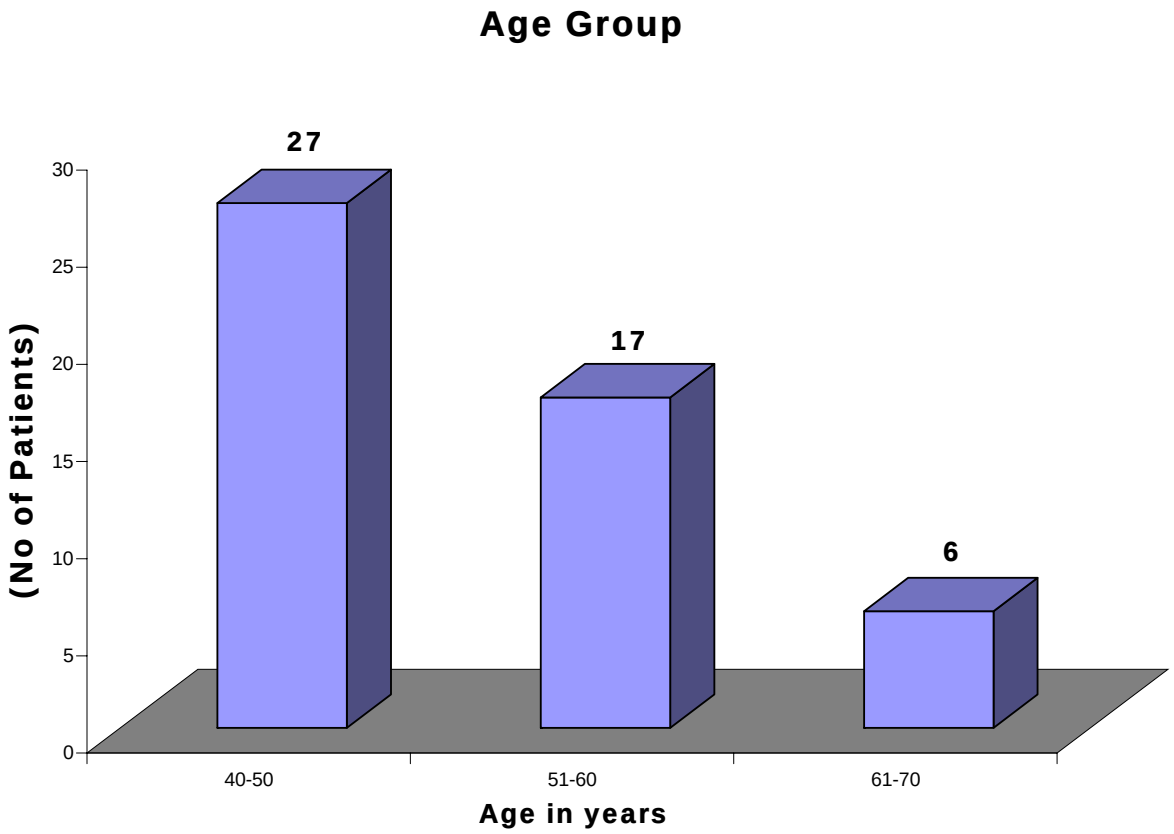
AGE DISTRIBUTION:

AGE	No OF PATIENTS
40-50 YEARS	27
51-60 YEARS	17
61-70 YEARS	6

Majority of the patients were in the age group of 40-50 years (54%).17 patients were in the age group of 51-60 years (34%) and the remaining 6 were in the age of 61-70 years (12%).

Occupation of the majority of the patients involved prolonged exposure to sunlight and dust .There were no patients using any forms of protection to sunlight in the above mentioned study group.

