"EVALUATION OF VISUAL ACUITY AND QUALITY OF LIFE AFTER CATARACT SURGERY IN KOLAR"

By

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Under the Guidance of
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LIST OF ABBREVIATIONS USED

PCO	»	Posterior capsular opacification
FGF	»	Fibroblast growth factor
EGF	»	Epidermal growth factor
IGF	»	Insulin-like growth factor
PDGF	»	Platelet-derived growth factor
TGF	»	Transforming growth factor
PTM	»	Post-translational modifications
GSH	»	Glutathione
IOL	»	Intra ocular lens
SMC	»	Senile Mature Cataract
SIMC	»	Senile Immature Cataract
SHMC	»	Senile Hyper-mature Cataract
AC	»	Anterior chamber
ICCE	»	Intra capsular cataract extraction
ECCE	»	Extra capsular cataract extraction
KPE	»	Kelman phacoemulsification technique
SK	»	Straite Keratopathy

ABSTRACT

BACKGROUND

Cataract has been consistently documented to be the most common cause of blindness in developing countries. Of the estimated 38-45 million blind in the world (vision <3/60 in the better eye, with available correction), nearly one fifth live in India alone. Senile cataract is the leading cause of blindness worldwide. Annual incidence of 3.8 million cataracts and over 9 million cataract blind people has been reported in India. The aim of cataract surgery is to improve visual function with the assumption that this will also improve overall quality of life. The gains from cataract extraction are usually demonstrated clinically by the change in the Snellen's visual acuity in the eye that had surgery.

METHODS

It is a hospital based prospective study of 500 patients with cataract attending RL Jalappa Hospital and research center, Tamaka, Kolar.

OBJECTIVES

1) . To evaluate visual acuity after cataract surgery.
2) To study the quality of life after cataract surgery.

RESULTS

In our study of 500 patients included, 228 were males and 272 were females. The mean age of the patient was 63 years. All patients were examined pre operatively as well as post operatively. Visual acuity was recorded on 1st day, 1st week and 6th week post operatively. All the complications were recorded post operatively. In this study 89.4% number of patients had a visual acuity of 6/18 or better and

complications encountered were iritis (12%), cystoid macular edema (6%), decentered IOL (11%), hyphaema (6%), PCO (1%) and straite keratopathy (8%). In this study pre operative general functions were 22.2+/- 3.25, social functions were 5.68+/- 1.07 and visual functions were 15.76+/-1.05. Post operatively general function score were 53.05+/-11.59,social functions were 13.35+/-3.07 and visual function were 23.23+/-3.18. As post operative score was high shows significant improvement in quality of life post operatively ($p<0.001$).

INTERPRETATION & CONCLUSION

Patients who underwent cataract surgery reported significant improvement in visual acuity and quality of life after cataract surgery.

KEYWORDS

Senile immature cataract, senile mature cataract, Intra ocular lens, Intra ocular pressure, Quality of life, Visual acuity.

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INTRODUCTION

Cataract has been consistently documented to be the most common cause of blindness in developing countries. Of the estimated 38-45 million blind in the world (vision <3/60 in the better eye, with available correction), nearly one fifth live in India alone.[1] Senile cataract is the leading cause of blindness worldwide. Annual incidence of 3.8 million cataracts and over 9 million cataract blind people has been reported in India. Cataract is a highly treatable condition due to dramatic advances in cataract surgery procedures ad intraocular lens design. In developing countries surgery is generally performed when cataract affects patient's daily activities.

The aim of cataract surgery is to improve visual function with the assumption that this will also improve overall quality of life. The gains from cataract extraction are usually demonstrated clinically by the change in the Snellen's visual acuity in the eye that had surgery. The impact on visual function or quality of life has not usually been considered as a separate issue, partly as these have been assumed to follow the improvement of visual acuity.

Visual impairment from cataract is associated with difficulties with activities of daily living and reduces quality of life. In rural low income settings, where the prevalence of unoperated cataract is highest, all household members regardless of age commonly contribute to basic needs of a household.

Post surgery improvements in social functionality could indicate an enhanced ability by patients to achieve greater autonomy due to the capacity to perform social activities more frequently. Relationships and social activities improve in accordance with the improvements in physical and emotional functions.

AIMS AND OBJECTIVES

1) To evaluate visual acuity after cataract surgery

2) To study the quality of life after cataract surgery.

REVIEW OF LITERATURE

Visual outcomes of 2369 cataract operated persons (3655 eyes) across seven major Indian states were assessed in 1998. This is the largest ever study over the past decade in the country. 9.54 percent of the examined population had undergone operation for cataract in one or both eyes[1].

An **Auckland study** of 488 eyes found that post cataract surgery visual acuity was 6/12 or better in 88% of eyes[2].

It is estimated that at least 105000 cataract operations are performed annually within the National Health Service, with at least a third of these being second eye procedures. As such, the finding indicate that significant additional gains in visual function and quality of life (health related and vision related), were achieved after second eye surgery[3].

Age effects regarding cataract surgery were studied by **Westcott et al** .This report examined 880 patients aged over 60 years who had cataract surgery. In patients with no co morbidities, the odds of achieving 6/12 or better acuity was 4.6 times higher among persons aged 60-69 years than among persons aged 80 years or older[4,].

Nursing home residents in USA have high rates of vision impairment with estimates ranging from 3 to 15 times higher than corresponding rates for community-dwelling older adults. Residents who underwent cataract surgery because of functional problems experienced short term significant improvements in their vision-targeted health related quality of life, in addition to improvement in their vision[5] .

Uncorrected refractive errors and Posterior Capsular Opacification (PCO) were primary causes of almost 40% of visual impairment in cataract operated participants. Uncorrected refractive errors can be treated easily by providing

corrective lenses. Posterior Capsular Opacification can be resolved easily with yttrium-aluminium-garnet capsulotomy[6.]

Several studies show that the patient's subjective assessment of visual function and vision related activities can reliably affect the visual impairment and quality of life.[7,8.]Quality of life assessments have been done in several other studies but these studies require a long time to assess and make the use of these questionnaires [9,10,11,12]. In our study, we used a simple, modified questionnaire to suit the environment and life style of our patients to compare quality of life and patient rehabilitation after cataract surgery using 7mm modified chevron, small incision cataract surgery. Reference

Another study conducted by **Owsley C et al[13]** on the impact of cataract surgery on health related quality of life in nursing home residence reported significant score improvement in the general vision (p=0.005), reading (p=0.001), psychological distress and social interaction of the nursing home vision targeted health related quality of life questionnaire.

As shown in other studies a study conducted by **Applegate W.B.[14]** et al showed that maximum improvement in vision and improvements in subjective measures of patient function occurred by 4 months and most improvement were maintained at 1 year.

The Cataract Outcome Study conducted by **P.Desai et al[15]** in 1993 and the Cataract Impact Study conducted by Polack S et al[5] in 2005 reported that visual function scores and health related quality of life scores obtained after cataract surgery resulted in less vision dependent activities. **Applegate MD et al[16]** showed that maximum improvement in vision occurred by 4 months and most improvements were maintained at one year. These surveys which have been conducted to assess quality of

life after cataract surgery have been utilized as a quantitative measurement of the cataract surgery results.

Huang F C[17] et al reported that during early post operative period (within 48 hours) 23.3% of all patients have some complications. The most frequent recorded were corneal edema (9.5%), raised IOP (7.9%) and uveitis (5.6%).

Khan M T[18] et al reported that 17(11.3%) eyes were found to have hyphaema at first post op follow up examination. Hyphaema resolved in 11(7.3%) cases in one week and in all cases 6- week post operatively. Post operative endopthalmitis was diagnosed in 2(1.3%) cases on first post op day, which was successfully treated. Corneal complications like corneal edema and striate keratopathy were noted in 19(12.7%) patients at first post op day. In 5(3.33%) cases, IOP was found to be more than upper level of normal (i.e. above 21mmHg) on first post op day.

In 2005 an observation study was published by **Lee[19] et al** indicating that cataract symptoms are highly associated to quality of life related to eyesight.

CATARACT

Cataract is a clouding that develops in the crystalline lens of the eye or in its envelope (lens capsule), varying in degree from slight to complete opacity and obstructing the passage of light. Early in the development of age-related cataract, the power of the lens may be increased, causing near-sightedness (myopia), and the gradual yellowing and opacification of the lens may reduce the perception of blue colors. Cataracts typically progress slowly to cause vision loss, and are potentially blinding if untreated. The condition usually affects both eyes, but almost always one eye is affected earlier than the other[20]. Often both eyes are affected. It is one of the leading causes of blindness in the world today, accounting for 50% of blindness worldwide[21].

Structure of lens and transparency -

Histologically the lens is composed of capsule, epithelium, and lens cells. The lens is derived from surface ectoderm cells overlying the optic vesicle. The embryonic nucleus develops by the sixth week of gestation which is surrounded by the fetal nucleus. At birth, the embryonic and fetal nuclei make up most of the lens. Later from birth to puberty infantile nucleus will be there, after that for rest of the life adult nucleus will be there. Postnatally, cortical lens fibers are laid down from the conversion of anterior lens epithelium into cortical lens fibers. The lens continues to grow throughout life through the regular addition of new lens fibers which are continually formed at the equatorial germinative zone. As the new fibers are formed, the old fibers move to the center and the new fibers occupy the outer part. [22]

Lens transparency depends on the regular arrangement of the lens fibers and of the cytoplasm within the fibers, and their disorganization results in the development of cataract. The proteins exist in the cytoplasm of the lens fibers as a complex protein

solution. Approximately 90% of these proteins are crystallins (alpha, beta, and gamma) and transparency is achieved as a result of tight packing of these molecules[22].

History of cataract-

Cataract is derived from the Latin word *cataracta* meaning "waterfall" and that from the Greek Ka1apak (*down*-rushing) [23]. As rapidly running water turns white, the term may later have been used metaphorically to describe the similar appearance of mature ocular opacities. In Latin, cataract had the alternate meaning "portcullis" and the name possibly passed through French to form the English meaning "eye disease" (early 15th century), on the notion of"obstruction". Early Persian physicians called the term *nazul-i-ah,* or "descent of the water"-vulgarised into waterfall disease or cataract-believing such blindness to be caused by an outpouring of corrupt humour into the eye.

Epidemiology of cataract -

World Health Organization calculated that the number of visually impaired people worldwide was in excess of 161 million. Cataract is the leading cause, accounting for 47.8% of all cases.[24]The estimated global costs of blindness and low vision was estimated at US$42 billion.[25] Each person who is blind requires a caregiver, placing demands on one tenth of their time so reducing their economic activity.[26] Over the next 20 years it is estimated that the world's population will increase by about one third, this growth occurring predominantly in developing countries. During the same period, the number of people over the age of 65 years will be more than double. Therefore, there will be approximate doubling in the incidence of cataract, visual morbidity, and need for cataract surgery In the developed world, the threshold for

cataract surgery is now 6/9 or less, which has resulted in a three- to fourfold increase in patients receiving surgery with an associated increased need for resources and funding.

Without trivializing the challenges for developed nations, the real challenges are in developing countries, which will bear an increased burden for cataract blindness. Cataracts occur earlier in life in developing countries, and the incidence is higher. In India, visually significant cataract occurs 14 years earlier than in the United States, and the age-adjusted prevalence of cataract is three times that of the United States. [27, 28.] In addition there are fewer ophthalmologists to carry out the surgery. It is possible for governmental and nongovernmental organization programs to reduce the prevalence of blindness, as illustrated by the Gambian Eye Care program, which reduced the prevalence of blindness from 0.7% to 0.42% between 1986 and 1996.[1] However, in the developing world, the shift from intracapsular to extracapsular cataract surgery has also resulted in a lower visual threshold for surgery and increased the number of operations that need to be done. Developing intraocular lens manufacturing facilities in these countries (such as the Fred Hollows Foundation in Eritrea and Nepal), will reduce costs and improve access to surgery.

Pathophysiology of cataract -

The lens transmits, filters, and focuses light onto the retina. The lens has a high refractive index and is transparent because of the high concentration and orientation of structural proteins: α, β, and γ crystallins. Lens cells are epithelial in origin. A single layer of cuboidal cells is found on the anterior surface of the lens, below the capsule. They are nucleated, actively divide, and account for almost all the metabolic activity of the lens. Cuboidal cells in the equatorial zone of the lens differentiate and

elongate into lens fiber cells, and lose their nuclei and intracellular organelles such as mitochondria. Thus, most of the lens consists of mature lens fibers, which lack the ability to perform metabolic functions such as protein synthesis and energy production. The fibers are forced toward the interior of the lens and are compressed as new fibers are deposited over them. As the lens fibers age, other biochemical, physiological, and structural changes occur. Aging changes share some similarities with age-related cataract changes; however, there are also unique cataract changes. [29,30]

The transparency of the lens is dependent on the regular organization of the lens cells and intracellular lens proteins. Genetic, metabolic, nutritional, and environmental insults and ocular and systemic diseases cause cataracts by affecting lens clarity. These factors disrupt cellular organization and intracellular homeostasis, eventually causing spatial density fluctuations, light scattering, and absorption, which compromise vision. Cataract is not a single disease. There are different causes, morphologies, and rates of opacification. Once damaged, the lens has limited means of repair and regeneration, and may lose its transparency by the formation of opaque lens fibers, fibrous metaplasia, epithelial opacification, accumulation of pigment, or formation of extracellular materials. Several interlinked mechanisms for cataract formation have been proposed, and no single theory completely explains age-related cataract (the commonest form).[4] Although much is still unknown about cataractogenesis, many of the important components are becoming clearer.

