PROSPECTIVE STUDY OF RETINOPATHY OF PREMATURITY IN LOW BIRTH WEIGHT AND PREMATURE BABIES AT KOLAR

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OF THE REQUIREMENT FOR THE DEGREE OF

M.S. IN **OPHTHALMOLOGY**

By

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UNDER THE GUIDANCE OF

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APRIL– 2011

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ABSTRACT

Background/Objectives:

To know the incidence, clinical spectrum and outcome following treatment of Retinopathy of Prematurity (ROP).

Materials and Methods:

Prospective study of infants with birth weight < or= 2000g and/or gestational age < or = 34weeks from the period of 1st December 2008 to 31st May 2010. They were screened by indirect ophthalmoscopy using +20D lens between 2 to 3 weeks after birth. The ROP was staged according to Revised International Classification of Retinopathy of Prematurity guidelines and followed up till the retinal vascularization was complete. Early Treatment Retinopathy of Prematurity guidelines were followed for laser treatment.

Results:

The overall incidence of ROP was 38.6%. The incidence of APROP was 13.1%. The incidence of classical ROP was 86.8%. The mean birth weight and mean period of gestation was significantly lower in babies with ROP compared to those without ROP (1555.91g vs 1672.50g) with t=2.851; (p value=0.005) and (32.23wks vs 34.58wks) with t=6.728 ;(p value <0.001) respectively. Few risk factors were present in higher proportion in babies with ROP when compared to babies without ROP, these were RDS (26.5% vs 14.6%), Oxygen (18.4% vs 8.7%), NNJ (32.7% vs 23.2%) and Sepsis (22.4% vs 15.9%) respectively. The above mentioned 4 risk factors may be clinically significant. Six infants (12 eyes, 13.1%) with Aggressive Posterior Retinopathy of Prematurity and six infants (12 eyes, 13.95%) with classical ROP underwent laser photoablation using 532nm green laser. All (100%) lasered eyes showed favourable

outcome following laser photoablation. 23.7% of our babies with ROP were weighing >1500g. 32.2% and 15.2% of babies with ROP had period of gestation >30 weeks and >32wks respectively.

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Conclusion:

Our prospective study in a level III NICU in a rural hospital reveals that timely and appropriate screening and treatment of Retinopathy of Prematurity results in excellent outcomes of babies with severe ROP. The Western screening guidelines are challenged by our results as we had 23.7% of infants with ROP whose birth weight was >1500g, 32.2% and 15.2% of infants with ROP with POG >30 and >32 weeks respectively and show that it may not be applicable in rural neonatal care centres of our country

Keywords: Retinopathy of Prematurity, period of gestation,

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INTRODUCTION

Retinopathy of prematurity (ROP) is a potentially blinding vasoproliferative retinopathy seen in premature infants with low birth weight.[1]

Prematurity, in terms of both gestational age (less than 32 weeks) and low birth weight (less than 2000 g) is the most important risk factor for the development of ROP. High concentration of oxygen administration for long duration is also considered an important risk factor. Newer risk factors continue to be defined, but overall the etiology remains unknown and multifactorial.

Since there are increased number of neonatal care units, large numbers of low birth weight premature babies are surviving and are at high risk of developing retinopathy of prematurity if not detected early and treated. There are very few studies being done on retinopathy of prematurity, its incidence and risk factors and also about the treatment and its outcome particularly in the semi urban and rural areas.

So, this study is being under taken to determine the incidence, risk factors, clinical spectrum and outcome following treatment of retinopathy of prematurity in our setup and also to increase the awareness about this condition.

AIMS AND OBJECTIVES

To know the incidence, clinical spectrum and outcome following treatment of Retinopathy of Prematurity.

REVIEW OF LITERATURE

Retinopathy of prematurity (ROP) is a disorder of premature low birth weight infants featuring abnormal proliferation of the developing blood vessels at the junction of vascular and the avascular retina.

ROP was first described by Theodore Terry in 1942 as retrolental fibroplasia.[2]

The term ROP was coined by Heath in 1951.[3] In the same year, Campbell suggested that uncontrolled oxygen was responsible for epidemic of ROP. [4]

Second epidemic occurred in late 1970s and 1980s due to increased survival of very low birth weight infants with assisted ventilation.[5-8]

In Asian countries like India, due to extremes of health care both first and second epidemic exists called the Third epidemic.[9]

Reasons for the "third epidemic" in middle income countries and India are

- High Birth Rates
- High rates of preterm births (2% vs 1% < 1500g)
- Improving survival rates
- Varying levels of neonatal care
  - Excellent – second epidemic picture
  - Poor – first epidemic picture
- Screening programs
  - Coverage is not 100%
  - More mature and heavier babies are not covered

The first blood supply to the inner retina appears in the form of "spindle cells" from the adventitia of the hyaloid artery at about 16 weeks of gestation. Spindle cells canalize and metamorphose into mature vessels.[10] and reach the nasal ora serrata by around 36weeks and temporal by around 39-41weeks. [5, 11]

Etiopathogenesis of ROP is described by 2 hypotheses:

1) the classical theory [12,13] - which states that raised pO_2 if sustained leads to permanent vasoconstriction and this in turn causes endothelial proliferation adjacent to it when neonate returns to room air thus neovascularization.

2) Spindle cell theory [14,15] - proposed by Kretzer et al, postulates that neovascularization following spindle cell insult by excess angiogenic factors that are produced in relatively hyperoxic retina in preterm infants, whose retina is thin, largely avascular and deficient in anti-oxidative property. Excess VEGF (vascular endothelial growth factor) which is up-regulated by hypoxia is considered as the most important factor causing ROP.[16]

There are very few studies being done in India. And all are reported from urban cities.

The first prospective study in India was done by Charan et al in the year 1995 in babies weighing <1700g at birth and the incidence was found to be 47.27%.[17]

In the same year, a study on ROP in babies <2000g in Indian subcontinent, by Gopal L et al revealed the incidence to be 38%.[18]

In Bangalore the study was done on the incidence and risk factors by Rekha SS and Battu RR in 1996 where the incidence was 46% and oxygen given at birth and anemia were found to be the independent risk factors.[19]

Next in 1996, Maheshwari et al found the incidence to be 20% in babies weighing <1500g.[20]

A study by Varughese in 2001 in babies weighing <1500g, showed the incidence as 51.89%.[21]

So, this study is being under taken to determine the incidence, clinical spectrum and outcome of treatment of Retinopathy of prematurity in our setup and also to increase the awareness about this condition.

The International Classification of Retinopathy of Prematurity (ICROP) [22] was published in 1984 under the leadership of John Flynn later expanded in 1987. It was developed to describe the severity of ROP based on 4 parameters:

1. Zone,

2. Extent of involvement by clock hours. (twelve 30 degree sectors involved)

3. Stage of the disease at the junction of vascular and avascular retina and the

4. Presence or absence of plus disease (dilated and tortuous posterior pole vessels)

Each zone is centered at the optic disc:

Zone I- consists of a circle whose radius is twice the distance from the optic disc to the macula.

Zone II- extends centrifugally from the edge of zone 1 peripherally to the nasal ora serrata.

Zone III- residual crescent of the retina temporally i.e. anterior to zone 2.

Staging of the disease:

Stage 1- demarcation line: a flat white line lies within the plane of the retina at the junction between the vascularized and the avascularized retina.

Stage 2- ridge at the junction between the vascularized and the avascularized retina. It extends above the plane of the retina.

Stage 3- extraretinal fibrovascular proliferation or neovascularization that extends from the ridge into the vitreous. The severity of the lesion can be subdivided into

a. Mild: presence of only limited amount of vascular tissue,

b. Moderate: significant amount of tissue infiltrating into the vitreous.

c. Severe: massive infiltration of tissue surrounding the ridge is seen.

Stage 4a- extra foveal partial retinal detachment (RD)

Stage 4b- partial RD involving the fovea.

Stage 5- total RD

A "Plus" (+) is added, if at least 2 quadrants (usually 6 or more clock hours) of dilation and tortuosity of the posterior retinal blood vessels is seen.[23]

Prethreshold ROP [23] was defined as:

- Zone I, any stage ROP without plus disease
- Zone II, stage 2 ROP with plus disease
- Zone II, stage 3 ROP without plus disease;
- Zone II, stage 3 ROP with plus disease but fewer than 5 contiguous or 8 cumulative clock hours.

Threshold disease is defined as five contiguous or eight non contiguous clock hours of involvement of stage III with plus disease in zone I or zone II.

ICROP was updated in 2005 [24] by adding

1. the concept of a more virulent form of retinopathy – APROP (Aggressive posterior Retinopathy Of Prematurity)
2. Description of an intermediate level of vascular dilatation and tortuosity of posterior pole vessels that are insufficient for the diagnosis of plus disease (Pre-plus disease)
3. Clarification of extent of zone I.

APROP previously called the Rush disease or type II or fulminate ROP is characterized by increased dilatation and tortuosity of posterior pole vessels in all 4 quadrants i.e. out of proportion to the peripheral retinopathy. It does not progress through the classic stages of 1 to 3. It may appear as only a flat network of

neovascularization at the deceptively featureless junction between the vascularized and the avascularized retina. There can be hemorrhages, nodes or loops present at that junction.

Fulminate retinopathy of prematurity is again classified into 3 stages by Shapiro [25]:

Stage 1- no definite opaque demarcation line at the junction of vascular and the avascular retina. There may be halo at the edge of the vascular junction.

Stage 2- fibrous tissue is transparent and arteriovenous anastomoses are seen in the avascular retina.

Stage 3A- new vessels are seen that appear flat as fronds or tangles in circumferential extent.

Stage 3B- neovascular frond is clearly above the retinal plane and is seen in isolated regions <2 clock hours

Stage 3C- new vessels continue to extend circumferentially, radially and in height.

Earlier, peripheral retinal ablation was performed when the ocular findings indicated a risk of approximately 50% for retinal detachment that is when there was a threshold disease as indicated by the CRYO-ROP study.[26]

Now the treatment is based on ETROP (Early Treatment Retinopathy of Prematurity) guidelines.[23]

In this study, infants with bilateral high-risk prethreshold ROP (n=317) had one eye randomized to early treatment with the fellow eye managed conventionally (control eye). In asymmetric cases (n=84), the eye with high-risk prethreshold ROP was randomized to early treatment or conventional management. Results showed a reduction in unfavorable visual acuity outcomes with earlier treatment, from 19.5% to 14.5% (*P*=0.01). Unfavorable structural outcomes were reduced from 15.6% to 9.1%

(P<0.001) at 9 months. Further analysis supported retinal ablative therapy for eyes with type 1 ROP, defined as

- Zone I, any stage ROP with plus disease
- Zone I, stage 3 ROP with or without plus disease
- Zone II, stage 2 or 3 ROP with plus disease

The analysis supported a wait-and-watch approach to type 2 ROP, defined as

- Zone I, stage 1 or 2 ROP without plus disease or
- Zone II, stage 3 ROP without plus disease.

These eyes should be considered for treatment only if they progress to type 1 or threshold ROP.

In the past decade, laser photoablation has almost supplanted cryotherapy as the standard treatment for ROP. [27]

Compared to cryotherapy, laser photoablation seems to be associated with better structural and visual outcomes, fewer post-operative complications and less myopia.

In the study by Sanghi G [28] showed, treatment with 532 nm green laser was possible in eyes with tunica vasculosa lentis and vitreous or preretinal haemorrhage, without inducing any cataract, anterior- segment ischemia or hyphaema and also that treatment with 532nm green laser had less pain than the diode laser, which has a deeper penetration.

Laser treatment can be done in a scattered pattern, a near-confluent pattern, or a confluent treatment pattern. A study [29] showed that patients treated with confluent laser photocoagulation had a low rate of progression to stage 4 or 5 retinopathy of prematurity. Also, the need for additional laser treatment was small, with rates of

complications and structural outcomes comparable to previous reports using a nonconfluent laser pattern.

When APROP is diagnosed immediate treatment is indicated. Circumferential laser ablation of the avascular peripheral retina has been shown to achieve retinal quiescence within hours.[30]

Eyes with APROP may follow an atypical course after laser ablation. Such eyes may still fare poorly, even when plus disease wanes. Additional laser to avascular retina may be necessary after the overlying flat stage 3 neovascularization regresses. The absence of obvious fibrosis at the time of laser should provide no reassurance as to reduced probability of progression to retinal detachment.[31]

Most ROP regresses spontaneously. One of the first signs of stabilization of ROP is failure of the retinopathy to progress to the next stage. [24] ROP develops gradually with the earliest stages appearing usually 6 to 8 weeks after birth. In some extremely low birth weight infants, development and progression may become very rapid, which is termed rush disease.

The most common natural outcome of ROP in early stages is regression of disease without any major sequelae in 80-90% cases. Gallo et al [32] reported frequency of regressed ROP with moderate to severe sequelae in 45.5 % cases.

The scenario in the developed and developing countries differs. In the latter ′larger′ and ′older′ infants are now more likely to develop ROP than their counterparts in western countries. The application of western screening guidelines for developing countries has been questioned.

There is a geographic variation in the incidence of ROP in babies born at even similar gestational ages. In the West, ROP at least of the threshold variety is not seen in

higher birth weight (BW) babies! Therefore, larger babies generally are not screened since the incidence of treatable ROP is low and these infants have been observed to have a generally good outcome even without treatment.

The American Academy of Pediatrics (AAP) recommends screening of infants born at ≤ 29 weeks gestational age (GA) and/or ≤ 1500 g BW (regardless of supplemental oxygen); 1500 to 2000 g BW if supplemental oxygen was administered and the infants had an unstable clinical course. However, Wright et al. recommended screening of all infants born at ≤ 32 weeks GA and/or ≤ 1500 g BW (regardless of supplemental oxygen) and predicted that it would lead to savings in excess of 1.5 million dollars annually in the United States; this was approved by Andruscavage et al. A recent study from the US found that no infant with birth weight greater than 1500 g developed treatable ROP.

Similar guidelines exist in the UK as laid out by the Royal College of Ophthalmologists and the British Association of Perinatal Medicine. All babies less than 32 weeks gestational age (up to 31 weeks and 6 days) or less than 1501g birth weight should be screened for ROP.