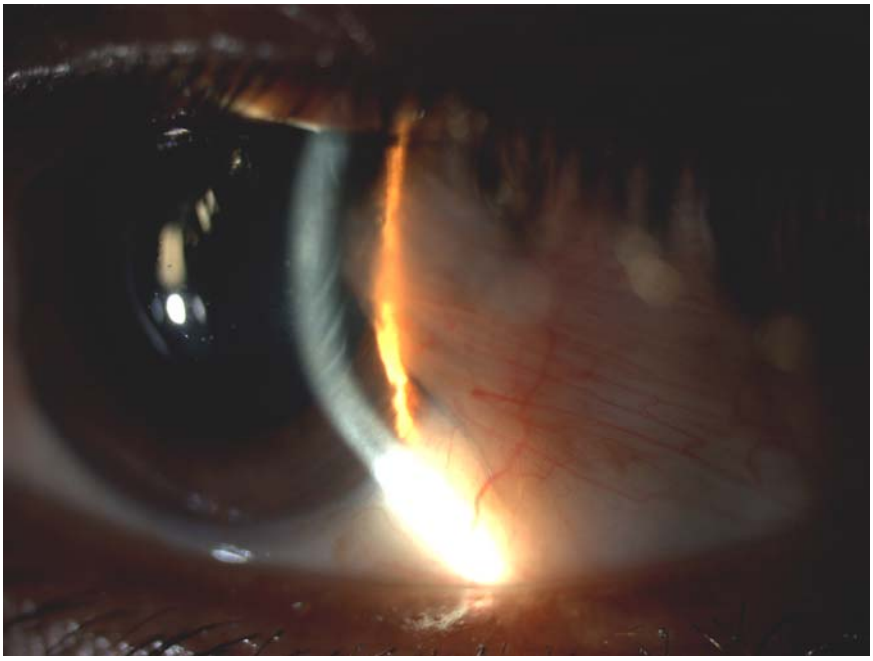
Graphical representation of age group distribution in the patients having pterygium.



PTERYGIUM LOCATION

Location of pterygium	No. of eyes	Percentage
Nasal	49	98%
Temporal	1	2%

49 eyes showed the presence of the nasal pterygium. 1 eye showed a temporal presence with Descemets folds radiating from the temporal portion of the limbus. Pterygium is known to cause traction on the cornea resulting in irregular hemimeridian with the rule astigmatism. The traction resulted in Descemet's membrane radial folds on the cornea.[26]

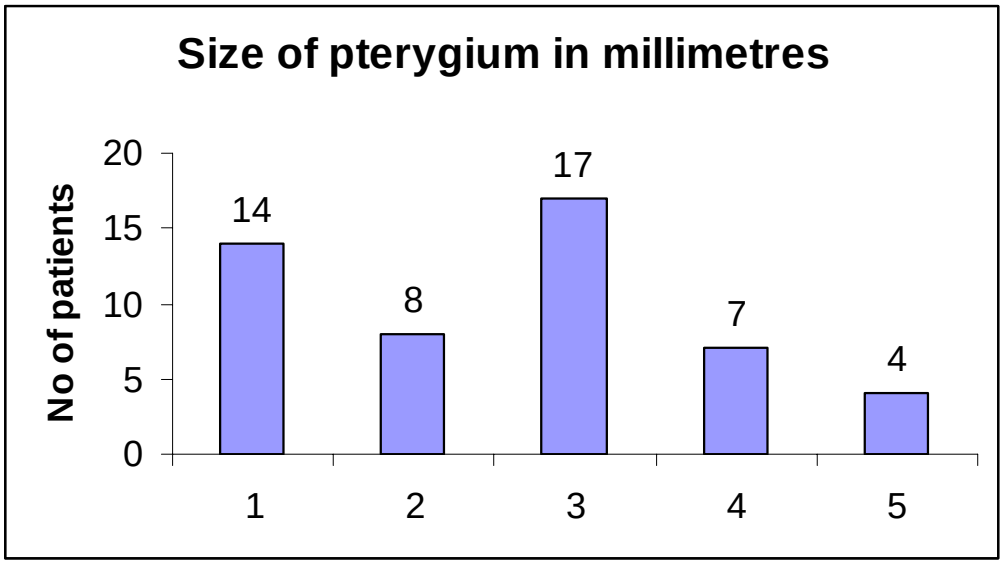


(Figure showing Descemet's folds on the temporal limbus)

