"A ONE YEAR CROSS SECTIONAL STUDY OF FUNDAL CHANGES IN PATIENTS WITH PREGNANCY INDUCED HYPERTENSION ATTENDING KLES' PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELGAUM"

By

DR. AYUSH SINGAL

Dissertation

SUBMITTED TO THE
KLE UNIVERSITY
BELGAUM, KARNATAKA

IN PARTIAL FULFILMENT
OF THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF SURGERY
IN
OPHTHALMOLOGY

Under the Guidance of

DR. REKHA B. K. M.S., D.O.M.S.

Professor

DEPARTMENT OF OPHTHALMOLOGY
J.N.MEDICAL COLLEGE, NEHRU NAGAR,
BELGAUM-590010.

MAY 2009

KLE UNIVERSITY BELGAUM, KARNATAKA

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled **"A ONE YEAR CROSS SECTIONAL STUDY OF FUNDAL CHANGES IN PATIENTS WITH PREGNANCY INDUCED HYPERTENSION ATTENDING KLES' PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELGAUM"** is a bonafide and genuine research work carried out by me, under the guidance of **Dr. REKHA B. K.** **M.S.,D.O.M.S.** Professor, Department of Ophthalmology, J. N. Medical College, Belgaum.

Date:

Place: Belgaum. **(Dr. AYUSH SINGAL)**

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled **"A ONE YEAR CROSS SECTIONAL STUDY OF FUNDAL CHANGES IN PATIENTS WITH PREGNANCY INDUCED HYPERTENSION ATTENDING KLES' PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELGAUM"** is a bonafide research work done by **Dr. Ayush Singal** in partial fulfilment of the requirement for the degree of MS Ophthalmology.

Date:

Place: Belgaum.

Dr. REKHA B. K. **M.S.,D.O.M.S.**

Professor

Department of Ophthalmology,

J.N. Medical College,

Belgaum – 590010.

ENDORSEMENT BY THE HOD, PRINCIPAL/HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled **"A ONE YEAR CROSS SECTIONAL STUDY OF FUNDAL CHANGES IN PATIENTS WITH PREGNANCY INDUCED HYPERTENSION ATTENDING KLES' PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELGAUM"** is a bonafide research work done by **Dr. Ayush Singal** under the guidance of **Dr. REKHA B. K.** M.S.,D.O.M.S., Professor, Department of Ophthalmology, J. N. Medical College, Belgaum.

Dr. R. K. Dandur M.S., D.O.M.S.
Professor & Head
Department of Ophthalmology,
J.N.Medical College,
Nehru Nagar,
Belgaum-590010

Date:
Place: Belgaum.

Dr.V.D.Patil MD, DCH
Principal
J.N.Medical College,
Nehru Nagar,
Belgaum-590010

Date:
Place: Belgaum.

COPYRIGHT

DECLARATION BY THE CANDIDATE

I hereby declare that the KLE University Belgaum, Karnataka shall have the rights to preserve, use and disseminate this dissertation in print or electronic format for academic / research purpose.

Date:

Place: Belgaum **Dr. AYUSH SINGAL**

ACKNOWLEDGEMENT

This dissertation work has been a learning experience during my postgraduation period. By the grace of the Almighty, it was carried out with ease and enthusiasm and for this I will always be indebted to him.

It has been my humble and blessed privilege to work under and carry out this study under guidance of **Dr. Rekha B. K.**, Professor, Department of Ophthalmology, J. N. Medical College, Belgaum. I express my sincere gratitude for her constant motivation, supervision, encouragement and support in carrying out this study and completing it successfully.

I am very thankful to **Dr. R. K. Dandur**, Professor and Head, Department of Ophthalmology, for his support during the period of my dissertation. I am very grateful to The Principal **Dr. V. D. Patil**, J. N. Medical College, Belgaum, for his support and permission to undertake this study.

I sincerely thank The Medical Director, K.L.E.S' Prabhakar Kore Hospital & MRC, Belgaum, for his valuable support and help, in permitting me to include the patients from K.L.E.S Hospital required for my study. I also thank **Dr. R. S. Mudhol**, Superintendent and co-ordinator, JNMC free ward section, K.L.E.S' Prabhakar Kore Hospital and MRC.

I owe an immense debt of gratitude to Professor **Dr. U. S. Dandavatimath,** Professor **Dr. S. B. Patil**, Professor **Dr. M.I. Magdum**, Department of Ophthalmology for their invaluable suggestions and support throughout the study. I express my gratitude to Associate Professor **Dr. Vinay Dastikop**, Associate Professor **Dr. Shilpa Kodakany**, Associate Professor **Dr. Arvind Tenagi** for their colossal support and help. I sincerely thank Assistant Professor **Dr. Arvind Yakkundi,** Assistant Professor **Dr. Umesh Harakuni**, Assistant Professor

Dr. Shivanand Bubanale, Registrars **Dr. Rohini Kolari** and **Dr. Jyoti Bali** for their immense help and constant encouragement.

I would also like to express my gratitude to faculty and postgraduates of **Department of Obstetrics and Gynaecology** for their unconditional support during the entire study period.

My deep sense of gratitude to all my **postgraduate colleagues** and **dear friends** for their relentless support in carrying out this dissertation. I am also thankful to paramedical staff **Mrs. Nagarkar** and **Mr. Pundalik**. I wish to offer my thanks to Department of Medical Education for their valuable information and support.

I would like to acknowledge the tireless and timely work done by **Miss. Veena** & **Mr. Deepak** of **Sai Xerox & D. T. P. Centre** for their excellent data processing and completion of this manuscript in a very short time.

No amount of words can measure up to the deep sense of gratitude and thankfulness that I feel towards **my parents, my sister, my loving wife Dr. Varuna and my in laws** whose cherished blessings and countless sacrifices are behind whatever success I have achieved in my life. Last but not the least, this acknowledgement is incomplete if I fail in my duty to thank all subjects who have whole heartedly participated in the study and have made the study complete.

Dr. AYUSH SINGAL

LIST OF ABBREVIATIONS

BM	:	Blurred Margins
BSP	:	Black Spots in Vision
BV	:	Blurred Vision
C	:	Clear
CBM	:	Can’t be made out
CO	:	Cup Obliterated
CON	:	Convulsions
CWS	:	Cotton Wool Spot
D	:	Diplopia
FAN	:	Focal Arteriolar Narrowing
FL	:	Flashes of Light
FUN	:	Fundus
GAN	:	Generalised Arteriolar Narrowing
GER	:	Greyish Elevated Retina
GR	:	Grade
GS	:	Gunn’s Sign
H	:	Headache
LS	:	Leg Swelling
ME	:	Macular Edema
N	:	Normal
P	:	Present
PIH	:	Pregnancy Induced Hypertension
RD	:	Retinal Detachment
RT	:	Retinopathy
SH	:	Superficial Hemorrhage
SPH	:	Splinter Hemorrhage
SS	:	Salu’s Sign
RPE	:	Retinal Pigment Epithelium

ABSTRACT

INTRODUCTION:

Pregnancy Induced Hypertension (PIH) is a systemic disease which usually occurs after 20th week of pregnancy in approximately 5% of pregnant females. Ocular involvement in patients with PIH may range from arteriolar narrowing to blindness due to retinal detachment and visual cortex ischemia. This one year study evaluated frequency and spectrum of retinal fundus changes in patients with PIH and relationship between degree of hypertensive retinopathy to severity of PIH.

METHODOLOGY:

130 patients with PIH were included in study after applying inclusion and exclusion criteria. Detailed ocular history and examination was done in each patient including fundoscopy. Investigations like urine protein, blood urea, serum uric acid, platelet count was done is all the patients to know the systemic severity of the disease. Data was collected in predetermined proforma and results were analysed.

OBSERVATIONS AND RESULTS:

130 patients with age range from 18 to 40 years were included in study. 56.15% of these were primigravidas while 79.23% were in more than 32 weeks of gestation. 77 patients had mild preeclampsia, 43 had severe preeclampsia and 10 patients had eclamptic convulsions. 57.69% of patients had grade I retinopathy, 3 patients each had grade II and III retinopathy. 2 patients had papilledema where as total of 4 patients developed exudative retinal detachment as a complication of PIH. 42 patients had normal fundus picture. Analysis of data showed that the risk of

developing hypertensive retinopathy increases with increasing severity of PIH from mild preeclampsia through severe preeclampsia to eclampsia.

CONCLUSION:

This study shows that even with increasing health awareness and improving standard of treatment; rare ocular complications of PIH like retinal detachment do occur along with less severe changes of retinal vasculature. Also, this study shows that with increasing severity of PIH there is a greater chance of developing of retinopathy.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF PHOTOGRAPHS

INTRODUCTION

Pregnancy Induced Hypertension (PIH), which includes preeclampsia and eclampsia occurs in approximately 5% of pregnancies usually after 20th week of pregnancy.[1] Preeclampsia is characterized by hypertension, proteinuria and generalized edema.[2] When preeclampsia progresses rapidly and convulsions develop, the condition is then termed as eclampsia.[2-4]

The incidence of PIH is 10% and eclampsia affects 5% leading to 17.2% of maternal mortality and 22% of foetal mortality. Early detection and treatment are therefore of paramount importance.[5]

This systemic disease, which can affect almost every organ system of the body, has potentially devastating consequences for both mother and baby.[6]

Abnormalities of the conjunctiva like capillary tortuosity, conjunctival hemorrhage, intravascular thrombi, localized ischemic necrosis of conjunctiva due to severe vasospasm have also been observed along with changes in retina, retinal vasculature, choroid, optic nerve and visual cortex.[4,5] Hence ophthalmologist should be aware of the changes that occur in the eye .

The eye serves as window through which the vessels of the brain can be studied. The retina acts as an index of the state of parenchyma of kidney. Since there exists close relationship between retinal, cerebral and renal vessels, fundoscopy gives the opportunity of observing the changes in the vascular tree and deducing there from the general condition of vascular system of the body.

Fundoscopic findings in PIH include a reduced arteriole to vein ratio. A-V crossing changes, hemorrhages, exudates in retina, exudative retinal detachments and choroidal infarcts.[7]

By detecting retinal arteriolar spasm the ophthalmologist might determine when immediate delivery of baby is required to reverse the preeclamptic state and prevent an adverse maternal outcome.[6]

Thus, the retinal fundoscopic examination has become the primary investigative procedure in assessment of ophthalmic changes in patients with PIH. The procedure is simple, non invasive, cost effective and easily performed either at outpatient department or at bedside.

Ocular changes may be the initial finding in an asymptomatic patient necessitating primary care referral.[8]

The focus of the current study is to find out the frequency and spectrum of fundal changes in one hundred and thirty cases of PIH presenting to tertiary health care centre (KLES' Prabhakar Kore Hospital & Medical Research Centre, Belgaum) in South India.

AIMS AND OBJECTIVE OF STUDY

1) To find out frequency and spectrum of fundal changes in patients with Pregnancy Induced Hypertension.
2) To find out relationship between degree of hypertensive retinopathy and severity of Pregnancy Induced Hypertension.

REVIEW OF LITERATURE

ANATOMY OF RETINAL VASCULATURE

Central retinal artery is a branch of the ophthalmic artery. The central retinal artery along with the central vein pierces the lamina cribrosa to appear at the optic nerve head. It lies superficially in the nasal part of physiological cup covered by glial tissue. Here it divides into two branches, a superior and an inferior, each of which sub divides into temporal and nasal branches. The four terminal branches of central retinal artery divide dichotomously as they proceed towards ora serrata where they end without anastomosis.[9]

By doing fundoscopy we can study central retinal artery along with central retinal vein with their branches.

SIGNIFICANCE OF FUNDOSCOPY IN PIH

Landesman R. et al, stated that the eye grounds are probably the best single indictor of the progress of the toxemia. In general retinal changes run parallel with severity of the hypertension and therefore of the toxemia.[6, 9-11]

Mussey RD & Mundell BJ, concluded that examination of fundus in patients with PIH permits an objective assessment of vascular changes and provides a basis for further obstetric management.[12]

Riss B. et al, accepted that the examination of ocular fundus permits the direct evaluation of fundal changes and early recognition of fetal jeopardy.[13]

The diagnosis of hypertensive disorders complicating pregnancy was out lined by working group of the National High Blood Pressure Education

Program (NHBEP), 2000;[14] based on many factors , one being the severity of hypertension; as follows :

GESTATIONAL HYPERTENSION:

1) BP $\geq$ 140/90 mm Hg for first time during pregnancy
2) No Proteinuria
3) BP returns to normal < 12 wks post partum.
4) Final diagnosis made only post-partum.
5) May have other signs of preeclampsia e.g. epigastric discomfort or thrombocytopenia.

PREECLAMPSIA:

1. Minimum Criteria
   a) BP $\geq$ 140/90 mmHg after 20 wks gestation.
   b) Proteinuria $\geq$300mg/24hr or $\geq$1+dipstick
2. Increased certainty of preeclampsia
   a) BP $\geq$160/110mmHg
   b) Proteinuria 2.0gm/24Hb or $\geq$2+dipstick
   c) Serum Creatinine $\geq$1.2gm/dl unless known to be previously elevated.
   d) Platelets < 1,00,000/mm^3
   e) Microangiopathic hemolysis (increase LDH)
   f) Elevated ALT or AST.
   g) Persistent headache or other cerebral or visual disturbance.
   h) Persistent epigastric pain.

Eclampsia:

Seizures that cannot be attributed to other causes in women with preeclampsia.

SUPER IMPOSED PREECLAMPSIA (ON CHRONIC HYPERTENSION):

New onset proteinuria $\geq$300mg/24hrs in hypertensive woman but no proteinuria before 20 weeks of gestation.

A sudden increase in proteinuria or blood pressure or platelet count <1,00,000/mm^3 in women with hypertension and proteinuria before 20 weeks gestation.

CHRONIC HYPERTENSION:

BP $\geq$140/90mmHg before pregnancy or diagnosed before 20 weeks gestation

Or

Hypertension first diagnosed after 20 weeks gestation and persistent after 12 weeks post-partum.

AETIOPATHOGENESIS

Inspite of vast amount of research the etiology of preeclampsia still remains unsolved. It is likely that abnormal changes which characterize toxemia of pregnancy might be precipitated by a number of different stimuli in individuals who by some unknown mechanism have appeared to respond by developing sodium and water retention, hypertension, altered renal functions and convulsions.

The incidence is more in women[14] :

- Who is exposed to chorionic villi for the first time
- With pre-existing vascular disease.
- With abundant chorionic villi i.e., twins, hydatiform mole.
- Who is genetically predisposed to the development of hypertension

Pre-eclampsia is associated with vasospasm and pathological vascular lesions in multiple organ systems including the uteroplacental vascular bed, increased platelet activation with platelet consumption and subsequent activation of coagulation system in microvasculature.

Normal Endothelial Cell function

The endothelium function by the mechanical separation of blood products from collagen and smooth muscles of the vascular wall. It simultaneously allows transport of nutrients, waste products, regulatory molecules and phagocytic cells across cellular basement membrane. Normally vascular endothelial surface resists platelet aggregation and coagulation.

Increase pressor responses

Increased vasopressor reactivity in women with early preeclampsia has been identified by Roab and co-workers (1956) and Talledo and associates (1968). Grant and co-workers (1973) demonstrated that increased vascular sensitivity to angiotensin II clearly preceded the onset of pregnancy-induced hypertension.[14]

Prostaglandin

It has been concluded that the blunted pressor response described earlier is due principally to decreased vascular responsiveness mediated in part by vascular endothelial synthesis of prostaglandins or prostaglandin like substances.[14]

Nitric Oxide

Previous termed endothelium derived relaxing factor is synthesized by endothelial cells from arginine.

Both PGI_2 and endothelium derived relaxing factors are the most important mediators of vasodilatation Endothelium dependant contractions induced by arachidonic acid are elicited through TxA_2 and PGE_2.[14]

Endothelins

These polypeptides are potent vasoconstrictors. Plasma endothelin-1 is increased in normotensive laboring and non-laboring women and even higher levels have been reported in preeclamplic women.