In contrast, ROP is seen in larger, bigger BW babies in Asia and other developing countries. In south India, threshold ROP has been seen in babies born with 2000 g birth weight. In India, even heavier babies (>1250 g) and older gestational age babies (>32 weeks) also fall prey to development of ROP and sometimes to severe ROP as shown by Vinekar et al [33] .While partly this might reflect the failure of very small infants to thrive, other factors such as perhaps the quality of neonatal care that has led to a decline of ROP in the West is lacking here. In the light of ETROP results that advocate treatment for even high-risk pre-threshold ROP, earlier screening becomes necessary. Efforts are already underway in the UK to modify the existing guidelines.

Region-specific screening criteria modified according to recent developments in the understanding and management of ROP need to be evolved in India as well. This could be made possible by a concerted and cooperative effort between the major eye institutes with an analysis of pooled data and auditing of delivery services to constitute.

And the study by Jalali S [34] revealed that ocular morbidity (vision-threatening severe retinopathy) related to retinopathy of prematurity was seen in bigger and more mature babies. This study provides a scientific basis for establishing screening criteria for retinopathy of prematurity in South India and other middle-income countries.

Severe ROP is often encountered in babies weighing greater than 1250 g at birth in developing countries. Western screening guidelines may require modifications before application in developing countries. The fact that screening of ′heavy′ babies may be missed when we adhere to the western guidelines is evident from the study by Vinekar et al [33]. When they applied the guidelines recommended by the American Academy of Ophthalmology on their study group, 11 babies (17.7%), with threshold or worse ROP would be either > 1500 g or > 32 weeks and would have been missed. Similarly, applying British screening guidelines, 14 babies (22.6%) would have been missed. These figures are comparable with a recent report from China, where 30.4% and 16.2% of infants with ROP Stages 3, 4 and 5 were reported to exceed the screening criteria of the United States and United Kingdom respectively.

More bigger and mature babies are developing severe ROP in South India than in industrialized countries [35]. The characteristics of babies affected are similar to those seen during the first epidemic of ROP which occurred during the 1950s in Europe and North America. Guidelines on oxygenation and screening policies should be jointly

developed by pediatricians and ophthalmologists to end this epidemic of avoidable blindness in India.

Valuable information regarding the incidence, clinical course and natural history of retinopathy of prematurity was gleaned from CRYO-ROP trial. In this prospective trial, 65.8% of infants developed some degree of retinopathy of prematurity and 6% reached threshold [36, 37].

The incidence and severity of disease were closely related with lower birth weights and earlier gestational (post-conceptional) ages. Whereas, the incidence of retinopathy of prematurity was 47% in infants with birth weight between 1000grams and 1251g; it rose to 81.6% for infants weighing less than 1000g at birth. Over 80% of infants born at <28weeks developed ROP, but only 60% of infants born at 28- 31weeks developed retinopathy. No infant born after 32 weeks developed retinopathy of prematurity and stage 3 disease was not seen in infants with birth weight greater than 1500g. [37, 38]

The CRYO-ROP investigators stressed that the timing of pathological vascular events correlated more closely with postconceptional age than the chronological age, independent of birth weight. Median onset of stage 1 ROP was 34 weeks after conception. Median onset of threshold disease was 37 weeks with a range of 33.6 to 42 weeks after conception. [36, 37]

Morbidity from retinopathy of prematurity continues to be an important cause of blindness through out the world. In a study by Gilbert et al [39] in 1997, they noted the proportion of ROP declined from 'the single commonest cause of blindness' in children in industrialized countries to a fraction of that number, between 16-18% of blindness registrations. The magnitude of the problem however is not known in the countries that do not maintain blindness registers that include developing and several of the middle income countries.

In all the African countries (except South Africa), there were virtually no children with severe visual impairment (SVI, corrected visual acuity of less than 6/60 in the better eye) caused due to ROP. The Asian countries showed a fluctuation between 0-16.9%, Eastern European countries varied from 0-25.9%, and 4.1-38.6% in Latin America.

Countries with infant mortality rates (IMR) above 60/1000 live births had very low proportions of SVI due to ROP, or no cases recorded. Countries with intermediate IMR (10-60 //1000 births) seemed to have the highest proportion of childhood blindness due to ROP. This was attributed to introduction and expansion of intensive neonatal care services in these countries.

On the other hand ROP in birth weights <1000 g indicates the increased survival of extremely low birth weight, akin to the 'second epidemic' of the developed world. This mixed picture, a unique and emerging problem of the middle income countries he christened, 'the third epidemic'. It reflects the varying levels of intensive neonatal care provided within these countries. India is no exception.

Gilbert concluded that the occurrence of blindness due to ROP in some of these countries with relatively high birth weights suggested an increased survival of larger premature babies who were probably given unmonitored supplemental oxygen, akin to the 'first epidemic' in the industrialized world.[40]

In one of the older reports, Purohit (1985), [41] explored the role of multifactorial etiologies in the development of ROP. In this group of 3025 premature infants, who weighed 1750 g or less at birth, he noted the incidence of ROP of any stage to be 11% at the time of hospital discharge. When they were viewed birth weight wise, the incidence was 43% in the group between 500 and 749 g, whereas it was only 3% in the 1500 - 1750 g category. This study however had the limitation of having

multiple centers involved with each center following different criteria for selection and risk factor recording.

A Danish study conducted by Fledelius (1990) [42] based on 2 Danish surveys suggested that the screening cut-off should be no lower than 1,750 g BW, and / or 32 weeks GA. Using these criteria only 7 (out of 210) infants who were heavier than 1750 g, developed ROP, and none of these 7 showed progression. In the subgroup 1501-1750 g, 14 (out of 77) showed ROP and 1 got blind.

In a retrospective study to document regressed ROP in children aged 5-10years, premature babies born in Sweden during the years 1976-81 were included by Gallo et al (1991), [33] who studied infants <1500g birth weight and <33 weeks gestation. Birth weights between 1501 - 1999g were also included if the period of gestation was <33 weeks. In this latter group of relatively heavier babies, 17% required any form of ophthalmic care. Only one baby in this heavy group showed severe regressed ROP (7.7% of all severe regressed ROP cases). Moderate regressed ROP was seen in 11 (22.9%), and combined regression in 12 (19.7%) more infants. In addition 16 (36.4% of all non-regressed) showed no regression, and hence major sequelae. The overall frequency of regressed ROP was 45.5%.

In another study that used gestational age as the sole criteria for ROP screening, Acheson (1991), [43] used < 30 weeks as the cut off in a prospective cohort. While out of the 35 infants born at < 25 weeks, 17 (48%) had stage III disease, this proportion seemed to decrease with successive increase in the gestational age, so that at 30 weeks there were no infants with stage III ROP. Breaking the stages down by birth weight, 37% of infants < 750 g had stage III ROP, whereas in the heavier group of > 1250 g., which had a total of 79 infants, 61 had no ROP, 5 and 3 had stage I and II respectively. Only 2 infants (2.5%) progressed to stage III.

In a prospective study from UK to study the natural history of ROP, Fielder et al in 1992 [44] noted that the age at onset and rate of progression of retinopathy were largely determined by the stage of development, but were also modified by systemic and local 'risk factors'. All the 572 infants included were < 1701g BW, and included 66 Indo-Pakistani infants. Only 27 infants (4.7%) showed ROP of stages III or IV. Only 2 infants with severe ROP (stage III/IV) were heavier than 1250g BW.

Regional variations of ROP at onset and the progress exhibited a propensity to involve the nasal and temporal retina rather than the superior and inferior regions. The onset was significantly related to the degree of prematurity as follows (median GA): no ROP 32weeks; onset of ROP 30 weeks; onset of temporal and nasal retina simultaneously 28 weeks; inferior retina 29 weeks; nasal retina alone 28 weeks; and superior retina 27.5 weeks. Thus relatively mature infants develop ROP initially in the temporal retina, but with decreasing GA there was a tendency to develop in nasal and temporal simultaneously or in the most immature infants, in the nasal solely. Stage I developed at a median post conceptual age of 34.6 weeks. Stage II at 35.1 weeks, and stage III at 37.1 weeks. In 2 infants stage III was first seen > 42 weeks. The likelihood of circumferential extension increased according to the retinal area of onset: temporal<inferior<nasal<superior. 20% of ROP confined to temporal retina at onset and 75-90% in nasal / superior retina extended later to involve the entire circumference.

In an Australian study, Keith et al (1995), [45] reviewed the incidence of ROP, by dividing the infants into groups based on birth weight, namely 1000-1249 g and 1250-1499 g. The lighter group had 657, and the heavier group had 716 infants. The incidence of any ROP was 14.6% and 6.4% respectively. However in the severe ROP group the difference was even greater at 5% and 0.8% respectively. Hence they

suggested the possibility of reducing the cut-off BW for screening to 1249g or less, so as to reduce the morbidity associated with examinations, nursing expenses, data management and inconvenience for the parents.

In a German prospective study conducted by Jandeck et al (1996), [46] which included 452 premature infants of birth weight <_1500 g, or > 1500 g if they had required additional oxygen supplementation or had undergone surgery under general anesthesia, reported possible risk factors for ROP in 'large babies'.

The 3 'heavy' babies weighed 2080, 2185 and 2325 g at birth respectively. In all three cases severe intrauterine bleeding with fetal blood loss had induced preterm birth. These infants had severe peripartal asphyxia, acidosis, and low / non-measurable blood pressure. The umbilical cord packed cell volume (pcv) values were (0.14-0.19) which was very low compared with the mean pcv of the other babies (0.48). All three received multiple blood transfusions. The 3 infants in addition were operated under general anesthesia for, jejunal atresia (day 2), duodenojejunal atresia (days 17 and 43), renal and cardiac failure (days 25and 45) respectively. Besides these 3 infants no other baby over 1650 g (BW) had developed any stage of ROP.

The authors therefore commented that infants that develop severe systemic illnesses may develop threshold ROP, 'even when they weigh > 2000 g at birth'.

In particular they mentioned that if umbilical cord blood packed cell volume (pcv) is <0.30 then the infant must be screened 'even if it weighed > 1500 g at birth'.

A similar study from the United Kingdom, was undertaken by Goble et al (1997) [47] to determine whether the inclusion criteria for screening could be safely altered without missing any stage III ROP. Out of 1611 infants examined between 1989 and 1995, 1429 fell within the existing guidelines for screening produced by the Royal College of

Ophthalmologists and the British Association of Perinatal Medicine, namely any baby < 1500 g birth weight or < 31 weeks gestational age.

This retrospective analysis included all infants <1700 g BW, or < 32 weeks POG. 182 babies (out of 1429) were heavier than 1500 g BW or were older than 31 weeks POG. The mean BW was 1199 g. Interestingly, they found that no baby > 1200 g developed stage III ROP except one which weighed 1375 g at birth, and that too did not need reach threshold. They hypothesized that by reducing the cut off for screening to < 1250 g (like the CRYO -ROP study) or < 29 weeks it would reduce the number of infants to be screened by 30%. They however agreed that it is not appropriate to attempt to alter national screening guideline based on the strength of a single study, and it needed confirmation from other centres within the United Kingdom itself. Hutchison et al (1998) [48] in a study to test a screening protocol that uses the dual criteria of postconceptional and chronological age, retrieved the records of 179 infants who had undergone argon laser treatment for threshold ROP, found that 8% (25 infants) were in the birth weight group of 1251 - 1500 g.

They commented that, using chronological age alone increased the risk of missing threshold ROP in large birth weight babies. Using post-conceptual age alone would increase the risk of missing it in smaller birth weight babies. They hence recommended that the guidelines for screening should be performed at 7 weeks chronological age or 34 weeks postmenstrual age whichever comes first, (irrespective of the birth weight). This they felt seemed to reliably detect the onset of threshold ROP while reducing the number of unnecessary early examinations.

In a study undertaken to determine the appropriate upper limits for gestational age and birth weight for ROP screening, Wright et al (1998) [49] prospectively included 707

infants conforming with ROP guidelines set by the AAP, and recorded their maximum stage of ROP with respect to birth weight and gestational age.

They found no ROP greater than Stage I in infants with birth weight >_1500g, or > 32 weeks gestation. All the cases of threshold ROP and stage IV that they saw were confined to infants with gestational ages < 30 weeks or birth weights<1200g. Hence they suggested a change in existing AAP screening guideline of < 28 weeks to < 32 weeks, to help avoid the undesirable consequence of missing several cases of ROP more than Stage I. In their study the proportion of infants was 13.5%, 7.9% and 25% of Stages II, III and IV respectively and would have been missed out if the cut off was taken < 28 weeks.

It is well known that world over the survival of babies born prematurely has increased greatly in the recent past and that low birth weight carries a significant morbidity for ophthalmic problems.

The Oxford Register of Early Childhood Impairment, which records ROP as an etiology in merely 6% of the cases certainly does not reflect the worldwide situation. There is a rising incidence of severe ROP associated with increased survival of preterm babies in middle-income countries such as Latin America and Eastern Europe, all of which have less than ideal standards of care.

Western guidelines for screening are clearly not appropriate to our setting, and it is ironic that countries such as ours with sparse resources have the greatest screening load. ROP just like cataract is important for they are two of the few conditions about which the clinician can influence outcome.

Andruscavage (2002) [50] in a more recent retrospective analysis, questions the validity of the existing screening guideline (USA) with respect to birth weight. Over a six year period 438 premature infants were screened for ROP. Of the eligible infants surviving

28 days, 276 (91.7%) of 301 infants with birth weights < 1500 g and 162 (52.3%) of 310 infants with birth weights between 1501 and 2500 g were screened for ROP.

Ten infants (3.9%) of the 310 infants with large birth weights developed stage I and II ROP. Two infants (0.6%) of the 310 infants with large birth weights progressed to threshold ROP and required treatment.

The author commented that restrictive criteria to identify premature infants eligible for routine ophthalmoscopic screening for ROP may be the cause for some infants going unexamined and their ROP going undetected.