Cell Proliferation and Differentiation –

The proliferation and differentiation of the epithelial cells are under the control of growth factors present in the media that bathe the lens. Fibroblast growth factor

(FGF), which stimulates epithelial proliferation, is produced in the ciliary epithelium and is present in low concentrations near the anterior lens surface. Higher concentrations near the lens equator induce differentiation into lens fibers. Other growth factors, such as epidermal growth factor (EGF), insulin-like growth factor (IGF), platelet-derived growth factor (PDGF), and transforming growth factor (TGF-β) are also involved in these processes. If the concentration of FGF is too low, the concentrations of other growth factors are incorrect, or differentiation is inhibited by a cytokine, differentiation of epithelial cells into fiber cells in the equatorial zone will not occur. The undifferentiated cells continue to migrate to the posterior pole. Therefore, incorrect cell maturation, differentiation, and proliferation can result in posterior subcapsular cataract formation.[29]

Metabolic Disturbance and Osmotic Regulation Failure –

Altered gene expression causes changes in enzyme, growth factor, membrane protein, and other protein levels. This causes a reduction in energy production, cytokine fluctuations, changes in ion transport, calcium metabolism and antioxidant pathways, and a breakdown in protective mechanisms.[29]

The lens maintains ion differentials between intra- and extracellular fluids (high potassium and low sodium internally; low potassium and high sodium externally) via the action of the sodium-potassium ATPase pump. Pump inactivation causes increased intracellular osmolality, which with membrane leakiness results in localized water accumulation and light scatter.[29]

The lens epithelial cells are bathed in aqueous humor, a source of nutrients and mineral ions including calcium (Ca^{2+}). Ca^{2+} is a versatile intracellular signal that regulates many functions including the permeability of the cell membranes. The

extracellular Ca^{2+} concentration is 10 times the intracellular Ca^{2+} concentration, and this gradient drives Ca^{2+} into the epithelial cell. Ca^{2+} pumps on the plasma and intracellular organelle membranes regulate cytoplasm Ca^{2+} levels. Within the cell, very little Ca^{2+} is free; most is bound to complex proteins including crystallins or sequestered in the intracellular organelles (the endoplasmic reticulum, golgi apparatus, and mitochondria). Extracellular Ca^{2+} can be bound to lipid molecules in the outer layer of the cell membrane. Reduced capacity of membrane lipids to bind Ca^{2+} affects cell membrane permeability and causes a deterioration of intracellular Ca^{2+} homeostasis, a rise in intracellular Ca^{2+} levels, the formation of calcium oxylate crystals, the formation of strong bonds between Ca^{2+} and insoluble lens proteins, increased light scattering, and nuclear cataract formation. Increased intracellular Ca^{2+} levels also affect lens epithelial cell terminal differentiation causing posterior subcapsular cataracts. Steroids have been shown to mobilize intracellular Ca^{2+} in other tissues. It is hoped that in the future Ca^{2+}-regulating drugs will be developed that prevent cataracts.[31]

Calpains –

Calpains are a group of intracellular cysteine proteases, which are activated by Ca^{2+}. The physiological roles of calpains in the lens are poorly understood, but they may be needed to degrade damaged lens proteins that accumulate during the life of the lens. Calpains can contribute to cataract in two ways. First, a lack of calpains can lead to pathologically elevated levels of damaged proteins, reduced optical performance, and cause cataract. Second, excessive stimulation of calpain activity by Ca^{2+} can also lead to unregulated proteolysis and cataract. Calpain inhibitors could therefore be useful in the nonsurgical treatment of cataract. However, calpain inhibitors of high molecular

weight are unable to cross membranes and are of no therapeutic use at present, while others have poor water solubility or are toxic to lenses.[32]

Protein Modification –

Post-translational modifications (PTMs) of lens proteins can be additive, neutral, or subtractive, which increase, cause no change, or decrease the molecular weight of lens proteins, respectively. Additive modifications are numerous and typically involve covalent bonding of small molecules to polypeptides. Methylation, acetylation, carbamylation, glycation in diabetics, and binding of ascorbate are examples of PTMs and may be responsible for the coloration of the lens. The addition of these small molecules to proteins occurs especially in disease and can alter the function or properties of a protein. Diabetes (reducing sugars), renal failure (cyanate generated from urea), aging (photo-oxidation products), and steroid use (ketoamines) have been linked to cataracts. PTMs can cause further protein reactions such as polymerization and make proteins more susceptible to photo-oxidation by UV light. [29, 30]

Subtractive PTMs include proteolysis of crystallins and other lens proteins by enzymes including calpains. Crystallin cleavage causes precipitation of lens proteins. Cleavage of channel proteins can affect intercellular communication or create the lens barrier. Neutral PTMs such as isomerization affects the function of the protein and can result in denaturation. Deamidation changes the charge and affects protein-protein interactions.

Proteins in the center of the lens are as old as the individual; therefore, despite being very stable, they can be modified over several decades. Protein modification causes conformational changes (unfolding) that expose thiol groups, which are usually

"hidden" in the folds of the protein. These groups are oxidized to form disulfide bonds such as oxidized glutathione (GSSG) causing aggregation of proteins. Further conformation changes and aggregation occur, which result in scattering and absorption of light.

Oxidation -

Oxidation is a key feature in the pathogenesis of most cataracts and low oxygen levels (O_2) are important for maintaining a clear lens. There is a steep oxygen gradient from the outer part of the lens to the center. Mitochondria in the lens cortex remove most of the oxygen, thus keeping nuclear O_2 levels low. However, in older people mitochondrial function diminishes and superoxide production by the mitochondria increases resulting in increased nuclear oxygen and superoxide levels. As the lens ages, a lens barrier develops at approximately the cortex-nuclear interface, which impedes the flow of molecules such as antioxidants (including glutathione) into the nucleus. Unstable nuclear molecules such as H_2O_2, which are generated in the nucleus or which penetrate the barrier, therefore have more time to cause protein oxidation. Also there is a lower concentration of antioxidants. Decomposition of UV filters in the nucleus also produces unstable, reactive molecules that bind to proteins, especially if glutathione (GSH) levels are low. Ascorbate also becomes reactive with proteins in the absence of GSH. These oxidative changes can be detected even in the earliest cataracts and are progressive. Elevated levels of superoxide or peroxide ($H_2O_{2)}$ in the aqueous (exogenous H_2O_2) may play more of a role in the development of cortical cataracts since the cortex is closest to the aqueous. Copper and iron also play a role in oxidative damage. They are present in higher concentrations in cataract lenses, and both are involved in redox reactions, which produce hydroxyl radicals.[30]

Defensive Mechanisms -

Primary defenses are provided by antioxidant enzymes and antioxidants such as ascorbate, glutathione, tocopherols, and carotenoids, which maintain lens proteins in the reduced state. A decrease in nuclear concentration of GSH can occur while the cortical levels remain normal even in advanced age-related nuclear cataracts. In advanced age-related nuclear cataracts more than 90% of protein sulfhydryl groups and almost half of all methionine residues in the nuclear proteins become oxidized. Secondary defenses include proteolytic and repair processes, which eliminate damaged proteins, UV filters, and other molecules such as glutathione reductase and free radical scavenging systems. Failure of these protective mechanisms, a shortage of antioxidants, and increased free radicals result in cell membrane and protein damage. [29, 30]

Other Factors -

Crystallins may have a number of functions. For example α-crystallin may be a chaperone that binds to other lens proteins to prevent precipitation. Decreased crystallin levels cause proteins to precipitate, which leads to cataract formation. Phase separation of proteins refers to the hydrophobic aggregation of lens proteins causing reversible protein rich and poor regions in the lens fibers, which results in light scatter. The lipid composition of the cell membranes alters dramatically with age. PE plasmalogen and phosphatidylcholine levels decrease and sphingolipid levels increase, which may have functional consequences.

Etiological classification-

Due to various etiologies and different morphological patterns, classification of cataract can be done in many ways. We have tried to classify cataract according to the etiology and further dividing into different morphological types depending upon the site, as a particular cause may predispose to a specific morphological type of cataract.

1. Congenital cataract
2. Acquired cataract
    a. Age-related cataract
    b. Toxic – smoking, drugs
    c. Radiation
    d. Systemic diseases
    e. Ocular diseases
    f. Trauma
    g. Metabolic

Etiology-

Age -

Many factors cause age-related cataract. The cumulative effect of environmental factors (UV light, x-irradiation, toxins, metals, steroids, drugs, and diseases including diabetes) plays a role. Gene expression changes result in altered enzyme, growth factor, and other protein levels. Protein modification, oxidation, conformational changes, aggregation and phase separation, formation of the nuclear barrier, increased proteolysis, defective calcium metabolism, and defense mechanisms are also important factors. Compromised ion transport leads to osmotic imbalances and

intercellular vacuolation. Abnormal cellular proliferation and differentiation also produces opacities

Trauma –

Blunt trauma, which does not result in rupture of the capsule, may cause an anterior and/or posterior subcapsular cataract, or both. Initially, fluid influx causes swelling and thickening of the lens fibers. Later the fibers become less swollen; the anterior subcapsular region whitens and may develop a characteristic flower-shaped pattern or an amorphous or punctuate opacity. A Vossius ring of iris pigment may be present on the anterior capsule. If the capsule is ruptured, it usually ruptures posteriorly; the lens is rapidly hydrated forming a white cataract. A small capsular penetrating injury may result in localized lens opacity. A larger rupture results in rapid hydration and complete opacification. Penetrating injuries can be caused by accidental or surgical trauma such as a peripheral iridectomy or during a vitrectomy

Electric shocks as a result of lightning or an industrial accident cause coagulation of proteins or osmotic changes. These cataracts are typically fern-like with a grayish white anterior and posterior subcapsular opacities.[33] Sources of ionizing radiation, such as from x-rays, damages the capsular epithelial cell DNA, affecting protein and enzyme transcription and cell mitosis. An enlarging posterior pole plaque develops. Nonionizing radiation, such as infrared, is the cause of cataract in glassblowers and furnace workers working without protective lenses. A localized rise in the temperature of the iris pigment epithelium causes a characteristic posterior subcapsular cataract, which may be associated with exfoliation of the anterior capsule.

Systemic Disorders -

In uncontrolled type 1 diabetes mellitus, a bilateral diabetic snowflake cataract may occur in young people. Hyperglycemia causes glucose to diffuse into the lens where it is converted to sorbitol by the enzyme aldose reductase. The cell membrane is impermeable to sorbitol, therefore it accumulates in the lens fiber. Water enters the lens to correct the osmotic imbalance and the lens fibers swell then rupture. The onset is usually quick with the development of white, anterior and posterior subcapsular and cortical opacities with vacuoles and water clefts.

In type 2 diabetic adults, an earlier onset age-related type of cataract occurs. They are more prevalent with longer duration of the diabetes, occur earlier, and progress more rapidly than other age-related cataracts. Many mechanisms are involved and include sorbitol accumulation, protein glycosylation, increased superoxide production in the mitochondria, and phase separation. During hyperglycemia, glucose is reduced to sorbitol using up antioxidant reserves, so less glutathione can be maintained in the reduced form, which causes oxidative stress. In diabetic cataractogenesis, levels of lens Ca^{2+} are elevated, which activates calpains causing unregulated proteolysis of crystallins. In animal models, aldose reductase inhibitors have been shown to prevent the development of cataracts, but no benefit has been demonstrated in humans. The cataracts are usually cortical or posterior subcapsular or less frequently nuclear. [34, 35]

Galactosemia is an autosomal recessive disorder where a lack of one of the three enzymes involved in the conversion of galactose into glucose causes a rise in serum galactose levels. There is an accumulation of galactitol within the lens and in a similar process to diabetes; the osmotic imbalance is corrected by water inflow. Anterior and posterior subcapsular opacities occur during infancy, which later become nuclear. Galactose 1-phosphate uridyltransferase galactosemia is also associated with failure to

thrive, mental retardation, and hepatosplenomegaly. Progression of the cataract can be prevented if galactose is removed from the diet. Galactokinase deficiency is associated with galactosemia and cataract but without the systemic manifestations.[36]

Fabry's disease is an X-linked lysosomal storage disorder that results in accumulation of the glycolipid ceramide trihexoside. The patient suffers from episodic fever, pains, hypertension, renal disease, and a characteristic rash. In the affected man and the carrier woman, a typical mild, "spoke-like," visually insignificant cataract develops.

Lowe's or oculocerebrorenal syndrome is a severe X-linked disorder that results in mental retardation, renal tubular acidosis, aminoacidosis, and renal rickets. Associated congenital glaucoma, congenital cataracts, and corneal keloids can all lead to blindness. The cataract is total, the lens being small and discoid. Female carriers may show focal dot opacities in the cortex.

Alport's syndrome is a dominant, recessive, or X-linked trait disease causing hemorrhagic nephropathy and sensorineural deafness. Ocular features include congenital or postnatal cortical cataract, anterior or posterior lenticonus, and microspherophakia.

Dystrophia myotonica is a dominantly inherited disorder and results in muscle wasting and tonic relaxation of skeletal muscles. Other features include premature baldness, gonadal atrophy, cardiac defects, and mental retardation. Cataract is a key diagnostic criterion and may develop early, but usually occurs after 20 years of age and progresses slowly, eventually becoming opaque. Early cataract consists of polychromatic dots and flakes in the superficial cortex. As the opacities mature, a

characteristic stellate opacity appears at the posterior pole. Other ocular features include hypotony, blepharitis, abnormal pupil responses, and pigmentary retinopathy.

Rothmund-Thompson syndrome is an autosomal recessive disorder characterized by poikiloderma, hypogonadism, saddle-shaped nose, abnormal hair growth, and cataracts, which develop between the second and fourth decades of life and progress rapidly.

Werner's syndrome is an autosomal recessive disorder with features that include premature senility, diabetes, hypogonadism, and arrested growth. Juvenile cataracts are common. The condition usually leads to death at about 40 years of age.

Cockayne's syndrome causes dwarfism, but with disproportionately long limbs with large hands and feet, deafness, and visual loss from retinal degeneration, optic atrophy, and cataracts.

Dermatological Disorders -

Both the skin and the lens share a common embryological origin, the ectoderm. Therefore, skin disorders may be associated with cataract formation.

Atopic dermatitis and eczema may affect any part of the body, especially the limb flexures. Cataract develops in some atopic adults, usually as a bilateral, rapidly progressive "shield cataract." This is a dense, anterior subcapsular plaque with radiating cortical opacities, and wrinkling of the anterior capsule because of localized proliferation of lens epithelium. Posterior subcapsular opacities may also occur.

Ichthyosis is an autosomal recessive disorder that features hypertrophic nails, atrophic sweat glands, cuneiform cataracts, and nuclear lens opacities.

Incontinentia pigmenti is an X-linked dominant disorder that affects skin, eyes, teeth, hair, nails, and the skeletal, cardiac, and central nervous systems. Blistering skin lesions occur soon after birth, followed by warty outgrowths. Ocular pathology includes cataract, chorioretinal changes, and optic atrophy.

Central Nervous System Disorders –

Neurofibromatosis type II is an autosomal dominant disorder causing numerous intracranial and intraspinal tumors and acoustic neuromata. Ocular features include combined hamartoma of the retina and retinal pigment epithelium, epiretinal membranes, Lisch nodules (a diagnostic sign), and cataracts that develop in the second or third decade of life. The cataracts are posterior subcapsular, or cortical.

Zellweger syndrome, also known as hepatocerebrorenal syndrome, is an autosomal recessive disorder, characterized by renal cysts, hepatosplenomegaly, and neurological abnormalities. Ocular features include corneal clouding, retinal degeneration, and cataracts.

Norrie's disease is an X-linked recessive disorder that causes leukokoria, congenital infantile blindness, and is associated with mental retardation and cochlear deafness. In the eye, vitreoretinal dysplasia, retinal detachment, vitreous hemorrhage, and formation of a white retrolental mass occur. Eventually, a cataract forms.

Ocular Disease and Cataracts -

Inflammatory uveitis (e.g., Fuchs' heterochromic cyclitis and juvenile idiopathic arthritis) usually results in posterior subcapsular or posterior cortical lens opacities. Infective uveitis (e.g., ocular herpes zoster and toxoplasmosis, syphilis, and

tuberculosis) can cause cataracts, but the organism does not penetrate the lens. However, in maternal rubella infection, after 6 weeks of gestation, the virus can penetrate the lens capsule causing unilateral or bilateral lens opacities at birth or they may develop several weeks or months later. The opacity is nuclear and has a dense, pearly appearance. Corticosteroid treatment can also cause cataracts. Retinal pigment degenerations such as retinitis pigmentosa, Usher's syndrome, and gyrate atrophy are associated with cataracts, which are usually posterior subcapsular opacities and may be caused by toxic retinal breakdown products or from the deficiency of a product necessary for normal retinal and lens metabolism. Retinal detachment and retinal surgery may cause a posterior subcapsular cataract particularly in association with silicone oil injection and tamponade or an anterior subcapsular form may develop because of metaplasia of the lens epithelium after vitreoretinal surgery. Degenerative myopia is associated with posterior cortical, subcapsular, and nuclear cataracts. Ciliary body tumors may be associated with cortical or lamellar cataract in the affected quadrant. Anterior segment ischemia may cause a subcapsular or nuclear cataract, which progresses rapidly.