Vascular endothelial growth factor (VEGF)

This is important in vasculogenesis and control of microvascular permeability and has been identified in human placenta. They found an increase in VEGF, parallel to increased uteroplacental vessel resistance in women with preeclampsia.[14]

Genetic Predisposition

The tendency for preeclampsia-eclampsia is inherited. Chesley and Cooper (1986) studied at Margaret-Haque Maternity Hospital from 1935 to 1984 and concluded that preeclampsia-eclampsia is highly heritable.[14]

Immunological factor

Dekker and Sibai (1988) have reviewed the possible role of immune maladaptation in the pathophysiology of preeclampsia. Antibodies against endothelial cells have been found in 50% of women with preeclampsia vs 15% of normotensive controls.[14]

Inflammatory factors

Redman and colleagues (1999) have proposed that the endothelial cell dysfunction associated with preeclampsia can result from generalized perturbation of normal generalized maternal intravascular inflammatory adaptation to pregnancy. In this hypothesis preeclampsia is considered a disease due to an extreme state of activated leucocytes in maternal circulation. The oxygen free radicals lead to the formation of self-propagating lipid peroxides that in turn propagate highly toxic radicals, which in turn injure endothelial cell. Such injury modifies endothelial cell production of nitric oxide as well as interfering with prostaglandin balance.[14]

Endothelial cell dysfunction

In normal individuals endothelial derived relaxing factor (EDRF) is constantly released at low levels and controls the blood pressure. In PIH the endothelial cells suffer considerable injury due to the toxic lipid peroxides and free radicals, which leads to reduction of EDRF.[14]

Injured endothelium produce vasoconstrictors, increased membrane permeability, clot formation, vasospasm and blood vessel remodeling This vital response to disruption of vascular integrity can cause serious physiologic disturbances.[14]

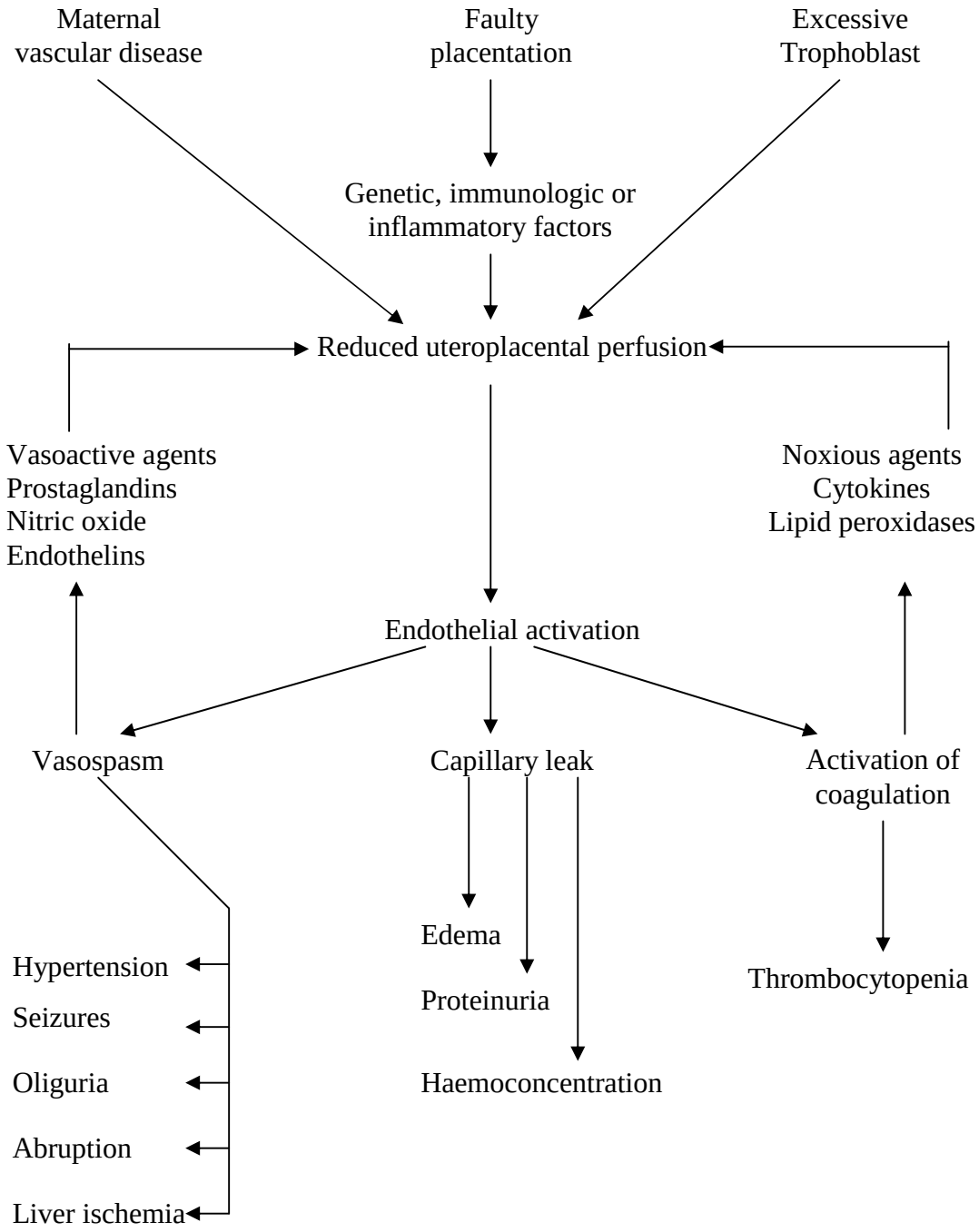
Vasospasm

Vasospasm is basic to the pathophysiology of preeclampsia-eclampsia [Volthard (1918), Himnselmann (1924) and Landermann (1954)]

Vasospasm causes resistance to blood flow and accounts for the development of arterial hypertension. Vasospasm itself exerts a damaging effect on vessels. Angiotensin-II causes endothelial cells to contract. These changes lead to endothelial cell damage and inter-endothelial cell leaks through, which blood constituents, including platelets and fibrinogen, are deposited subendothelially (Brunner and Gauras, 1975). The vascular changes together with local hypoxia of the surrounding tissue lead to hemorrhage, necrosis and other end organ disturbances.

Due to vasospasm, the vascular endothelial damage in systemic and uteroplacental circulation causes enhanced platelet activation and fibrin generation and there is significant decrease in platelet levels in preeclampsia as platelets are used in intravascular coagulation.

Pathophysiological considerations in the development of hypertensive disorders due to pregnancy



OCULAR CHANGES IN PIH

SPASM OF RETINAL ARTERIOLES:

Retinopathy occurring during toxemia of pregnancy generally shows the features of hypertensive retinopathy.[15]

The first changes observed in the normal retinal arterioles during the toxemia of pregnancy is attenuation of retinal arterioles.[16] It is associated with a rise in the diastolic blood pressure. This attenuation usually occurs first in the nasal periphery but gradually spreads towards the disc and become generalized. Once it has developed it usually persists until pregnancy is terminated.[17-19]

In generalized arteriolar constriction instead of the normal diameter ratio of vein to arteries i.e. 3:2, in toxemia the arterioles are constricted so that the ratio is 2:1. As the severity of the toxemia progress, the arterioles are usually seen to become more constricted until the ratio increases to 3:1 or more. In estimating the vein arteriole ratio, the examiner compares the diameter of a principal vein and its corresponding arteriole.[19]

Arteriolar narrowing is the defining quality of hypertensive retinopathy and is due to vasoconstriction as an autoregulatory response. Vasoconstriction can be regarded as an autoregulatory attempt to control the volume of blood received by the retinal capillary bed. It is most commonly seen in the early phase of hypertension.[19]

Haselhorst and Mylius in 1928, and Masters and Hallum later recognized marked retinal spasm which was at times transient or persistent, localized or diffuse. They associated this with severe toxemia and the arterial

spasm was found to antedate retinopathy. After the pregnancy the spasm disappeared in many cases.[10]

Wagener in 1933, reported the study of a series of 40 cases of hypertensive toxemia of pregnancy and concluded that spastic lesions of the arterioles were the most frequent and usually the primary sign of retinal involvement.[18]

Retinal vasoconstriction is the most obvious fourth sign in manifest preeclampsia along with classic symptom triad of hypertension, edema and proteinuria.[17]

Toxemia of pregnancy in its earlier stages is associated with angiospasm and increased tonus of the central retinal arterioles. The retinopathy, which develops in its later stages, is characterized by the presence of hemorrhages and exudates frequently accompanied by edema.[20, 21]

Masters in 1933, found a passive congestion of the retinal veins in the early months of pregnancy. He noted arteriolar spasm in most toxemias with systolic blood pressure over 150 mmHg and in only several was spasm noted with a blood pressure below 150.[10]

Mussey and Mundell in 1939 concluded that persistent arteriolar spasm was a guide to the further management of the toxemia. These authors found varying retinal changes with similar blood pressure and in 44% of toxemia cases, the progressive retinal vessel spasm was the deciding factor in the decision to terminate pregnancy.[10]

Wagener and Keith in 1939, further stressed the importance of arteriolar retinal spasm. They stated that diffuse angiospasm may produce

sufficient vascular injury to contraindicate further pregnancy.[10]

Addis accepted the hypothesis that arterial spasm is a frequent pathogenic factor underlying and uniting the various symptoms of late toxemia. He suggested that these manifestations are the result not of varying causes but of the kind of tissue on which the single cause acts and that vascular spasm is the factor, which underlies and unites all the manifestations of preeclampsia and eclampsia.[11] Arteriolar narrowing is the defining quality of hypertensive retinopathy and is due to vasoconstriction as an autoregulatory response. Vasoconstriction can be regarded as an autoregulatory attempt to control the volume of blood received by the retinal capillary bed. It is most commonly seen in the early phase of hypertension.[19]

Based on different autoregulatory responses in different age groups Robert Leishman[22] proposed a classification for hypertensive retinopathy :

CLINICAL CLASSIFICATION OF FUNDUS APPERANCES IN HYPERTENSION AND ARTERIOSCLEROSIS (MODIFIED AFTER ROBERT LEISHMAN):

OLD ARTERIES:

Group 1

- Normal old age
- Involutionary sclerosis
- Average age 64 years
- Usually raised systolic pressure without corresponding rise of diastolic pressure.

- Fundus – Optic disc pale and showing a shallow depression. Arteries straight, narrow, pale and branched at acute angle walls not easily visible. Reduced light reflections, arteriolar walls of normal and diminished thickness.

Group 2

- Involutionary sclerosis with hypertension (B.P. 200/94-120) i.e. hypertension in relatively fibrous vessels with passive stretching of walls.
- Age – 64 yrs
- Fundus – Arteries, the large vessels are wide and curvilinear, the smaller once narrow or straight. Some congestion of veins distal to A-V crossings. Later the arteries become tortuous and wide.

Group 3

- Advanced involutionary sclerosis with hypertension
- Age 64 years.
- High level of diastolic pressure seems to be well tolerated.
- Fundus- Arteries, main vessels wide, red and tortuous, peripheral vessels pale and narrow congestion of veins distal to AV crossings.

Group 4

- Normal youth
- Fundus – optic disc pink. Arteries wide of bright color, walls visible, light reflection bright.

Group 5

EARLY HYPERTESNION

- Age 39 years
- May be transient condition in acute glomerulonephritis or in toxemia of pregnancy.
- Fundus – Arterioles straight and narrow veins. No concealment of veins at AV crossing but congestion of veins distal to crossings.

Group 6

- Fulminating malignant hypertension. Severe hypertonus acting on vessels undefined by fibrosis.
- Age 45 years
- BP – 250/150mm Hg
- Fundus – Oedema of optic disc and retina. Arteries pale, large, tortuous and elongated small arteries straight and narrow hard while exudates, superficial cotton wool exudates, small capillary hemorrhages, arteriolar thrombosis.

Group 7

- Severe hypertension with reactive sclerosis. Hypertonus and hyperplasia giving rise to replacement fibrosis and sclerosis.
- Age 59 years
- BP – 250/130 mm Hg.

- Fundus – Arteries, tortuous, distal arterioles straight and narrow in parts but red and full in others. Impediment and concealment of veins at A-V crossings.

Addis, Herrick, Irying and others emphasized the role of vascular spasm in the production of syndrome of toxemia.[11]

Alton V. Hallum in a span of 14 years studied the eye grounds of approximately 2500 women suffering from hypertension during pregnancy. The one outstanding and consistently reliable change observed in the eye grounds was the degree of general and localized spastic constriction of the retinal arterioles. According to him during the course of toxemia of pregnancy the most information is gained from the eye grounds by making repeated examination daily or every few days.[23]

Glenn Jaffe and Howard Schatz, performed a prospective, controlled masked study designed to evaluate the ocular manifestations of preeclampsia in 56 patients including 25 controls, 17 mild preeclamptic and 14 severe preeclamptic patients. There was a statistically significant correlation between reduction in arteriole to vein ratio and a diagnosis of severe preeclampsia. There was also a significant correlation between the number of focal constrictions and a diagnosis of severe preeclampsia. The results of this controlled masked study indicate that the role of the ophthalmologists in the diagnosis and management of preeclamptic patients is limited.[6]

CHANGES AT ARTERIOVENOUS CROSSINGS

Due to vasospastic changes occurring in vasculature of retina, changes do occur at arteriovenous crossings.

DEFLECTION OF VEIN:

The artery and vein shares a common adventitial sheath surrounded by a glial tissue with fusion of basement membrane and surrounding muscular layer. Normally at crossing of the vessel there is no evidence of their depression or elevation. In hypertension, there is a deflection in the course of the vein – SALU'S SIGN.

The usually type of deflection in hypertension is lateral, instead of crossing under artery obliquely the veins do so at right angles taking apparently the shortest possible route.[19, 24, 25]

COMPRESSION OF THE VEIN:

Apparent narrowing of the vein when it is crossed by the artery is called GUNN'S SIGN. Banking of blood column distal to the crossing is seen in the advanced stages of hypertension.[25]

Simple nicking – when there is simple deflection of the vein out of its course.[25]

Moderate nicking – when vein appears to be partially cut and blood column is obstructed and banked upon the peripheral side of the crossing.[25]

Definite nicking – when the vein is apparently completely cut leaving a blank space on either side of the artery. The narrowing of the lumen is partly due to encroachment of the common vascular wall into the lumen of the vein and partly due to constriction of venous lumen partly due to an obstruction of the blood column by optical effects of thickened walls.[25]

RETINOPATHY DUE TO PIH

If the hypertension continues hemorrhages and cotton wool exudates develop. Hemorrhages are of small size, superficial, striated or flame shaped. They mostly occur along the vessel and near the optic disc. There are superficial hemorrhages that lie in the nerve fibre layer – striate hemorrhage follows the pattern of nerve fibre bundle. Cotton wool exudates are typical of hypertension and are rounded, creamy color and with soft edge. Striate texture and frayed edge of the exudates indicate it's situation at the nerve fibre layer. They lie underneath the vessel or over it.[24-26]

Silex in 1895 reported 35 cases of advanced retinal changes in pregnancy.[10]

Schiotz in 1921, found 40 cases of retinopathy associated with pregnancy in an extensive survey of 8400 women at Christiania Clinic.[10]

The great majority of the earlier authors associated retinopathy with latent or an actual nephritis. Miller in 1915 went so far so as to say that the presence of retinopathy was an important diagnostic factor suggesting the presence of nephritis.[10]

Wolff and Zade evaluated 13 cases clinically with advanced retinal changes, 10 were of chronic nephritis.[10]

The fetal mortality associated with retinopathy was as high as 80%. Rochon Du Vigneaud showed that uninterrupted pregnancy associated with retinopathy resulted in blindness or serious visual damage in 75% of the cases.[10]

Wagener description of the retinal changes in cases of toxemia of pregnancy was divided into 4 stages.[11]

Grade 1: Spastic narrowing of arterioles of the retina.

Grade 2: Irregular constriction of the lumen.

Grade 3: Narrowing and constriction are more fixed with cotton wool patches and hemorrhages.

Grade 4 Diffuse retinitis.

Wagner, Schiotz, Masters and Hallum all agreed that retinopathy may be a definitive indication for interruption of the pregnancy.[10]

PAPILLOEDEMA

The optic nerve is enclosed upto lamina cribrosa within meningeal sheaths common to the brain. Hence any rise in intracranial pressure becomes equally evident. There is blurring of the margins of the optic disc. The blurring starts at upper and lower margin and extend around nasal side and at the end the temporal margin becomes blurred.[27]

HYPERTENSIVE OPTIC NEUROPATHY

Malignant hypertension can lead to optic nerve head swelling with plasma leakage and disruption of nerve fibres, which ultimately lead to loss of axons with subsequent gliosis. The pathophysiologic mechanism of hypertensive disc edema is controversial. Few propose that secondary encephalopathy is the mechanism of hypertensive papilloedema. Others believe that disc edema is secondary to ischemic changes of optic disc. Despite its complex vasculature, the optic nerve head is susceptible to

ischemia by virtue of its tightly arranged nerve fibres within non expandable intrascleral canal. Both the mechanical factors and ischemia may play role in the development of disc edema in hypertensive optic neuropathy.[28, 29]

Keith and Wagener had put forward a grading system for hypertensive retinopathy which is most frequently used in clinical practice:

GRADING OF HYPERTENSIVE RETINOPATHY KEITH WAGENER'S CLASSIFICATION:[30, 31]

Grade 1 - Mild-to-moderate narrowing or sclerosis of the arterioles

Grade 2 - Moderate to marked narrowing of the arterioles
Local and/or generalized narrowing of arterioles
Exaggeration of the light reflex
Arteriovenous crossing changes.