PTERYGIUM LENGTH

Size in mm	No of eyes	Percentage (%)
0-1	14	28
1.1-2	8	16
2.1-3	17	34
3.1-4	7	14
>4.1	4	8

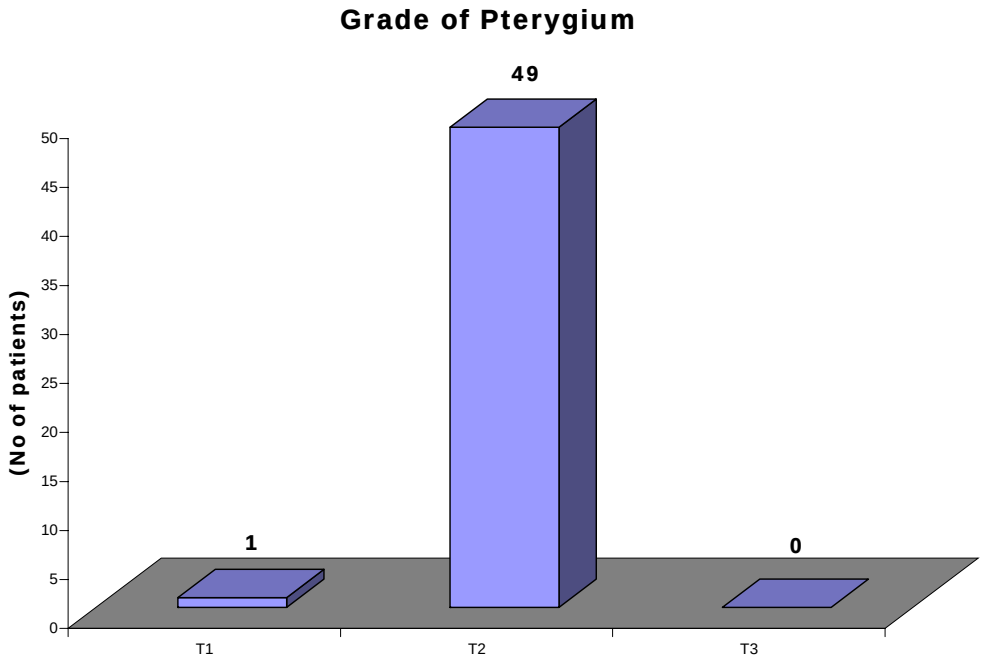
Majority of the eyes (34%) presented with a pterygium of size between 2.1mm to 3mm. 16% of the eyes had a length between 1.1mm to 2mm. 14% of the patients had a size ranging between 3.1mm to 4mm and only 8% of the patients had a pterygium of size greater than 4.1mm.