Mathew et al (2002), [51] from the United Kingdom, in contrast to the previous study, claims that the birth weight criteria may be lowered based on his more recent retrospective findings. Over a 8 year period infants were screened based on the existing UK guideline of BW < 1500 g. A POG of < 32 weeks was used. Of the 205 eligible infants, 64 were found to have ROP of any stage (incidence 31.2%). The maximum stages reached were 13.2%, 11.7% and 4.8% for stages I, II and III respectively. Three babies (1.5%) reached threshold.

If however the cut-off, < 1251 g and < 30 weeks were to be applied on the same group:- 150 / 205 (73%) fewer babies would have needed screening. In their group there were only 5 babies (8%) with birth weight > 1250 g and 8 babies (12%) with POG of > 30 weeks amongst the babies with ROP but all were stages I or II. All the babies with threshold had BW < 1000g and POG of < 28 weeks.

The authors recommended refinement in the existing screening guidelines to include only those < 1251 g and < 30 weeks POG, in order to focus on 'vision threatening stages of ROP'.

Archambault (1987), [52] retrospectively analyzed the incidence of ROP in babies < 2000 g at birth. 157 babies over a two year period were eligible. Of these 24 (15%) had ROP,

75% of who were < 1000 g BW. This incidence was eight times more in this birth weight group than between 1001- 2000 g. Only one case of grade IV ROP was diagnosed, in an infant > 1000 g (1020g, BW).

Stressing the multifactorial basis of ROP development, Charles et al (1991) [53] undertook a study in a predominantly poor, inner city population in the United States. The mean birth weight of all the premature babies (< 36 weeks, gestation) was 1295 g (450-4175g). They found the incidence of ROP to be 46%. Of those babies who were < 1200 g the incidence rose to 72%.When comparing the mild ROP and severe ROP groups, birth weight ($p<0.001$), O2 administration ($p<0.01$), intraventricular hemorrhage ($p<0.01$) and sepsis ($p<0.01$) were found to be significant.

More recent reports on risk factor analysis [19,20 , 40, 46, 54 ,55] include those that were traditionally thought to be associated with ROP and a few newer factors. The past two years has seen few reports in the literature relating to risk factor analysis. They include birth weight, gestational age, apgar score at one minute (> 6 low risk) and time of oxygen exposure (> 25 days high risk, < 5 days low risk), number of blood transfusions, level of anemia (Hb < 8 g/dl or Hct <_25% have milder ROP than that of less severely anemic infants), history of bronchopulmonary dysplasia (BPD), sepsis, length of hospital stay, ventilator days, prenatal steroids, oxygen dependency at day 60 of life and necrotizing enterocolitis More recent studies in the Western literature refer to the 'lowering of incidence of ROP'.

Rowlandste al (2001), [56] in a 9 year prospective study, screened babies according to the UK guidelines published by the Royal College of the 383 babies examined they found a decrease in the number of infants with Stage III - V disease over the 9 year period despite a significant decrease in the mean gestational age (29.8 to 29 weeks) and mean birth weights (1361 to 1134 g)during that same period.

The felt that the reduction in incidence was attributable to improved ventilation techniques, use of prenatal steroids, and use of surfactant and overall improvement of neonatal services.

Fledelius (2000) [57] reporting on the changing trends of ROP incidence in Denmark, found that there was a drop in the incidence of ROP in babies weighing between 1251 and 1750 g, but no change for those weighing less than 1251 g.

The explanation for this queer change has been debated by Donahue in the BJO editorial, [54] where he proposes that it is due to the dichotomous fashion in which the recent advances in neonatology have influenced incidence trends of ROP. He observed that while children who would have fought for survival 2 decades ago now remain healthy throughout their neonatal period, others for whom death was certain now fight for survival with reasonable odds. Hence older babies are showing a 'decreased risk' for ROP whereas smaller children who are alive are at 'high risk'. The need of the hour he says is to identify with more accuracy children deemed to be at 'highest risk'.

Quinn in the same editorial [58] categorically states that, 'there is no agreed policy on what should be the screening criteria in babies larger than 1250 g'. He agrees that the current database of the natural history of ROP has been collected from the nurseries of industrialized nations and similar data from 'industrializing nations' are largely unknown.

He states that with the current level of understanding, it is not possible to estimate the cost effectiveness of screening heavy birth weight babies due to three reasons: 1) the population at risk has not been defined. 2) the natural history of babies weighing > 1251 g has not been documented 3) an effective strategy to screen these heavy babies before they are discharged from the hospital has to yet be evolved. Fearing that 'older

and larger babies' may be at risk in these 'industrialising nations', screening guidelines must 'not be generalised' and must take into account' regional differences'.

The ROP scenario in India is relative new, but like other middle-income group countries, we too are on the brink of a major public health problem. On one hand our infant mortality is in the mid-range of 60/1000 LB (with trends of further reduction), our neonatal care centres are expanding and general level of care has improved over the last decade. On the other hand however factors such as inequitable distribution of medical resources in particular neonatal services, lack of awareness amongst neonatologists and ophthalmologists alike and lack of special training and resources for treatment has made the 'at risk' infant in India very susceptible to the danger of ROP.

As mentioned earlier in one of the first reports on the incidence of ROP, Charan et al (1995) [17] published the first prospective data of our country. 165 babies weighing ,<1700 g (BW) were included. The incidence of any stage of ROP was a staggering 47.27%. Group wise, 16.97 %, 17.58%, 11.52% and 1.21% reached Stage I, II, III and IVB respectively. Although babies with a lower birth weight and lower gestational age had a significantly higher incidence of ROP, yet, the difference in mean birth weight and gestational age at birth for various stages of ROP was not significant. The problem of ROP in 'heavier' babies' was recognised even in that study wherein 29 babies (17.6%) weighing between 1251 - 1500 g and 14 babies (8.5%) weighing 1501 - 1700 g developed ROP. The latter group included 3 babies with stage III. The authors hence recommended that all babies <1700 g BW must receive screening.

Almost simultaneously, Gopal et al (1995) [18] reported the incidence of ROP in 50 babies < 2000 g (BW). Their incidence was 38%. The mean birth weight in the study was 1477 g and mean POG was 32 weeks. 23.5% of the babies weighing > 1500 g developed ROP. Of the 19 cases with ROP, 2 did not receive oxygen during the postnatal course. 8

infants developed threshold, all of whom had received oxygen and 75% of whom had received blood transfusions.

In another South Indian study, Rekha et al (1996) [19] reported the incidence and risk factors for ROP. In this prospective study over a 4 year period, all babies < 1500 g and <_34 weeks POG were included. The incidence of any stage of ROP was 46%. This was 73% in babies < 1000 g (BW) and 47.3% among the < 1500 g group. Of the 100 babies 21 had stage I, 14 had stage II, 8 had stage III and 3 *had stages* IV and V. The *significant* risk factors were POG < 32 weeks, blood tranfusions, apnoea, anemia and exposure to oxygen, with the last two risk factors being independent predictors for the development of ROP.

In a similar study, Maheshwari et al (1996)[20], prospectively screened babies of <1500 g BW, POG < 35 weeks who required oxygen for > 24 hours. 66 infants were eligible after a follow-up period of 15 months. The incidence was comparatively lower than the other Indian studies at that time, namely 20% in this cohort and 27 % among VLBW babies. The incidence of threshold ROP was 7%. Blood transfusion and clinical sepsis emerged as separate risk factors in this study group.

In a retrospective study by Vinekar et al [33], where they the included the premature babies with BW >1250g the mean birth weight was 1533.9 g and the mean period of gestation was 30.9 weeks. One hundred and twenty-four of 275 eyes (45.1%) had threshold or worse ROP. Statistically significant risk factors for threshold or worse disease were, ′outborn babies′ ($P < 0.001$), respiratory distress syndrome ($P = 0.007$) and exchange transfusion ($P = 0.003$).

Studies[59] have shown that mutations occurring in the FZD4 gene affect patients diagnosed with both FEVR and ROP and that clinical picture often overlaps and may require a detailed birth and family history for diagnosis. Genetic testing confirms

inherited vitreoretinopathy and helps direct clinical management. Patients diagnosed with ROP may have a mutation in the FZD4 gene and display characteristics consistent with FEVR.

Another study by Vinekar et al[60], reports the possible role of thrombocytopenia in the pathogenesis of APROP. The index case described in this study showed spontaneous resolution of APROP with plus disease within 3 days of correcting thrombocytopenia and did not require laser treatment. The retrospective cohort of nine consecutive Asian Indian infants with APROP with similar stage and plus disease as the index case was studied. The mean platelet count of these infants before laser treatment was compared with 21 age- and birth weight-matched control subjects. The role of low platelets in the etiopathogenesis of APROP has not been previously elucidated. This study shows that a platelet count of <100,000 was associated with severe disease. Recently, platelets have been reported to play a key role in angiogenic regulatory protein delivery. It is possible that premature infants who develop retinopathy of prematurity in the setting of low platelet counts may lack the function of either delivering the optimal level or incompletely scavenging the excess of vascular endothelial growth factor A present in APROP. The spontaneous resolution of disease in the index case with platelet correction alone needs additional studies to correlate the timing and magnitude of correction that may play a role.

In a prospective cohort study by Chowdari S [61], preterm infants with BW <1500 g and POG <or=32 weeks and who also had additional risk factors were screened for ROP at 4 weeks after birth or 31-33 post conceptional age, whichever was later. The incidence of ROP in the 552 infants who were screened was 22.3%. No ROP was found in infants weighing>or=2000 g or with a gestational age more than 36 weeks.

Significant risk factors predisposing to ROP in their study were septicemia, apnea, oxygen therapy and use of blood products.

The incidence from our country has been reported by Varughese et al (2001) [21],which included babies with both gestational age < 34 ; and birth weight < 1500 g. Out of 123 babies which were eligible using these criteria, 79 survived and received screening. 38 of these had no ROP, 7 had stage III, 5 babies progressed to threshold, 3 of which received cryotherapy and 2 were referred for laser outside. The overall incidence of any ROP in this study was 51.89%.

A study by Azad et al [62] which reinforces the fact that screening of all babies is necessary in cases of multiple births, and birth weight alone cannot be relied upon as a single factor to predict the severity and course of ROP, as even heavier siblings can develop severe ROP and may present with variable course.

Shah et al [63], have said that Retcam may replace Binocular indirect ophthalmoscopy (BIO) for screening of ROP as the positive and negative predictive values were 96.43% and 70.97% respectively when Retcam was compared with the BIO in their study in 2006-Screening for retinopathy of prematurity--a comparison between binocular indirect ophthalmoscopy and RetCam 120 In another study it was shown that sensitivity of WFDRI (wide field digital retinal imaging) in detecting any ROP, stage 3 ROP and plus disease to be 60%, 57% and 80%, respectively, with a specificity of 91%, 98% and 98%, respectively. There was excellent proportional agreement between the two screening methods for detecting stage 3 ROP and plus disease (0.96 and 0.97) and very good agreement on management decisions [64].

It provides state-of the art wide field pediatric retinal imaging (130 degrees). It has instant & accurate documentation, avoiding time-consuming retinal drawings In just a few minutes one can do an entire exam and the images are stored

permanently on a 50 GB DVD. It allows easy accessible imaging even by non-ophthalmologists (NICU nurses) and also allows the transmission of these digital images to centers where ROP expertise is available via telemedicine. It serves as a good teaching tool for others, also helps in teaching the parents. Comprehensive database keeps track of each image, session and patient allowing for later side-by-side comparison of the case images. FFA can also be done. Newer Retcam II & III have the same features but with a flat screen monitor. Retcam shuttle is laptop based.

MATERIALS AND METHODS

Study design: Prospective study

Study period: 1st December 2008 to 31st May 2010

Source of data:

Neonatal care units of department of Paediatrics, RLJH & RC, attached to Sri Devaraj Urs Medical College, Tamaka, Kolar.

Inclusion criteria:

1. All neonates with birth weight < or = 2000g
2. All neonates with gestational age < or = 34weeks.
3. Any neonate outside these criteria if the treating pediatrician believed it was at special risk for ROP due to post natal systemic illness.

Exclusion criteria:

1. Family history of familial exudative vitreoretinopathy,
2. Family history or features suggestive of Norries disease
3. Congenital hydrocephalous.
4. Any baby lost to follow up before the outcome of the disease could be ascertained.

Method of collection of data:

Eyes fulfilling the above criteria framed, from the period of 1st December 2008 to 31st May 2010 were included in this study.

All babies who require ROP screening according to the inclusion criteria mentioned above, and also the infants outside these criteria if the attending paediatrician seeks a

referral for this purpose that is if the infant was “at risk” for ROP were registered and screened in the ROP clinic.

Informed consent was taken from the parents prior to the examination and also prior to the treatment.

The records maintained in the file include neonatal data.

Data collected included variables like date of birth, birth weight, period of gestation, history of oxygen exposure and associated neonatal illnesses that were present.

Other risk factors specially looked for were the presence of respiratory distress syndrome, hyaline membrane disease, sepsis, neonatal jaundice, multiple births, apneic episodes, anemia, intraventricular hemorrhage, pneumonia, polycythemia, thrombocytopenia, blood transfusion, hypoglycemia, shock, necrotizing enterocolitis, hydrocephalous, congenital heart disease, and meconium aspiration syndrome. Maternal complications e.g. pregnancy induced hypertension were noted wherever such details were available.

Anterior segment examination was carried out in all the babies prior to dilatation to look for engorgement of iris vessels, pupillary rigidity in particular.

Posterior segment examination was carried out under full pupillary dilatation using phenylephrine 2.5% and cyclopentolate 0.5% eye drops instilled 2 to 3 times with an interval of 15 to 20 minutes before the examination.

All babies included for ROP screening were subjected to wide-field digital retinal imaging using a portable pediatric retinal camera (Retcam Shuttle, Clarity, MSI, CA, USA). This was done in collaboration with the Department of Pediatric Retina, Narayana Nethralaya Postgraduate Institute of Ophthalmology, Bangalore, whose team would visit the NICU on a fixed schedule every week.