Toxic Causes –

Topical, inhaled, and systemically administered steroids can cause posterior subcapsular cataracts. The mechanisms are poorly understood but direct and indirect mechanisms are involved. Direct interaction of steroids with enzymes may affect their function, e.g., steroid modulation of Na^+,K^+-ATPase may cause sodium-potassium pump inhibition affecting osmotic regulation. Steroids may induce crystallin conformational changes causing aggregation and may affect intracellular Ca^{2+} homeostasis causing protein bonding. Indirectly, steroids affect DNA/RNA synthesis

of proteins and enzymes causing metabolic changes, and may also affect ciliary body growth hormone levels responsible for lens cellular differentiation causing posterior subcapsular opacities.[29]

Chronic use of long-acting anticholinesterases previously used in the treatment of chronic open-angle glaucoma may cause anterior subcapsular vacuoles and posterior subcapsular and nuclear cataracts. Pilocarpine, a shorter acting agent, causes less marked changes. The mechanism of action is unknown. Phenothiazines, such as chlorpromazine, may cause deposition of fine, yellow-brown granules under the anterior capsule in the pupillary zone and may develop into large stellate opacities but are not usually visually significant. The development of the opacities may be related to the cumulative dose of the medication, and photosensitization of the lens may play a role. Allopurinol used in the treatment of gout is also associated with cataracts.[37]Psoralen-UV-A therapy for psoriasis and vitilligo has been shown to cause cataracts in very high doses in animal studies, but is rare in humans; concomitant UV exposure may be a risk factor. Antimitotic drugs, such as busulfan, used in the treatment of chronic myeloid leukemia, may cause posterior subcapsular cataract. The antimalarial chloroquine (but not hydroxychloroquine), which is also used in the treatment of arthritis, may cause white, flake-like posterior subcapsular lens opacities. Amiodarone is used to treat cardiac arrhythmias and causes insignificant anterior subcapsular opacities and corneal deposits.[38]

Siderosis, which follows retention of a foreign body, causes iron deposits in the lens epithelium and iris, and results in a brown discoloration of the iris and a flower-shaped cataract may occur. Wilson's disease, an autosomal recessive disorder of copper metabolism, causes a brown ring of copper deposition in Descemet's

membrane and the lens capsule, resulting in a sunflower cataract - an anterior and posterior capsular disc-shaped polychromatic opacity in the pupillary zone with petal-like spokes that is not usually visually disabling.[33] Hypocalcemia in hypoparathyroidism is associated with cataracts. In children, the cataract is lamellar; in adults it produces an anterior or posterior punctate subcapsular opacity.

Congenital and Juvenile Cataracts -

Congenital cataracts are noted at birth, infantile cataracts occur in the first year, and juvenile cataracts develop during the first 12 years of life. Hereditary cataracts may be associated with other systemic syndromes, such as dystrophia myotonica. About one third of all congenital cataracts are hereditary and unassociated with any other metabolic or systemic disorders.

Trisomy 21, or Down's syndrome, is the most common autosomal trisomy, with an incidence of 1 per 800 births. Systemic features include mental retardation, stunted growth, mongoloid facies, and congenital heart defects. Ocular features include visually disabling lens opacities in 15% of cases, narrow and slanted palpebral fissures, blepharitis, strabismus, nystagmus, light-colored and spotted irides (Brushfield spots), keratoconus, and myopia.[40] Cataract is also associated with trisomy 13 (Patau's syndrome), trisomy 18 (Edwards' syndrome), Cri du chat syndrome (deletion of short arm of chromosome 5), and Turner's syndrome (X chromosome deletion).

A total cataract is a complete opacity present at birth. It may be hereditary (autosomal dominant or recessive) or associated with systemic disorders such as galactosemia, rubella, and Lowe's syndrome. Infantile cataracts cause amblyopia if unilateral and

may cause strabismus and nystagmus if bilateral. The incidence is about 0.4% of newborns, but the majority of cases are not associated with poor vision. Amblyopia depends on the size, location, and density of the cataract. The causes of infantile cataracts are many and include maternal infections (such as rubella), systemic diseases, hereditary disorders, and ocular disease.

Visual effects of cataracts -

The effect of cataract on vision varies according to the degree of the cataract and the cataract morphology.

Visual Acuity -

Visual acuity has been the standard tool by which to measure the visual effect of cataracts. However, visual acuity can remain good despite other lens opacity related effects on vision, which compromise the patient's ability to function. Therefore, visual acuity should not be assessed in isolation when considering the functional effect of a cataract in a particular patient.

Contrast Sensitivity, Glare, and Wavefront Aberrometry –

Patients who have a cataract-related reduction in contrast sensitivity notice difficulty with detailed visual tasks at low ambient light levels because of loss of contrast at higher spatial frequencies. Contrast sensitivity measurements based on linear sine-wave gratings have resulted in improved understanding and quantifying of visual quality and function. Contrast sensitivity measures the total visual system quality in terms of contrast. Wavefront aberrometry measures the optical quality in terms of spatial distortion. Both measurements are useful to understand the effects of cataracts

on vision.[41]Contrast sensitivity data can be processed by digital imaging software to demonstrate the quality of a patient's vision. Glare, which occurs as a result of forward scatter of light, may be produced by opacities that do not lie within the pupil diameter and therefore also affects visual function.[42]

Other effects-

The natural aging of the human lens produces a progressive hyperopic shift. Nuclear changes induce a modification of the refractive index of the lens and produce a myopic shift, which improves uncorrected near vision. Cortical opacities may cause localized changes in the refractive index of the lens, which may result in monocular diplopia or even polyopia. As the lens nucleus becomes more yellow, it absorbs blue light. The slow change is not apparent to the patient until after cataract surgery. The morphology, density, and location of lens opacities may cause changes in the visual field. These changes may be progressive and may obscure the disc; therefore, diagnosis and monitoring of glaucoma may be compromised.

Investigations-

Preoperative evaluation -Apart from a general medical examination, a patient due to undergo cataract surgery requires a detailed and pertinent ophthalmic examination.

I. **Visual acuity**- Although most patients will have visual acuity assessed using a Snellen chart; this is probably rather a crude approach as only high contrast vision is tested- usually in a darkened room. Contrast sensitivity charts and/or the use of LogMAR visual acuity measurement can give a better understanding of the quality of vision. Brightness acuity testing. When a chart is viewed through a glare source, also adds practical information about the patient's visual disability

2. **Cover test**- A heterotropia may indicate amblyopia,which carries a guarded visual prognosis, or the possibility of diplopia if vision is improved.

3. **Pupillary responses**- Because a cataract never produces an afferent pupillary defect. Its presence implies additional pathology, which may influence the final visual outcome.

4. Ocular adnexa. Dacryocystitis, blepharitis, chronic conjunctivitis. lagophthalmos, ectropion, entropion and tear film abnormalities may predispose to endophthalmitis and require effective preoperative treatment.

5**. Cornea-** A wide arcus senilis or stromal opacities may prejudice a good surgical view. Eyes with guttata and decreased endothelial cell counts are vulnerable to postoperative decompensation secondary to operativetrauma. In these cases special precautions should be taken to protect the endothelium during surgery with viscoelastics. Eyes with significant endothelial cell loss may be cadidates for simultaneous penetrating keratoplasty. The amount of pre-existing corneal astigmatism should be measured .A decision as to whether astigmatism should be corrected can be made after discussion with the patient.

6. **Anterior segment**-A shallow anterior chamber can render cataract surgery difficult. Pseudoexfoliation indicates a weak zonule, with the possibility of problems during surgery. A poorly dilating pupil can make cataract surgery difficult. Recognition of this allows intensive preoperative mydriatic drops, planned stretching of the pupil prior to capsulorrhexis or intracarneral injection of mydriatic. A poor red reflex compromises the performance of a good capsulorrhexis. This can be overcome by staining the capsule with a dye such as trypan blue.

7. **Lens**- The type of cataract is relevant. uclear cataracts tend to be harder and may require more phaco power, while cortical opacities tend to be softer. The colour of the nucleus depends on the age of the cataract and varies from transparent - grey - grey-

yellow - amber - brown - black. The latter occupies most of the lens and is the hardest.

8. **Fundus examination** pathology such as age-related macular degeneration may affect the visual outcome. **Ultrasonography** may be required in eyes with very dense cataracts that preclude fundoscopy.

Biometry-

Surgical removal of the crystalline lens subtracts approximately 200 from the refracting system of the eye. The aphakic eye is grossly hypermetropic; modern cataractsurgery therefore involves the implantation of an intraocular lens (10L), ideally in the same location as the surgically removed crystalline lens. Biometry affords calculation of the lens power likely to result in the desired postoperative refractive error. In its simplest form, biometry involves two parameters: (a) keratometry - the curvature of the anterior corneal surface (steepest and flattest meridians), expressed in dioptres or mm of radius of curvature and (b) axial length - the anteroposterior dimension of the eye in millimetres. There are two main methods used to measure axial length.

1**. A-scan ultrasonic biometry**- in which an ultrasonic wave is passed through the eye and the time of its return after hitting intraocular structures produces a trace. Biometry can be either by direct contact or more accurately, using a water bath. The display allows ocular structures to be identified and the distance from thefront of the cornea to the retina determined. The sound beam must be aligned with the visual axis for maximal echo reflection. Each echo shows up as a spike on the oscilloscope screen. A certain amount of skill is required to obtain accurate measurements

2**. Zeiss IOL Master** - is a non-contact method that utilizes two coaxial laser beams which are partially coherent and produce an interference pattern (partial coherence

inferometry). Measurements have high reproducibility and generally require less skill than ultrasonic biometry. The IOLMaster is a complete biometry system which also performs keratometry, anterior chamber depth, corneal white to white and comes with formulae for calculating IOL power. Storage of data and validation of the A-constant is another useful feature. Aphakic. pseudophakic and silicone filled eyes can also be measured but eyes with dense posterior subcapsular opacities present a problem.

3. **IOL power calculation formulae-** Numerous formulae incorporating additional parameters such as anterior chamber depth and individualized surgeon factors. Have been developed to optimize the accuracy of preoperative prediction.

4. **Personalized A-constant-** is the process of fine-tuning 10L calculation. It is done by back calculating the Aconstant using at least 20 cases of average eyes. Most modern biometry machines have this facility already Programmed into them. If a constant error of say plus 1D is found in most cases, a new personalized A- constant can be used to ensure a better outcome.

Treatment – surgical

HISTORY OF CATARACT SURGERY

Daviel (1696-1762), a Normandy-born French oculist, started a revolution of surgical innovation (which continues to the present time) by describing a new, planned method for extraction of the cataract from the eye. In 1753, he published details of this innovative surgery [43].

1. Daviel faced his seated patient and made his incision at the lower limbus with a

keratome. (Recall that surgery during this time was done without anesthesia, and the patient's defensive Bell's phenomenon would afford the surgeon only this inferior approach.)

2. The incision was extended with scissors right and left above the level of the pupil.
3. The lens capsule was incised with a sharp needle.
4. The lens contents were loosened by a spatula.
5. The cataract (nucleus) was expressed by gentle pressure.
6. Lens material (cortex) was removed by curette.
7. The cornea was replaced (no sutures were used) and the eye patched.

Thus, Daviel described a planned extracapsular extraction. The surgery did not immediately catch hold. Oculists continued to couch. Still, the new concept was not lost, and others started to write about improvements on Daviel's approach. Between 1753 and 1862, three milestones took place that profoundly affected the direction of cataract surgery:

1. Pierre–Francois–Benezet Pamard of Avignon shifted the surgical incision to the upper part of the eye. He had the patient lie on his or her back and operated from the head of the table.
2. Carl Himly, a German oculist, improved the surgeon's view by introducing pharmacologic mydriasis.
3. Albert Mooren of Düsseldorf added a preliminary iridectomy to combat the complication of pupillary block.

THE INTRACAPSULAR CATARACT EXTRACTION -

Slowly, between 1760 and 1860, surgeons recognized that Daviel's operation had inherent dangers; vitreous loss was high and inflammation with bound-down pupils common place. Skilled surgeons, including Sharp, who had mastered Daviel's technique, began to think that the eye would have a better chance if the whole lens were to be removed *in toto* from the eye.[44,45]

Many surgeons contributed to the shift to intracapsular surgery. Samuel Sharp (1753) described surgery that introduced the subject of taking the entire lens out of the eye with the capsule intact. Albrecht von Graefe (1867) devised his long, thin, sword-like corneal knife to facilitate the corneal incision. Christiaen (1845) wrote on breaking the zonules with a curved blunt probe passed into the anterior chamber (AC). Von Graefe (1867) and A. Terson (1871) removed the cataract *in toto* with a spoon introduced behind the lens. G Reuling (1879) used a loop instead of a spoon.[46] In 1867; Boston's Henry W Williams introduced suture closure of the cataract wound. This significant advancement was further endorsed by Suarez de Mendosa (1891), Eugene Kalt (1894), and Frederick Verhoeff (1916).

Finally, the time was ripe for another monumental step forward. In 1895, Colonel Henry Smith advocated performing the intracapsular extraction without internal AC manipulation. He utilized external pressure with a muscle hook on the peripheral inferior cornea to loosen zonules. With sufficient mechanical zonulolysis, he then expressed the lens using pressure from the muscle hook creeping superiorly over the cornea. Because the zonules were still attached at the

12-o'clock position, they would act as a hinge causing the lens to tumble. The tumbled lens would thus present its inferior pole to the wound first. Further expression would fially expel the lens from the eye. Later, Smith would describe a modification of his mechanical zonulolysis that would allow for an actual linear sliding of the whole lens without tumbling.[47]

Thus was born the famed Smith–Indian linear sliding maneuver. (The 'Indian' comes from the fact that Smith's surgery was performed during military duty while in India. He learned the technique from Lieutenant Colonel Mulroney in 1894. Smith picked up on the procedure, popularized it, and published in 1926.)

The search for a safer intracapsular extraction continued unabated. The next idea devised to remove the lens, traction, did improve its safety. A forceps was introduced to grasp the inferior pole of the lens. Gentle traction coupled with side-to-side movements enabled E Kalt, G Stanculeann, and Arnold Knapp (1910) to lyse inferior zonules.[48] With a continuous grasp on the capsule, a Smith muscle hook was used to help the tumbling lens.

Verhoeff at the Massachusetts Eye and Ear Infirmary was not satisfied with the tumbling maneuver. He designed the Verhoeff capsular forceps (1916) with open-ended half rings at the tips. Through a superior-sector iridectomy, Verhoeff grasped the tilted-up 12-o'clock pole of the lens and gently slid out the cataract. The forceps was designed in such a way that its tips were gentle on the capsule, thus reducing risk of capsular rupture. Because the lens was actually pulled out of the eye without tumbling and with less external pressure, vitreous loss was less of a threat.