CLASSIFICATION OF PTERYGIUM

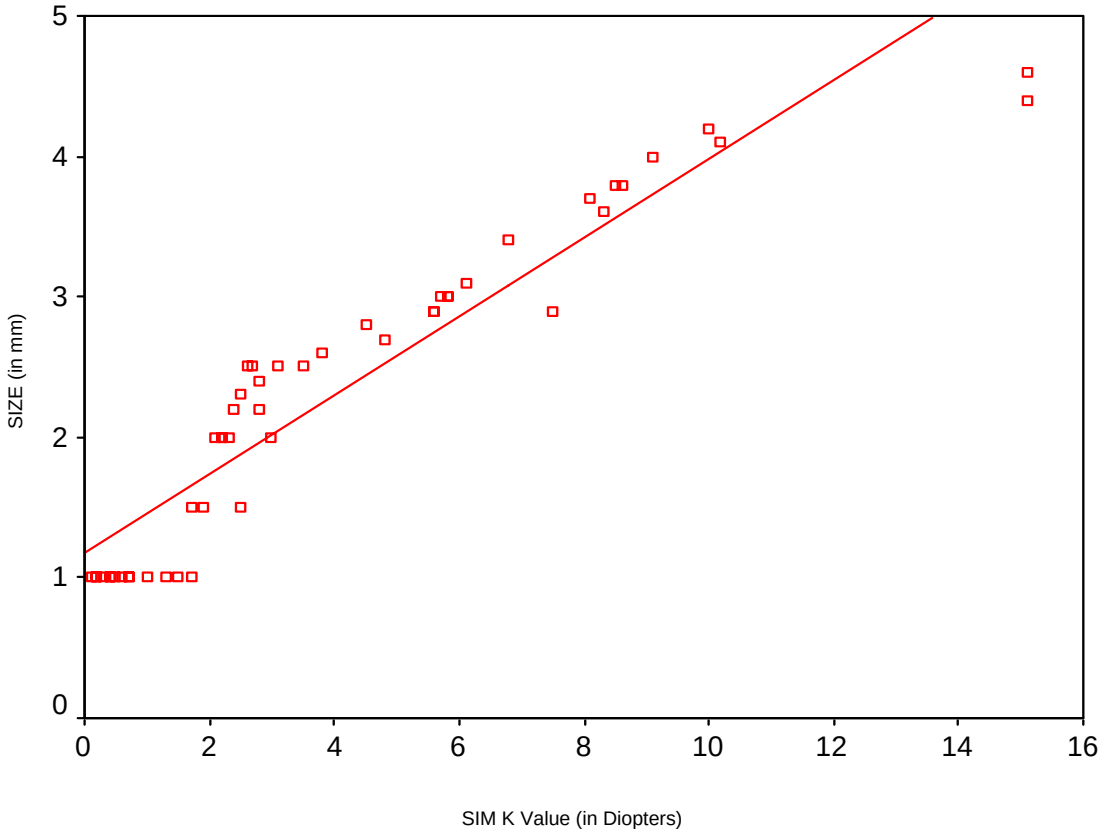
GRADE	
T1	1
T2	49
T3	0

Of the 50 patients 49 eyes were in the grade T2 as classified before and 1 eye belonged to the group T1. There were no patients in the group T3 in our study.



PTERYGIUM SIZE AND ASTIGMATISM

There was a significant correlation found between the size of the pterygium and the induced astigmatism (r-value 0.940 and p value of 0.00) as by Pearson correlation.