Grade 3 - Retinal arteriolar narrowing and focal constriction
Retinal edema
Cotton-wool patches
Hemorrhage

Grade 4 - As for Grade 3, plus papilledema

RETINAL DETACHMENT

Soon after the invention of the ophthalmoscope by Helmholtz, Albrecht. Von Graefe in 1855, described the first case of retinopathy with retinal detachment.[10] He was the first one to describe the retinal changes in preeclampsia.[16]

Though the retinal detachment itself is uncommon, sub clinical damage to the choriocapillaries may occur in preeclamptic patients.

A retinal detachment complicating the retinopathy of the toxemia of pregnancy is not common, but since the first case was reported by Von Graefe (1855) a considerable number has appeared in the literature.[19]

In 1928, Mylius obtained the first photograph of this complication.[16]

Helbron (1902) collected records of 21 cases, SeJuotz (1921) 50 cases and Fry (1929) 57 cases. In recent years however, prophylactic measures have made its occurrence rare.[19]

Ballantyne and Michelson suggested that the subretinal exudates causing detachments originated in choroid.[27]

Moore[32] and Friedenwald[33] proposed that retinal detachments resulted from damage to retinal vasculature.

Nonrhegmetogenous retinal detachment in toxemia of pregnancy appear to be caused by fluid from choriocapillaries pouring through necrotic retinal pigment epithelium.[34]

In 1980, Mabie and Ober had implicated both choroidal and retinal vasculature as a source of subretinal fluid in retinal detachment complicating toxemia of pregnancy.[35] These detachments are bilateral. They are self-resolving and get reattached rapidly.[36]

HYPERTENSIVE CHORIODOPATHY

The relationship of choroidal changes and retinal detachment in toxemia of pregnancy was first described by Verdehaeme in 1911.[3]

Hypertensive choriodopathy is seen typically in young patients with pliable vessels that are not yet sclerotic from long term hypertension.

Toxemia of pregnancy, renal disease, pheochromocytoma, accelerated hypertension and connective tissue disorder can manifest hypertensive choriodopathy.[28, 29]

In accelerated or malignant hypertension the arteries and arterioles of the choroids undergoes fibrinoid necrosis. Fibrinoid necrosis represents replacement of smooth muscle fibres by fibrin platelet and other plasma protein materials. It occurs when severe hypertension causes vessel wall damage from severe narrowing. This results in patchy non-perfused areas of choriocapillaries.

The overlying RPE appears yellow (focal ischemic infarcts) in acute phase and with time becomes irregularly pigmented with depigmented halos (Elschings spots). They are typically in mid periphery and in vicinity of the optic disc. In fluorescein angiography, choroidal vasculopathy is demonstrated by irregular filling patterns, delay in filling time and areas of diffuse leakage or window defects depending on the stage of Elschings spot formation.[28, 29]

In patients with chronic hypertension Siegerts streak are seen. They represent area of hyperpigmentation overlying sclerotic choroidal arteries in compressed and attenuated choricapillaries. Seigerts lesion imply advanced generalized vascular sclerosis with poor prognosis.

Acute hypertension has greater effect on the choroidal circulation than on retinal circulation. Much of the damage to the endothelium and musculature of the choroidal arterioles is the result of this increase in blood pressure that overwhelms the compensatory tone.[28,29]

CLASSIFICATION OF HYPERTENSIVE RETINOPATHY MODIFIED KEITH WAGENER'S CLASSIFICATION:[28,29]

Classifi –cation	RETINOPATHY			ARTERIOLES		ARTERIO SCLEROSIS	
	Hemor rhages	Exu- dates	Disc Edema	A:V Ratio	Focal Spasm	Light Reflex	AV crossing defect
Stage 0	-	-	-	3:4	1:1	Fine Yellow	None
Stage 1	-	-	-	1:2	1:1	Broad Yellow	Mild Vein, Depression A-V Nicking
Stage 2	-	-	-	1:3	2:3	Copper wire	Vein depression
Stage 3	+	+	-	1:4	1:3 cotton wool sports	Silver wire	Right angle Disappearance of vein, distal dilatation
Stage 4	+	+	+	Fibrous Fine Cord	Elschings spots No distal flow	Stage 3	Stage 3

CORTICAL BLINDNESS

Cortical blindness is one of the important causes of blindness in toxemia of pregnancy. It is a complication of severe preeclampsia. The pathophysiology of cortical blindness is debated to be from cerebral vasospasm and ischaemic injury or vasogenic edema caused by increased capillary permeability. The incidence of cortical blindness manifested by hypertensive encephalopathy in preeclampsia is 1 to 15%.[37]

This has been attributed to cerebral edema of the occipital cortex that develops in some cases of toxemia due to vascular spasm. The cerebral edema of occipital cortex has been documented in such cases by CT scans. Complete visual recovery is common in such cases and usually parallels the resolution of cerebral edema.[38]

Also one study concluded that cortical blindness associated with toxemia resulted from petechial hemorrhages and focal edema in occipital cortex.[39]

PROGNOSIS

If the retinal changes are confined to arterial attenuation and patient is well within the pre-organic stage of her toxemia then patient will probably respond to adequate conservative treatment and the pregnancy may justifiably be continued although under the closest supervision.[19]

The changes seen in the retina of patients with toxemia of pregnancy if properly interpreted from clinical standpoint is a valuable aid in the estimation of patients immediate and future course. These ophthalmic examination should be made at frequent intervals if the patient has a rapidly rising blood pressure and angiospastic changes are present. Absence or regression of retinopathy has been used as a sign to allow a pregnancy to continue.[40]

Other Methods of investigation Ophthalmic artery velocimetry

Maternal ophthalmic arterial blood flow velocimetry can be assessed by Doppler velocimetry.

Ophthalmic arterial pulsatility index shows significant correlation with gestational age, which might be caused by decreasing vascular resistance during pregnancy. In preeclamptic women it was found that there was significant lower ophthalmic arterial pulsatility index and higher mean velocity compared with those in normotensive gravidas.[41]

MATERIALS AND METHODS

MATERIAL

Patients with pregnancy induced hypertension (PIH) who were admitted in the Department of Obstetrics and Gynaecology in KLES' Dr. Prabhakar Kore Hospital & Medical Research Centre, Belgaum from January 2007 to December 2007 were included in the study.

METHODS

INCLUSION CRITERIA: One hundred and thirty consecutive patients who were referred to Department of Ophthalmology diagnosed as having PIH from January 2007 to December 2007 were included in the study.

EXCLUSION CRITERIA:

1. Subjects who had a positive history of hypertension, diabetes mellitus, cardiovascular disease and collagen vascular disease prior to pregnancy were excluded from the study.
2. Patients with ocular media opacity in both eyes which might interfere with detailed examination of fundus were also excluded from the study.

All patients were informed in detail about the procedure and informed consent was taken. After obtaining history and presenting complaints, patient was examined for palor, pedal edema, pulse, blood pressure at admission and at the time of fundus examination.

Patients visual acuity was checked clinically at bed side.

Torch light examination was done to rule out any gross anterior segment pathology.

One to two drops of 0.5% of tropicamide was instilled into the cul-de-sac. The patient was instructed to apply digital pressure on lacrimal sac for 2 to 3 min after drug instillation to avoid systemic absorption. 15 to 20 minutes later, fundoscopy was done by experienced surgeon and the postgraduate student.

Initial examination was carried out with direct ophthalmoscope followed by indirect ophthalmoscopy.

The fundus picture was described and documented with the help of color coded fundus diagrams.

Patients who were stable were mobilized to ophthalmic department and fundus photographs were taken with the help of fundus camera.

INTERPRETATION OF FUNDOSCOPY

The ophthalmoscopic findings were documented and staging and grading was done according to Keith Wagener's classification of Hypertensive retinopathy.

GRADING OF HYPERTENSIVE RETINOPATHY KEITH WAGENER'S CLASSIFICATION:

Grade 1 - Mild-to-moderate narrowing or sclerosis of the arterioles

Grade 2 - Moderate to marked narrowing of the arterioles

Local and/or generalized narrowing of arterioles

Exaggeration of the light reflex

Arteriovenous crossing changes.

Grade 3 - Retinal arteriolar narrowing and focal constriction

Retinal edema

Cotton-wool patches

Hemorrhage

Grade 4 - As for Grade 3, plus papilledema

After doing staging and grading as per Keith Wagener's classification of Hypertensive retinopathy diagnosis of retinopathy was made and further obstetric management was decided based on fundoscopic findings.

RESULTS

This study titled A ONE YEAR CROSS SECTIONAL STUDY OF FUNDAL CHANGES IN PATIENTS WITH PREGNANCY INDUCED HYPERTENSION ATTENDING KLES' PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELGAUM.

The data was tabulated and analysed as follows:

Table 1 : Age Distribution

Age (in years)	No. of Patients	Percentage	Retinopathy
≤ 19	12	9.23%	8 (67%)
20-29	96	73.85%	62 (64.6%)
≥30	22	16.92%	17 (77%)

MEAN AGE : 25 yrs

In this study of 130 patients, 73.85% were in age group of 20 to 29 years followed by 17% in age group of ≥30 years.

Graph -1: Showing Distribution according to Age

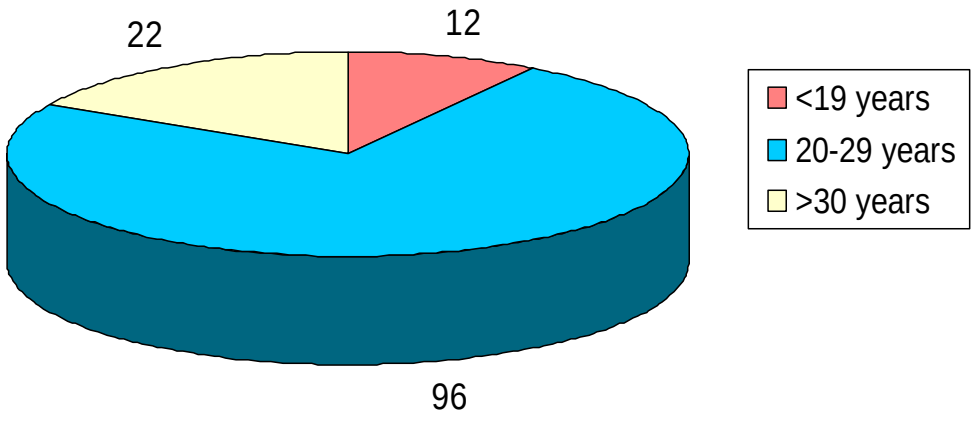


Table 2: Gravida

Gravida	No. of Patients	Percentage
G_1	73	56.13%
G_2	32	24.62%
G_3	17	13.10%
G_4	7	5.38%
G_7	1	0.77%

In the present study of 130 cases, 56.13% were primigravidas followed by 24.62% were Gravida II.

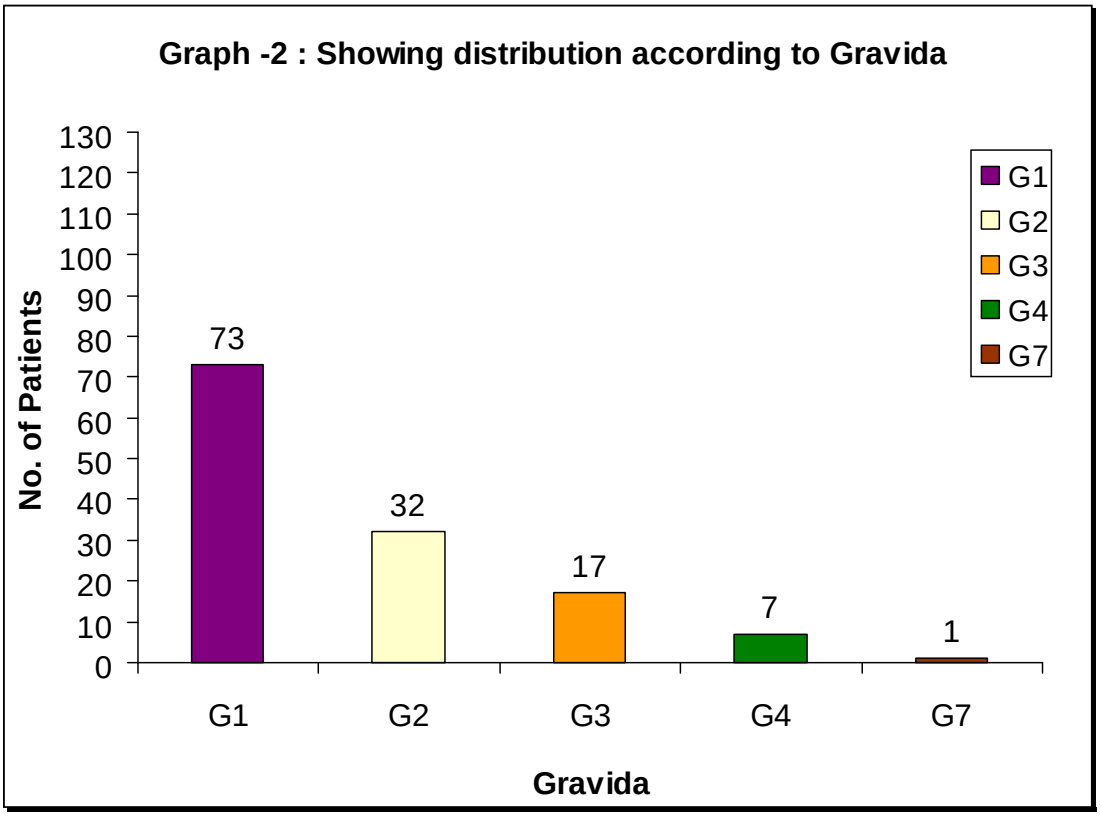


Table 3: Duration of Pregnancy

Duration of Pregnancy in weeks	No. of Patients	Percentage
20-28	9	6.92%
29-32	18	13.85%
32 Onwards	103	79.23%

MEAN GESTATIONAL AGE – 36Weeks

In this study of 130 patients, 79.23% were in 32 to 40 weeks of gestation followed by 13.85% in 29 to 32 weeks of gestation.

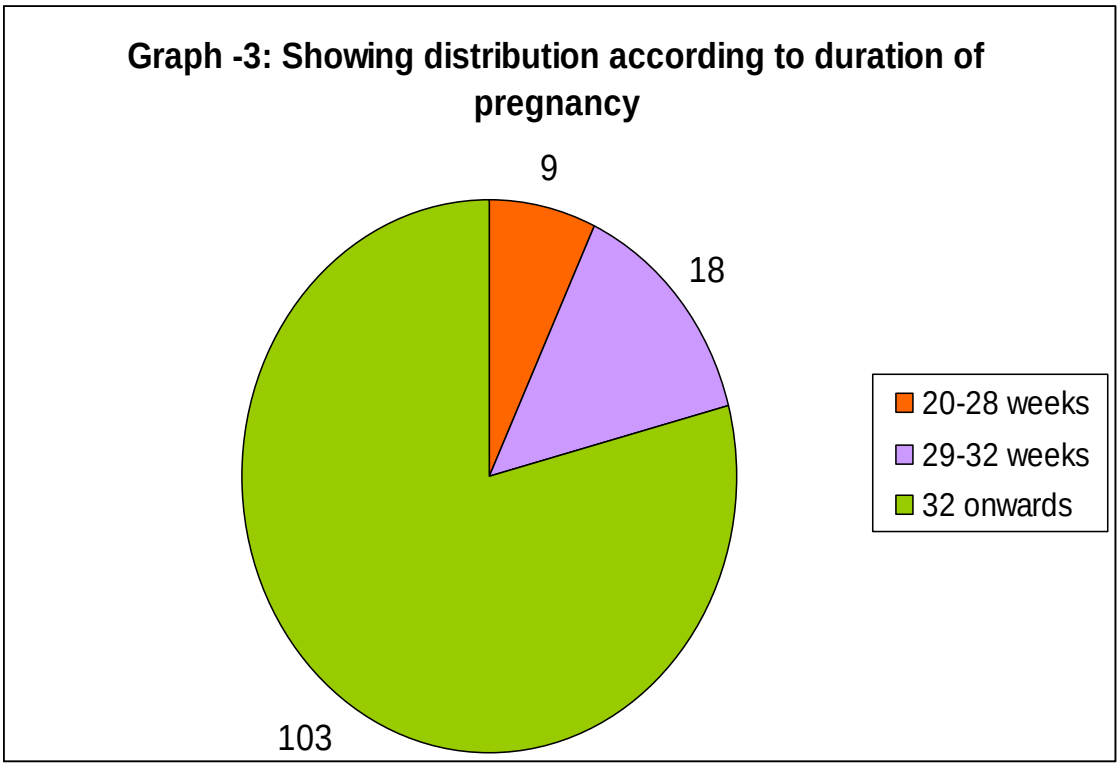


Table 4: Symptoms

Symptoms	No. of Patients	Percentage
Blurred Vision	51	39.23
Convulsions	10	7.70
Headache	78	60
Leg swelling	130	100
Flashes of light	1	0.77
Black spots in visual field	1	0.77
Diplopia	1	0.77

In the present study of 130 cases, 100% of the patients complained of leg swelling followed by 60% of patients had headache. 39.23% of patients had blurring of vision as initial symptoms.