The timing of first examination:

- At 2wks of birth for the babies born with birth weight <1000g and/or <29wks.
- At 3wks of birth for the babies born with birth weight >1000g and/or >29wks of gestation.

All the babies were screened by the trained ophthalmologist using indirect ophthalmoscope and +20D lens with scleral depressor after applying infant lid speculum and topical anaesthesia (proparacaine 0.5%). Oculocephalic reflex was made use of, by rotating the infant's head, to visualize the required area of retina standing opposite the quadrant of the retina to be examined. Wire vectis is used as a scleral indenter whenever required.

An anterior segment photograph of the maximally dilated pupil and 7 or more images of the retina (disc centred, macula centred, macula temporal, and 4 quadrants) were captured. When required or if there was ROP, more number of images of that quadrant was also captured.

Depending upon the severity, retinopathy of prematurity was classified according to revised International Classification of Retinopathy of Prematurity, published in 2005.

Frequency of follow up was determined by retinal findings at first examination:

Temporal avascular retina was followed up after 2 weeks

Stage 1 or 2 without plus disease in zone 1 or 2 was followed up after a week.

Any stage meeting the criteria of ETROP guidelines was treated with 532 nm green laser using indirect ophthalmoscope, at R.L.Jalappa Hospital, Kolar NICU under the monitoring of treating Paediatrician.

Near confluent to confluent laser spots were applied. Supplement laser was given wherever required.

Follow up was done until the retina was completely vascularized.

Laser treatment, the accepted modality of treatment was done for the babies with ROP who met the criteria of ETROP guidelines, after taking the informed consent from the parents.

Study has been approved by the Ethical Committee of Sri Devaraj Urs University, Kolar

RESULTS AND ANALYSIS

157 infants who fulfilled the inclusion criterion of our study were screened and registered in the ROP clinic, Department of Ophthalmology in association with neonatal unit of department of Paediatrics, R.L.Jalappa Hospital Tamaka, Kolar, between 1st December 2008 and 31st May 2010. This is a level III NICU in a rural set up.

Of the 157 babies, 39 babies did not follow up till the retinal maturity was diagnosed. Of these 39 babies, 34 had immature retina up to zone 3, 2 had stage 1 and 3 babies had stage 2.

All the babies who had retinal vessels reaching up to mid zone 3 were considered mature, despite having missed the follow up. Hence, 118 babies (236 eyes) were included for the analysis and is presented below.

The mean number of screening visits for the infants was 4.30± 2.95 visits (Mean ± SD) (range: 1 to 14) and the median was 4.

DEMOGRAPHIC DETAILS –

Of the 118 babies, 69 (58.5%) were male and 49 (41.5%) were female.

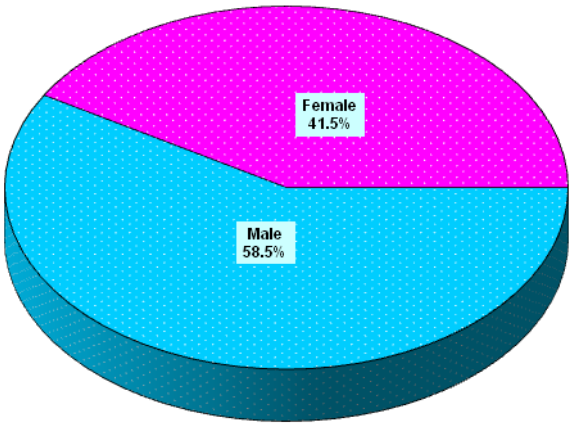


fig 1: Gender distribution

The overall mean birth weight of the babies was 1627.78g (±SD 308.21), ranged from 940g to 2700g.

The overall mean gestational age was 33.65weeks (±SD 2.83), ranged from 28weeks to 40weeks.

CLINICAL CHARACTERISTICS OF THE BABIES SCREENED WERE AS FOLLOWS:

The birth weight ranged from 940g to 2700 g.

The overall mean birth weight of the whole study group was 1627.78g (±SD 308.54).

Median birth weight of babies screened in one year was found to be 1600g.

Mean birth weight of babies with ROP was 1555.91 grams (±SD 278.23) (range- 940 to2300g). Median birth weight of the babies with ROP was 1570g.

The mean birth weight of the babies without ROP was 1672.50(±SD 317.23), the median being 1630g.

The mean birth weight was significantly lower in babies with ROP compared to those without ROP (1555.91g vs 1672.50g) with t=2.851; P=0.005.

Table 1:

	Number of eyes	**Birth weight**	
		Mean	**SD**
No ROP	**145**	**1672.50**	**317.23**
Any ROP	**91**	**1555.91**	**278.23**

The overall mean gestational age of the babies was 33.65weeks (±SD -2.83).

Median period of gestation of all the babies was 33.

Mean period of gestation of babies with ROP was 32.23weeks (±SD2.34), ranged from 28 to 39 weeks. Median was 32weeks.

The mean period of gestation of the babies without ROP was 34.58(±SD2.74) and median was 35.

The mean period of gestation was significantly lower in babies with ROP compared to those without ROP (32.23wks vs 34.58wks) with t=6.728 ;(p value <0.001).

Table 2

	Number of eyes	**POG**	
		Mean	**SD**
Without ROP	**145**	**34.58**	**2.74**
Any ROP	**91**	**32.23**	**2.34**

CLASSIFICATION OF ROP AND DISEASE PROFILE**:**

Of the 118 babies included in the study 49 babies had Retinopathy of prematurity.

Overall incidence of ROP was found to be 38.6%.

ROP Sub classification –

49 babies had some ROP. 43 babies had classical ROP and 6 babies had APROP.

Only 91 eyes of these 49 babies showed some stage of ROP, remaining 7 eyes resolved to mature retinas without developing ROP at any time during follow up.

Of the 91 eyes, who had some ROP 79eyes (86.8%) had classical ROP and 12 eyes (13.1%) had APROP.

Anterior segment involvement was not seen in any of our babies.

Classical ROP**:**

ROP stage1 was seen in 26 eyes (28.57%), stage2 was seen in 51eyes (56%) and stage3 was seen in 2 eyes (2.1%).

Plus disease was seen in 8 babies (12 eyes)

13.95% (six babies) with classical ROP met the threshold for treatment based on ETROP guidelines and received laser photocoagulation.

Aggressive Posterior Retinopathy of Prematurity (APROP)**:**

Of the 118 babies 12 eyes of 6 babies (13.1%) had APROP.

All 100% had plus disease at the time of treatment.

All 100% of APROP had zone1 involvement.

All eyes (100%) with APROP received laser photocoagulation at the time of diagnosis.

Table 3: Stage distribution of ROP

ROP	**Number of eyes**	**%**
• Stage I	26	28.57
• Stage II	51	56
• Stage III	2	2.1
• Stage APROP	12	13.1

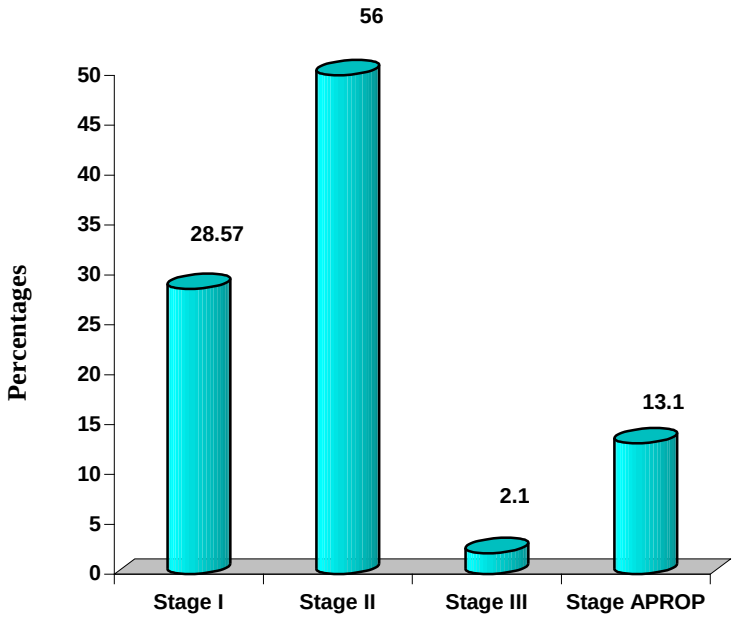


fig 2: stage distribution of ROP

Zone sub classification –

Of the 91 eyes with ROP, Zone I disease was observed in 14 (15.3%) eyes, Zone II in 29 (31.8%) eyes and Zone III in 48 (52.74%) eyes.

2 eyes of classical ROP and 12 eyes (100%) of APROP had zone I disease.

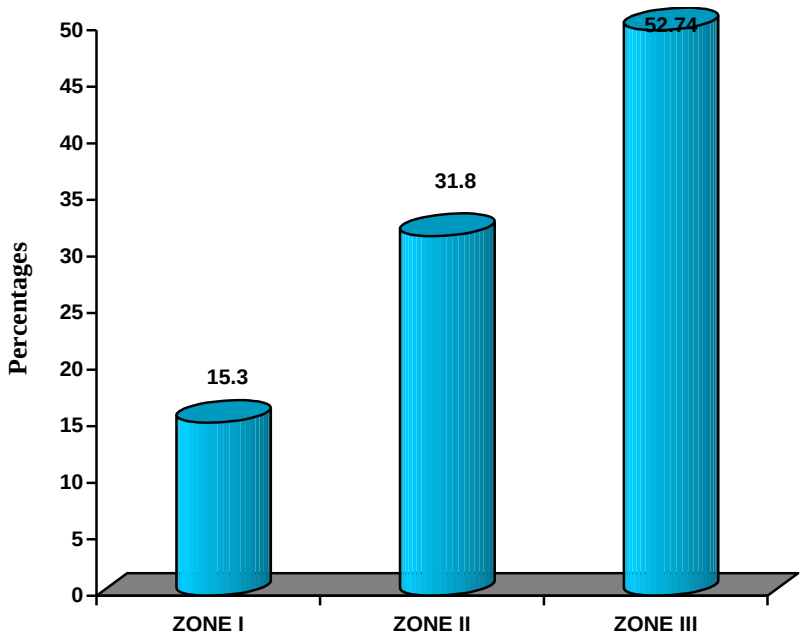


fig 3**: ZONE Distribution of ROP**

TREATED ROP**:**

12 babies, 24 eyes reached threshold for treatment during screening. Of 24 eyes of 12 babies, 6 babies had classical ROP and remaining 6 had APROP.

All (100%) were treated with 532nm green laser using indirect ophthalmoscopic delivery system at SDUMC, Kolar neonatal intensive care unit under the monitoring of Pediatrician.

ETROP (Early Treatment Retinopathy of Prematurity) guidelines were followed for the treatment.

Near confluent to confluent laser burns were delivered in all eyes. All eyes had favourable outcome after laser treatment (100%). Favourable outcome was defined using CRYO-ROP guidelines.

12 eyes (50%) required supplement laser after a median of 1 week. Of the 12 eyes who needed supplement laser 10 eyes had APROP and 2 eyes had stage 2 ROP with plus disease (classical ROP)

Overall mean number of laser spots was 2756.96 (±SD 1526.52), median was 3092.

The mean number of laser spots was 1504.17(±SD1061.72), median was 1085.5 in the eyes treated for classical ROP.

The mean number of laser spots was 4009.75(±SD566.38), median was 4141.50 in the eyes treated for APROP.

The mean number of supplement laser spots was 353.17(±SD266.95) and the median 360.50.

Table 4 : Mean and median laser spots

Laser spots	Mean	SD	Median
12 cases			
• **RE**	2546.25	1429.87	3057.00
• **LE**	2967.67	1652.56	3345.00
• **both eyes**	2756.96	1526.52	3092.00
Classical ROP			
• **RE**	1423.33	1135.91	1085.50
• **LE**	1585.00	1083.51	1080.00
• **both eyes**	1504.17	1061.72	1085.5
APROP			
• **RE**	3669.17	425.99	3648.00
• **LE**	4350.00	495.86	4226.60
• **both eyes**	4009.75	566.38	4141.50

Table 5: Number of supplementary laser spots

Supplementary laser spots	Mean	SD	Median
• **RE**	402.00	288.55	378.50
• **LE**	304.33	260.33	229.50
• **Both eyes**	353.17	266.94	360.50

RETCAM IMAGING**:**

All babies could be successfully imaged in every visit (100%). The images were obtained based on the protocols described in the methods.

RISK FACTOR ANALYSIS**:**

We noted the presence or absence of 17 risk factors. They are enumerated in the table below.

In our NICU, considering the cohort of all babies the common risk factors were Neonatal jaundice (27.11%), Twins (24.5%), Respiratory distress syndrome (RDS) (19.49%), Sepsis (18.6%), Pregnancy induced hypertension (PIH) (18.6%). However, none of the risk factors reached statistical significance.

We compared the incidence of all risk factors between the group that had ROP and the group that did not have ROP and we found that none of the risk factors reached statistical significance. However, clinically we noticed a trend of few risk factors being present in higher proportion in babies with ROP when compared to babies who did not have the disease. These were

1.Respiratory distress syndrome (26.5% vs 14.6%)

2.Oxygen therapy (18.4% vs 8.7%)

3.Neonatal jaundice (32.7% vs 23.2%)

4.Sepsis (22.4% vs 15.9%) respectively.

Multivariate analysis was not done as no risk factor was significant on univariate analysis. The above mentioned 4 risk factors may be clinically significant.