Another ingenious idea was proposed by P Stoewer (1902). He used a suction device attached to the lens for lifting it out of the eye.[49] Ignacio Barraquer performed phacoerysis with a pneumatic forceps. His son, Jose Barraquer, developed an electric vacuum pump machine with a special erysiphake handle for suction removal of the cataract.[50.] American surgeons invested in the machine but found it bulky and awkward; they still preferred the forceps delivery with the Verhoeff forceps, the Kalt smooth forceps, or the Arruga forceps.

The next breakthrough came to intracapsular surgery with the development of chemical zonulolysis. Mechanical zonular destruction was first used by Christiaen (1845) and Luca (1866). The latter used a curved probe to press down on the limbus. Jose Barraquer (1958) demonstrated the dramatic efficacy of chemical zonulolysis using an enzyme α-chymotrypsin.[51] Surgeons fially had a new, simple method to loosen the lens using absolutely no distorting pressure.

The final significant improvement arrived when T Krawawicz in Poland (1961) introduced the cryoextractor.[52] A small, cold probe could be frozen to the surface of the lens forming an ice ball, fusing the lens capsule, cortex, and nucleus, lessening thus the risk of capsule rupture during extraction. With gentle teasing, the lens could be delivered without any pushing on the eye whatsoever.Intra capsular surgeons now had a simplified extraction method and round pupil delivery became the standard versus the old sector iridectomy.The intra capsular cataract extraction (ICCE) was in its heyday in the early 1970s , but its demise was rapidly approaching .

THE RETURN TO ECCE

THE POSTMODERN ERA-

The intracapsular cataract extraction (ICCE) was in its heyday in the early 1970s, but its demise was rapidly approaching. Despite the encouraging results, there remained a substantial rate of potentially blinding complications, including aphakic retinal detachment and cystoid macular edema, which could be reduced by keeping the posterior capsule intact.[53]Moreover; smaller incisional cataract wounds were sought.

But the major concern was the optical rehabilitation of the aphakic patient with glasses. Intraocular lens (IOL) development from the 1940s through the 1970s enhanced aphakic rehabilitation during this period. Harold Ridley performed his first artificial lens implant at St Thomas' Hospital in London on Nov 29, 1949, [54] but the approach was not correct for the popular ICCE movement. The ICCE left nothing to support Ridley's posterior chamber lens. The intact zonular–capsular diaphragm was the safe scaffolding Ridley was looking for, and after trying only two posterior chamber lenses in patients after ICCE; he put all the rest in ECCE patients.[55]

In 1960, the tide began to change. Harold Scheie described a procedure for aspirating a soft congenital cataract from the eye through small incisions.[56] He performed aspiration through a single needle and irrigation with AC maintenance through a separate needle. With this technique for removing the lens contents, the dangers of secondary cataract, glaucoma, and uveitis were significantly reduced

for children or young adults with ‘soft’ cataracts.

Between 1965 and 1972, Cornelius Binkhorst of Holland was modifying the IOL concept. He believed that an intact posterior capsule would provide better anatomic support for the IOL. He first set out to refine the extracapsular cataract extraction (ECCE) so that all cataracts, hard and soft, could be removed. He devised the technique of removing a window of anterior capsule with toothed forceps, by aspirating a soft nucleus or by expressing a hard nucleus, and by irrigating and aspirating a portion of the remaining cortex through a bent olive-tip cannula. The Binkhorst IOL had two or four loops (haptics) that protruded through the pupil and rested on the posterior capsule. The optic was positioned in front of the iris, sitting atop the loop pedestal. The miotic pupil held the pedestal central until the loops stuck or scarred down to the posterior capsule.

Another famous European, Jan Worst, from the Refaja clinic, also in Holland, was also converting to ECCE at about the same time that Binkhorst was doing his work. Worst devised the ‘Worst Medallion’ IOL. His IOL required suture fixation to the iris as well as two loops protruding through the pupil to rest on the posterior capsule. By 1977, Worst and colleagues reported on a large series of 2000 cases using this new lens.[57]

While Binkhorst and Worst were working and teaching in Europe, Kelman was inventing his phacoemulsification instrument. Kelman impressed an incredulous ophthalmic community by publishing his landmark description of the ultrasonic breakup of the nucleus coupled with the Scheie concept of irrigation–aspiration of the cortex in 1967.

Kelman was the invited guest of national medical meetings and showed films of his revolutionary work. He conducted courses at his local New York hospital and published an instructional text.[58] Increasing numbers of surgeons gathered to observe and learn the Kelman phacoemulsification technique (KPE). The transition was not easy. Few surgeons actually stuck to the technique because it was technically difficult and dangerous (high risk of corneal damage, capsular rupture, vitreous loss, and nucleus dislocation into the vitreous). The procedure also failed to catch on because a primary capsulotomy was taught, thus negating the advantage of compartmentalizing the anterior and posterior segments. The KPE's final flaw was that an IOL had yet to be developed that safely took advantage of the intact posterior capsule and that could be implanted through a small incision.

While Kelman was developing his emulsification, John Shock (1972) introduced the alternative phacofragmentation and irrigation system.[59] The concept was simple in that a small irrigation needle powered by a standard ultrasound machine was used to break up the nucleus and cortex material, and the irrigation stream was used to wash out the lens remnants without reliance on more sophisticated aspiration equipment. Shock's phacofragmentation developed slowly, and 2 years later he had performed only 47 procedures. His results were best on soft cataracts (under age 40 years). He also introduced an adjunctive method of rapid freezing and thawing (‘phacocryolysis’) of the nucleus in senile cataracts.[60] Shock's technique was not widely adopted; the KPE proved to be more efficacious and popular.

Intracapsular surgeons were not convinced of the need for change until the

modern posterior chamber IOL was developed by S P Shearing (the Shearing lens with J loops; 1977). Richard Kratz published the results of his first cases of KPE with the Shearing posterior chamber IOL in 1978. Surgeons around the world were impressed when Kratz proclaimed that the 'J-loop IOLs tend to stabilize intraocular contents postoperatively, thus reducing' iritis, cystoid macular edema, vitritis, and retinal detachment.[61]

Norman Jaffe of Miami, Florida was also planting the seeds of ICCE discontent. In 1979, he sealed the fate of the pupillary-supported IOL by reporting that 'after ~800 Copeland implants, I was not satisfied. I was disturbed by' many complications.[62] Later, Jaffe and co-workers made a major contribution by pointing out that the extracapsular procedure carried a lower incidence of complications. Norman Jaffe, Henry Clayman, and Marc Jaffe showed in a prospective study that angiography-proven cystoid macular edema was lower in uncomplicated ECCEs than in uncomplicated ICCEs.[63]

A stronger, more convincing article came out by the same authors in 1984; this showed in a retrospective consecutive series that the incidence of retinal detachment in the myopic eye was much lower with an intact posterior capsule after an ECCE than after the ICCE.[64] The figures were dramatic; in high myopes, the rate of retinal detachment was 0.66% in ECCEs versus 5.74% in the ICCEs (follow-up of 1-4 years). No one could ignore this significant revelation. The national conversion to ECCE accelerated.

Pioneers in the ECCE movement were many. William Simcoe (1977) introduced his Simcoe curved 23-gauge cannula connected to a small irrigating bulb.[65] Later;

Simcoe devised a closed-chamber vacuum stripping system utilizing a suction syringe in one hand connected to an aspirating cannula in the other hand. For nucleus delivery, he popularized the Simcoe lens loop; a curved loop with serrations to ‘grasp’ the undersurface of the nucleus for extraction from the capsular bag.[66] Simcoe was a master at simplifying the ECCE in that he required few instruments, no assistants, and no machines.

McIntyre was another pioneer. In 1976, he introduced the simple low-flow double-bore irrigation–aspiration cannula connected directly to a small aspirating syringe.[67]

James Gills was also leading the way in Florida by perform-ing high-volume surgery and perfecting the Gills method (with help from Robert Welch). The Gills method was a simple manual technique of nucleus expression followed by cortical cleanup with an end-opening Gills 25-gauge cannula attached to a 3-mm syringe. His cortical cleanup in a semiclosed chamber utilized the concept of engaging the cortex in the cannula port and then wiggling and teasing the cortex free from its capsular adherence. Repeated segments of cortex were teased out of the eye by cycles of insertion of the Gills needle with irrigation fluid, then suction and teasing of the cortex, and fially deliverance of the needle with the cortex out of the eye. The 12-o'clock cortex was exposed for cleanup by using a forceps to reflect back the anterior capsular edge. The method was simple and effective, but automated systems gradually became more popular. Automated irrigation/aspiration could be performed better with a closed, more controlled AC and with fewer AC entries and exits.

In 1980, a new adjunct was introduced to ophthalmic surgery that would greatly aid surgeons in their conversion to ECCE and that would contribute to safer and more highly successful surgery. That new product was the AC -maintaining viscoelastic gel, hyaluronic acid (Healon).

Balazs in 1972 isolated and purified a hyaluronic acid gel for vitreous replacement.[68] But it was not until 1980 that two Boston surgeons, David Miller and Robert Stegman, demonstrated that the same viscoelastic product, Healon, could be used advantageously in the AC. It was described as useful to stabilize the AC depth, to protect the endothelial cells, and to facilitate the insertion of the IOL.[69] Surgeons tried this new viscoelastic material and discovered that it made their surgery easier and safer. By the mid-1980s, it was in almost universal use.

RESURGENCE OF PHACOEMULSIFICATION -

Kelman introduced his phacoemulsifier in 1967, [70] but the potential for complications concerned many intracapsular surgeons. Seeing the nucleus manipulated into the AC and then blasted apart by the sonification tip was enough to dissuade many surgeons from straying from their refied and safe whole lens removal or ECCE nucleus expression.

There were a few interested surgeons, however, who did like the Kelman concept. In the early 1970s, Sinskey employed a one-handed technique to bowl out the central nucleus, followed by collapsing down and aspiration of the peripheral nuclear shell.[71] Little was the first to advocate a two-handed technique to gain better control of the nucleus.[72] He inserted a second instrument through the phacoemulsification incision. Kratz improved the concept of two-handed

phacoemulsification by changing the position of the second instrument paracentesis site to the 2- or 3-o'clock limbal position. This simple change made nondominant hand work more comfortable and effective. Kratz unveiled a series in 1979 that made surgeons stand up and take notice.[73] He described his phaco technique, which included bowling out of the nucleus, two-handed tipping up of the 12-o'clock nuclear edge, and phacoemulsification of the bowl periphery. He did elegant work in the era of 'can-opener' capsulotomy and no viscoelastic gel. Surgeons went to observe and learn from Kratz, and he became at that time the surgeon to operate on fellow ophthalmologists. A prized pupil, Maloney, traveled the country teaching the Kratz tilting technique. The method gradually took on a new name – 'the Maloney three-step' technique.[74] In retrospect, this method was not as easy as it sounded. The learning curve was difficult, and surgeons were not happy with their frequent ruptured capsules, dropped nuclei, and damaged endothelium.

Emery and Little published a classic text that described with graphic detail their version of the Kelman–Cavitron phacoemulsifier-aspiration system.[75] As a follow-up to this landmark text, Emery and McIntyre published a text of extracapsular surgery and phacoemulsification.[79] The phacoemulsification section was well illustrated and detailed.

SMALL INCISION CATARACT SURGERY -

Smaller phacoemulsification probes and foldable implants finally allowed surgeons to realize their next quest for astigmatically neutral surgical wounds. Early in 1982, Kraff and Sanders proved that smaller incisions were better than

large, producing less early-induced astigmatism and less late-healing astigmatic shift.[76] Colvard and co-workers in 1980 advocated a scleral-to-AC single plane entry and use of the Terry keratometer to reduce astigmatism.[77] Fenzl used a scleral 'flap' for phacoemulsification and then enlarged it to 7 mm for the IOL.[78]Girard was the first to name and describe the true scleral tunnel.[79] This tunnel started through half-thickness sclera and entered the AC central to the scleral spur; it was 5 mm long and 7 mm wide and was used to implant an IOL after pars plana lensectomy.

Surgeons seized on the scleral tunnel concept. It was a neat operation with incision entry far from the cornea. It was generally agreed that a linear incision line or a 'frown' configuration would result in more stable healing and less slippage. Singer advocated the frown incision [80] and Pallin patented the 'chevron' incision.[81]

With better constructed tunnels, attention turned to devising the proper closure. Shepherd contributed a large breakthrough with the astigmatically neutral horizontal suture.[82] Others were quick to offer modifications; Fine with the 'infiity suture', [83] Masket with the 'horizontal anchor suture', [84] and Fishkind with the 'horizontal overlap suture'.[85] These were all mattress-like sutures which closed the incision without inducing radial traction forces.

In 1990, McFarland shocked surgeons' conservative nature by demonstrating that a properly created scleral tunnel would make a corneal valve effect that sealed any egress of AC fluid.[86] Operators were just getting used to one-stitch surgery and now were being thrust into sutureless surgery.

The next advance to phacoemulsification was the revolutionary concept of moving the incision to clear cornea. Fine in February 1992 described a new concept of a planar temporal clear-corneal sutureless incision, [87] which was a self-sealing incision positioned farthest away from the corneal center on the temporal meridian. Fine claimed this incision was easier, quicker, and astigmatically neutral. Others recognized the elegant simplicity of the clear-corneal incision and ventured their modifications. In March 1992, Kellan described a 'scleral less' clear-corneal incision under a short conjunctival flap.[88] Ernest supported this concept because he found that corneal healing was faster (7 days compared with 1 month) when the fibroblastic healing response was allowed to start at the conjunctival limbus. Williamson and Langerman[89] introduced hinged clear-corneal incisions to improve the corneal seal as an answer to critics who said the risk of retrograde bacterial flow into the AC was too great.

During the evolution into phacoemulsification, there were some surgeons who for various reasons decided not to follow. They graduated into the small incision era under the leadership of Peter Kansas. Kansas devised a manual phaco section technique that encompassed prolapsing the nucleus into the AC and then sectioning the endonucleus with a Kansas trisecter in front and a 'cutter board' vectis behind. The three pieces of nucleus were manually removed through a 5- to 6-mm incision. Kansas introduced this technique to the 1986 ASCRS meeting, and some faithful followers continue to use the method today.

BASIC CONCEPTS AND PRICIPLES

BASIC STEPS OF MANUAL SMALL INCISION CATARACT SURGERY-

The steps are:

1) Birdle suture and conjunctival section.

2) Sclerocorneal tunnel.

3) Anterior capsulotomy : Can opener or continuous curvilinear capsulorhexis

4) Hydrodissection and hydrodelineation

5) Prolapse of nucleus into anterior chamber

6) Epinucleus and cortex aspiration

7) Implantation of IOL in the bag.

8) Wound closure.

1) **Birdle suture-** Placed to maneuver the globe and to fix the same during various surgical procedures .For the usual superior tunnel the suture is placed beneath the tendon of superior rectus muscle. For temporal approach lateral rectus is used.

2) **Conjunctival flap-** Small fornix based flap is preferredAmm in width and 8 mm in length is sufficient .Tenons capsule is cut and adequate cautery is applied.