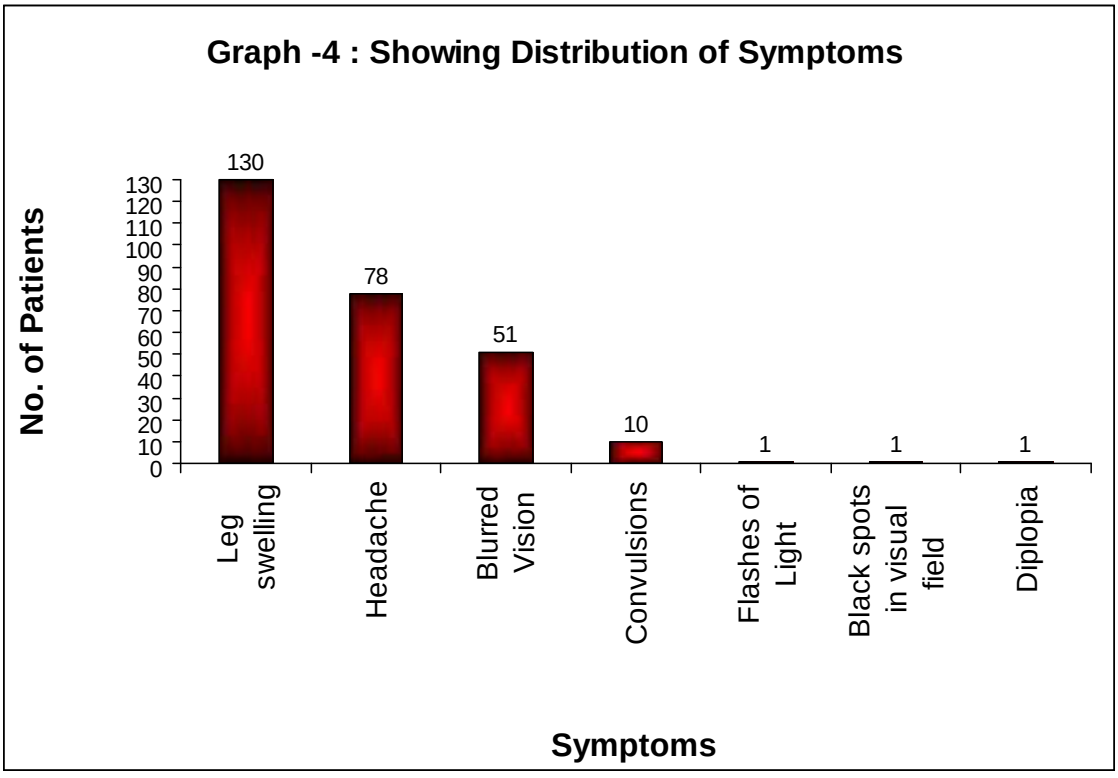


Table 5: Severity of PIH

Severity of PIH	No. of Patients	Percentage
Mild Preeclampsia	77	59.23
Severe Preeclampsia	43	33.07
Eclampsia	10	7.70

In the present study of 130 cases, 59.23% of the patients had mild preeclampsia i.e. blood pressure < 160/110 and 33.07% had severe preeclampsia i.e. blood pressure ≥ 160/110. Only 10 patients in the present study had convulsions.

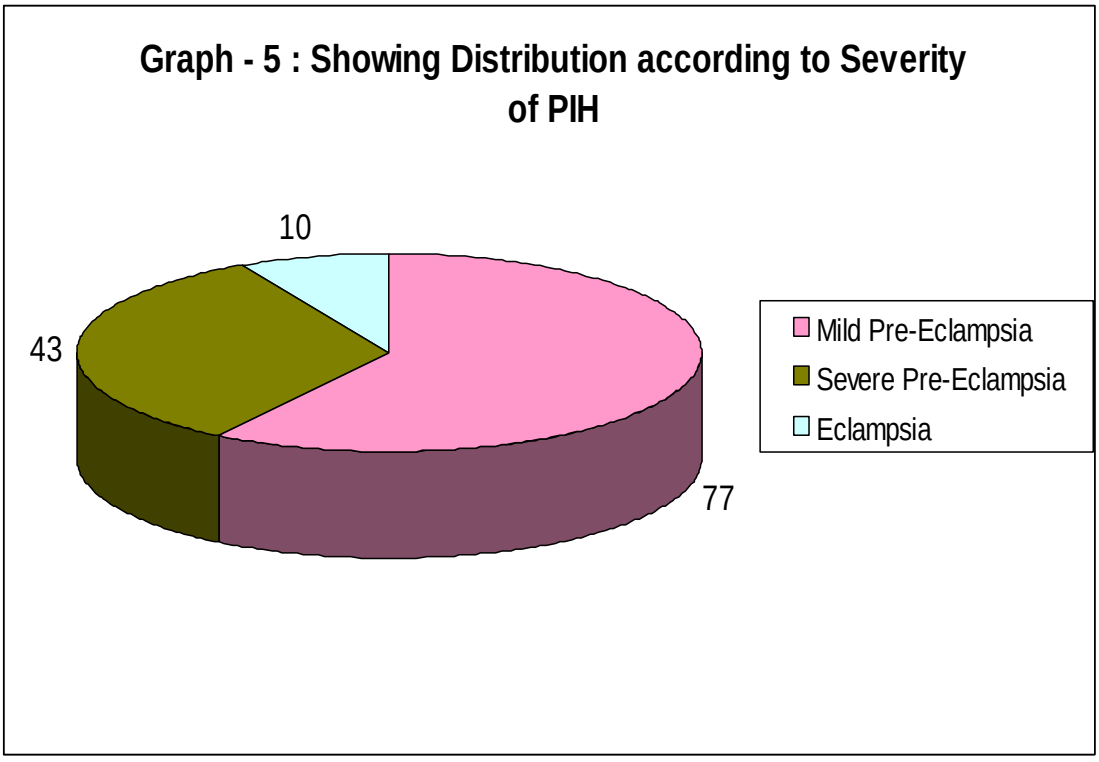


Table 6: Fundus changes according to Grades of Retinopathy

Grades	No. of Patients	%
Normal Fundus	42	32.30
Grade-I Hypertensive Retinopathy	76	58.47
Grade-II Hypertensive Retinopathy	3	2.30
Grade-III Hypertensive Retinopathy	3	2.30
Grade-IV Hypertensive Retinopathy	2	1.53
Retinal Detachment	4	3.1

In the present study of 130 cases, 58.47% had Grade-1 hypertensive retinopathy followed by 32.30 of patients having normal fundus with no evidence of any hypertensive retinopathy changes. Retinal detachment occurring as a complication of PIH was seen in 3.1% of total patients.

Graph - 6 : Showing Fundus changes according to Grades of Retinopathy

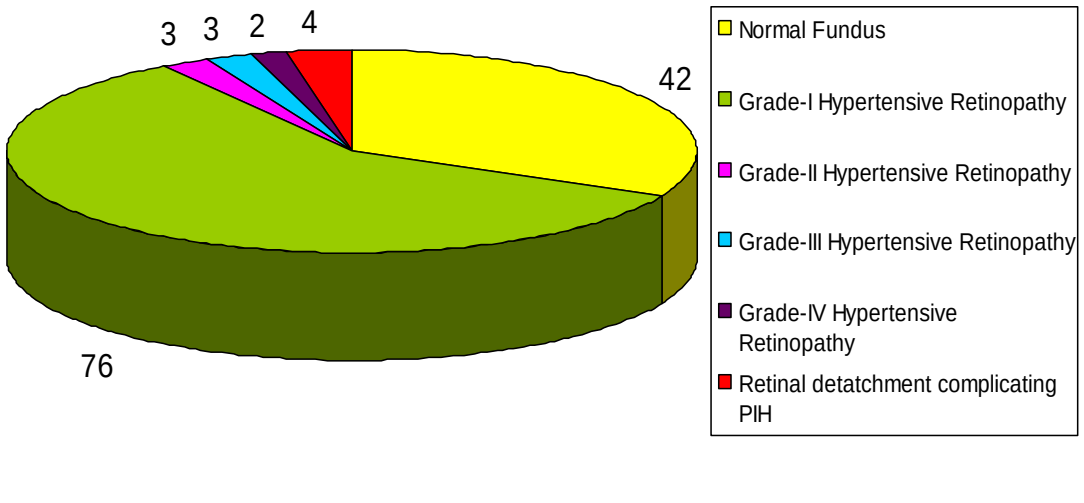


Table 7: Severity of Arteriolar Narrowing

Severity of Arteriolar Narrowing	No. of Patients	Percentage
Normal	44	33.85%
Focal arteriolar constriction	05	3.85%
Mild generalized arteriolar constriction	39	30%
Severe generalized arteriolar constriction	42	32.30%

In the present study of 130 cases, 86 patients had some degree of arteriolar narrowing. 42 patients had severe generalized arteriolar narrowing while 39 patients had mild generalized arteriolar narrowing. Only 5 patients had focal arteriolar constriction.

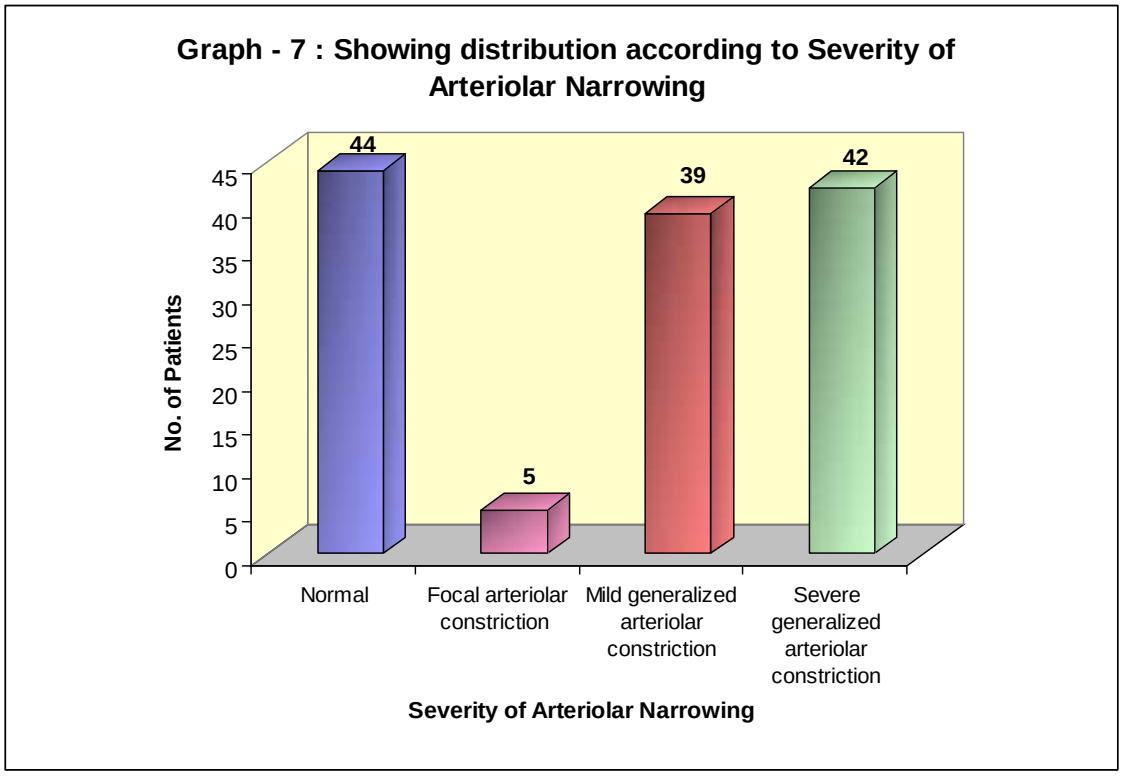


Table 8: Distribution of severity of retinopathy in mild pre-eclampsia group

Grade of Retinopathy	No. of Patients	Percentage
Normal	30	38.96%
Grade – I	44	57.14%
Grade – II	0	0%
Grade – III	1	1.30%
Grade – IV	1	1.30%
RD	1	1.30%

In the present study of 130 cases, 77 patients had mild preeclampsia, of which 57.14% had Grade I hypertensive retinopathy. Out of total 77 patients with mild preeclampsia 38.96% patients had normal fundus at the time of fundoscopic examination.

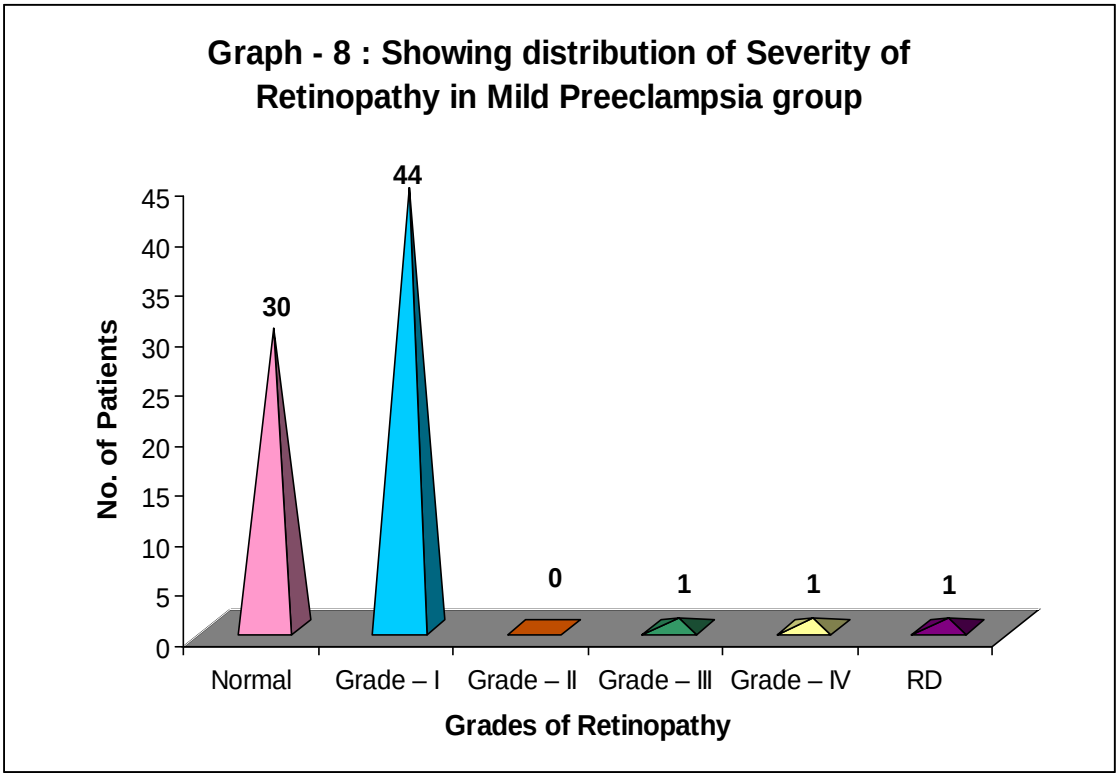


Table 9: Distribution of severity of retinopathy in Severe Pre-eclampsia group

Grades of Retinopathy	No. of Patients	Percentage
Normal	10	23.26%
Grade – I	27	62.79%
Grade – II	2	4.65%
Grade – III	2	4.65%
Grade – IV	0	0%
RD	2	4.65%

In the present study of 130 cases, 43 patients had severe preeclampsia. Out of these 43 patients, 62.79% had Grade I Retinopathy while normal fundus was seen in 23.26% of patients. Grade II and III Retinopathy and retinal detachment complicating preeclampsia was seen in 2 patients each.