Table 6:

Risk factors	NO ROP		ANY ROP		P value
	No	%	No	%	
Oxygen	6	8.7	9	18.4	0.162
Sepsis	11	15.9	11	22.4	0.473
Anemia	1	1.4	2	4.1	0.569
Polycythemia	1	1.4	0	0.0	1.000
Thrombocytopenia	6	8.7	3	6.1	0.734
DIC	1	1.4	0	0.0	1.000
NNJ	16	23.2	16	32.7	0.296
MSAF	3	4.3	1	2.0	0.640
RDS	10	14.5	13	26.5	0.156
Birth Asphyxia	11	15.9	7	14.3	1.000
Pneumonia	0	0.0	1	2.0	0.415
PIH	12	17.4	10	20.4	0.811
LSCS	14	20.3	7	14.3	0.470
Hydrocephalous	0	0.0	1	2.0	0.415
Meningitis	2	2.9	0	0.0	0.510
Plasma Transfusion	2	2.9	0	0.0	0.510
Twins	15	21.7	14	28.6	0.356

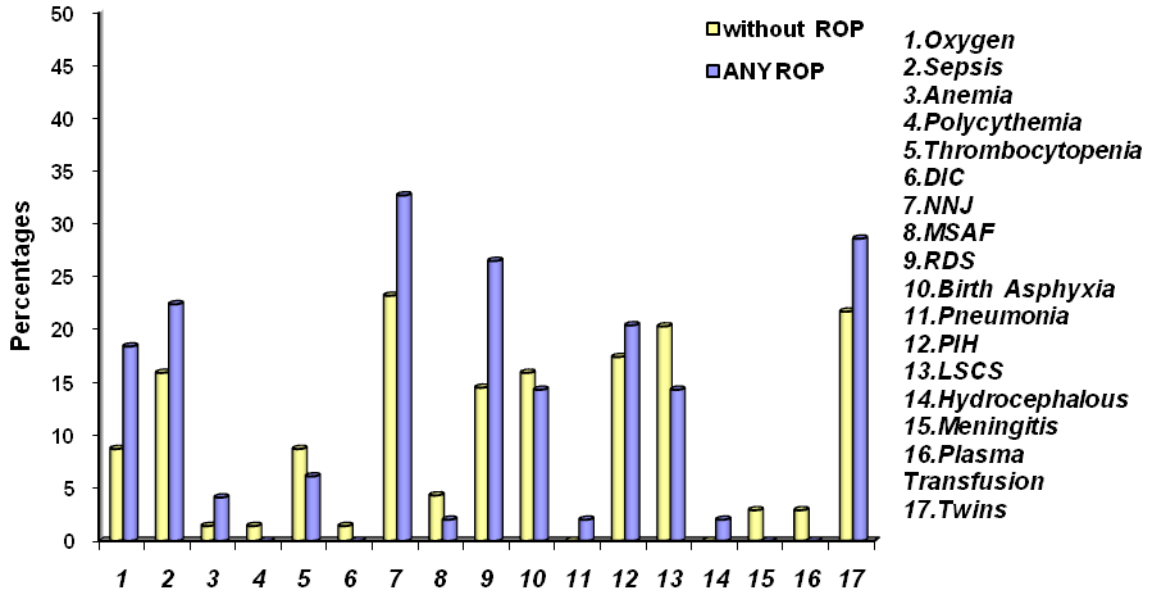


fig 4: Risk factors

BIRTH WEIGHT AND PERIOD OF GESTATION ANALYSIS:

We analyzed the cohort based on the birth weight and period of gestation with respect to screening guidelines prevalent in developed countries.

And are represented in the tables below:

Table 7:

Birth weight	NO ROP (n=69)	ANY ROP (n=49)
• ≤1500 grams	24	21
• >1500 grams	45	28

If the criteria of < 1500 g BW was used 28(23.7%) of babies with ROP would have been missed.

Table 8:

POG	No ROP (69)	Any ROP (n=49)
	No	**No**
• ≤30 weeks	4	11
• >30 weeks	65	38

If <30 weeks POG was used as the criteria then 38 (32.2%) babies with ROP would have been missed.

Table 9:

POG	No ROP	Any ROP
	No	**No**
• ≤32 weeks	17	31
• >32 weeks	52	18

If the criteria <32 weeks POG was followed for screening then 18(15.2%) babies with ROP would have been missed.

If we had the combined the criteria of BW <1500g and ≤30wks POG, we would have missed 2(4.08%) babies with ROP who required treatment. (US Guidelines)

If we had the combined the criteria of BW <1500g and ≤32wks POG, we would have missed 1(2.04%) baby with ROP who required treatment as (UK Guidelines) depicted in the table below.

Table 10:

Criteria	***with ROP***	***Treatable ROP***
	NO.	***NO.***
US guidelines		
≤1500g BW & ≤ 30 weeks POG	*14*	*10*
>1500 g BW & >30 weeks POG	*23*	*2*
UK guidelines		
≤1500g BW & ≤ 32 weeks POG	*23*	*11*
>1500 g BW & >32 weeks POG	*14*	*1*