3) **Scleral tunnel construction-** Consists of the following steps

a) **External scleral incision**: A one- third to half- thickness external scleral groove is made 2.5 to 3mm from the surgical limbus. It may be linear or frown shaped. Length of the incision is dependent on nuclear density. Usually an external scleral groove of 6- 6.5mm length is made.

b) **Sclerocorneal tunnelling**: The actual tunnelling is done with a crescent up blade. Uniform thickness and extended upto 1-1.5 mm into the clear cornea. A side port entry is made with a lance tip at 10 0 clock position. It is done parallel to the iris and 2mm wide. Viscoelastic is injected through this. Other uses are aspiration of cortical matter, reformation of anterior chamber at end of surgery and also capsulorhexis through the sideport entry.

c) **Internal corneal incision**- Done with a 3mm angled keratome. Anterior chamber is entered with the keratome and the internal wound is visualised as a straight line. The incision is extended in the same plane using an enlarger. Instead of a superior tunnel, a temporal approach can also be used.

4) **Capsulotomy-** Either can opener or continuous curvilinear capsulorhexis or envelope technique. Can opener is done by using a sharp cystitome or bent 26 G needle. Multiple small tears are preferred to avoid capsular tags .l0 - 20 punctures are made in each quadrant circumferential to the equator to avoid damage to the zonular insertion. Continous curvilinear capsulrhexis has various advantages. It ensures a permanent verifiable and secure fixation of "in the bag" IOL implantation. The absence of anterior capsular tags ensures a safe and easy aspiration of peripheral cortical matter. Minimal trauma to the zonules is exerted. In case of posterior capsular rupture, it still provides a good support for the sulcus fixation of intraocular lens implant. Presence of visible anterior capsular rim helps in carrying out polishing of the anterior subcapsular epithelial cells and thus preventing posterior capsular opacification later on. In case of Morganian hypermature cataract the envelope technique is used.

5) **Hydroprocedure and prolapse of nucleus into the A.C** - Depends on the

type of capsulotomy done. In capsulorhexis hydrodisscetion and hydrodelineation is required. It is a 2 step procedure .One is to inject fluid and the second is to press downwards while injecting with the cannula. This combination of mechanical and hydrostatic forces causes the opposite pole of the nucleus to pop out of the capsulorhexis margin. The rest of the nucleus is dialled out. With can opener technique there is no need for hydro procedures. Nucleus is mechanically prolapsed into the AC with the help of a Sinskey hook.

6) **Nucleus extraction**- Either by one of the following methods.

-Irrigating vectis method

-Phacosandwich method

-Phacofracture method.

-Blumenthal method.

7) **Cortex aspiration**- A thorough wash with a Simcoes cannula decreases the incidence of postoperative iritis, after cataract formation and cystoid macular oedema.

8) **IOL implantation**- As the size of the wound is close to 6mm it is preferable to place 6.0mm optic rigid PMMA IOLS. Proper use of viscoelastic and tilting of IOL during insertion enables in the bag lodgment.

8) **Wound closure-** The primary function of a three plane incision is to provide water tight self sealed valve effect. After IOL implantation the lips of the wound are tested by applying pressure with a sponge against the posterior lip of the wound to make it incisional leak. In the absence of leak the conjunctival flap is unfolded back over the incision.

COMPLICATIONS OF MSICS

INTRA OPERATIVE-

A)**Wound construction**-

Incision length- Small incision can cause difficulty in nucleus delivery leading to endothelial damage and iris damage. Long incision can cause poor approximation leading to wound leak and against the rule astigmatism (ATR).

Incision placement- Anterior incision can cause poor self sealing effect leading to wound leak and ATR. Posterior incision results in wide tunnel, increases bleeding and increases risk of premature entry and hence leading to difficult nucleus delivery and instrument manipulation.

Incision depth: Ideally half to one third of scleral depth. Button holing can occur if superficial dissection of scleral flap. Premature entry may occur. Scleral disinsertion may occur if a very deep groove is made.

B) **Descemet's membrane (DM) stripping on entry** -

Any penetrating incision through the cornea and any object being introduced through a corneal incision may cause a DM stripping. Complete stripping may occur when fluid or viscoelastic is injected through the paracentesis and cannula tip not placed into the AC but rather into the corneal canal thus causing hydro dissection of the cornea.

C) **Paracentesis**-

The additional self sealing stab incision could have complications if it is placed too far central into the cornea causing DM stripping. If made too far peripheral into the sclera could cause bleeding. If it is too small for instruments it could cause DM stripping. If too large it could cause leakage. If it is not done parallel to the plane of iris it could cause injury to the lens and iris.

D) **Capsulotomy** - A small capsulorhexis can cause difficulty in prolapsing the nucleus and cortical aspiration predisposing to PC tear, zonular dialysis and residual cortex. A large capsulorhexis can cause problems for 'in the bag placement' of IOL.

E) **Hydro dissection** - Ideally it is done in 4 quadrants. If hydrodissection is done forcefully in one quadrant it will cause undue pressure in the posterior capsule leading to posterior capsular rupture and chances of nucleus drop.

F) **Nucleus prolapse**- Important points to be noted before nucleus prolapse are corneal status, pupillary size, cataract density, integrity of zonules and size of tunnel .Any failure to pay attention to these will result in endothelial damage, iridodialysis, damage to iris, zonular dialysis or posterior capsule tear.

G) **Nucleus delivery**- Inadequate tunnel size can lead to endothelial damage, iris sandwich when 6 0' clock iris gets trapped between the vectis inferiorly and nucleus superiorly.

H) **Hyphema**- Due to injury to iris or wound.

I) **Iris injury**: Direct injury causing sphincter tears and iridodialysis and Iris prolapse.

J) **Intraoperative miosis**- This could cause difficulty in nucleus delivery.

K) **Zonular Dialysis**- This could be encountered during nucleus delivery when the while rotating the nucleus to the anterior chamber excessive pull is employed. It could also be as a result of the type of cataract like hypermature cataract or conditions like pseudoexfoliation.

L) **Posterior capsular tear**- This depends on the type of cataract like hypermature cataract. In case the cataract is a posterior polar cataract and hydrodissection is done along with a hydrodelineation.

M) **Residual cortex**: If improper or inadequate wash is given. This could lead to uveitis.

N) **Dropped nucleus**- This is the most dreaded intra ocular complication. A trained vitreo retinal surgeon is the best to handle this.

O) **Posterior vitreous pressure**- Possible causes are speculum or instrument exerting pressure on the globe, excessive pull of the birdIe suture, poor facial block, valsalva manoeuvre (coughing, straining) specially in short necked people, obese patients, large volume anesthetic agents used in ciliary block.

P) **Expulsive hemorrhage**- It is a rare but may occur. Manifests as tissue prolapsed through the wound, loss of red reflex and hard globe.

POST OPERATIVE-

Immediate complications-

Wound leak- Due to excessive episcleral cautery, tearing or button holing of roof of tunnel, premature entry or shallow entry, Nuclear / cortical fragment in tunnel, incorrect suture placement.

Late complications-

Corneal complications -like corneal edema, striate keratopathy, Bullous keratopathy, DM stripping (missed at time of surgery), recurrent erosions.

Uveitis- due to surgical manipulations

Post operative rise of IOP- Due to retained viscoelastics which gets locked up between the posterior surface of IOL and the posterior capsule.

Retinal detachment- Very rare these days.

Posterior dislocations of IOL- Due to unrecognised posterior capsule tear.

Vitreous hemorrhage.

Vitritis

Anterior ischemic optic neuropathy.

Cystoid macular edema (CME) - After posterior capsular rupture the anterior chamber pressure is the major determinant for the amount of vitreous that would prolapse. The stretch forces of vitreous prolapse may result in CME.

Posterior capsular opacification (PCO) - Capsular opacification is of 3 forms. i.e. fibrosis, elschnig pearls and capsular wrinkling.

Post surgical astigmatism- Long incision can cause poor approximation leading to wound leak and against the rule astigmatism (ATR).Anterior incision can cause poor self sealing effect leading to wound leak and ATR. The long term effects of cautery may give rise to- unacceptable levels of post-operative astigmatism by two mechanisms: a) Heat induced scleral shrinkage and b) Closure of capillaries & small vessels which affect the wound healing process. Hence cautery should be used sparingly if at all during cataract surgery. Lens tilt has been suggested as a cause of post surgical astigmatism. Significant tilting is required to induce clinically significant astigmatism. A 20D IOL must be inclined 10 degrees from the vertical plane to cause I D of cylinder. Another cause for IOL induced astigmatism is anterior chamber IOLs. Over sized rigid AC IOLs have been suggested as a cause of postoperative cylinder, owing to the stretching of the corneoscleral ring.

QUALITY OF LIFE

Quality of life related to health has two aspects which involve different problems for validation[90, 91] One is the objective part, which is the functional status of 'the individual and the other is the subjective feeling of health and welfare [92]. It includes:

Physical functionality*:* the possibility of carrying out daily routines involving hygiene and personal care, walking" etc. The physical side of quality of life also refers to disease symptoms and the treatment thereof.

Psychological functionality*:* the mental and emotional welfare, satisfaction and happiness.

Social functionality*:* social relations, participation in activities. Perception of health, pain and above all satisfaction with life. In recent years there has been a trend involving the assessment of visual function vis-a-vis quality of life related to eyesight. This increased interest has led to the develop men surveys –which attempt to measure said concepts. If it were possible to make a quantitative measurement of the effect of cataracts on quality of life, we would have an objective parameter to assess its development, the indication of surgery and subsequent improvements. The goal of cataracts surgery is to improve visual acuity and therefore the visual function, considering that it entails improvements in quality of life. Cataract surgery is done to get good quality of life (related to health and eyesight) after cataract surgery [93]. Our work aims at a comprehensive attention to patients, searching not only technical improvements but also an enhanced quality of their lives. MSICS with IOL implant is an effective and safe method for improving quality of life, mainly in the physical dimension. Reduced role limitations due to physical health problems after surgery matches the physical improvement, thus enhancing the functionality of patients in their routine activities. The improvements in social relationships confirms the importance of measuring quality of life aspects in the results of any medical intervention[94]

MATERIALS AND METHODS

TITLE OF THE STUDY:

"Prospective Two Years Study of evaluation of visual acuity and quality of life after cataract surgery in kolar".

SOURCE OF DATA: 500 patients of cataract from RLJH opd and kolar opd, are selected at R.L.J. HOSPITAL AND RESEARCH CENTRE, TAMAKA, KOLAR attached to SRI DEVARAJ URS MEDICAL COLLEGE from November 2010 to December 2012 after taking informed consent.

INCLUSION CRITERIA:

Patients with Senile Cataracts- Immature, mature and hyper mature stages.

EXCLUSION CRITERIA:

1. Those associated with raised intra ocular pressure or glaucoma
2. Evidence of previous inflammation or trauma
3. Other types of acquired cataract like metabolic, radiational, complicated etc.
4. Patients having Diabetes Mellitus, Hypertension or Age Related Macular Degeneration.
5. Patients having senile cataract with Pseudoexfoliation syndrome.

The indication for cataract surgery in present scenario is defective vision enough to interfere with daily activities.

METHOD OF COLLECTION OF DATA -A prospective study was conducted on five hundred patients fulfilling the criteria. All the patients underwent similar protocol for standard cataract evaluation, which consists of -

Pre-operative evaluation:

All the patients were admitted into the hospital the day prior to surgery. All these patients underwent, the following pre-operative evaluation, and complete eye examination, including a full history of any, previous ocular disease or surgery examination both direct and indirect, intraocular pressure recording by Visual acuity recording by Snellens or E or Kannada charts, fundus examination, Schiotz tonometry and detailed slit-lamp examination.

Keratometry by Bausch and Lomb Keratometer, A Scan biometry, (using DGH Jed med instrument) and IOL power calculation using the SRK formula. General examination including CVS and RS examination, BP recording, urine analysis and blood sugar evaluation were done:

.Written informed consent was taken from all the patients.

. Xylocaine test dose.

Pre operative preparation:

The night before the day of surgery and three hours before surgery one drop of ciprofloxacin eye drops hourly was instilled in the eye. The pupil was dilated with cyclopentolate 1%, tropicamide 0.5% and fluribrofen 0.03%, used 3 times in one hour for two hours before surgery.

Procedure

All the surgeries were performed under local anesthesia (peri bulbar block.) using 4ml mixture of 2% xylocaine with adrenaline , hyaluronidase and 4ml 0.75% bupivacaine. A good massage was given to the eye ball for ten minutes to achieve

adequate hypotony. Just before the start of the surgery, the skin around the eye was painted with 5% povidone iodine and the same drops was installed topically.

MSICS:

After peribulbar block the patients were shifted on to the operating table. Under aseptic precautions the patients were draped. Under the operating microscope the surgeries were performed. Initially wire speculum was placed to secure the lids. Superior rectus bridle suture was applied. Fornix based conjunctival flap was made. Bleeding vessels were cauterized with wet cautery. Blade no. 15 was used to make a straight 6mm incision was made 2mm behind the limbus. Scleral tunnel was done with a crescent blade. Side port entries were made. Viscoelastics was injected to form the AC. A keratome blade 2.8 mm was used to enter AC. Viscoelastics was injected again. With a bent insulin needle capsulorrhexis was done.Hydrodissection was perfomed. An enlarger was used to enlarge the scleral tunnel. The nucleus was prolapsed into the AC. Viscoelastics was injected under the nucleus and above the nucleus. An irrigation vectis was introduced underneath the nucleus and the nucleus delivered. Cortical wash was given. Viscoelastics injected. PCIOL placed in the capsular bag. Patient was given a wash to remove the excess viscoe1astics and the side port entries were hydrated. Patient was given subconjunctival injection of gentamicin and deaxamethasone.

Post-operative evaluation

On the first post-operative day, all the patients were submitted to detailed slit-lamp'

examination and fundus examination. Visual acuity unaided and with pinhole vision will be recorded in each patient postoperatively on day one, one week, one month and third month . Quality of life is accessed by asking standard questionnaire. Quality of life is improved or remains the same or worsened after cataract surgery is judged by asking standard questionnaire and the score they obtained.

- Quality of life has improved – high score after surgery
- Quality of life remained the same –same score before and after surgery
- Quality of life has worsened- worst score after cataract surgery

Descriptive statistical methods like mean and proportion will be used for evaluation. Comparison between proportions will be done through Chi square method and 'Z'

OBSERVATION AND RESULTS

AGE DISTRIBUTION

A total of 500 patients were included in the study. The mean age of the patients was 63.