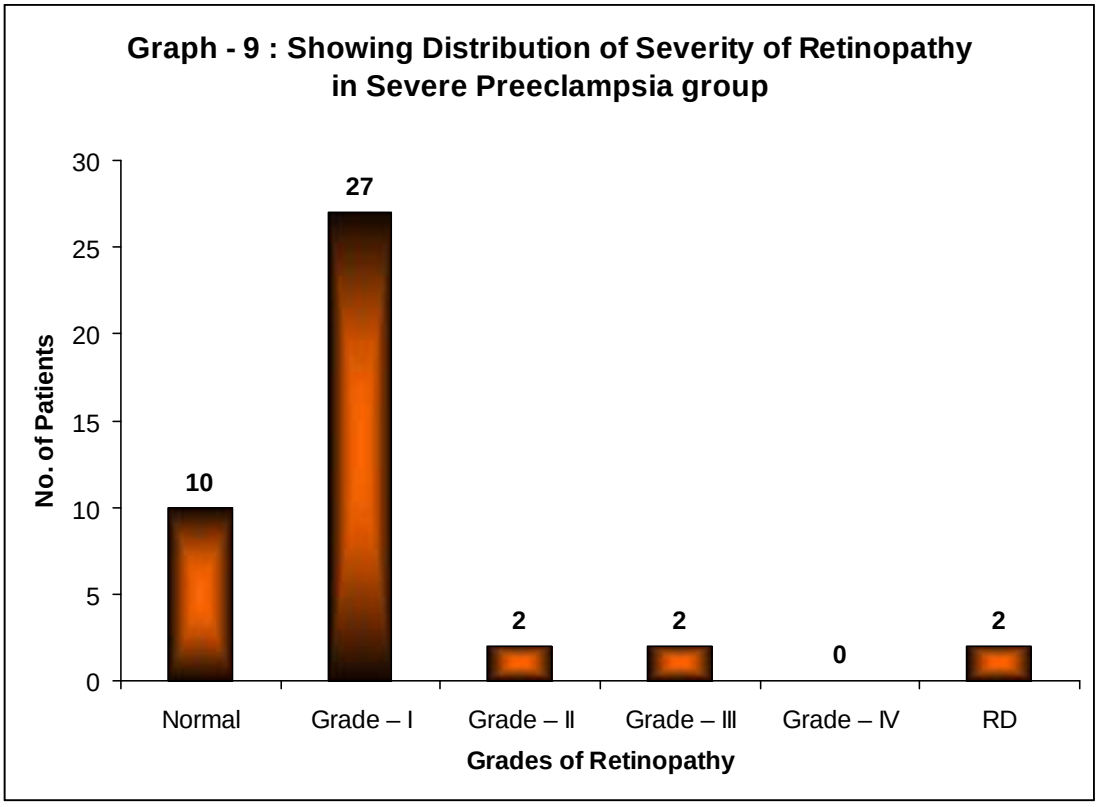


Table 10: Distribution of severity of retinopathy in eclampsia group

Grade of Retinopathy	No. of Patients	Percentage
Normal	2	20%
Grade – I	5	50%
Grade – II	1	10%
Grade – III	0	0%
Grade – IV	1	10%
RD	1	10%

In the present study of 130 cases, 10 patients had eclampsia. Of these 80% had retinopathy while only 20% had normal fundus on examination. Grade I Retinopathy was seen in 50% of patients where as Grade II & IV Retinopathy and retinal detachment complicating eclampsia was seen in 1 patient each.

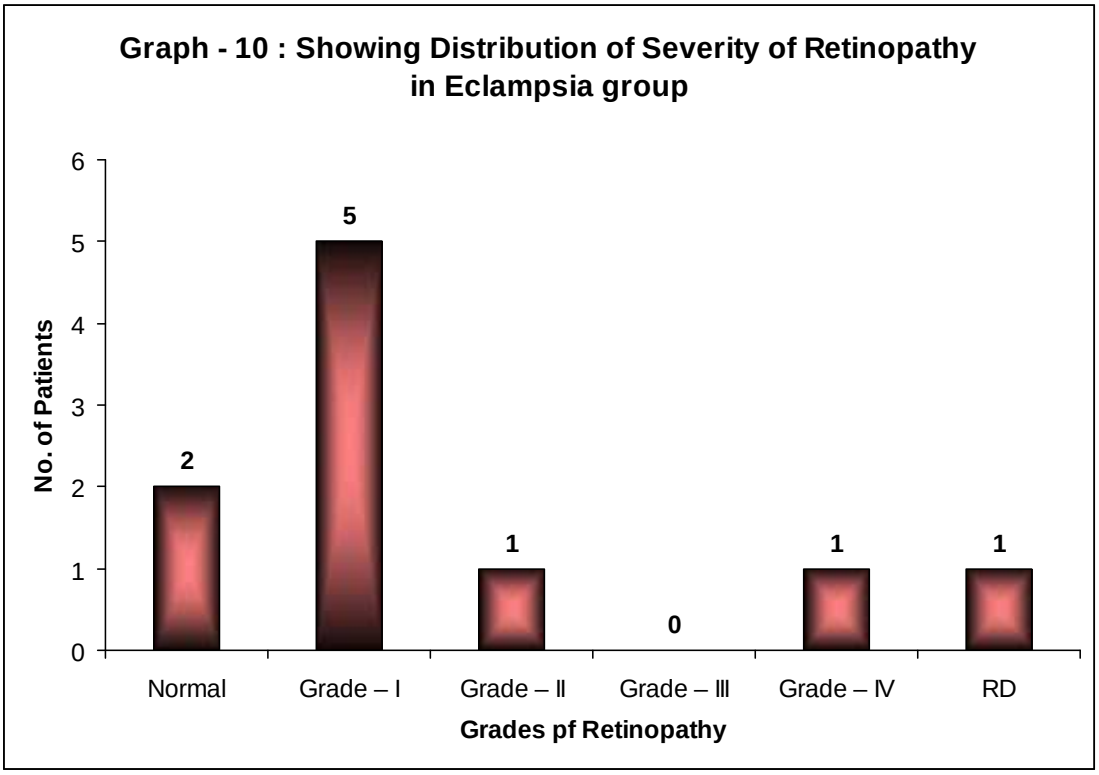


Table 11: Severity of Proteinuria

Proteinuria	No. of Patients	Percentage	Retinopathy
+	89	68.46%	60 (67.42%)
++	27	20.77%	16 (59.25%)
+++	8	6.15%	5 (62.5%)
++++	6	4.62%	5 (83.33%)

In the present study of 130 cases, 68.46% of patients had 1+ proteinuria on urine dipstick test followed by 20.77% patients having 2+ proteinuria. Patients with 3+ and 4+ proteinuria constituted 6.15% and 4.62% respectively.

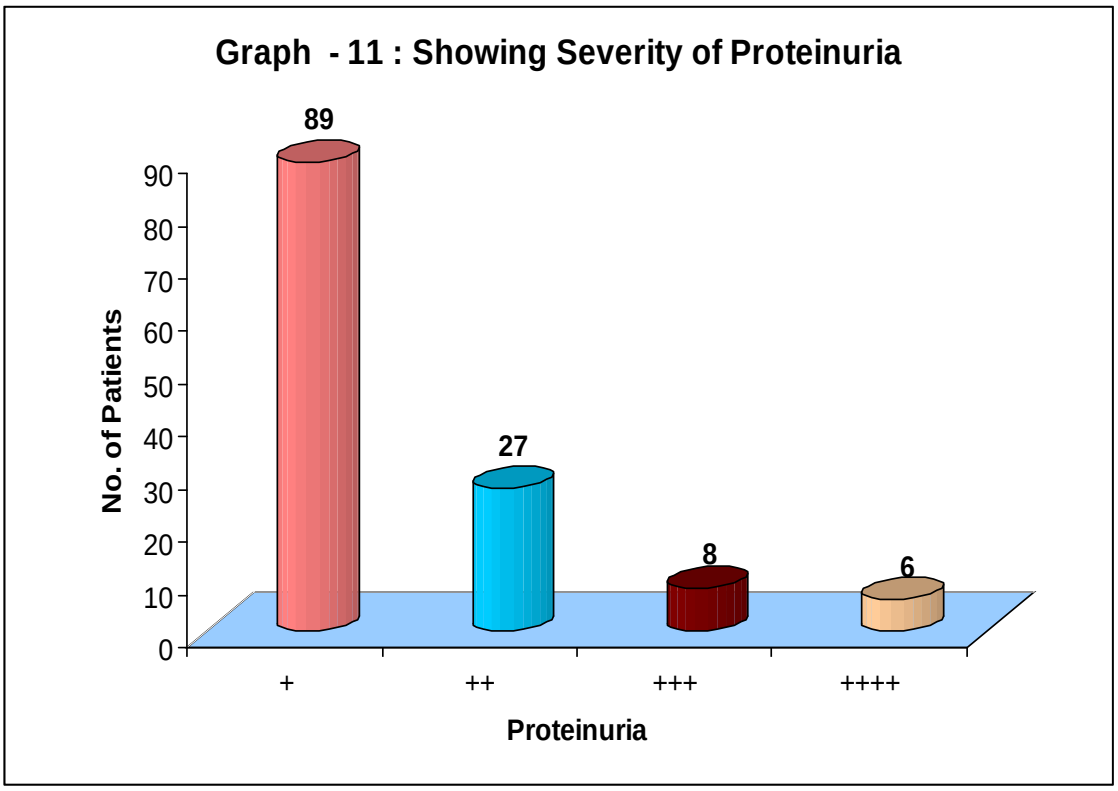


Table 12: Relationship between blood urea value in milligram % & severity of PIH

Severity of PIH	Maximum	Minimum	Mean
Mild Pre-Eclampsia	43.0	7.0	17.66
Severe Pre-Eclampsia	71.0	6.0	22.07
Eclampsia	44.0	10.0	27.4

In present study, blood urea in mild pre-eclampsia group ranged from 7.0 to 43.0 mg.% with an average of 17.66mg.% whereas range of blood urea in severe pre-eclampsia was from 6.0 to 71.0 mg.% with an average value of 22.07 mg.%. In patients having eclampsia blood urea ranged from 10.0 to 44.0 mg.% with an average of 27.4mg.%.

Table 13: Relationship between uric acid value in milligram % & severity of PIH

Severity of PIH	Maximum	Minimum	Mean
Mild Pre-Eclampsia	11.0	2.5	5.32
Severe Pre-Eclampsia	8.9	3.0	5.94
Eclampsia	13.5	5.0	7.75

In present study, serum uric acid in mild pre-eclampsia group ranged from 2.5 to 11.0 mg.% with an average of 5.32mg.% whereas range of serum uric acid in severe preeclampsia was from 3.0 to 8.9 mg.% with an average value of 5.94 mg.%. In patients having eclampsia serum uric acid ranged from 5.0 to 13.5 mg.% with an average of 7.75mg.%.

DISCUSSION

AGE DISTRIBUTION: (Table 1)

In Tadin et al, study of 40 women with pre-eclampsia 45% (18) showed abnormalities of the fundus. The average age of 40 patients was 29.1 years.[42] Many studies showed that younger and older age group are associated with risk factors.[43]

In another study by Jaeffe and Schatz, mean age of patients with pre-eclampsia was 28 years.[6]

Mean age of the patients in this study was 25 years.

GRAVIDA: (Table 2)

Pre-eclampsia in an otherwise healthy women is a disease of first pregnancy.[43]

In the present study of 130 patients with pregnancy induced hypertension 56.13% of patients were primigravidas.

This result of present study is comparable to previous studies, which have concluded that PIH is more common in primigravidas.

DURATION OF PREGNANCY: (Table 3)

In Cunnigham et al, study of patients with pre-eclampsia and eclampsia the gestational age when pre-eclampsia and eclampsia developed ranged from 18-40 weeks with average gestational age of 34 weeks.[39]

In this study, range of gestational age at which patients in present study developed PIH was from 23-40 weeks. 79.23% of patients developed PIH after 32 weeks of gestation with an average or mean gestational age of 36 weeks.

This result of present study is comparable with the earlier study.

SYMPTOMS: (Table 4)

Visual disturbances such as scotoma, diplopia and dimness of vision are seen in 30-50% of patients with eclampsia and 20-25% of patients with preeclampsia.[43] Headache has long been known to be harbinger of eclamptic convulsions.[44] Headache is most common symptom among patients with preeclampsia.[24]

In the present study, 60% of patients had headache as one of the complaints while approximately 39.23% of patients complained of visual symptoms like blurred vision, flashes of light, black spot in visual field and diplopia.

This result of the study co-relates with earlier study as similar percentage of patients had visual symptoms as is in earlier studies. Also after legs swelling; headache was most common symptom among the patients in this study.

SEVERITY OF PIH: (Table 5)

In one study the overall incidence of PIH in obstetric patients was found out to be 5%. Approximately 5% of these patients developed eclamptic seizures.[24, 43]

In the present study, 7.70% of all PIH patients had eclampsia which is slightly higher compared to previous study. Further most of the patients(59.23%) in this study had mild pre-eclampsia with blood pressure < 160/100mm of Hg.

FUNDUS CHANGES ACCORDING TO GRADES OF RETINOPATHY: (Table 6)

Retinal changes have been observed in 40-100% patients of preeclampsia.[43, 45] The most common ocular finding is constriction of arterioles occurring in approximately 60% of patients with pre-eclampsia in one study.[18] The hallmark of abnormal ocular findings is terminal arteriolar vasospasm.[24, 45] Wagener reported spastic lesions of retinal arterioles in 70% cases of PIH.[40]

Arteriolar narrowing of generalized nature is seen later and may resolve following pregnancy.[43] Retinal detachment is seen in 1-2% of all patients with PIH.[36]

In the present study, 67.7% of patients with PIH had retinal involvement which co-relates with 40-100% described in previous study. Further Grade I Hypertensive Retinopathy which consists of retinal arteriolar narrowing is most common Grade of retinopathy seen in more than half

(58.47%) of the patients included in the study. This also co-relate with previous study which states arteriolar narrowing is the most common fundus finding in patients with PIH. Patients who had retinal detachment constituted 3.1% of all 130 patients with PIH. This is comparable to the results of other studies.

SEVERITY OF ARTERIOLAR NARROWING: (Table 7)

The most common ocular finding is constriction of retinal arterioles, occurring in approximately 60% of patients with preeclampsia.[1, 18]

The most prominent finding is terminal arteriolar vasospasm, associated with the development of systemic hypertension.

At first, focal areas of spasm may be observed which progress to more generalized narrowing as preeclampsia worsen.[6, 18]

In the present study, 66.15% of patients had some degree of vasospasm. Most of these patients i.e. 62.30% of total 130 cases had generalized arteriolar constriction while only 3.85% had focal arteriolar constriction.

DISTRIBUTION OF SEVERITY OF RETINOPATHY IN MILD PREECLAMPSIA GROUP: (Table 8)

The most common abnormality seen in visual system is spasm and narrowing of retinal vessels.[6, 24, 43] The earliest finding is focal constriction of retinal arterioles which may progress to generalized narrowing.[24]

The result of present study co-relates with earlier studies as majority of patients (57.14%) with mild preeclampsia has Grade I Retinopathy i.e. focal and generalized arteriolar narrowing.

DISTRIBUTION OF SEVERITY OF RETINOPATHY IN SEVERE PREECLAMPSIA GROUP: (Table 9)

In one study the degree of hypertensive retinopathy was directly proportional with severity of preeclampsia.[42]

Landesman R et al, have found a co-relation between the degree of retinopathy and severity of preeclampsia.[9, 10]

Mussey R D et al, have found a co-relation between frequency of retinal changes and the level of blood pressure.[11]

The result of present study co-relates with previous studies as more percentage of patients developed retinopathy as well as more severe grade of retinopathy as compared to mild preeclampsia group.