Fig 5: +20 D lens, infant lid speculum and sclerel depressor

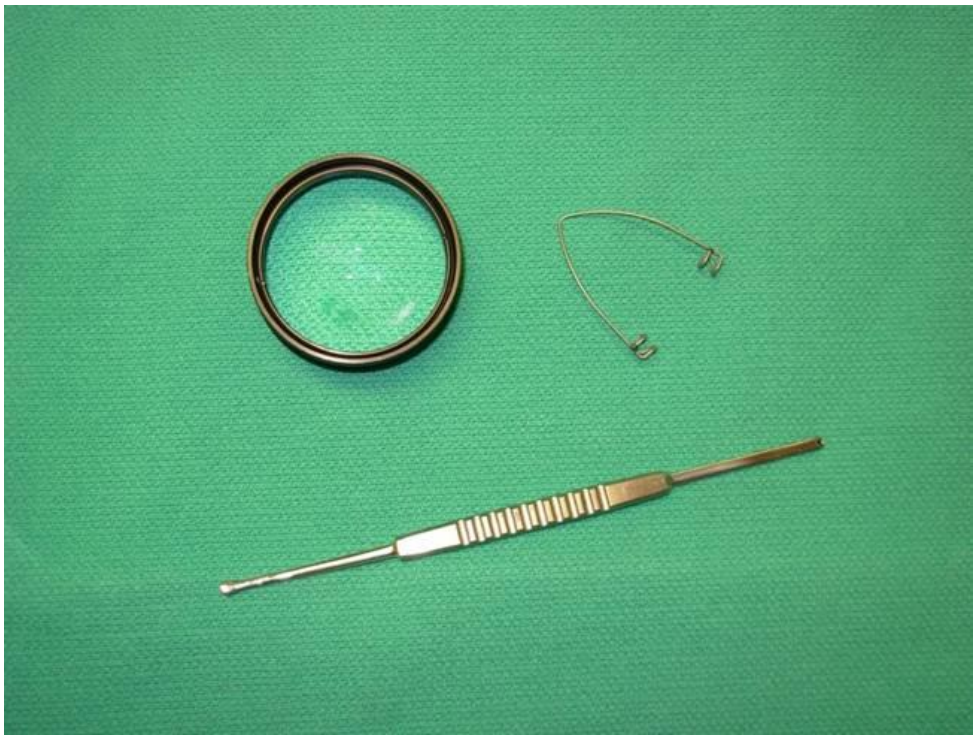


Fig 6: screening of the infant

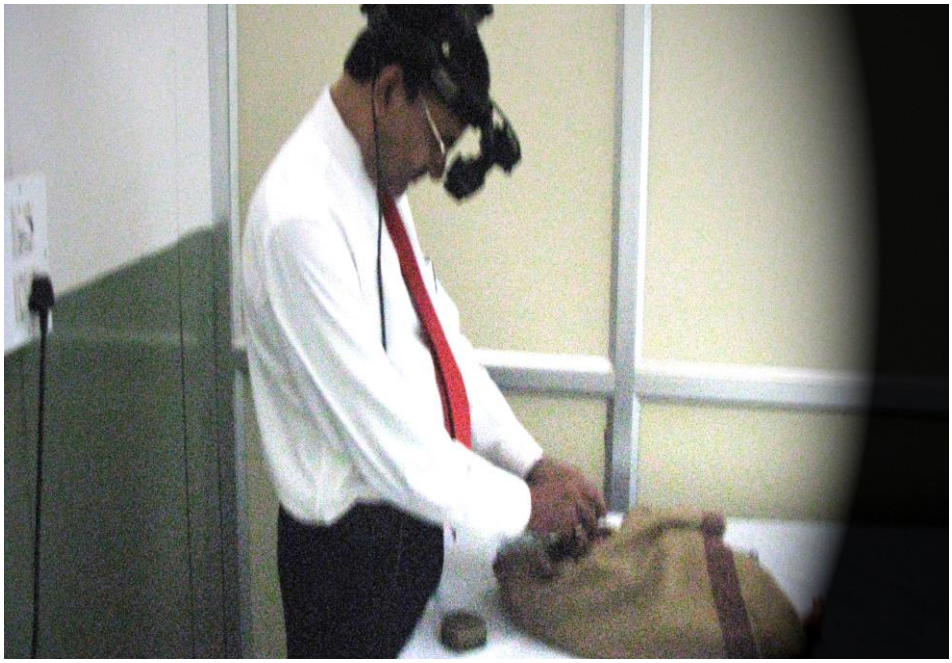


Fig7: stage 1 ROP in right eye

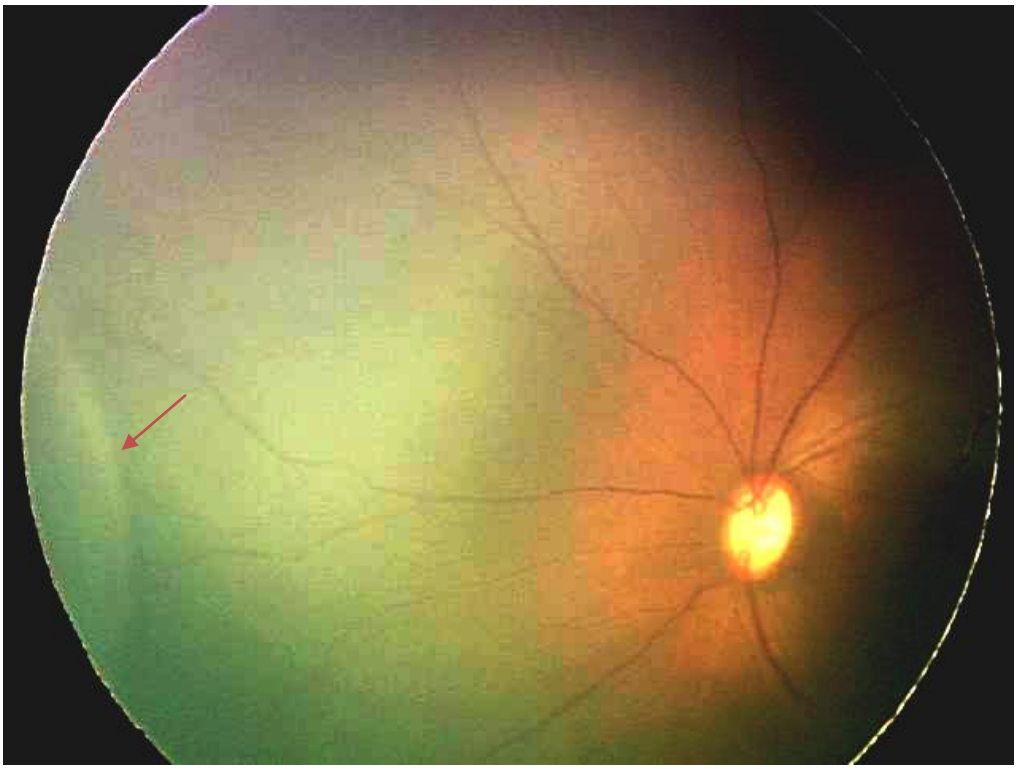


Fig 8: stage 2 ROP in left eye

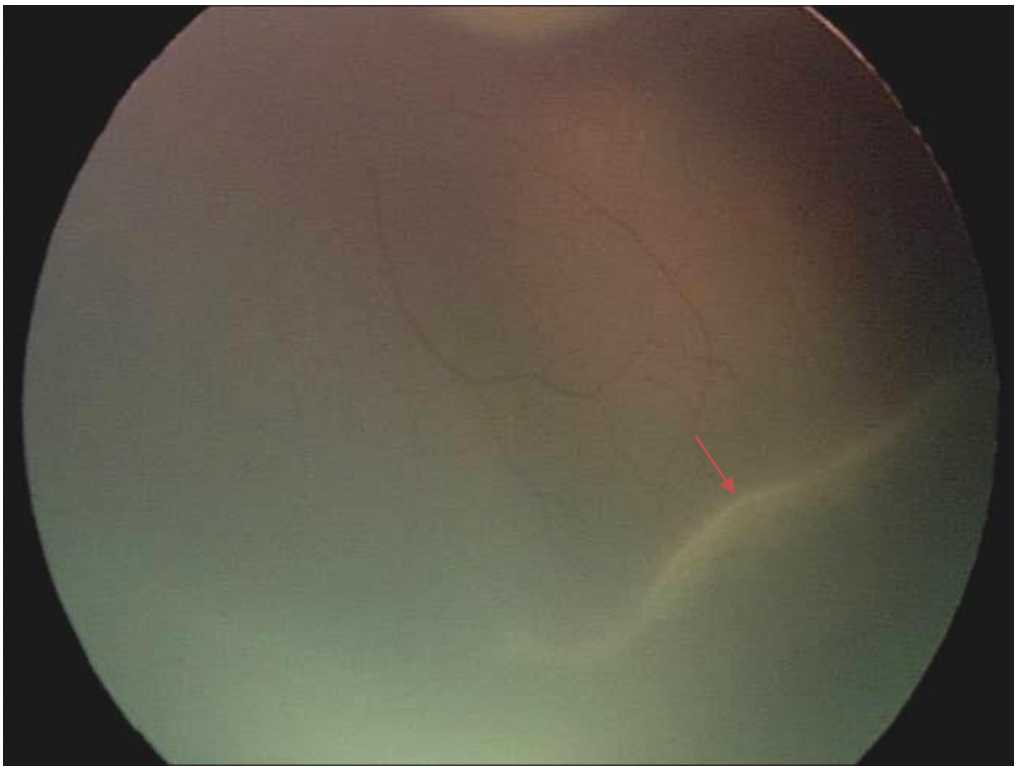


Fig 9: stage 3 in left eye

Fig 10: APROP in zone 1 right eye

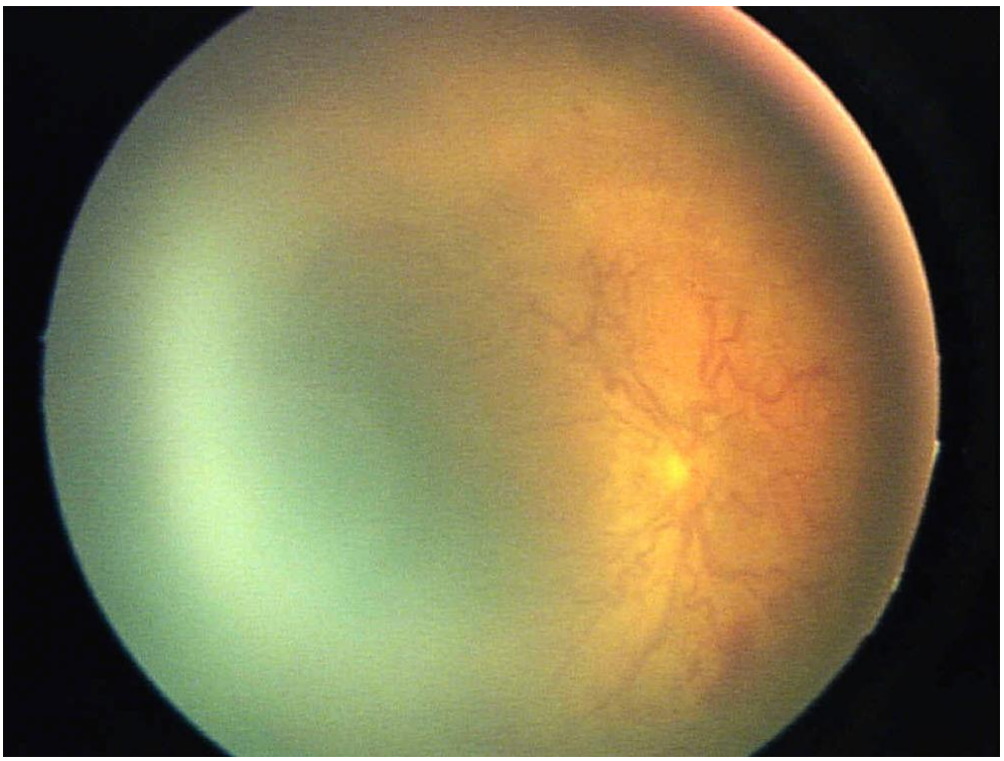


Fig 11: Laser treated

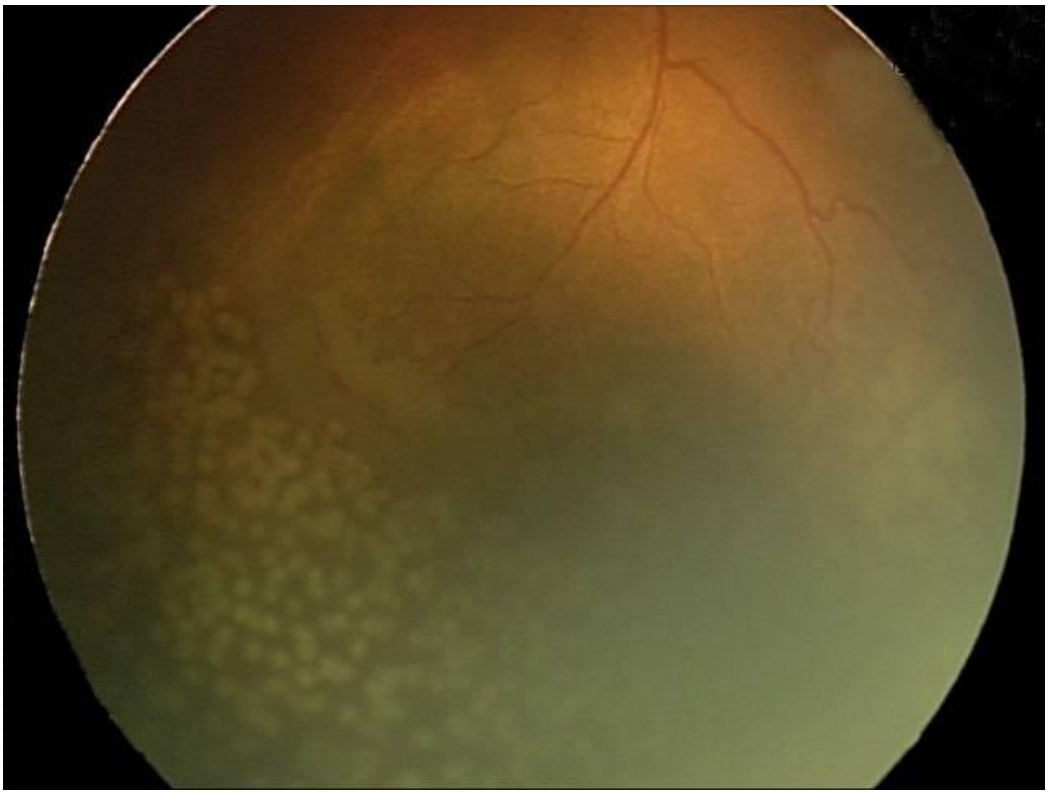
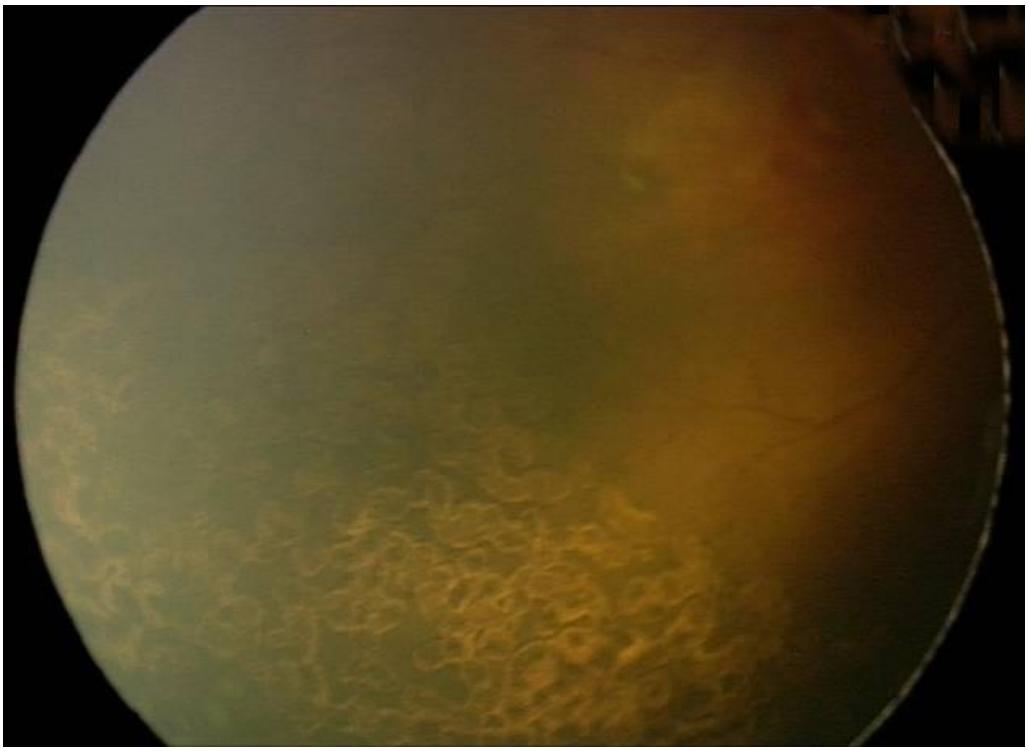


Fig 12: Supplement laser treated



DISCUSSION

Our NICU is a level III NICU in a rural area.

Even though there is no special criterion for admission of pregnant women (with or without risk factors) for delivery, most of them are "high risks" (around 60%-70%). The commonest systemic diagnoses of mothers admitted in our labour room are PIH and Anemia.

We screened 157 babies in 18months period between 1st December 2008 and 31st May 2010. Of these 157 babies, we selected 118 babies who had complete follow up. Of others some of them did not follow up until the retina was completely vascularized, some of them were discharged against medical advice and few died. The mean number of screening visits for the infants, whom we selected in our study was 4.30± 2.95 visits (Mean ± SD) (range: 1 to 14) and the median was 4.

Our study, the first rural prospective cohort study with the screening criteria of ≤2000g at birth, reports the incidence of any stage of ROP as 38.6% which corresponds to the previously published incidences in urban area that is shown in the table no. 11

Table 11: INCIDENCES OF ROP

Study	Year	Incidence	BW Inclusion criteria(g)
Charan et al [17]	1995	47.27%	≤1700
Gopal et al[18]	1995	38%	<2000
Rekha.S et al [19]	1996	46%	<1500
Maheshwari et al [20]	1996	20%	<1500
Varughese et al [21]	2001	51.89%	<1500
Chaudhari S[61]	2008	22.3%	<1500g
PRESENT STUDY	2010	38.6%	≤2000

We screened all the babies <2000g and/or <34weeks and found the incidence of ROP in118 babies which is 38.6%.

In 1995, a first single largest prospective study in India by Charan et al[17] reported the incidence of ROP to be 47.27% which is more when compared to our study.

In the same year, another prospective study by Gopal et al[18]revealed the incidence of ROP to be 38%, similar to our study.

A prospective cohort study conducted by Rekha S et al[19] in the year 1996, revealed the incidence as 46%, higher incidence when compared to the present study.

Maheshwari et al[20] in the same year reported the incidence to be 20% that is lesser than that of our study.

In 2001, Varughese et al[21] reported the incidence of ROP as 51.89%, which is much higher when we compare it to our study.

Our study included the babies with BW < or = 2000g and POG < or = 34 weeks and mean POG and BW was 33.65weeks (± 2.83) and 1627.78g (± 308.21) respectively that is more when compared to other studies with respect to both mean POG and mean BW, as we included even the "heavier babies" upto 2000g.

Comparison of Period of gestation with other studies

Table 12: COMPARISON OF MEAN POG AND MEAN BW OF OUR STUDY WITH OTHER STUDIES:

Study	Mean POG (weeks)	Mean BW (grams)
Charan et al [17]	32.47±0.2	1382± 17.8
Gopal et al [18]	32.4	1477.6
Vinekar et al[33]	30.9± 1.8	1533.9± 286
PRESENT STUDY	33.65weeks (± 2.83)	1627.78g (± 308.21)

In the study by Charan et al[17], the overall mean POG was 32.47±0.2 weeks. There was no cut off criterion for POG for screening in the study but birth weight cut-off was ≤ 1700g and the mean BW was found to be 1382± 17.8g. In the study by Gopal et al[18], which included all the premature babies with BW <2000g, reported the mean POG and mean BW to be 32.4weeks and 1477.6g respectively. In another study by Vinekar et al[34], that included all the premature babies with BW >1250g who had ROP, reveals the mean POG and BW to be 30.9± 1.8 and 1533.9± 286 respectively.

Aggressive posterior retinopathy of prematurity (APROP) is a severe form of zone 1 ROP, has been reported to be an important part of ROP in Indian studies [28, 30]. Incidence of APROP in our study was 13.1%. The mean BW of the babies with APROP was found to be 1448g, ranged from 1300g to 1640g and mean POG was 31.16 weeks ranged from 28wks to 32wks. All our babies with APROP had a BW >1250g. All eyes had zone I involvement. All eyes underwent confluent laser ablation and all (100%) had favourable outcome.

A study[28] from a large tertiary care center in North India between September 2005 and March 2007 revealed the Mean BW and gestational age to be 1,259.66 +/- 310.51 g (range, 660-2,000 g) and 29.75 +/- 2.35 weeks (range, 26-36 weeks), respectively. Twenty-one infants (47.72%) had a birth weight > 1,250 g. Thirty-three (40.74%) eyes had Zone I, and 48 (59.26%) had posterior Zone II disease. Mean follow-up was 12.8 months (range, 6-24 months). At the last follow-up visit, 55 (71.4%) of 77 eyes had a favorable outcome.

In another study by Shah PK[30], the incidence of APROP was found to be 66.67%. Mean gestational age was 31.75 weeks (range 28-34 weeks) and the mean birth weight was 1554 g (range 850-2290 g). It was comparable with our studies.

Table 13: COMPARISON OF MEAN BW AND MEAN POG IN APROP GROUP OF OUR STUDY WITH OTHER STUDIES

Study	Mean BW	Mean POG
Shah P K [30]	1554g	31.75weeks
Sanghi G [28]	1,259.66g(±310.51)g	29.75(±2.35)wks
PRESENT STUDY	1448g	31.16 weeks

In our study stage I disease was seen in 26 eyes (28.57%), stage2 was seen in 51eyes (56%) and stage3 was seen in 2 eyes (2.1%).

Table 14: STAGE DISTRIBUTION OF ROP COMPARED WITH OTHER STUDIES

	Stage I	Stage II	Stage III
Charan R [17]	28(16.97%)	29(17.58%)	19(12.1%)
Rekha S [19]	21	14	8
Vinekar A [33]	30(19.9%)	101(66.9%)	20(13.2%)
PRESENT STUDY	26 (28.57%)	51eyes (56%)	2 eyes (2.1%).

Outcome following treatment using 532nm green laser compared to other studies:

The outcome following laser treatment is favourable following adoption of ETROP guidelines as shown by some studies. We had 100% favourable outcome and we followed the ETROP guidelines. In the study by Lira et al[65] 31 eyes with threshold disease were treated using 532nm green laser and favourable outcome was 96.7%. Another study by Sanghi G [65] treated 100 eyes with threshold and high risk prethreshold, had a favourable outcome of 97%. They have included the eyes with tunica vasculosa lentis, preretinal hemorrhage. But we did not have any babies with

TVL or preretinal hemorrhage. A study by Alme AM et al[68] shows that after the ETROP guidelines were implemented, there was a decrease from 10.3% to 1.9% of eyes developing Stage 5 retinal detachment, despite post-ETROP group having a lower average GA and lower average birth weight.