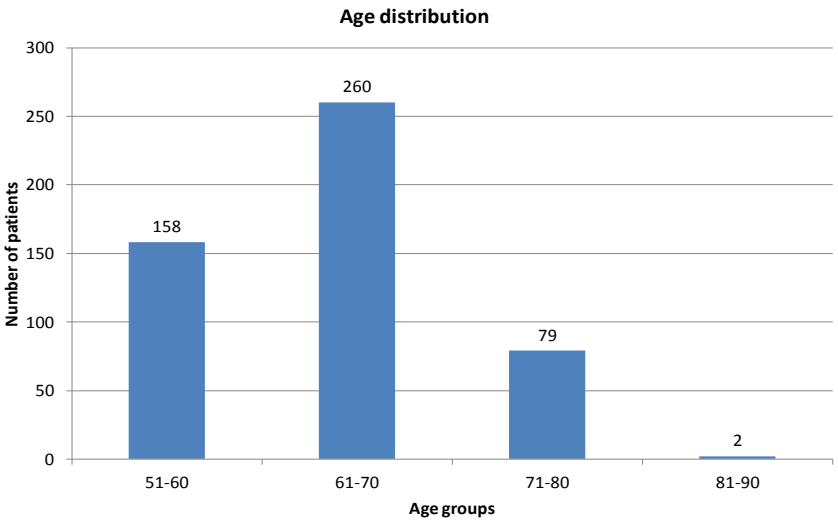
TABLE 1-

Age in years	Number of patients	%
51-60	158	31.6
61-70	260	52
71-80	79	15.8
81-90	2	0.4

Mean ± SD: 63.06±8.99

The above table shows that in our study there were a total of 500 patients .Out of these 500 patients 2 were aged 81-90, 79 were 71-80,260 were aged 61-70 and 158 was aged between 51-60.

FIGURE 1-



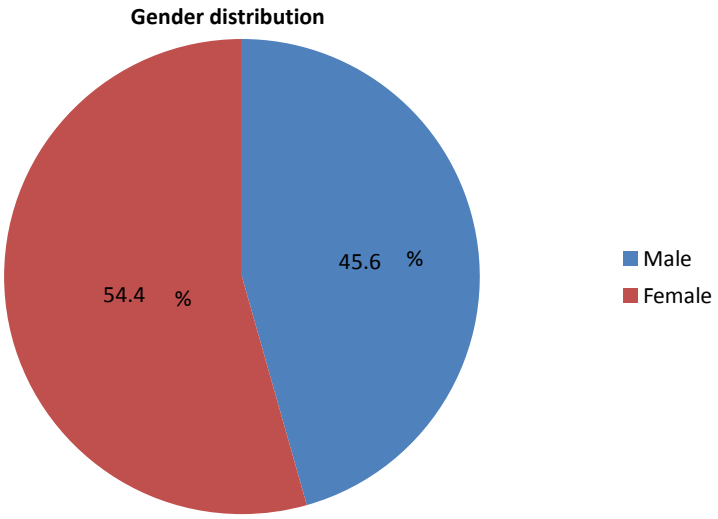
GENDER DISTRIBUTION

TABLE- 2

Gender	Number of patients	%
Male	228	45.6
Female	272	54.4
Total	500	100.0

The table here shows that in our study males were 45.6% and females were 54.4%.

FIGURE -2



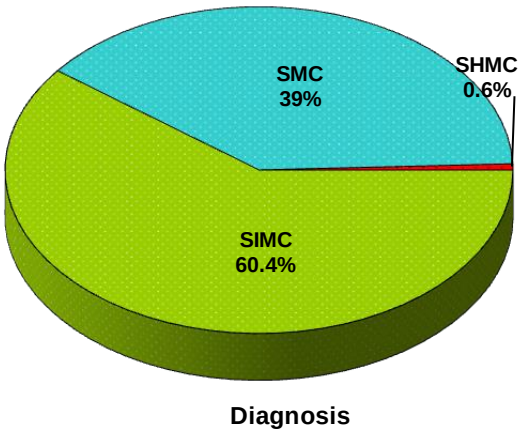
DISTRIBUTION OF DIAGNOSIS OF PATIENTS STUDIED

TABLE-3

Diagnosis	Number of patients (n=500)	%
SIMC	302	60.4
SMC	195	39.0
SHMC	3	0.6

The table and chart here shows that that in our study ,out of 500 patients 60.4% were having Senile immature cataract , 39% were having Senile mature cataract and 0.6% were having Senile hyper mature cataract.

FIGURE - 3



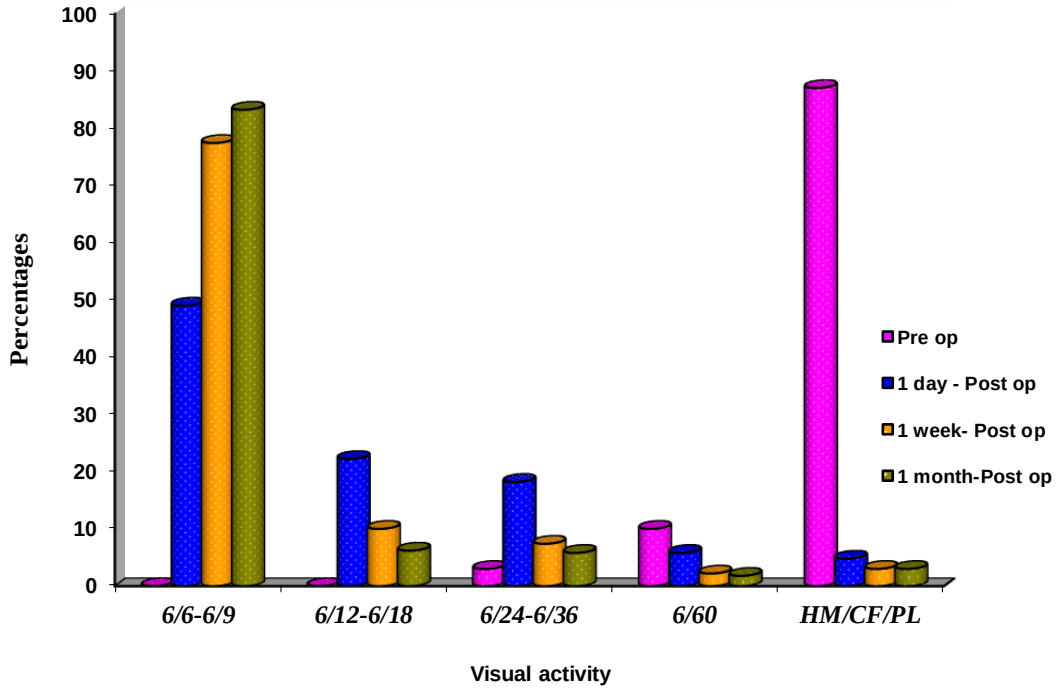
COMPARISON OF VISUAL ACTIVITY OF PATIENTS STUDIED

TABLE -4

Visual activity	Pre op (n=500)	Post op 1 day (n=500)	Post op 1 week (n=500)	Post op 1 month (n=500)	% change
6/6-6/9	0	245(49%)	387(77.4%)	416(83.2%)	+83.2%
6/12-6/18	0	111(22.2%)	50(10%)	31(6.2%)	+6.2%
6/24-6/36	15(3%)	91(18.2%)	37(7.4%)	29(5.8%)	+5.8%
6/60	50(10%)	29(5.8%)	11(2.2%)	9(1.8%)	+1.8%
HM/CF/PL	435(87%)	24(4.8%)	15(3%)	15(3%)	-97.0%

The above chart shows that maximum number patients having vision of hand movement, counting finger and perception of light preoperatively. Post operatively the percentage of patients with BCVA of 6/6 to 6/9 on day one is 49%, on first week is 77.4% and on one month is 83.2%.

FIGURE -4



POST OPERATIVE COMPLICATIONS

TABLE -5 **EARLY COMPLICATIONS**

Early Complications	Number and Percentage
Iritis	60 (12%)
Straite keratopathy	40 (8%) Mild-25(5%), Severe-15(3%)
Hyphaema	30(6%)
Decentered IOL	5(1.0%)

The above chart shows 37% of patients were having some early complication and in that iritis was most commonly present (12%).

FIGURE-5

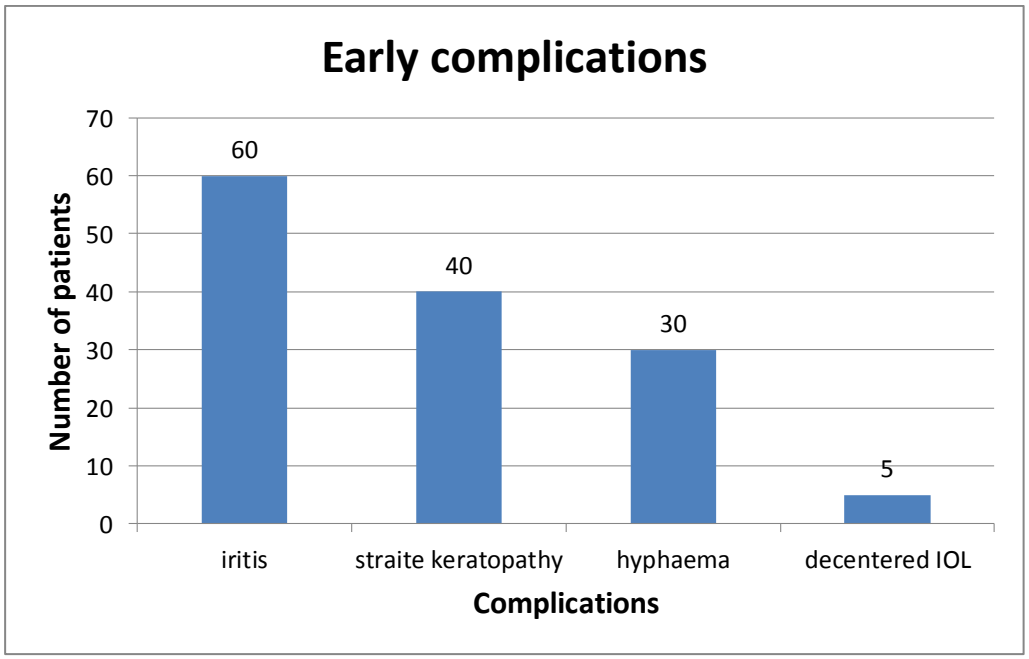
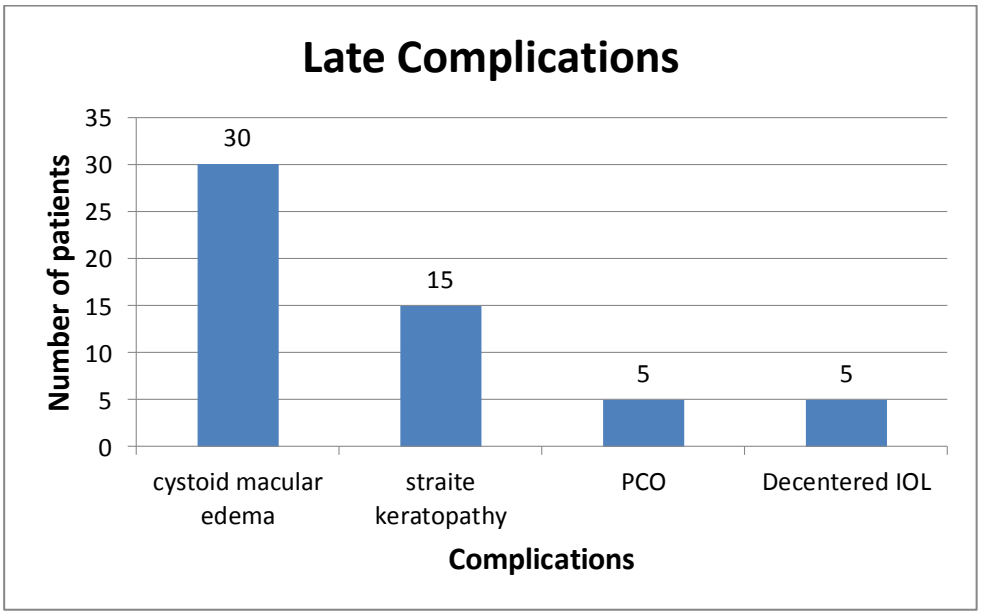


TABLE - 6 **LATE COMPLICATIONS**

Late complications	Number and Percentage
Cystoid macular edema	30 (6%)
Straite keratopathy	15 (3%)
PCO	5 (1%)
Decentered IOL	5 (1%)

The above charts show that 11 % of patients were having some complications which were causing less vision, even after six weeks post operatively. In that most common complication encountered was CME which was present in 6 % of patients.

FIGURE -6



DISTRIBUTION OF GENERAL FUNCTIONING,VISUAL FUNCTIONNING AND SOCIAL FUNCTIONNING OF PATIENTS STUDIED

TABLE-7

General Functioning	Pre op (n=500)	Post op (n=500)	% change
Q.1			
• 1.cannot see	33(6.6%)	24(4.8%)	-1.8%
• 2.can see slight a bit	437(87.4%)	22(4.4%)	-83.0%
• 3.can see with moderate difficulty,	30(6%)	7(1.4%)	-4.6%
• 4.can see with some difficulty	-	72(14.4%)	+14.4%
• 5.can see	-	375(75%)	+75.0%
Q.2			
• 1.cannot see	84(16.8%)	21(4.2%)	-12.6%
• 2.can see slight a bit	390(78%)	21(4.2%)	-73.8%
• 3.can see with moderate difficulty,	26(5.2%)	14(2.8%)	-2.4%
• 4.can see with some difficulty	-	116(23.2%)	+23.2%
• 5.can see	-	328(65.6%)	+65.6%
Q.3			
• 1.cannot see	83(16.6%)	20(4%)	-12.6%
• 2.can see slight a bit	367(73.4%)	26(5.2%)	-68.2%
• 3.can see with moderate difficulty,	50(10%)	10(2%)	-8.0%
• 4.can see with some difficulty	-	113(22.6%)	+22.6%
• 5.can see	-	331(66.2%)	+66.2%
Q.4			
• 1.cannot see	176(35.2%)	20(4%)	-31.2%
• 2.can see slight a bit	290(58%)	10(2%)	-56.0%
• 3.can see with moderate difficulty,	34(6.8%)	32(6.4%)	-0.4%
• 4.can see with some difficulty	-	142(28.4%)	+28.4%
• 5.can see	-	296(59.2%)	+59.2%
Q.5			
• 1.cannot see	105(21%)	13(2.6%)	-18.4%
• 2.can see slight a bit	355(71%)	34(6.8%)	-64.2%
• 3.can see with moderate difficulty,	40(8%)	10(2%)	-6.0%
• 4.can see with some difficulty	-	122(24.4%)	+24.4%
• 5.can see	-	321(64.2%)	+64.2%
Q.6			
• 1.cannot see	204(40.8%)	35(7%)	-33.8%
• 2.can see slight a bit	267(53.4%)	9(1.8%)	-51.6%
• 3.can see with moderate difficulty,	29(5.8%)	13(2.6%)	-3.2%
• 4.can see with some difficulty	-	129(25.8%)	+25.8%
• 5.can see	-	314(62.8%)	+62.8%
Q.7			
• 1.cannot see	151(30.2%)	18(3.6%)	-26.6%