DISTRIBUTION OF SEVERITY OF RETINOPATHY IN ECLAMPSIA GROUP: (Table 10)

The result of this study matches with other studies as greater percentage of patients developed more severe retinopathy as compared to mild and severe preeclampsia. Also, there is increased chances or greater risk (80%) to patient with eclampsia to develop retinopathy as compared to mild (61.04%) and severe (76.74%) preeclampsia.

SEVERITY OF PROTEINURIA: (Table 11)

Proteinuria is an important sign of preeclampsia. The minimum criteria for diagnosis of preeclampsia are hypertension and minimal proteinuria. Proteinuria may be minimal or severe.[46]

In the present study, all 130 patients had proteinuria and range of proteinuria varied from 1+ to 4+. This co-relates with previous study. Also, result of present study indicated that patients with severe proteinuria (4+) have greater chance of developing retinopathy than less severe proteinuria.

RELATIONSHIP BETWEEN BLOOD UREA VALUE IN MILLIGRAM % & SEVERITY OF PIH: (Table 12)

In study by Tandon and Kishore, blood urea level in mild preeclampsia ranged from 19.5 to 30.0 mg% with an average of 24.6 mg% while in severe preeclampsia group value of blood urea ranged from 24.0 to 103.0 mg% . In eclamptic women values ranged from 30.0 to 57.0 mg% with an average value of 43.4mg%.[47]

Knowledge of blood urea level offers information which is useful in complicated cases of toxemia. The rising level is almost always associated with increasing severity of toxemia and falling levels with improvement. In complicated cases this additional means of assessment can be of considerable assistance in management.[47]

The result of present study correlates with the previous study as increasing level of mean blood urea level is seen with increasing severity of Pregnancy Induced Hypertension.

RELATIONSHIP BETWEEN SERUM URIC ACID VALUE IN MILLIGRAM % & SEVERITY OF PIH: (Table 13)

In study by Tandon and Kishore, serum uric acid level in mild preeclampsia ranged from 4.6 to 6.4 mg% with an average of 5.2 mg% whereas in severe preeclampsia group value of serum uric acid ranged from 4.2 to 8.0 mg% with a mean value of 5.63 mg%. In eclamptic women values ranged from 6.2 to 11.2 mg% with an average value of 7.2 mg%.[47]

High values of uric acid have been reported by Stender and Cadden[47] (1939), Crawford (1941),[47] Moshe, Lancet and Fisher (1956), Prabhawati (1957) and Pollak (1960).

There is definite clinico-biochemical correlation between serum uric acid levels and blood pressure.

In the present study, the mean value of blood uric acid in mild and severe preeclampsia is 5.32 mg% and 5.94 mg% respectively. In eclamptic patients the mean blood uric acid level was 7.75 mg%. This result of the study matches with the previous study. Further, there is increase in the mean value of blood uric acid with increase in severity of PIH.

Thus it may be inferred that there is a clinico-biochemical correlation with respect to blood uric acid levels and severity of PIH and that blood uric acid level are of considerable value in predicting severity of PIH.

CONCLUSION

1. Fundoscopy is simple, non invasive, safe and reliable procedure, which can be done in OPD or at bedside any number of times. It should be done routinely in all patients of pregnancy induced hypertension to interpret the vascular changes in PIH.
2. Though the definite diagnosis is made by considering other factors, a suggestive diagnosis by fundoscopy guides the subsequent obstetrics management of the patient.
3. In this study, majority of patients showed grade I hypertensive retinopathy. Retinal detachment complicating PIH was seen in four patients.
4. This study shows that even with increasing health awareness and improving standard of treatment ; rare ocular complications of PIH like retinal detachment do occur along with less severe changes of retinal vasculature. Also, this study shows that with increasing severity of PIH there is greater chances of developing of retinopathy.

SUMMARY

One hundred and thirty patients with Pregnancy Induced Hypertension (PIH) who were referred to Department of Ophthalmology from January 2007 to December 2007 were included in this study.

Detailed ocular examination including fundoscopy was done in all 130 patients and results interpreted.

Of these 130 patients , 73.85% were in the age group of 19-29 years followed by 16.92% in age group of ≥ 30 years.

Seventy three (56.13%) of these patients were primigravidas among the 130 patients.

79.23% were in 32-40 weeks of gestation period.

All 130 patients (100%) complained of leg swelling followed by 78 patients (60%) who also had headache along with leg swelling. Fifty one patients (39.23%) complained of blurring of vision out of these 130 patients.

77 (59.23%) had mild preeclampsia while 43 (33.07%) had severe preeclampsia. Ten (7.70%) patients had eclampsia out of total 130 included in study.

Seventy six (58.47%) patients had grade I hypertensive changes while 42 (32.30%) patients had normal fundus with no evidence of any hypertensive changes on fundoscopy. Grade II and Grade III changes were seen in 3 (2.30%) patients each while Grade IV changes was seen only two (1.53%) patients. Exudative retinal detachment complicating PIH was seen in four (3.10%) patients.

The percentage of patients developing retinopathy due to PIH increased as we move from mild preeclampsia group to severe preeclampsia to eclampsia group.

All patients had proteinuria of varying severity ranging from 1+ to 4+ with patients with severe proteinuria of 4+ having greater chance of developing retinopathy.

In this study, it was found that with increasing severity of PIH the mean value of biochemical investigations like blood urea and serum uric acid is also increased. Thus, these investigations can be of additional advantage in assessing the severity of PIH.

Thus in this study, we tried to assess frequency and spectrum of fundal changes in patients with PIH and also the relationship between the degree of fundal changes and severity of PIH.

BIBLIOGRAPHY

1. Sheth BP, Mieler WF. Ocular complications of pregnancy. Curr Opin Ophthalmol 2001 Dec;12(6):455-63.

2. Valluri S, Adelberg DA, Curtis RS, Olk RJ. Diagnostic indocyanine green angiography in preeclampsia. Am J Ophthalmol 1996 Nov;122:672-77.

3. Fastenberg DM, Fetkenhour CL, Chromokos E, Shoch DE. Choroidal vascular changes in toxemia of pregnancy. Am J Ophthalmol 1980 Mar;89(3):362-68.

4. Schultz KL, Birnbaum AD, Goldstein DA. Ocular diseases in pregnancy. Curr Opin Ophthalmol 2005 Oct;16(5):308-14.

5. Chap 18. Hypertensive disorders of pregnancy. In: Mudaliar & Menon, editor. Clinical Obstetrics. 9th ed. Chennai: Orient Longmann; 1999. p. 133.

6. Jaeffe G, Schatz H. Ocular manifestation of preeclampsia. Am J Ophthalmol 1987 Mar;103(3):309-15.

7. Capoor S, Goble RR, Wheatley T, Casswell AG. White-centered retinal hemorrhages as an early sign of preeclampsia. Am J Ophthalmol 1995 Jun;119(6):804-6.

8. Barbazetto IA, Pizzarello LD. Ocular changes during pregnancy. Compr Ophthalmol Update 2007 May;8(3):155-67.

9. Landesman R, Douglas RG, Synder SS. Retinal changes in toxemia of pregnancy. Am J Obstet Gynecol 1952;63(1):16-27.

10. Landesman R, Douglas RG, Synder SS. Retinal changes in toxemia of pregnancy. Am J Obstet Gynecol 1951;62(5):1020-33.

11. Mussey RD, Mundell BJ, Rochester. A guide in management of toxic hypertensive syndrome of pregnancy. Am J Obstet Gynecol 1939;37:30-6.

12. Riss B, Drobec P, Riss P. Incidence and significance of fundus changes in EPH gestosis. Klin Monatsbl Augenheilkd 1983 Sep;183(3):180-3.

13. Riss B, Riss P, Metka M. Prognostic value of changes in fundus oculi in EPH gestosis. Z Geburtshilfe Perinatol 1983 Nov;187(6):276-9.

14. Chap 34. Hypertensive disorders in pregnancy. In: Cunningham, Gant Leveno, Bloom SL, Hauth JC, Gilstrap LC, editor. Williams Obstetrics. 22nd ed. NewYork: Mc-Graw Hill; 2004. p. 761-787.

15. Brancato R, Menchini U, Bandello F. Proliferative retinopathy and toxemia of pregnancy. Ann Ophthalmol 1987 May;19(5):182-3.

16. Kenny GS, Cerasoli JR. Color fluorescein angiography in toxemia of pregnancy. Arch Ophthalmol 1972 Apr;87:383-88.

17. Seidman DS, Serr DM, Ben-Rafael. Renal and ocular manifestations of hypertensive diseases of pregnancy. Obstet Gynecol Surv 1991;46(2):71-6.

18. Hallum AV. Eye changes in hypertensive toxemia of pregnancy: a study of 300 cases. JAMA 1936;106(9):1649-51.

19. Chap IV. Retinopathies associated with general diseases. The vascular diseases. In: Duke Elder SS, editor. System of ophthalmology. Diseases of the retina. New Delhi: Jaypee Brothers; 1990. p. 277-356.

20. Beer PM, Cooper HM, Krohel GB. Indocyanine green angiographic findings in toxemia of pregnancy. The vitreous society online journal 1997 Jan;1-2

21. Klein R, Klein BE, Moss SE. Hypertension and retinopathy. Arteriolar narrowing and arteriovenous nicking in a population. Arch Ophthalmol 1994;112:92-8.

22. Chap 11. Diseases of retina. In: Lyle TK, Cross AG, Cook CAG, editor. May and Worths diseases of the eye. CBS Publishers and distributors; 1985. p. 315

23. Hallum AV. Changes in retinal arterioles associated with the hypertensions of pregnancy. Arch Ophthalmol 1947;37:472-90.

24. Ober RR. Pregnancy induced hypertension. In: Ryan SJ, editor. Retina . Mosby publications; 1994. p. 441-7.

25. Murphy RP, Chew EY. Hypertension. In: Ryan SJ, editor. Retina. Mosby publication; 1994. p. 449-55.

26. Chap IV. Haemorrhages , exudates and neovascularisation in fundus. In: Pahwa JM, Billore OP, editor. Retinal diseases. 3rd ed. Oxford and IBH publishing Co. Pvt Ltd.; 1978. p. 30-2 .

27. Ballantyne AJ, Michelson IC. Hypertensive retinopathy. In: Textbook of fundus of eye. Williams and Wilkins; 1962. p. 146.

28. Davis EA, Dana MR. Pregnancy and the eye. In: Albert DM, Jakobeic FA, Azae DT, editor. Principles and practice of ophthalmology. 2nd ed. W.B.Saunders company; 2000. p. 4767-74.

29. Kim SK, Christlieb, Mieler WF. Hypertensive and tis ocular manifestations. In: Albert DM, Jakobeic FA, Azae DT, editor. Principles and practice of ophthalmology. 2nd ed. W.B.Saunders company; 2000. p. 4506-23.

30. Chap 12. Retinal vascular disease. In: Kanski JJ, editor. Clinical ophthalmology. 4th ed. Butteworth-Heinmann; 1999. p. 495-7.

31. Rogers AH. Vascular disorders hypertensive retinopathy. In: Yanoff M, Duker JS, editor. Ophthalmology. 2nd ed. Mosby publications; 2004. p. 852.

32. Moore F. Medical ophthalmology. 2nd ed. London Balkistan Co.; 1925. p. 189-90.

33. Friedenwald JS. Pathogenesis of albuminuric retinitis. In: The Libman anniversary volumes. Volume 2. NewYork International press; 1932. p. 453-58.

34. Folk JC, Weingeist TA. Fundus changes in toxemia. Ophthalmology 1981 Nov;88(11):1173.

35. Mabie WC, Ober RR. Fluorescein angiography in toxemia of pregnancy. Br J Ophthalmol 1980;64:666-71.

36. Prado RS, Figueiredo EL, Magalhaes TV. Retinal detachment in preeclampsia. Arq Bras Cardiol 2002;72(2):185-6.

37. Apollon KM, Robinson JV, Schwartz RB, Norwitz ER. Cortical blindness in severe preeclampsia. Obstet Gynecol 2000 Jun;95(6):1017-9.

38. Gupta P, Gupta R, Bajaj S. Effects of pregnancy on eye. Ophthalmology Today 2003 May-Jul;4(3):102-6.

39. Cunningham FG, Fernandez CO, Hernandez C. Blindness associated with preeclampsia and eclampsia. Am J Obstet Gynecol 1995 Apr;172(4): 1291-8.

40. Wagener HP. Arterioles of the retina in toxemia of pregnancy. JAMA 1933;101(8):1380-4.

41. Ohno Y, Kawai M, Wakahara Y, Kitagawa T, Kakihara M, Arii Y. Ophthalmic artery velocimetry in normotensive and preeclamptic women with or without photophobia. Obstet Gynecol 1999 Sep;94(3):361-3.

42. Tadin I, Bojic L, Mimica M, Karelovic D, Dogas Z. Hypertensive retinopathy and preeclampsia. Coll Antropol 2001;25:77-81.

43. Sunness JS. The pregnant woman's eye. Surv Ophthalmol 1988 Feb;32(4):219-38.

44. Belfort MA, Saade GR, Grunewald C, Dildy GA, Herd JA, Nissell H et al. Association of cerebral perfusion pressure with headache in women with pre-eclampsia. Br J Obstet Gynecol 1999 Aug;106:814-21.

45. Beck RW, Gamel JW, Willcourt RJ, Berman G. Acute ischemic optic neuropathy in severe preeclampsia. Am J Ophthalmol 1980;90:342-6.

46. Prasad GN, Shukla BK. Retinal changes in eclampsia and pre-eclampsia. JAMA 1976 Jan;66(1):8-10.

47. Kishore N, Tandon S. Significance of biochemical and ophthalmoscopic changes in toxemia of pregnancy. J Obstet Gynec India 1965 Dec;15 (6): 551-59.

PROFORMA

1) NAME OF THE PARTICIPANT: I.P.No:

2) AGE:

3) SEX:

4) ADDRESS:

5) DURATION OF PREGNANCY (IN WEEKS):

I) HISTORY PRIOR TO PREGNANCY

a) Vision prior to pregnancy	Normal / Decrease
b) H/O spectacle use prior to pregnancy	YES / NO
c) H/O blurring of vision	YES / NO
d) H/O flashes of light	YES/NO
e) H/O black spots in visual field	YES / NO
f) H/O Convulsions	YES/NO
g) H/O seeing single object as two	YES / NO
h) H/O episodes of transient loss of vision	YES / NO
i) H/O swelling of legs	YES/NO
j) H/O Diabetes	YES/NO
k) H/O Hypertension	YES/NO
l) H/O Rash, joint pains	YES/NO

II) HISTORY - DURING PREGNANCY {After 20 weeks)

a) Vision at present Normal/Decrease

b) Use of spectacles now YES / NO

c) H/O Convulsions YES/NO

d) H/0 blurring of vision YES/NO

e) H/O Flashes of light YES / NO

f) H/O Headache YES / NO

g) H/O black spots in visual field YES / NO

h) H/O seeing single object as two YES / NO

i) H/O episodes of transient loss of vision YES /NO

j) H/O leg swelling YES / NO

k) Any of the above complaints in previous pregnancy :

l) OBSTETRICS HISTORY G P A L

III) EXAMINATION :

i)PALOR YES/NO

ii) PEDAL EDEMA YES / NO If YES, PITTING / NON-PITTING

iii) PULSE

iv) B.P AT ADMISSION:_____ PRESENT: _____

A) OCULAR EXAMINATION: **OD** **OS**

1. ADENEXA
2. SCLERA
3. CONJUNCTIVA
4. CORNEA
5. ANTERIOR CHAMBER
6. IRIS
7. PUPIL
8. LENS:

B) FUNDUS EXAMINATION

1. GLOW
2. MEDIA
3. DISC
4. CUP : DISC RATIO
5. BLOODVESSELS

    a) Focal constriction of arterioles:

    b) Generalized arteriolar narrowing:

    c) Arterio-venous crossing changes:

    d) A : V Ratio:

    e) Any other positive finding :

6. BACKGROUND:

a) Haemorrhages:

b) Cotton-wool spots;

c) Hard Exudates:

d) Any other positive finding :

7. MACULA:

IV) INVESTIGATION

1) URINE PROTEIN

2) HAEMOGLOBIN%

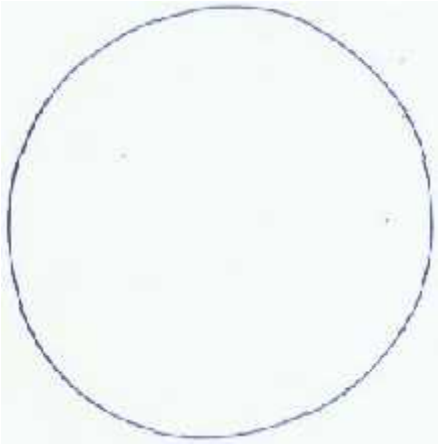
3) PLATELET COUNT

4) BLOOD UREA

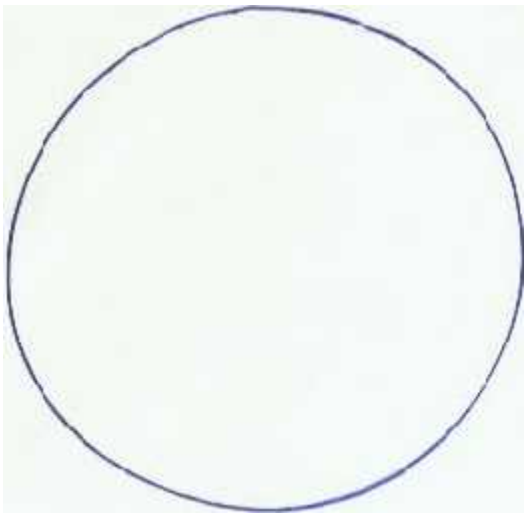
5) SERUM CREATININE

6) SERUM URIC ACID

V) FUNDUS PICTURE **OD**



OS



V) DIAGNOSIS

VI) REMARKS

VII) ANY OTHER COMMENTS :

Signature of examiner **Signature of patient** **Signature of guide**

CONSENT FOR PARTICIPATION IN RESEARCH STUDY

A ONE YEAR CROSS SECTIONAL STUDY OF FUNDAI, CHANGES IN PATIENTS WITH PREGNANCY INDUCED HYPERTENSION ATTENDING KLE'S PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELGAUM

Principal Investigator; Dr. Ayush Singal.