Table 15: OUTCOME FOLLOWING LASER TREATMENT

STUDY	Type of ROP	Number of eyes treated	Favourable outcome
Lira et al [65]	Threshold	31	96.7%
Sanghi G [65]	Threshold and high risk prethreshold	100	97%
PRESENT STUDY	Threshold and high -risk prethreshold	24	100%

Near-confluent to Confluent laser scars were produced in our babies who required treatment. In the study by Gonzalez VH et al [29] revealed that progression to stage 4 or 5 occurred only in a total of 6 eyes (6%) who received confluent laser that is a low rate of progression to stage 4 or 5 retinopathy of prematurity. Also, the need for additional laser treatment was small, with rates of complications and structural outcomes comparable to previous reports using a nonconfluent laser pattern. Another study by Fallah N [27] reported that confluent laser treatment almost eliminated the need for supplemental laser treatment. In the study by Banach et al [27] showed that rate of progression to RD was less in near confluent laser burns when compared to scatter pattern of laser burns((3.6% of eyes vs 29.4%)

We analyzed the cohort based on the birth weight and period of gestation with respect to screening guidelines prevalent in developed countries.
If the criteria of < 1500 g BW was used 28(23.7%) of babies with ROP would have been missed. (Table No 7)

If <30 weeks POG was used as the criteria then 38 (32.2%) babies with ROP would have been missed. (Table No 8)

If the criteria <32 weeks POG was followed for screening then 18(15.2%) babies with ROP would have been missed. (Table No 9)

The screening criteria of ROP according to UK guidelines is <31weeks and 6days POG and/or <1501g BW. If we had followed the UK guidelines for screening there were chances of missing 1 baby (2.04%) with ROP who required treatment. (Table No 10)

If we had the combined the criteria of BW <1500g and ≤32wks POG, we would have missed 1(2.04%) baby with ROP who required treatment as depicted in the table 10 .

RISK FACTORS

We tend to look for risk factors in our babies as previously done in urban ROP subset and we found no significant risk factor. We compared the incidence of all risk factors between the group that had ROP and the group that did not have ROP and we found that none of the risk factors reached statistical significance. However, clinically we noticed a trend of few risk factors being present in higher proportion in babies with ROP when compared to babies who did not have the disease. These were RDS (26.5% vs 14.6%), Oxygen (18.4% vs 8.7%), NNJ (32.7% vs 23.2%) and Sepsis (22.4% vs 15.9%) respectively. The above mentioned 4 risk factors may be clinically significant. This was compared with other studies.

Table 16:

STUDY	YEAR	RISK FACTORS	URBAN /RURAL
Rekha S[19]	1996	Anemia, Blood Transfusions, Apnea, Duration of exposure To Oxygen	Urban
Maheshwari R[20]	1996	Blood transfusion, Sepsis	Urban
Dutta S [66]	2004	Packed cell transfusions, DVET, Ventilation, Apnea	Urban
Gupta VP [67]	2004	Oxygen, Apnea, Sepsis	Urban
Vinekar A[33]	2007	Sepsis, exchange transfusion, Outborn, RDS	Urban
Chaudhari S [61]	2009	Septicemia, apnea, oxygen, use of blood products	Urban
PRESENT STUDY	2010	No statistically significant risk factor. Clinically significant- RDS, Oxygen, NNJ, Sepsis	Rural

A South Indian study, by Rekha et al [19], reported the risk factors of ROP in 1996, found Anemia, Blood transfusions, Apnea and Exposure to oxygen significantly increased the risk of developing ROP and also found anemia and duration of oxygen therapy to be independent risk factors on multivariate analysis. Oxygen administration is the risk factor that is common with our study.

A North Indian study,by Maheshwari et al [20], reported in the same year found blood transfusion and clinical sepsis to be independent risk factors. Our results regarding risk factors are similar to these reports from India. Sepsis was a common risk factor in our study when compared.

Another study by Dutta S et al [66] reported that Packed cell transfusions, DVET, Ventilation, Apnea to be most common risk factors for developing threshold ROP. On multivariate analysis found packed cell transfusion, DVET as the independent risk factors.

In the same year Gupta VP [67], reported Oxygen, Sepsis and Apnea to be independent risk factors for ROP. When compared to this our study had Oxygen, Sepsis in common.

In 2007, a study by Vinekar A[33] found Sepsis, Exchange Transfusion, Outborn and RDS to be significant risk factors for developing severe ROP. And Outborn, RDS and Exchange Transfusion were the independent risk factors. Our study had Sepsis, RDS and Oxygen as the common risk factors. However none of the risk factors were statistically significant in our study.

Digital ROP Imaging / WFDI (wide field digital imaging):

Studies have already shown that the RetCam screening programme can detect all cases of 'ROP requiring treatment'. In addition, digital imaging requires significantly less time as compared to BIO. A study in 2006 by Shah et al [63], shows that Retcam may replace Binocular indirect ophthalmoscopy (BIO) for screening of ROP as the positive and negative predictive values were 96.43% and 70.97% respectively, sensitivity of RetCam was 85.71% (54/63) and specificity was 91.66% (22/24) when Retcam was compared with BIO.

In another study [64] it was shown that sensitivity of WFDI (wide field digital imaging)

in detecting any ROP, stage 3 ROP and plus disease to be 60%, 57% and 80%, respectively, with a specificity of 91%, 98% and 98%, respectively. There was excellent proportional agreement between the two screening methods for detecting stage 3 ROP and plus disease (0.96 and 0.97) and very good agreement on management decisions. Some studies have suggested that the store-and-forward protocol of telemedicine in ROP management provides a quality tool, which is accessible and cost-effective.

We used WFDI (wide field digital imaging) for all our babies using a Retcam shuttle to document case at each visit in addition to BIO.

We found certain advantages of Retcam imaging:

1. It was instant & accurate documentation, avoiding time-consuming retinal drawings and images could be stored permanently.
2. It served as a good teaching tool for educating the parents and also trainees.
3. Can track image sessions for side-by-side comparison of sessions, helping in better monitoring of the disease progression, with or without laser treatment.
4. It is Cost-effective.

We found we were able to screen all 100% of our babies with WFDI.

SUMMARY

- Total of 157 infants were screened and registered in the ROP clinic, Department of Ophthalmology and department of Paediatrics, R.L.Jalappa Hospital, Tamaka, Kolar between 1st December 2008 and 31st May 2010. This is a level III NICU in a rural set up.118 babies (236 eyes) were included for the analysis.
- The mean number of screening visits for the infants was 4.30± 2.95 visits (Mean ± SD) (range: 1 to 14) and the median was 4.
- Our study, the first rural prospective cohort study reports the incidence of any stage of ROP as 38.6% which corresponds to the previously published incidences in urban area. We screened all the babies with BW ≤2000g and/or POG ≤34weeks.
- The mean birth weight was significantly lower in babies with ROP compared to those without ROP (1555.91g vs 1672.50g) with t=2.851; P=0.005.
- The mean period of gestation was significantly lower in babies with ROP compared to those without ROP (32.23wks vs 34.58wks) with t=6.728 ;(p value <0.001).
- Of the 91 eyes, who had some ROP 79 eyes (86.8%) had classical ROP and 12 eyes (13.1%) had APROP.
- ROP stage1 was seen in 26 eyes (28.57%), stage2 was seen in 51eyes (56%) and stage3 was seen in 2 eyes (2.1%).
- Of the 91 eyes with ROP, Zone I disease was observed in 14 (15.3%) eyes, Zone II in 29 (31.8%) eyes and Zone III in 48 (52.74%) eyes. 2 eyes of classical ROP and 12 eyes (100%) of APROP had zone I disease.
- 28(23.7%) babies with BW >1500 g had ROP. 38 (32.2%) babies with ROP were >30 weeks of POG and 18(15.2%) babies with ROP were >32 weeks of

POG.

- 4.08% of our babies with treatable ROP were >1500g BW and >30weeks POG.
- 2.04% of our babies with treatable ROP were >1500g BW and >32weeks POG.
- We found we were able to screen all 100% of our babies with WFDI.
- Common risk factors found in our study were: Neonatal jaundice (27.11%), Twins (24.5%), Respiratory distress syndrome (RDS) (19.49%), Sepsis (18.6%), Pregnancy induced hypertension (PIH) (18.6%). However, clinically we noticed a trend of 4 risk factors i.e. RDS, Oxygen therapy, NNJ and Sepsis being present in higher proportion in babies with ROP when compared to babies who did not have the disease.
- We treated 12 babies and had 100% favourable outcome indicating that treatment outcome is very good if ETROP is followed.

CONCLUSION

Our prospective study in a level III NICU in a rural hospital reveals that timely and appropriate screening and treatment of Retinopathy of Prematurity results in excellent outcomes of babies with severe ROP. The Western screening guidelines are challenged by our results as we had 23.7% of infants with ROP whose birth weight was >1500g, 32.2% and 15.2% of infants with ROP with POG >30 and >32 weeks respectively and show that it may not be applicable in rural neonatal care centres of our country.

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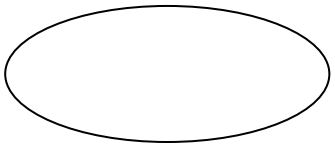
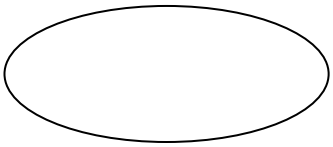
PROFORMA

Name: B/O MRD no.: Sl.no:
DOB: BW: POG:
Sex: Birth Order: Due Date:
Address: Phone no.: Date of First Exam:

OD	0S
 Mature Immature 360^0 ROP 1 2 3 4A 4B 5 No Plus Pre Plus Plus Zone 1 2 3 APROP	 Mature Immature 360^0 ROP 1 2 3 4A 4B 5 No Plus Pre Plus Plus Zone 1 2 3 APROP

RISK FACTORS:

Apnea	anemia	Blood transfusion	HMD	hydrocephalous	hypoglycemia
IVH	Meconium peritonitis	meningitis	metabolic	NEC	NNJ
Oxygen	Polycythemia	PDA	Pneumonia	PT	RDS
Sepsis	shock	surfactant	ventilation	Exchange transfusion	CHD

FOLLOW UP RECORD:

Sl.no.	Date	PCA	Right eye	Left eye	Comments	Next visit

Treatment details:
OD: Details
OS: Notes

Post treatment record:

Date	Age	Visual acuity	Refraction	Squint	Retinal sequelae	Comments
	OD OS					
	OD OS					
	OD OS					

CONSENT FORM FOR ROP LASER

B/O: **DOB:** **Hosp No.**

POG & PCA: **DIAGNOSIS:**

It has been explained in a language understood by me the diagnosis (Retinopathy of Prematurity, ROP) and need for laser photoablation for baby's eyes**.**

I understand that the laser is primarily being done to ablate the avascular retina and to slow or halt the progression of the disease (ROP) into advanced stages including retinal detachment and not primarily for visual gain.

The chances of possible 'unfavorable outcome', supplement laser, re-treatment, cryo supplementation have been explained. The fact that 'unfavorable outcome' may result despite adequate and appropriate laser treatment has been explained.

The intra-operative risks including hypoxia, de-saturation, apnea and possible need for resuscitation and ventilator care have been explained.

The increased incidence (as a consequence primarily of the disease) of refractive errors (20%), strabismus (20 - 30%), cataract (7-15%), glaucoma (>5%) and amblyopia in the long term follow-up of the child have been explained.

The need for long term follow-up for the above reasons has been explained, as long term visual rehabilitation is required in this condition whether treated or not.

We give this consent under no compulsion or coercion but with our own free will having understood and reasonably clarified our doubts.