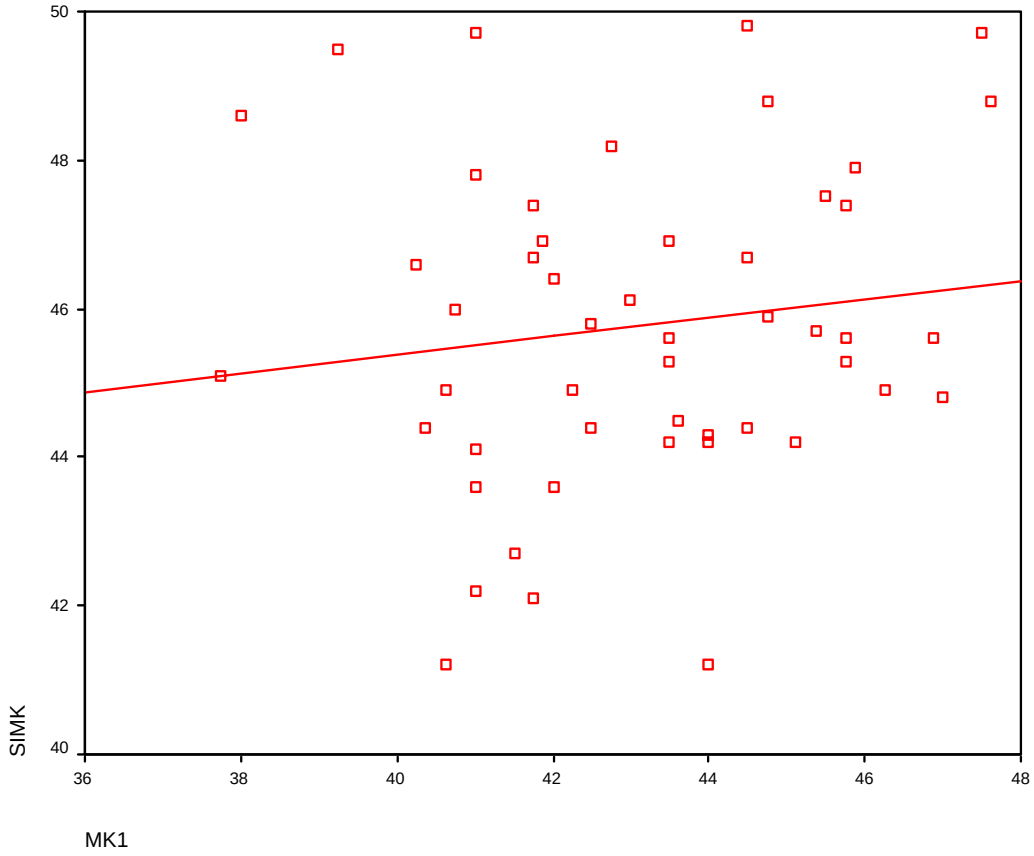


Pearson correlation

	SIM K
R- Value	.940
P- Value	.000

CORRELATION BETWEEN SIM K VALUES AND MANUAL KERATOMETRY VALUES:

There was no correlation found between the values of the K readings measured by using SIM K and by manual keratometry (r-value 0.140 and p-value 0.332) by Pearsons correlation.



Pearson correlation

	MK1
R- Value	.140
P- Value	.332

DISCUSSION

Pterygium induced astigmatism can often be the cause of subjective visual complaints. Previous studies have shown increased with the rule astigmatism in patients with pterygia by either refraction or keratometry. In this study this correlation was quantified by using the orbscan machine which provides the computer analyses of multiple rings (often 16 or 32), producing color-coded dioptric maps of the corneal surface. We also calculated the SIM K (simulated keratometry) value, providing the power and location of the steepest and flattest meridians for the 3 mm optical zone. The manual keratometry is limited in that it only measures the central cornea and the optical zone it measures is dependnt on the steepness of the cornea.

The amount of astigmatism measured in our patients represents the naturally occurring atigmatism plus the induced effect of the pterygium. The induced astigmatism is always with the rule whereas naturally ocuring astigmatism can occur in any axis.

Of the 50 patients in the study males had a higher incidence of pterygium. Majority of the patients were in the age group of 40-50 years (54%). 98% of the eyes showed the presence of the nasal pterygium.

Majority of the eyes (34%) presented with a pterygium of size between 2.1mm to 3mm.

Occupation of the majority of the patients involved prolonged exposure to sunlight and dust .There were no patients using any forms of protection to sunlight in the above mentioned study group.

There was a significant correlation found between the size of the pterygium and the induced astigmatism (r-value 0.940 and p value of 0.00) as by Pearson correlation.

CONCLUSION

Pterygia are fibrovascular growths extending from the bulbar conjunctiva onto the cornea. Once the pterygia reach a critical size they induce visually significant central with the rule astigmatic changes that may not be apparent by subjective refraction.

1. Of the 50 patients in the study group 52% were males and 48% were females.
2. 54% of the patients were in the age group of 40-50 years, 34% in the age group of 51-60 years and 12% were in the age group of 61-70 years.
3. Majority of the patients 98% had a nasal pterygium and were of the Grade T2.
4. There was a significant correlation found between the size of the pterygium and the induced astigmatism (r-value 0.940 and p value of 0.00) as by Pearson correlation.

   The amount of astigmatism measured in our patients represents the naturally occurring astigmatism plus the induced effect of the pterygium. Correlation between these two can only be estimated

when the pterygium has been removed surgically and the corneal topography done post operatively.