• 2.can see slight a bit	316(63.2%)	26(5.2%)	-58.0%
• 3.can see with moderate difficulty,	33(6.6%)	19(3.8%)	-2.8%
• 4.can see with some difficulty	-	137(27.4%)	+27.4%
• 5.can see	-	300(60%)	+60.0%
Q.8			
• 1.cannot see	147(29.4%)	18(3.6%)	-25.8%
• 2.can see slight a bit	325(65%)	28(5.6%)	-59.4%
• 3.can see with moderate difficulty,	28(5.6%)	17(3.4%)	-2.2%
• 4.can see with some difficulty	-	125(25%)	+25.0%
• 5.can see	-	312(62.4%)	+62.4%
Q.9			
• 1.cannot	76(15.2%)	23(4.6%)	-10.6%
• 2.with severe difficulty	375(75%)	8(1.6%)	-73.4%
• 3.with moderate difficulty,	49(9.8%)	28(5.6%)	-4.2%
• 4.with some difficulty	-	107(21.4%)	+21.4%
• 5.can	-	334(66.8%)	+66.8%
Q.10			
• 1.always	81(16.2%)	14(2.8%)	-13.4%
• 2.most of the time	376(75.2%)	35(7%)	-68.2%
• 3.some of the time	43(8.6%)	11(2.2%)	-6.4%
• 4.a little of time	-	77(15.4%)	+15.4%
• 5.never	-	363(72.6%)	+72.6%
Q.11			
• 1.always	88(17.6%)	14(2.8%)	-14.8%
• 2.most of the time	372(74.4%)	33(6.6%)	-67.8%
• 3.some of the time	40(8%)	17(3.4%)	-4.6%
• 4.a little of time	-	93(18.6%)	+18.6%
• 5.never	-	343(68.6%)	+68.6%
Q.12			
• 1.always	127(25.4%)	14(2.8%)	-22.6%
• 2.most of the time	336(67.2%)	33(6.6%)	-60.6%
• 3.some of the time	37(7.4%)	17(3.4%)	-4.0%
• 4.a little of time	-	115(23%)	+23.0%
• 5.never	-	321(64.2%)	+64.2%

Social	**Pre op (n=500)**	**Post op (n=500)**	**% change**
Q.13			
• 1.always	93(18.6%)	32(6.4%)	-12.2%
• 2.most of the time	362(72.4%)	12(2.4%)	-70.0%
• 3.some of the time	45(9%)	21(4.2%)	-4.8%
• 4.a little of time	-	81(16.2%)	+16.2%
• 5.never	-	354(70.8%)	+70.8%
Q.14			
• 1.always	99(19.8%)	13(2.6%)	-17.2%
• 2.most of the time	356(71.2%)	31(6.2%)	-65.0%

• 3.some of the time	45(9%)	29(5.8%)	-3.2%
• 4.a little of time	-	81(16.2%)	+16.2%
• 5.never	-	346(69.2%)	+69.2%
Q.15			
• 1.always	96(19.2%)	11(2.2%)	-17.0%
• 2.most of the time	360(72%)	33(6.6%)	-65.4%
• 3.some of the time	44(8.8%)	18(3.6%)	-5.2%
• 4.a little of time	-	95(19%)	+19.0%
• 5.never	-	343(68.6%)	+68.6%
Visual	**Pre op(n=500)**	**Post op(n=500)**	**% change**
Q.16			
• 1.very severe sensitivity	75(15%)	12(2.4%)	-12.6%
• 2.severe sensitivity	365(73%)	25(5%)	-68.0%
• 3.moderate sensitivity	60(12%)	22(4.4%)	-7.6%
• 4.mild sensitivity	-	109(21.8%)	+21.8%
• 5.no sensitivity	-	332(66.4%)	+66.4%
Q.17			
• 1.always	74(14.8%)	18(3.6%)	-11.2%
• 2.slightly	380(76%)	28(5.6%)	-70.4%
• 3.moderate	46(9.2%)	12(2.4%)	-6.8%
• 4.quite a bit	-	98(19.6%)	+19.6%
• 5.never,	-	344(68.8%)	+68.8%
Q.18			
• 1.poor	122(24.4%)	28(5.6%)	-18.8%
• 2.slightly fair	334(66.8%)	18(3.6%)	-63.2%
• 3.fair	44(8.8%)	11(2.2%)	-6.6%
• 4.good,	-	106(21.2%)	+21.2%
• 5.very good	-	337(67.4%)	+67.4%
Q.19			
• 1.very severe pain	-	-	-
• 2.severepain	-	-	-
• 3.moderate pain	-	-	-
• 4.slight pain	-	-	-
• 5.nopain,	500(100%)	500(100%)	+100%
Q. 20			
• 1.very severe redness	-	-	-
• 2.severe redness	-	-	-
• 3.moderate redness	-	-	-
• 4. Mild redness	-	-	-
• 5. no redness/ discharge	500(100%)	500(100%)	+100%

The above table shows the response of patients to the quality of life questionnaire given to them pre operatively and post operatively. Here it shows significant

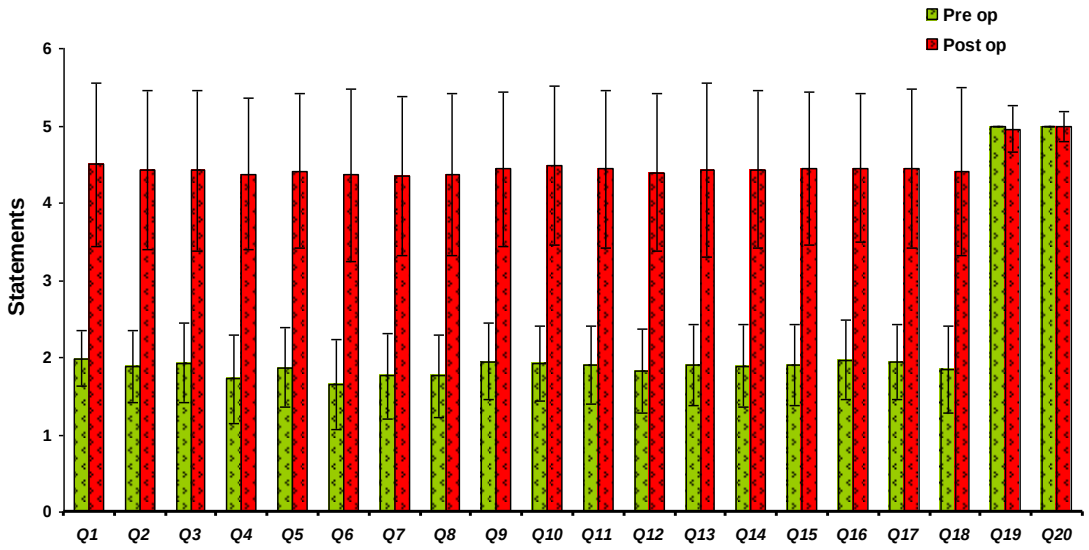
improvement in scores post operatively as compared to pre operative scores. Maximum patients opted option 4 or 5 post operatively which shows improvement in score hence improvement in quality of life.

COMPARISON OF PRE & POST OPEERATIVE DISTRIBUTION OF SCORES IN PATIENTS STUDIED

TABLE -8

Statements	Pre op (n=500)	Post op (n=500)	Significance
Q1	1.99±0.36	4.50±1.06	t=50.558;p<0.001**
Q.2	1.88±0.46	4.42±1.03	t=50.572;p<0.001**
Q.3	1.93±0.51	4.42±1.04	t=46.406;p<0.001**
Q.4	1.72±0.58	4.37±0.98	t=52.550;p<0.001**
Q.5	1.87±0.52	4.41±1.00	t=49.945;p<0.001**
Q.6	1.65±0.59	4.36±1.11	t=48.528;p<0.001**
Q.7	1.76±0.56	4.35±1.02	t=48.777;p<0.001**
Q.8	1.76±0.54	4.37±1.04	t=49.126;p<0.001**
Q.9	1.95±0.50	4.44±1.00	t=49.091;p<0.001**
Q.10	1.92±0.49	4.48±1.03	t=48.484;p<0.001**
Q.11	1.90±0.50	4.44±1.02	t=48.626;p<0.001**
Q.12	1.82±0.54	4.39±1.02	t=50.068;p<0.001**
Q.13	1.90±0.52	4.43±1.12	t=46.348;p<0.001**
Q.14	1.89±0.53	4.43±1.02	t=49.008;p<0.001**
Q.15	1.90±0.52	4.45±0.99	t=50.970;p<0.001**
Q.16	1.97±0.52	4.45±0.96	t=52.270;p<0.001**
Q.17	1.94±0.49	4.44±1.03	t=48.424;p<0.001**
Q.18	1.84±0.56	4.41±1.09	t=45.675;p<0.001**
Q.19	5.00±0.00	5.00±0.00	-
Q.20	5.00±0.00	5.00±0.00	-

FIGURE -7



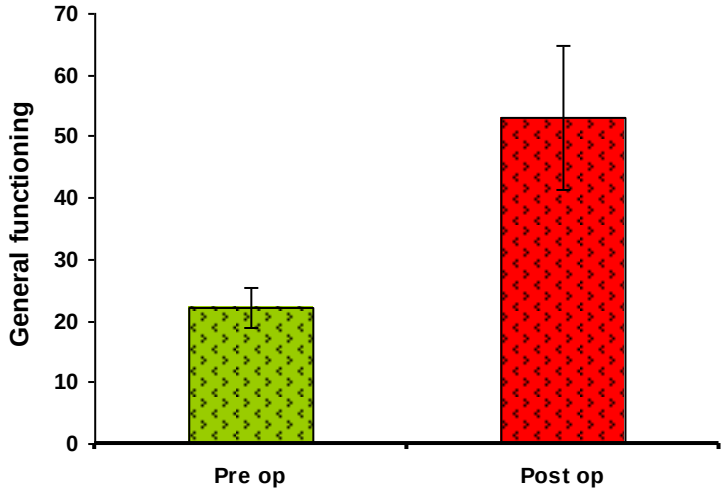
The above table and chart shows significant improvement in quality of life ($p<0.001$) as the scores are increased post operatively.

COMPARISON OF PRE & POST OPERATIVE GENERAL, SOCIAL AND VISUAL FUNCTIONING TOTAL SCORES

TABLE- 9

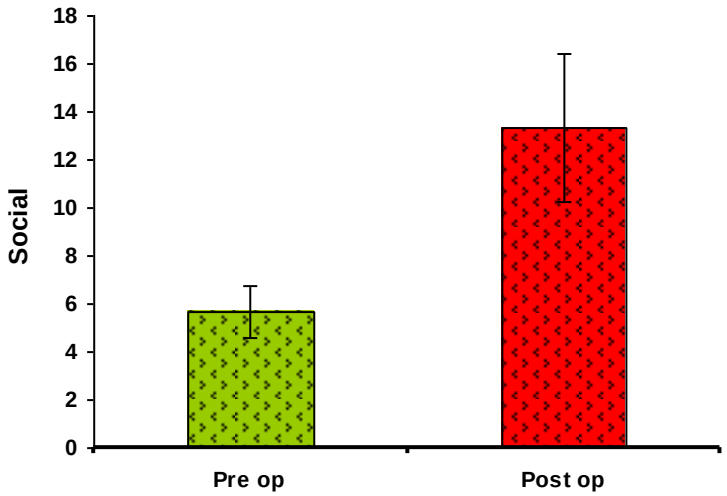
Variables	Pre op score	Post op score	Significance
General functioning	22.2±3.25	53.05±11.59	t=56.482;p<0.001
Social functioning	5.68±1.07	13.35±3.07	t=53.054;p<0.001
Visual functioning	15.76±1.05	23.23±3.18	t=49.637;p<0.001
Total	43.65±4.52	89.22±17.2	t=56.511;p<0.001

FIGURE – 8 COMPARISON OF PRE & POST OPERATIVE GENERAL FUNCTIONING SCORES



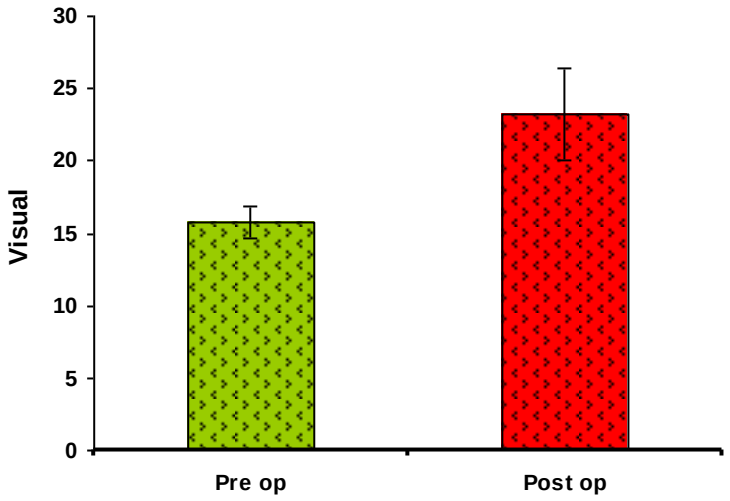
This chart shows significant improvement in general functioning post operatively as compared to pre operative scores as p value is < 0.001.

FIGURE- 9 COMPARISON OF PRE & POST OPERATIVE SOCIAL FUNCTIONING SCORES



This chart shows significant improvement in social functioning post operatively as compared to pre operative scores as p value is < 0.001.

FIGURE-10 COMPARISON OF PRE & POST OPERATIVE VISUAL FUNCTIONING SCORES



This chart shows significant improvement in visual functioning post operatively as compared to pre operative scores as p value is < 0.001.

DISCUSSION

In our study of 500 patients included, 228 were males and 272 were females. The mean age of the patient was 63 years. In that 158 were aged between 51-60, 260 were aged between 61-70, 79 were aged between 71-80 and 2 were aged between 81-90 years. Out of total, 302 patients were having senile immature cataract, 195 were having senile mature cataract and 3 patients were having senile hyper mature cataract.

All patients were examined pre operatively as well as post operatively. Visual acuity was recorded on 1st day, 1st week and 6th week post operatively. All the complications were recorded post operatively.

In this study 89.4% number of patients had a visual acuity of 6/18 or better. This correlates well with other studies.

In the study done by **Dandona L[95] et al**, 1390 patients underwent cataract surgery at Mudhol center and 954 patients underwent cataract surgery at Toodukurthy center. At Mudhol center 1213(87.30%) of patients have a visual acuity of 6/18 or better and at Toodukurthy center 798(83.6%) of patients have a visual acuity of 6/18 or better.

In the study done by **Bachani D[96] et al**, 2369 patients underwent cataract surgery and 71.40% of patients have a visual acuity of 6/18 or better.

In the study done by **Khan M T[18] et al**, 134 patients underwent cataract surgery and 86.80% of patients having a visual acuity of 6/18 or better.

FINAL BCVA COMPARISION BETWEEN STUDIES

Sl.No	Study	No .of patients operated	Percentage of patients having BCVA of 6/18 or better
1.	Our study	500	89.4%
2.	Dandona L	1390 (Mudhol center) 954 (Toodukurthy center)	87.3% (Mudhol center) 83.6% (Toodukurthy center)
3.	Bachani D	2369	71.40%
4.	Khan M T	134	86.80%

Post operative complication comparative study

In our study complications encountered were iritis (12%), cystoid macular edema (6%), decentered IOL (11%), hyphaema (6%), PCO (1%) and straite keratopathy (8%).

In the study conducted by **Khan M T[18] et al**, the complications encountered were hyphaema (11.3%), corneal edema/ straite keratopathy (12.7%), raised IOP (3.3%), CME (0.7%), endopthalmitis 2% of patients postoperatively.

In the study done be **Rangaraj[97] and co authors**, corneal straite are seen in 7% of patients post operatively.

In the study done by **Riley A F[98] et al**, CME is seen in 3.8% of patients and endopthalmitis in 0.2% of patients.