x _____ x _____

Signature of patient's attender Doctor's Signature

Name

LIST OF ABBREVIATIONS USED

ROP- Retinopathy of Prematurity

pO_2 - partial pressure of oxygen

VEGF- Vascular Endothelial Growth Factor

APROP- Aggressive Posterior Retinopathy of Prematurity

ETROP- Early Treatment Retinopathy of Prematurity

G or g- Grams

BW- Birth Weight

POG- Period of Gestation

GA- Gestational age

PCA- Post Conceptual Age

Wks- weeks

SVI- Severe Visual Impairment

Hct- Hematocrit

BPD- Bronchopulmonary Dysplasia

FEVR- Familial Exudative Vitreoretinopathy

BIO- Binocular Indirect Ophthalmoscope

NICU- Neonatal Care Unit

FFA- Fundus fluorescein Angiography

AAP- American Academy of Pediatrics

NNJ- Neonatal Jaundice

MSAF- Meconium Stained Amniotic Fluid

PIH- Pregnancy Induced Hypertension

RDS- Respiratory Distress Syndrome

LSCS- Lower Segment Caesarian Section

WFDI- Wide Field Digital Imaging

CHD- congenital heart disease

HMD- Hyaline Membrane disease

PDA- Patent Ductus Arteriosus

PT- Preterm

NEC- Necrotizing Enterocolitis

DVET- Double Volume Exchange Transfusion

ICROP- International Classification of Retinopathy of Prematurity

ETROP- Early Treatment Retinopathy of Prematurity

nm - nanometer

KEY TO MASTER CHART

Sl.No – Serial Number

POG – in weeks

BW – in grams

PCA – in weeks

RE – right eye stage of ROP

LE – left eye stage of ROP

In RE and LE – 1: stage 1

2: stage 2

3: stage 3

6: APROP

Zone – 1- zone I

2- Zone II

3- zone III

Rx RE – Right eye laser treated

Rx LE – Left eye laser treated

MASTER CHART

														RISK FACTORS																												
SI.NO	**NAME**	**HOSP NO**	**SEX**	**POG**	**BW**	**RE**	**zone**	**preplus**	**plus**	**LE**	**zone**	**preplus**	**plus**	**Oxygen**	**sepsis**	**anamia**	**polycythemia**	**thrombocytopenia**	**DIC**	**NNJ**	**MSAF**	**RDS**	**B asphyxia**	**pnemoniua**	**hypoglycemia**	**blood trans**	**PIH**	**LSCS**	**Hydrocephalous**	**meningitis**	**plasma transfusion**	**ascites**	**twins**	**no. of visits**	**Rx R**	**Rx L**	**supplemant laser(RE)**	**supplemant laser(LE)**	**no. of laser spots(RE)**	**no. of laser spots(LE)**	**supplemant laser(RE)**	**supplemant laser(LE)**
1	Mukthamba	467446	F	34	1600	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	2	-	-	-	-	0	0	0	0
2	Munirathna	467902	M	29	1560	1	3	-	-	1	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	7	-	-	-	-	0	0	0	0
3	Shashikala C	468659	M	31	1700	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	3	-	-	-	-	0	0	0	0
4	Shashikala 1	468980	M	30	1400	2	2	-	-	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	12	+	+	-	-	954	1238	0	0
5	Shashikala 2	468981	F	30	940	2	2	-	-	2	2	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	14	+	+	-	-	610	594	0	0
6	Anjum taj	468382	M	32	2070	1	2	-	-	1	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	2	-	-	-	-	0	0	0	0
7	Roopa G	470952	M	37	1300	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2	-	-	-	-	0	0	0	0
8	Pramila 1	470391	M	28	1300	6	1	-	+	6	1	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	4	+	+	-	-	3268	3674	0	0
9	Radha 2	473399	F	32	1350	2	2	-	-	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	+	13	+	+	-	-	1217	922	0	0
10	Renukamma	475107	F	38	1060	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	1	-	-	-	-	0	0	0	0
11	Rajamma G	470915	M	37	1890	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2	-	-	-	-	0	0	0	0
12	Geetha R	475907	M	33	2000	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	4	-	-	-	-	0	0	0	0
13	Gayathri	476472	F	37	1750	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	3	-	-	-	-	0	0	0	0
14	Nagaveni C	476454	M	33	1600	1	3	+	-	1	3	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	5	-	-	-	-	0	0	0	0
15	Saraswathi R	476672	F	37	2000	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	3	-	-	-	-	0	0	0	0
16	Suvarna R	476196	M	29	1500	2	2	-		2	2	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	7	+	+	-	-	2673	3016	0	0
17	Rajamma 1	473416	M	33	1750	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	1	-	-	-	-	0	0	0	0
18	Savitha S	477420	M	34	1700	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	1	-	-	-	-	0	0	0	0
19	Suma R	475291	F	35	1500	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	3	-	-	-	-	0	0	0	0
20	Usha S 2	478934	M	33	1710	2	3	-	-	1	3	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	+	6	-	-	-	-	0	0	0	0
21	Usha S 1	478933	F	33	1570	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	4	-	-	-	-	0	0	0	0
22	Saraswathi S	478621	F	30	1580	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	+	+	-	-	-	-	-	4	-	-	-	-	0	0	0	0
23	Nagarathna	478128	M	32	1630	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	0	0	0	0
24	Vidya rani	479887	M	36	1330	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	+	-	-	-	-	-	-	2	-	-	-	-	0	0	0	0
25	Hemalatha	480956	M	32	1640	6	1	-	+	6	1	-	+	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	7	+	+	+	+	4177	4594	386	89
26	Meena	481749	M	32	1290	2	3	-	-	2	3	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	7	-	-	-	-	0	0	0	0
27	Lavanya	480181	M	29	1480	1	3	-	-	1	3	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	8	-	-	-	-	0	0	0	0
28	Sunandamma	482773	M	34	1250	-	-	-	-	1	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	3	-	-	-	-	0	0	0	0
29	Susheelamma	486711	M	37	2000	2	2	-	-	2	2	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-	5	-	-	-	-	0	0	0	0
30	Prathima	486700	F	37	1500	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	2	-	-	-	-	0	0	0	0
31	Suvarna 1	491036	M	29	1500	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	2	-	-	-	-	0	0	0	0
32	Suvarna 2	491037	M	29	1000	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	2	-	-	-	-	0	0	0	0
33	Nagin taj	491808	M	34	1500	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	+	+	-	-	-	+	-	-	-	-	-	-	1	-	-	-	-	0	0	0	0
34	Prabhavathi M	491587	F	38	1600	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-	-	4	-	-	-	-	0	0	0	0

35	Bharathi	498601	F	37	1600	1	3	-	-	1	3	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	-	-	-	-	-	-	4	-	-	-	-	0	0	0	0
36	Shabina S 1	423576	F	32	1100	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	+	4	-	-	-	-	0	0	0	0
37	Shabina S 2	423577	F	32	1300	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	4	-	-	-	-	0	0	0	0
38	Shamalamma	502437	F	36	1600	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	4	-	-	-	-	0	0	0	0
39	Sujatha S	214122	M	36	1054	1	3	+	-	1	3	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	6	-	-	-	-	0	0	0	0
40	Manjamma	503048	F	28	1300	2	2	-	+	2	2	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-	-	10	+	+	+	+	140	841	87	61
41	Manjula K	502214	M	29	1400	1	3	-	-	1	3	-	-	+	+	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	4	-	-	-	-	0	0	0	0
42	Shakunthala N	503085	M	29	1100	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	7	-	-	-	-	0	0	0	0
43	Somamma	499106	M	40	2009	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	0	0	0	0
44	Poornima S	506196	F	33	1900	2	3	-	-	2	3	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	6	-	-	-	-	0	0	0	0
45	Shanthamma 1	504810	M	33	1730	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	1	-	-	-	-	0	0	0	0
46	Gayathri .N	506519	F	32	1390	2	2	+	-	2	2	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-	-	9	-	-	-	-	0	0	0	0
47	Manjula R	506523	M	34	1750	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	+	-	-	-	-	-	-	1	-	-	-	-	0	0	0	0
48	Rajashree	507652	M	32	1300	6	1	-	+	6	1	-	+	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	11	+	+	+	+	4106	4236	130	190
49	Chaitra 2	509115	F	31	1600	-	-	-	-	1	3	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	5	-	-	-	-	0	0	0	0
50	Chaitra 1	509114	F	31	1600	2	2	-	-	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	7	-	-	-	-	0	0	0	0
51	Mamatha U	508300	F	31	1350	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	5	-	-	-	-	0	0	0	0
52	Mamathaz 1	510615	M	32	1689	2	3	-	-	2	3	-	-	-	+	-	-	+	-	+	-	+	-	-	-	-	-	-	-	-	-	-	+	5	-	-	-	-	0	0	0	0
53	Mamathaz 2	510616	M	32	1500	2	3	-	-	2	3	-	-	-	+	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	5	-	-	-	-	0	0	0	0
54	Amreen	513513	F	36	1670	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	5	-	-	-	-	0	0	0	0
55	Rachamma	517530	M	34	2300	-	-	-	-	1	3	-	-	-	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	6	-	-	-	-	0	0	0	0
56	Rathnamma	519143	F	34	1810	2	3	-	-	2	3	-	-	+	-	+	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	8	-	-	-	-	0	0	0	0
57	Radha S	522604	M	32	1800	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	5	-	-	-	-	0	0	0	0
58	Divya	522093	F	34	1690	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	0	-	-	-	-	0	0	0	0
59	Anasuya	522095	M	36	1740	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	3	-	-	-	-	0	0	0	0
60	Premkumari 2	523304	M	35	1540	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	+	+	-	-	-	-	+	4	-	-	-	-	0	0	0	0
61	Premkumari 1	523303	F	35	1670	-	-	-	-	2	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-	+	4	-	-	-	-	0	0	0	0
62	Pramila	520333	F	30	1900	2	3	-	-	2	3	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	6	-	-	-	-	0	0	0	0
63	Surekha S	520827	M	40	2300	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	+	-	-	-	-	-	1	-	-	-	-	0	0	0	0
64	Anitha 2	528263	F	32	1200	2	2	+	-	2	2	+	-	-	-	-	-	-	-	-	-	-	+	-	-	-	+	+	-	-	-	-	+	11	-	-	-	-	0	0	0	0
65	Anitha 1	528262	F	32	1570	1	3	-	-	1	3	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	+	+	-	-	-	-	+	9	-	-	-	-	0	0	0	0
66	Prathima 1	511163	M	37	1670	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	1	-	-	-	-	0	0	0	0
67	Bhagyalakshmi	512121	F	33	1200	2	2	-	-	2	2	-	-	+	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	10	-	-	-	-	0	0	0	0
68	Kasturi	530432	M	36	1380	-	-	-	-	-	-	-	-	-	+	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	4	-	-	-	-	0	0	0	0
69	Varalakshmi	530419	M	32	1600	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	4	-	-	-	-	0	0	0	0
70	Tasmeen taj	532486	M	36	1600	1	2	-	-	1	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	6	-	-	-	-	0	0	0	0
71	Venkatalakshmi	535915	M	37	2700	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	0	0	0	0
72	Naseeema banu	539500	F	32	1800	2	3	-	-	2	3	-	-	-	+	-	-	+	-	+	-	-	+	-	-	-	-	-	-	-	-	-	-	9	-	-	-	-	0	0	0	0
73	Kalpana 1	231717	F	32	1730	2	2	-	-	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	8	-	-	-	-	0	0	0	0
74	Kalpana 2	231718	F	32	1790	2	2	-	-	2	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	8	-	-	-	-	0	0		0
75	Renuka A	554468	F	32	1750	-	-	-	-	1	3	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	6	-	-	-	-	0	0	0	0
76	Sujathamma	554906	M	32	1400	3	1	-	-	3	1	-	-	+	-	-	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	4	+	+	-	-	2946	2899	0	0
77	Shanthamma	558266	M	35	1600	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	0	0	0	0
78	Revathi	557096	F	34	2000	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	+	-	-	-	-	-	1	-	-	-	-	0	0		0
79	Sudha M	558259	F	39	1500	-	-	-	-	-	-	-	-	+	-	-	-	+	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	0	0	0	0
80	Devubai	562141	M	34	1860	2	3	+	-	2	3	+	-	-	+	-	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	10	-	-	-	-	0	0	0	0

81	Sujatha K	564320	F	37	1260	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	0	0	0	0
82	Sandhya Rani	562136	M	30	1820	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	+	+	-	-	-	-	+	-	-	-	-	7	-	-	-	-	0	0	0	0
83	Shruthi	562748	M	37	2000	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	2	-	-	-	-	0	0	0	0
84	Geetha A	565690	M	34	2000	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	2	-	-	-	-	0	0	0	0
85	Venkatrathnamma	571224	M	32	1700	1	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	4	-	-	-	-	0	0	0	0
86	Nagarathna N	570908	F	39	2000	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	-	-	-	-	-	4	-	-	-	-	0	0	0	0
87	Shilpa	571135	M	36	2000	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-	1	-	-	-	-	0	0	0	0
88	Aruna	571180	M	33	1600	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	2	-	-	-	-	0	0	0	0
89	Munirathna L	571919	F	39	1660	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	0	0	0	0
90	Sabeera	570850	M	32	1500	6	1	-	+	6	1	-	+	+	+	-	-	-	-	+	-	+	-	-	-	-	-	+	-	-	-	-	-	9	+	+	+	+	3799	4217	862	545
91	Swathi	579660	F	35	2250	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	5	-	-	-	-	0	0	0	0
92	Lakshmi Devi	575482	M	34	2000	2	3	-	-	2	3	-	-	+	-	-	-	-	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	5	-	-	-	-	0	0	0	0
93	Sarasamma	580216	F	37	1250	2	3	-	-	2	3	-	-	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	5	-	-	-	-	0	0	0	0
94	Menaka 1	587562	M	35	1450	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	1	-	-	-	-	0	0	0	0
95	Menaka 2	587044	M	35	1270	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	+	-	+	-	1	-	-	-	-	0	0	0	0
96	Arifa	586444	F	34	2000	-	-	-	-	-	-	-	-	-	+	-	-	+	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	0	0	0	0
97	Narmada	587562	M	33	2200	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	4	-	-	-	-	0	0	0	0
98	Pushpa G	588845	M	33	1400	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	2	-	-	-	-	0	0	0	0
99	Kavitha S 2	591103	M	32	1400	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	5	-	-	-	-	0	0	0	0
100	Veena M	590044	F	39	1700	-	-	-	-	1	3	-	-	-	+	-	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	4	-	-	-	-	0	0	0	0
101	Shammen Taj	576301	M	29	1200	2	3	-	-	1	3	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	6	-	-	-	-	0	0	0	0
102	Parvathamma	594710	F	39	2200	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	1	-	-	-	-	0	0	0	0
103	Uma	594702	M	33	1600	6	1	-	+	6	1	-	+	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	6	+	+	+	+	3497	4216	576	672
104	Manula T R	594415	M	31	1810	2	3	-	-	2	3	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	5	-	-	-	-	0	0	0	0
105	Sarmu	591568	M	34	1760	2	2	-	-	2	2	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	+	-	-	-	-	-	-	6	-	-	-	-	0	0	0	0
106	Manjula V	594610	F	35	1750	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-	-	4	-	-	-	-	0	0	0	0
107	Lakshmi Devi C	505276	M	35	1600	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	-	-	-	-	-	-	2	-	-	-	-	0	0	0	0
108	Anjum A	595289	M	35	1600	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	3	-	-	-	-	0	0	0	0
109	Rani A 2	598789	M	37	1800	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	3	-	-	-	-	0	0	0	0
110	Yasmeen Taj	596221	M	32	1500	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	2	-	-	-	-	0	0	0	0
111	Bhagyamma S	597453	F	37	2000	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	3	-	-	-	-	0	0	0	0
112	Chandrika	597448	F	34	1900	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	3	-	-	-	-	0	0	0	0
113	Bhagyavathi 1	599231	F	32	1400	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	2	-	-	-	-	0	0	0	0
114	Bhagyavathi 2	599230	F	32	1200	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	2	-	-	-	-	0	0	0	0
115	Manjula N	596529	M	30	1500	6	1	-	+	6	1	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	6	+	+	+	+	3168	5165	371	350
116	Devamma	599604	M	32	1500	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2	-	-	-	-	0	0	0	0
117	Geetha M	599597	M	36	2150	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	2	-	-	-	-	0	0	0	0
118	Radhamma	596527	F	33	1500	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	1	-	-	-	-	0	0	0	0