To conclude of the 50 patients in our study all the patients had induced with the rule astigmatism and the amount of astigmatism increases with the increase in the size of the pterygium.

SUMMARY

Title **:** Effect of pterygium on corneal astigmatism.

Sample size **:** 50 eyes

Study objective **:** To determine the effect of pterygium on corneal astigmatism.

Study conclusion **:** There was a significant correlation found between the size of the pterygium and the induced astigmatism (r-value 0.940 and p value of 0.00) as by Pearson correlation.

In this study there was a positive correlation between the size of the pterygium and the amount of induced astigmatism measured by using corneal topography.

Based on the results of this study we believe that the corneal topography analysis is an additional tool in the evaluation of the patient with pterygium allowing the measurement of optical changes in the cornea.

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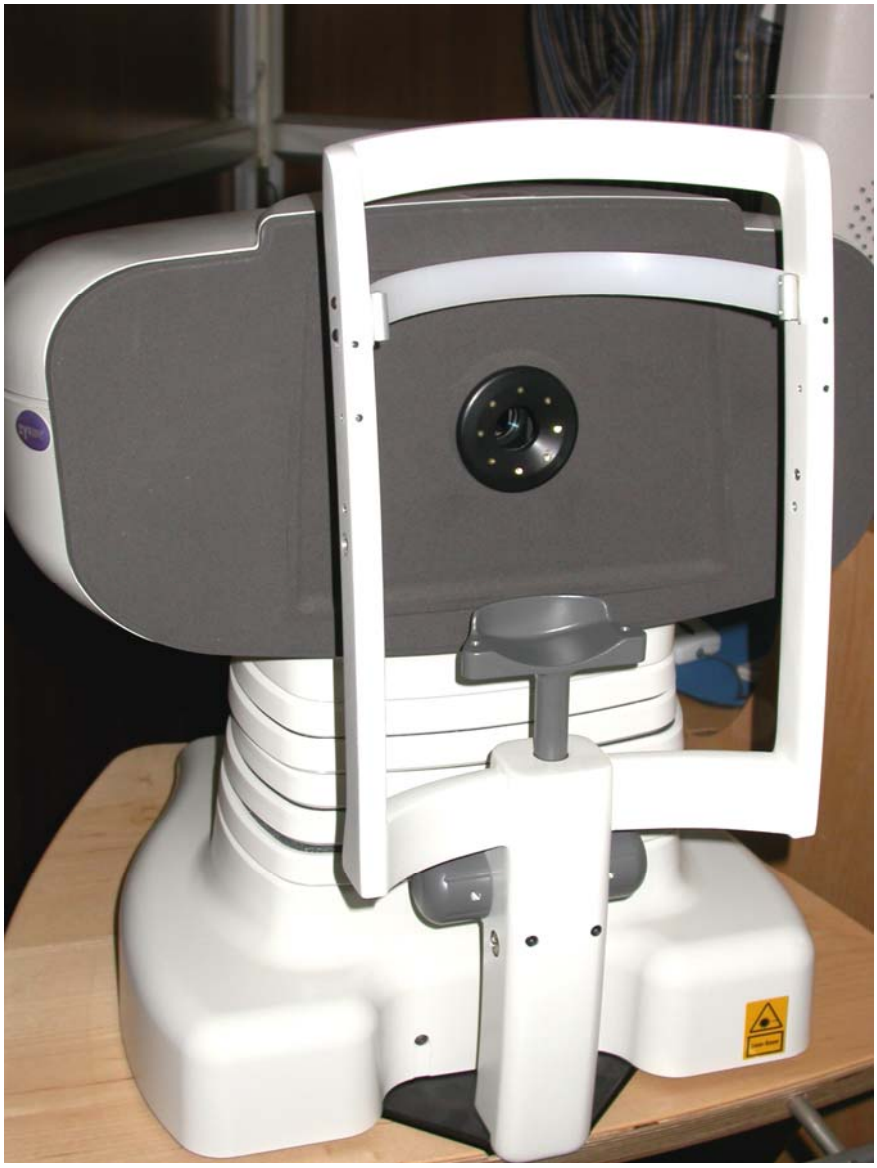
IMAGE OF THE ORBSCAN MACHINE