In the study done by **Desai P, Minassian D C[17] et al**, the complication encountered were raise IOP (7.9%),wound leak (1.2%), corneal edema(9.5%), hyphaema (1.1%),CME (0.05%), vitreous in ac in 0.3% of patients.

STUDIES

		Our	Khan M T	Rangaraj	Riley A F	Desai P
S.No	Complications					
1.	Iritis	12%	-	-	-	-
2.	CME	6%	-	-	3.8%	0.05%
3.	SK	8%	12.7%	7%	-	9.5%
3.	Hyphaema	6%	11.3%	-	-	1.1%
4.	Decentered IOL	1%	-	-	-	-
5.	PCO	1%	-	-	-	-

Post operative Quality of life comparison studies

A total of twenty questions were asked to all patients included in the study, pre operatively and post operatively. Comparing the self reported quality of life scores between unoperated and operated individuals provides assessment of the quality of life operations. In this study pre operative general functions were 22.2+/- 3.25, social functions were 5.68+/- 1.07 and visual functions were 15.76+/-1.05. Post operatively general function score were 53.05+/-11.59,social functions were 13.35+/-3.07 and visual function were 23.23+/-3.18. As post operative score was high shows significant improvement in quality of life post operatively ($p<0.001$). This correlates well with other studies.

In the study done by **Mercedes C L[94] et** al shows that the social function, general health, perception, role limitation due to physical problems and physical functions significantly improved between first and third consultations.

In the study done by **Rohatgi J[99] et al** shows quality of life improved significantly after cataract surgery. Visual improvements after cataract surgery paralleled improvement in quality of life and rehabilitation scores.

In the study done by **Owsley C[13] et al** on the impact of cataract surgery on health related quality of life in nursing home residence reported significant score improvement in the general vision ($p=0.005$), reading ($p=0.001$), psychological distress and social interaction of the nursing home vision targeted health related quality of life questionnaire.

In the study conducted by **Applegate W.B.[14] et al** showed that maximum improvement in vision and improvements in subjective measures of patient function

occurred by 4 months and most improvement were maintained at 1 year.

The Cataract Outcome Study conducted by **P.Desai [17]et al** in 1993 and the Cataract Impact Study conducted by **Polack S et al[100]** in 2005 reported that visual function scores and health related quality of life scores obtained after cataract surgery resulted in less vision dependent activities.

Applegate MD et al[16] showed that maximum improvement in vision occurred by 4 months and most improvements were maintained at one year.

CONCLUSION

Cataract has been consistently documented to be the most common cause of blindness in developing countries. Of the estimated 38-45 million blind in the world (vision <3/60 in the better eye, with available correction), nearly one fifth live in India alone.[1] Senile cataract is the leading cause of blindness worldwide

The aim of cataract surgery is to improve visual function with the assumption that this will also improve overall quality of life. The gains from cataract extraction are usually demonstrated clinically by the change in the Snellen's visual acuity in the eye that had surgery. The impact on visual function or quality of life has not usually been considered as a separate issue, partly as these have been assumed to follow the improvement of visual acuity.

Quality of life related to health involves different problems for validation. One is the objective part, which is the functional status of the individual, and the other is the subjective feeling of health and welfare.

Patients who underwent cataract surgery reported improvements in their vision-targeted health related quality of life , in addition to improvements in their vision They experienced improved general vision, fewer limitations because of vision , less difficulty with reading , less worry, frustration or upset over their vision, and greater ease and likelihood of engaging in social interactions.

SUMMARY

This is a prospective study conducted between Nov 2010 –Dec 2012 at R.L.Jalappa Hospital and Research Center. During the above mentioned period a total of 500 cases were included in the study. This study was done to evaluate visual acuity and quality of life after cataract surgery.

In our study, out of 500 patients 228 were males and 272 were females. The mean age of the patient was 63years. In this 153 were aged between 51-60 years, 260 were aged between 61-70 years, 79 were aged between 71-80 years and 2 were aged between 81-90 years.

Senile immature cataract was present in 302 patients, senile mature cataract present in 195 patients and senile hyper mature cataract was present in 3 patients,

All the patients were examined post operatively. Visual acuity was recorded on 1st day, 1st week and 1 month following surgery. All complications were recorded post operatively.

Visual acuity analysis on first day showed 49% of patients having visual acuity of 6/9 or better , on first week 77.40% of patients having visual acuity of 6/9 or better and after one month post operatively 83.2% of patients were having visual acuity of 6/9 or better.

Quality of life scoring showed significant improvement in quality of life post operatively.Pre operatively general functioning sore was 22.5+/-3.25 but post operatively it is increased to 53.05+/-11.59, visual functioning score pre operatively was 5.68+/-1.07 but post operatively it is 13.25+/-3.07 and visual functioning score pre operatively was 15.76 +/-1.05 and post operatively it is increased to 23.23 +/-3.18.

In our study complications reported post operatively were iritis (12%), cystoid macular edema (6%), decentered IOL (11%), hyphaema (6%), PCO (1%) and straite keratopathy (8%).

Visual acuity and quality of life were significantly improved in maximum number of patients post operatively.

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ANNEXURE I:

PROFORMA FOR EVALUATION OF VISUAL ACUITY AFTER CATARACT SURGERY.

CASE NUMBER:

NAME-

AGE-

SEX-

ADDRESS-

OCCUPATION-

I P NO-

DATE OF ADMISSION-

DATE OF SURGERY-

DIAGNOSIS-

TYPE OF SURGERY-

OCULAR EXAMINATION-

HEAD POSTURE-

	RE	LE
OCULAR POSTURE		
LIDS		
CONJUNCTIVA		
CORNEA		
ANTERIOR CHAMBER		
IRIS		
PUPIL		
LENS		
FUNDUS		

PRE OPERATIVE EVALUATION- RE/LE

KI-

AXIAL LENGTH-

K2-

INTRAOCULAR TENSION-

INTRAOCULAR LENS POWER-

REFRACTION -

PREOPERATIVE VISUAL ACUITY-

RE LE

DISTANT VISION-

WITH PIN HOLE-

SPECTACLES-

POST OPERATIVE VISUAL ACUITY- RE/LE

1st DAY	1st WEEK	1stMONTH
DISTANT- WITH PINHOLE-		

EXAMINATION FINDINGS - RE/LE

	1st DAY	1ST WEEK	1st MONTH
CLEAR CORNEA			
CORNEAL EDEMA			
STRIATE KERATOPATHY			
HYPHAEMA			
IRITIS			
IRIS PROLAPSE			
SHALLOW AC			
CORTICAL MATTER PRESENT			
DECENTERED IOL			
REFRACTIVE ERROR			
APHAKIA			
SECONDARY GLAUCOMA			

ENDOPTHALMITIS			
FUNDUS			

CORRECTION AFTER ONE MONTH- RE/LE

	SPHERICAL	CYLINDRICAL	AXIS
DV			
NV			

ANNEXURE II:

PROFORMA FOR EVALUTION OF QUALITY OF LIFE AFTER CATARACT SURGERY

Name -

S/O, W/O -

Occupation –

I.P. No. - Date:-

1. Do you experience difficulties seeing cars, traffic lights or road lights in the street?
   (3- can see with moderate difficulty, 2-can see slight a bit, 1- cannot see)
   (4-can see with some difficulty, 5-can see)

2. Do you experience difficulties in recognizing people's faces and gestures across the street? (3- can see with moderate difficulty, 2-can see slight a bit, 1- cannot see)
   (4-can see with some difficulty, 5-can see)

3. Do you experience difficulties in recognizing shops, banks and other places across the Street?
   (3- can see with moderate difficulty, 2-can see slight a bit, 1- can see,
   4-can see with some difficulty, 5-can see)

4. Do you experience difficulties in recognizing the numbers of destinations of approaching buses/vans or whether the taxi is vacant?
   (3- can see with moderate difficulty, 2-can see slight a bit, 1- cannot see)
   (4-can see with some difficulty, 5-can see)

5. Do you experience difficulties in walking or running because of visual impairment?
   (3- can see with moderate difficulty, 2-can see slight a bit, 1- can see,
   4-can see with some difficulty, 5-can see)

6. Do you experience difficulties in reading small prints in newspapers, books or magazines because of visual impairment?
   (3- can see with moderate difficulty, 2-can see slight a bit, 1- cannot see)
   (4-can see with some difficulty, 5-can see)

7. Do you experience difficulties in seeing price tags in markets, bank slips, water bills, food or medicine levels because of visual impairment?
   (3- can see with moderate difficulty, 2-can see slight a bit, 1- cannot see)
   (4-can see with some difficulty, 5-can see)

8. Do you experience difficulties in making telephone calls, taking medicines

or signing your name because of visual impairment?
(3- can see with moderate difficulty, 2-can see slight a bit, 1- cannot see)
(4-can see with some difficulty, 5-can see)

9. Do you experience difficulties in going down the stairs, curbs or slope because of visual impairment? (5- can, 4- with some difficulty, 3- with moderate difficulty, 2- with severe difficulty, 1- cannot)

10. Do you have experiences of bumping into objects and other people or falling down because of visual impairment?
(5- never, 4- a little of time, 3-some of the time, 2- most of the time, 1- always)

11. Is your ability to take care of yourself impaired because of visual impairment?
(5- never, 4- a little of time, 3-some of the time, 2- most of the time, 1- always)

12. Is your working capability affected by visual impairment?
(5- never, 4- a little of time, 3-some of the time, 2- most of the time, 1- always)

13. Are your usual entertainments affected by visual impairment?
(5- never, 4- a little of time, 3-some of the time, 2- most of the time, 1- always)

14. Do you avoid going out, visiting people or traveling because of visual impairment?
(5- never, 4- a little of time, 3-some of the time, 2- most of the time, 1- always)

15. Do you need assistance from others because of visual impairment?
(5- never, 4- a little of time, 3-some of the time, 2- most of the time, 1- always)

16. Do you experience sensitivity to light, seeing shadows around the light or multiple
images? (, 5- no sensitivity, 4- mild senstivity, 3- moderate sensitivity, 2-severe sensitivity, 1- very severe sensitivity)

17. Do you feel worried emotionally or frustrated by your visual impairment?
(5-never, 4-quite a bit, 3-moderate, 2-slightly, 1- always)

18. What is your objective overall visual function?
(5-very good, 4-good, 3-fair, 2-slightly fair, 1-poor)

19. Do you experience pain or discomfort in your eye?
(5-nopain, 4- slight pain, 3-moderate pain, 2-severepain, 1-very severe pain)

20. Do you experience redness or discharge from your eye?
    (5-very severe redness, 4-severe redness, 3-moderate redness, 2-mild redness,1-no redness/ discharge)

ANNEXURE III:

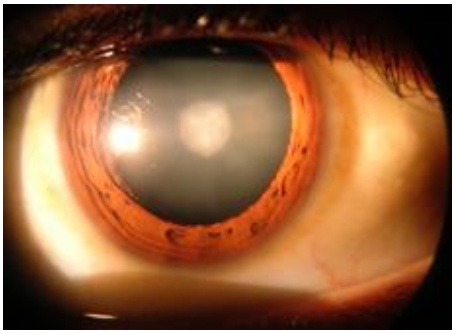

Figure 1- Senile immature cataract

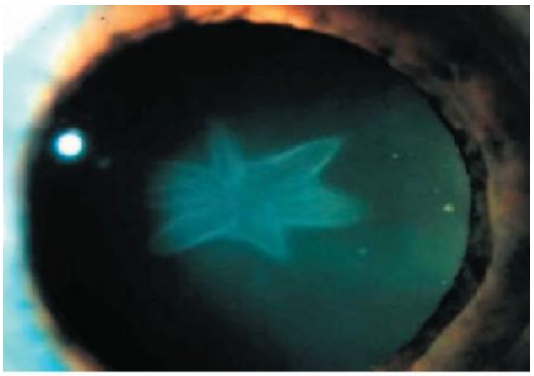

Figure 1: Traumatic cataract (note the flower shaped appearance)

Figure 2- Traumatic cataract

Figure 3- Post operative corneal edema

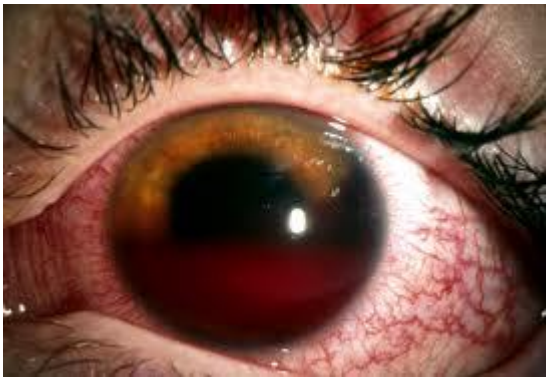

Figure 4 - Hyphaema

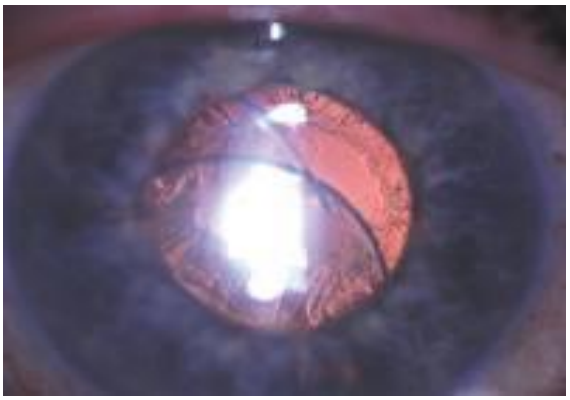

Figure 6 - Decentered IOL

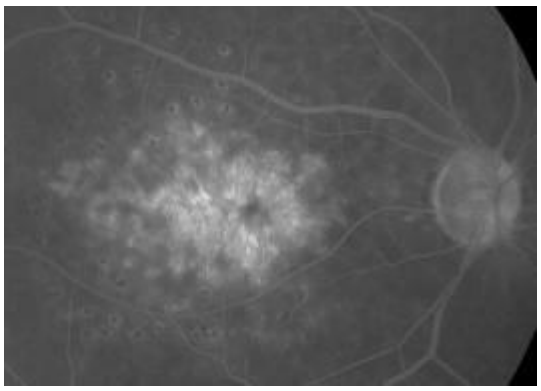

Figure 7- Cystoid macular edema

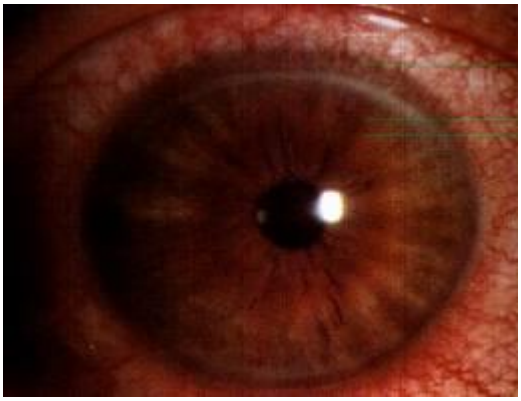

Figure 7- Iritis

KEY TO MASTER CHART

SI No: Serial Number

IP No: In Patient Number

M: Male

F: Female

SIMS: Senile immature cataract

SMC: Senile mature cataract

SHMC: Senile hyper mature cataract

PSP: Pseudophakia