Since you have been diagnosed as a case of raise in blood pressure due lo pregnancy (Preeclampsia), you are eligible to be a part of the above study and hence asked lo participate. This research is about retinal changes occurring in eye due to raise in blood pressure caused by pregnancy. The result of this study may be helpful in preventing ocular problems which ranges from slight burning of vision to total loss of vision.

If you agree to participate we would ask you relevant clinical history and do examination, especially fundus examination for which we have to dilate both your pupils by putting eye drops 2 or 3 times so as to have a clear view of entire retina.

Your decision to participate or not in the study will not affect the quality of treatment you receive. Further you may withdraw from this study at any time.

All the information regarding the subject of research or that collected will be informed to you. This information is kept confidential to extent permitted by the law. Any information which identifies you will not be released without your written consent. This study does not have any damaging aspects and there is no chance of injury. There is no extra cost incurred by you but however you will have to pay for the investigations and the procedure which is a part of the protocol of treatment. There is no

commitment for any reimbursement or compensation for your participation. Your participation in this study is entirely voluntary. You may withdraw from the study at any time or can be excluded from the study.

If you have any questions about your rights on research as research participant, you may contact Dr. V.D. Patil, Principal and Chairman, J,N. Medical College Institutional Ethical Committee for Human Subjects Research, Phone no,O831-2471350 at J. N Medical College, Belgaum.

If any complications and questions regarding this study, you may contact Dr.Ayush Singal, Postgraduate, Department of Ophthalmology, J. N. Medical College, Mobile : 9986594520, OR

Dr. Rekha B.K., Professor, Department of Ophthalmology, J.N. Medical College, Belgaum.

Signature of subject Date:

Name:

Signature of authorized representative Date:

Name:

Relation to the subject:

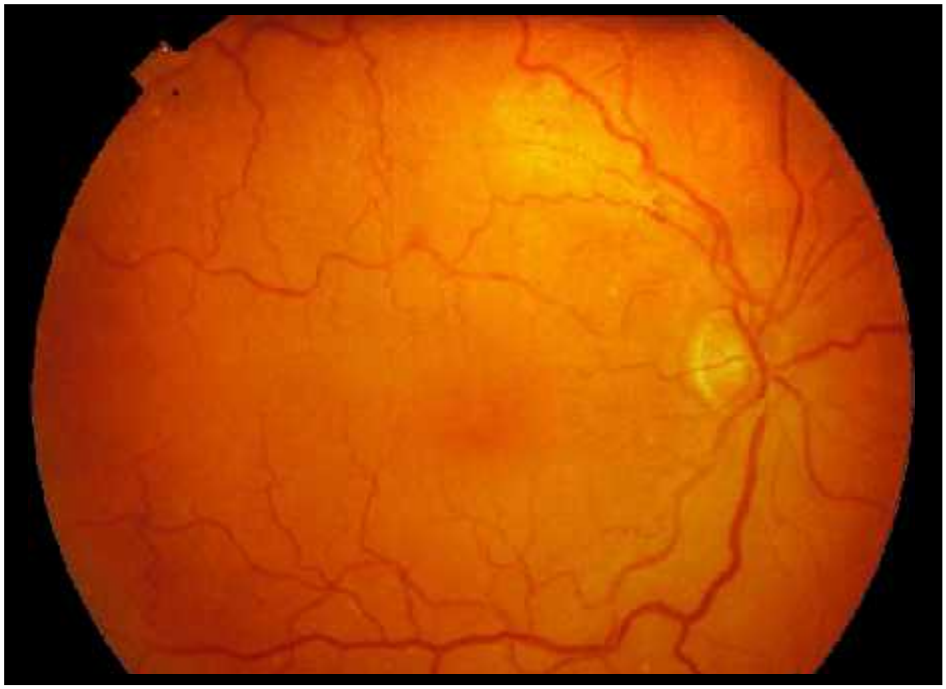
Signature of Witness: Date:

Name: _____.

Signature of the Investigator Date:

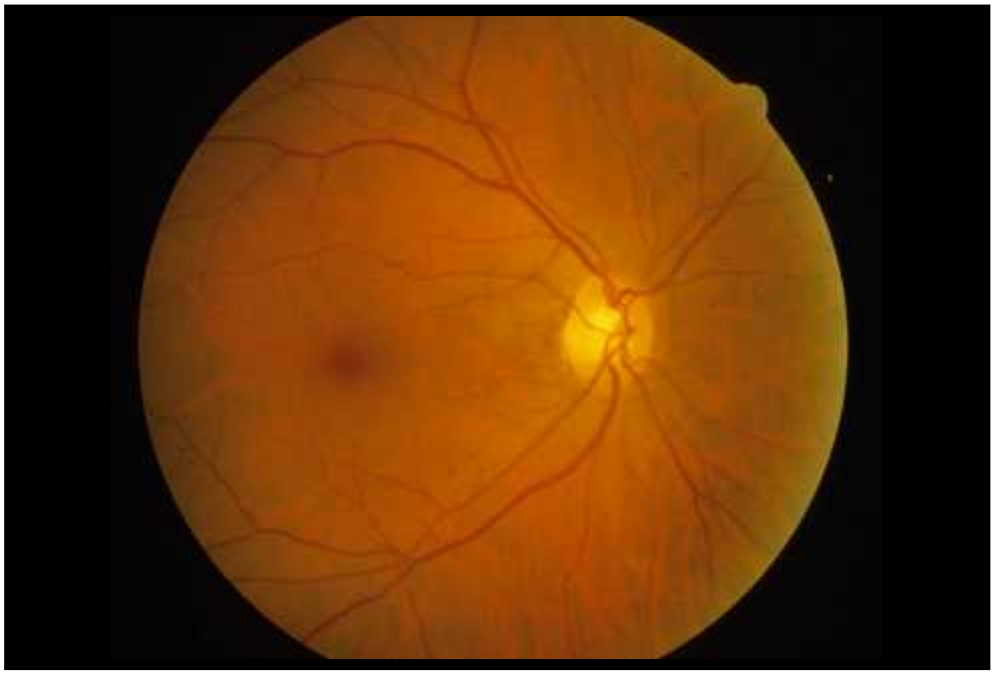
Name:

PHOTOGRAPH NO: 1



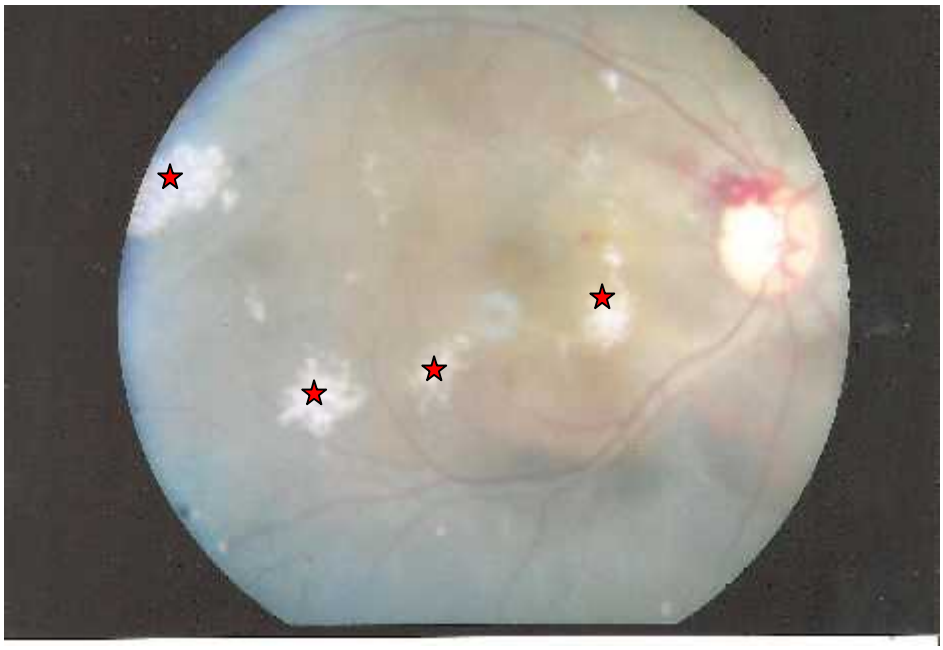
Fundus photograph of a patient showing generalized arteriolar narrowing

PHOTOGRAPH NO: 2



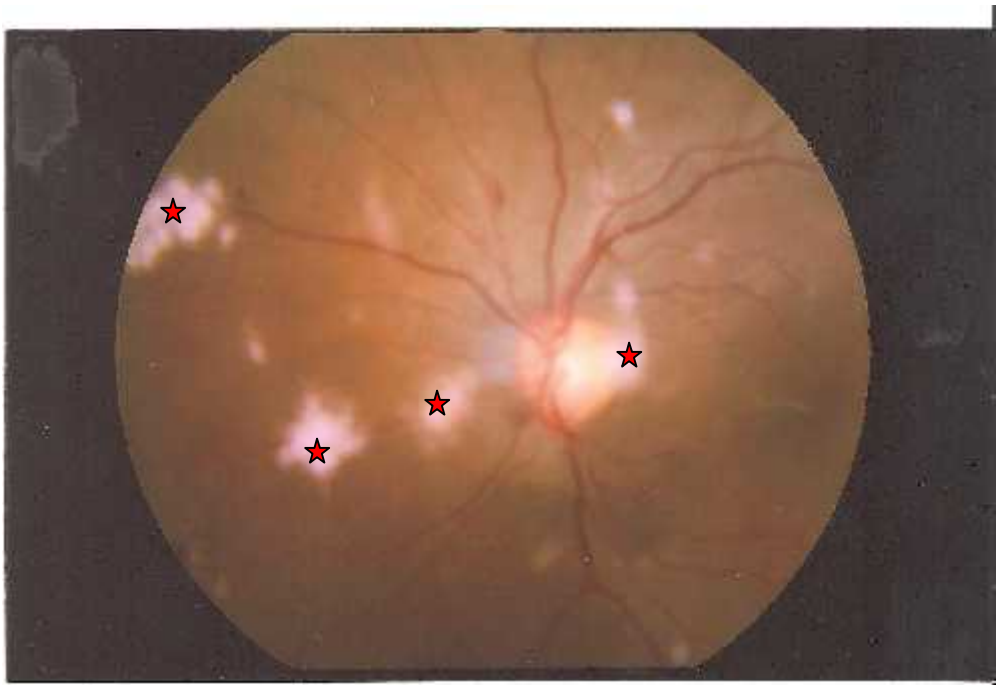
Fundus photograph of a patient showing arterio-venous crossing changes along superotemporal vascular arcade

PHOTOGRAPH NO: 3



Fundus photograph of a young patient showing splinter hemorrhage at superior disc margin

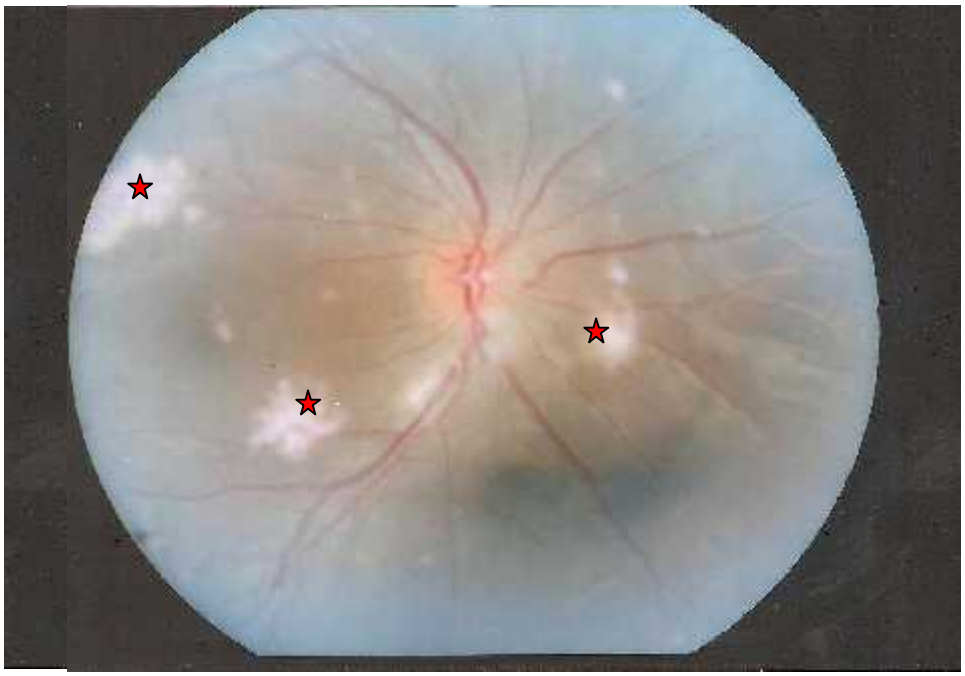
PHOTOGRAPH NO: 4



Fundus photograph of a young primigravida showing superficial flame shaped hemorrhage along superotemporal vascular arcade.

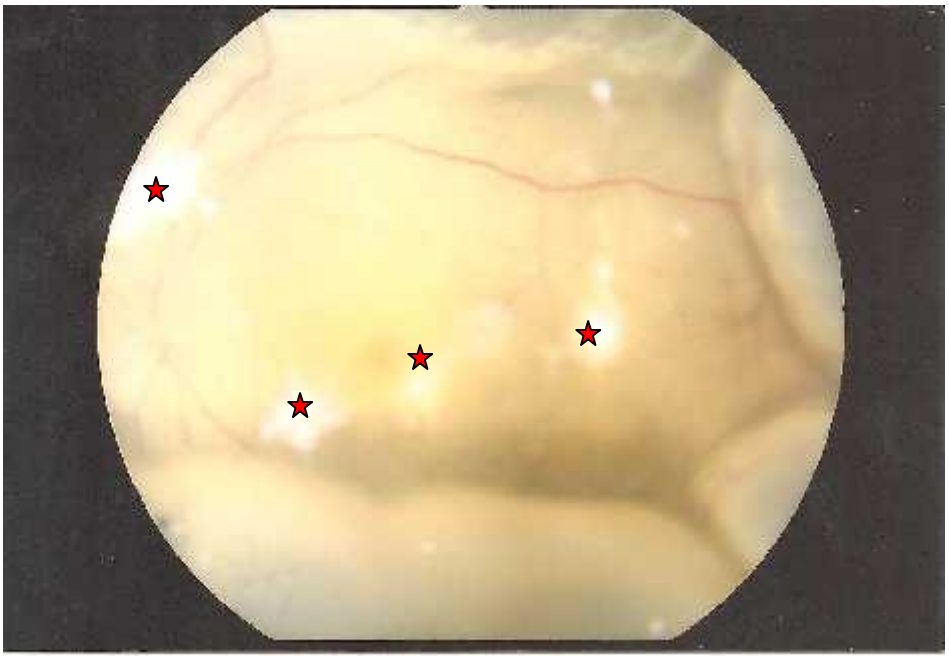
★ Please ignore artifacts in the photograph (marked with red star)

PHOTOGRAPH NO: 5



Fundus photograph of a full term primigravida showing blurring of disc margin.