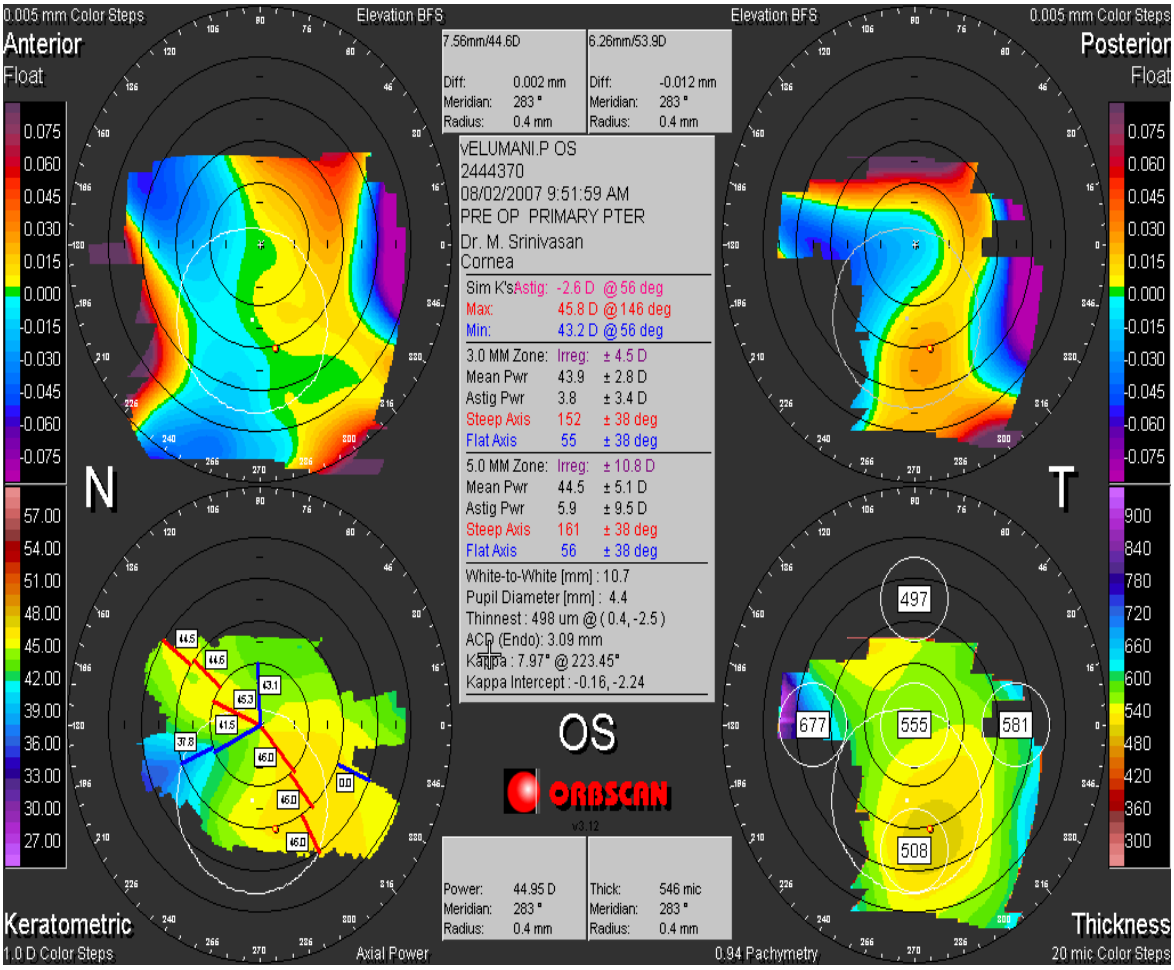


ORBSCAN MACHINE

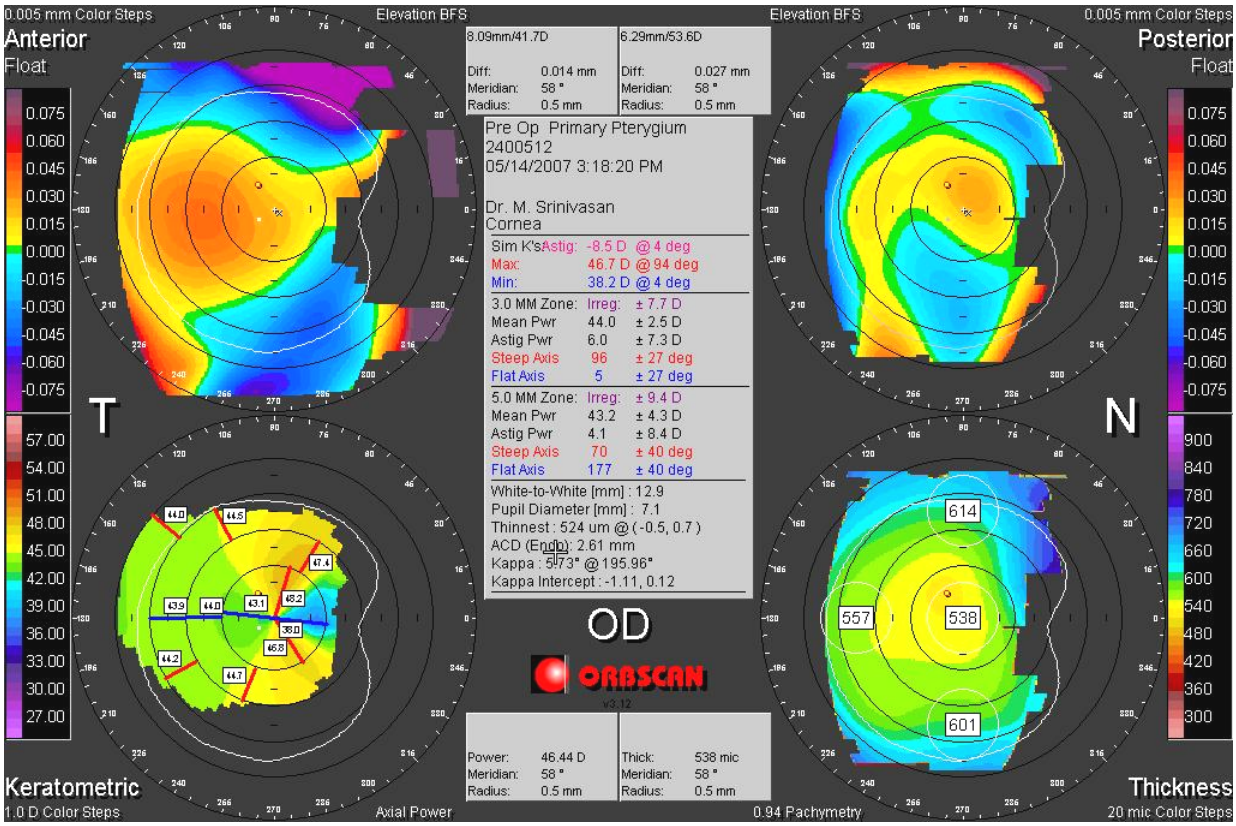
(Mires as in the Placido Disk)

(Mires as in the Orbscan)

ORBSCAN OF A PATIENT



Name	Age	Sex	Grade	Size in mm	Astigmatism
Annakodi	52	F	T2	3.8	-8.5D @ 4deg



Name	Age	Sex	Grade	Size in mm	Astigmatism
Murugesan	68	M	T 2	4.4	-15.1 D @ 154deg