PHOTOGRAPH NO: 6



Fundus photograph of a young primigravida showing bullous exudative retinal detachment.

★ Please ignore artifacts in the photograph (marked with red star)

MASTER CHART

S.No.	Name	Age (in yrs)	I.P. No.	Duration of pregnancy (in weeks)	Positive history prior to pregnancy	Complaints during pregnancy	Obstretric history	Palor	Pedal Edema	Pulse (in beats/min)	B.P at Admn(in mm Hg)	BP at time of fundoscopy (in mm of Hg)	Vision	OCULAR EXAMINATION										INVESTIGATIONS						Diagnosis
														Anterior or segment	FUNDOSCOPY									Urine Protein	HB% (in gram %)	Platelet counts (in lakhs /cu. mm)	Blood urea (in mg%)	S. Creatinine (in mg%)	S. Urica acid (in mg%)	
															Glow	Media	Disc	C:D Ratio	Blood Vessels	A-V Crossing changes	A:V Ratio	Background	Macula							
1	SATYAWWA.B.T	28	206545	39	NIL	CON,BV,FL,H, BSP, LS	G2P1A0L1	+	+	98	160/100	140/100	>6/60	NIL	P	C	N	0:3	GAN +++	SS	1:2	GER	N	++	6.4	1.52	43	1	8.4	RD
2	MADHURI M. PATIL	23	207571	40	NIL	BV,H,LS	G1P1A0L0	+	+	78	160/100	160/100	>6/60	NIL	P	C	BM	CO	GAN++	NIL	1:2	N	N	P	8	1.94	8.3	0.4	6	GR IV RT.
3	RANJANA.P.G	25	213398	28	NIL	BV,H,LS	G2P1A0L1	-	+	92	180/120	160/110	>6/60	NIL	P	C	SPH	O:3	GAN++	NIL	1:2	HG+	N	++	13.5	0.96	28	0.7	8.6	GR III RT.
4	BHARATI .S.H	20	217636	32	NIL	LS	G2P1A0L1	+	+	80	160/94	160/80	>6/60	NIL	P	C	N	O:3	GAN++	NIL	1:2	N	N	+	3.5	1.52	19	0.9	9.1	GR I RT.
5	MANISHA.N.M	24	217891	37	NIL	H, LS	G1P1A0L0	-	+	86	150/90	150/90	>6/60	NIL	P	C	N	O:3	GAN+++	NIL	1:2	N	N	+	11.4	2.45	14	0.6	4.4	GR I RT.
6	NEETU.B.P	20	218203	32	NIL	BV,H,D,LS	G1P1A0L0	-	+	94	220/130	180/110	>6/60	NIL	P	C	BM	0:2	GAN +	NIL	1:2	HG+, CWS+, GER	ME+	+++	13	1.6	46	1.1	8.9	RD
7	JAYASHREE.G.B	20	219114	39	NIL	BV, LS	G1P1A0L0	-	+	88	150/100	150/100	>6/60	NIL	P	C	N	0:3	FAN +	NIL	2:3	N	N	+	11.2	1.52	12	0.5	3.1	GR I RT.
8	SARITA .R.P	22	218960	37	NIL	BV,H LS	G1P1A0L0	-	+	86	140/100	130/80	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	++	10.8	2.69	22	1.1	6.2	N FUN
9	PREMA.P.H	20	219179	39	NIL	H, LS	G1P1A0L0	-	+	88	140/90	130/90	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	11.8	2.13	11	0.6	5.5	N FUN
10	VEENA .V.H	24	218982	29	NIL	H, LS	G3P1A1L1	-	+	84	160/120	150/100`	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	++	12.7	2.04	36	0.5	6.9	GR I RT.
11	MAYA.B.P	27	219035	29	NIL	NIL	G4P1A2L1	-	+	96	170/120	140/90	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	++++	10.6	1.62	28	0.7	5	N FUN
12	ANJUM.S	31	219718	32	NIL	H, LS	G1P1A0L0	-	+	88	180/120	180/120	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	++	12	2.6	28	0.4	6.1	N FUN
13	GEETA.D.J	20	219745	39	NIL	NIL	G2P1A0L1	-	+	86	130/110	130/90	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	12	1.98	12	0.4	5.8	N FUN
14	RASHMI.P.R	19	220387	40	NIL	H, LS	G1P1A0L0	-	+	90	150/106	130/80	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	13.9	2	12	0.4	5.3	N FUN
15	NAGAWWA	30	220371	40	NIL	LS	G4P2A1L2	-	+	98	200/120	160/100	>6/60	NIL	P	C	N	0:3	GAN++	NIL	1:2	N	N	++++	10.4	2.12	24	0.8	6.5	GR I RT.

S.No.	Name	Age (in yrs)	I.P. No.	Duration of pregnancy (in weeks)	Positive history prior to pregnancy	Complaints during pregnancy	Obstretric history	Palor	Pedal Edema	Pulse (in beats/min)	B.P at Admn(in mm Hg)	BP at time of fundoscopy (in mm of Hg)	Vision	OCULAR EXAMINATION										INVESTIGATIONS						Diagnosis
														Anterior or segment	FUNDOSCOPY									Urine Protein	HB% (in gram %)	Platelet counts (in lakhs /cu. mm)	Blood urea (in mg%)	S. Creatinine (in mg%)	S. Urica acid (in mg%)	
															Glow	Media	Disc	C:D Ratio	Blood Vessels	A-V Crossing changes	A:V Ratio	Background	Macula							
16	KAVITA.R.C	20	220534	34	NIL	H, LS	G1P1A0L0	-	+	86	150/112	130/90	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+++	13.6	4.11	24	0.6	6.6	N FUN
17	SHALAN.S.D	27	221039	28	NIL	BV, H	G4P2A1L2	+	+	92	160/96	160/90	>6/60	NIL	P	C	N	0:3	GAN +	NIL	1:2	N	N	+	4.9	2.29	70	2	11	GR I RT.
18	JYOTI S.D	28	221527	23	NIL	BV , H, LS	G3P1A1L1	-	+	80	170/110	170/100	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	++	12	4	20	0.6	5.7	N FUN
19	NIRMALA.V.H	27	222351	40	NIL	H, LS, HAD PIH AN IUD IN PP	G2P1A0L0	-	+	80	140/90	130/80	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	++	11.5	3.7	13	0.4	4.6	N FUN
20	SAVITA.C.M	19	222032	39	NIL	B.V. , H, LS	G1P1A0L0	-	+	80	160/100	150/100	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	10.5	1.92	19	0.4	10	N FUN
21	PRAVEEN.D.D	18	222054	29	NIL	H, LS	G1P1A0L0	-	+	82	140/100	130/90	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	++++	10.9	2.12	38	1.1	5.9	GR I RT.
22	SHIVALILA	23	222231	40	NIL	LS	G3P0A2L0	-	+	98	140/90	130/80	>6/60	NIL	P	C	N	O:3	GAN+	NIL	3:5	N	N	+	12.4	1.78	9	0.4	4.9	GR I RT.
23	LAXMI.B	20	222691	40	NIL	LS	G1P1A0L0	-	+	88	150/80	140/90	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	++	12	2.4	10	0.4	2.5	N FUN
24	GAYATRI	25	222716	40	NIL	H, LS	G1P1A0L0	-	+	92	170/120	160/110	>6/60	NIL	P	C	N	0:3	GAN+	GS	3:5	N	N	++	8	2.3	12	0.6	2.7	GR II RT.
25	PAVITRA	21	222898	41	NIL	BV, LS	G3P0A2L0	-	+	88	150/94	150/90	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	++	12	1.96	10	0.3	3.2	N FUN
26	MEENAL	22	221786	39	NIL	CON, H , LS	G1P1A0L0	-	+	110	180/100	150/90	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	++	9.8	4.42	17	0.9	9	GR I RT.
27	SOUMYA	27	223419	32	NIL	CON, BV, H, LS	G2P0A1L0	-	+	90	150/90	150/90	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	+++	12	2.96	18	0.8	5.6	GR I RT.
28	NEETA.D	20	223513	41	NIL	LS	G2P0A1L0	-	+	104	150/96	150/90	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	12.5	3.62	10	0.5	3.8	N FUN
29	JAYSHREE	32	223572	39	NIL	LS	G1P1A0L0	-	+	86	150/90	150/90	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	11.9	1.92	22	0.7	4.5	N FUN
30	CHITRA.C	20	223491	40	NIL	LS	G1P1A0L0	-	+	86	142/94	140/90	>6/60	NIL	P	C	N	0:3	GAN +	NIL	1:2	N	N	++++	10.5	3.5	12	0.4	4.8	GR I RT.

S.No.	Name	Age (in yrs)	I.P. No.	Duration of pregnancy (in weeks)	Positive history prior to pregnancy	Complaints during pregnancy	Obstretric history	Palor	Pedal Edema	Pulse (in beats/min)	B.P at Admn(in mm Hg)	BP at time of fundoscopy (in mm of Hg)	Vision	OCULAR EXAMINATION										INVESTIGATIONS						Diagnosis
														Anterior or segment	FUNDOSCOPY									Urine Protein	HB% (in gram %)	Platelet counts (in lakhs /cu. mm)	Blood urea (in mg%)	S. Creatinine (in mg%)	S. Urica acid (in mg%)	
															Glow	Media	Disc	C:D Ratio	Blood Vessels	A-V Crossing changes	A:V Ratio	Background	Macula							
31	DILSHAD	20	223587	37	NIL	BV, LS, H	G1P1A0L0	-	+	86	170/90	150/90	>6/60	NIL	P	C	N	0:4	GAN ++	NIL	1:2	N	N	+	12	2.6	12	0.6	6.3	GR I RT.
32	TRISHALA	23	223598	38	NIL	LS	G4P3A0L1	-	+	92	140/100	140/100	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	11	2	14	0.6	5.6	GR I RT.
33	MUMTAZ	22	223559	36	NIL	BV, H, LS	G2P1A0L1	-	+	96	170/110	160/100	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	13	3.66	10	0.4	3.8	N FUN
34	SUNITA.B	31	223765	37	NIL	LS, HAD PIH WITH IUD IN PP	G2P1A0L0	-	+	92	146/104	140/100	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	++	12	3.06	10	0.4	5.1	N FUN
35	SUMAN.S	19	223320	39	NIL	LS	G1P1A0L0	-	+	90	150/90	140/90	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	10.5	1.98	15	0.6	4.3	GR I RT.
36	JYOTHI.I	28	224337	29	NIL	BV, H, LS	G3P2AOL2	-	+	84	180/110	170/100	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	+	13.4	1.98	25	0.4	5.1	GR I RT.
37	ANUSUYA	25	224807	38	NIL	BV , H, LS. 1ST P-ECLAMPSIA. 2ND P-ABORTION	G3P1A1L1	-	+	86	140/90	140/90	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	9.8	2.5	12	0.5	5.1	GR I RT.
38	SAPNA.N	22	224348	39	NIL	H , LS	G1P1A0L0	-	+	86	150/100	140/90	>6/60	NIL	P	C	N	O:3	N	NIL	2:3	N	N	+	11.9	1.97	31	0.5	6.6	N FUN
39	ANNAPURNA.M.S	22	224364	40	NIL	LS	G1P1A0L0	-	+	92	150/100	150/100	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	10.8	1.64	11	0.5	5.1	N FUN
40	PARVATI	25	224416	39	NIL	LS	G2P1A0L1	-	+	86	150/100	140/90	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	11	2.04	10	0.4	4.9	N FUN
41	MANGAL	36	224565	35	NIL	BV, LS	G2P1A0L1	+	+	88	150/100	140/90	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	12.2	1.74	23	0.8	5.6	N FUN
42	SUNITA	25	222769	32	NIL	LS	G3P0A2L0	-	+	84	140/90	140/90	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	12	2.76	10	0.6	2.9	N FUN

S.No.	Name	Age (in yrs)	I.P. No.	Duration of pregnancy (in weeks)	Positive history prior to pregnancy	Complaints during pregnancy	Obstretric history	Palor	Pedal Edema	Pulse (in beats/min)	B.P at Admn(in mm Hg)	BP at time of fundoscopy (in mm of Hg)	Vision	OCULAR EXAMINATION										INVESTIGATIONS						Diagnosis
														Anterior or segment	FUNDOSCOPY									Urine Protein	HB% (in gram %)	Platelet counts (in lakhs /cu. mm)	Blood urea (in mg%)	S. Creatinine (in mg%)	S. Urica acid (in mg%)	
															Glow	Media	Disc	C:D Ratio	Blood Vessels	A-V Crossing changes	A:V Ratio	Background	Macula							
43	SAVITA	22	224691	40	NIL	H, LS	G1P1A0L0	-	+	90	140/100	140/100	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	++	11	2.04	20	0.7	5.3	N FUN
44	ANUSUYA.C.S	25	224807	40	NIL	H, LS	G3P1A1L1	+	+	80	150/80	140/80	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	11	1.92	15	0.4	5.6	GR I RT.
45	SANTOSHI.G.P	29	225239	34	NIL	H, LS	G1P1A0L0	-	+	92	150/110	150/100	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	10	1.86	10	0.6	5.2	GR I RT.
46	AFIRINA	18	225393	39	NIL	CON, BV, H, LS,, BS	G2P1AOL 1	-	+	98	140/100	140/100	>6/60	NIL	P	C	BM	CBM	GAN ++	NIL	1:2	N	N	+	12.1	3.4	12	0.5	6.5	GR IV RT.
47	SHILPA.M	26	225361	34	NIL	LS, HAD PIH WITH IN PP	G2P1A0L1	-	+	86	140/100	140/100	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	+	11.3	3.28	15	0.3	3.6	GR I RT.
48	SHEETAL	24	225735	41	NIL	LS	G2P1A0L1	+	+	88	170/110	150/100	>6/60	NIL	P	C	N	0:3	FAN +	NIL	1:2	N	N	+	7.4	1.66	20	0.6	5.9	GR I RT.
49	SUNITA.Y	21	225614	41	NIL	LS	G1P1A0L0	-	+	82	150/100	150/100	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	8	1.78	12	0.4	3.6	N FUN
50	RASHIDA	27	225957	39	NIL	BV, H, LS	G2P1A0L1	-	+	102	170/120	160/100	>6/60	NIL	P	C	N	0:3	GAN +	NIL	3:5	N	N	+	9.4	2.02	11	0.6	7	GR I RT.
51	TEJASWINI. P. C	20	226045	40	NIL	LS	G1P1A0L0	-	+	98	150/100	150/100	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	++	10	1.64	12	0.4	3.9	N FUN
52	SHEHNAZ	22	635617	40	NIL	H, LS	G1P1A0L0	-	+	90	170/116	132/98	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	12.3	1.96	16	0.7	5.8	GR I RT.
53	LAXMIBAI.K.C	38	226402	29	NIL	H, LS	G7P2A4L1	-	+	92	160/110	150/100	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	+	10.9	1.6	11	0.6	7.4	GR I RT.
54	SANGEETA.P	21	635930	36	NIL	LS	G1P1A0L0	-	+	96	150/100	150/100	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	10.2	1.91	13	1	6.6	N FUN
55	SUJATA.M	28	226586	40	NIL	LS	G1P1A0L0	-	+	88	150/110	150/110	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	11.8	1.62	12	0.4	4.1	N FUN
56	BASAWWA.R.M	25	226570	39	NIL	H, LS	G3P2AOL 2	-	+	100	168/84	150/80	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	+	10	1.96	17	0.6	5.2	GR I RT.
57	NIKHITA.N	22	226507	36	NIL	BV, H, LS	G1P1A0L0	-	+	108	154/96	150/90	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	GER	N	++	9	1.4	43.3	1.2	6.4	RD

S.No.	Name	Age (in yrs)	I.P. No.	Duration of pregnancy (in weeks)	Positive history prior to pregnancy	Complaints during pregnancy	Obstretric history	Palor	Pedal Edema	Pulse (in beats/min)	B.P at Admn(in mm Hg)	BP at time of fundoscopy (in mm of Hg)	Vision	OCULAR EXAMINATION										INVESTIGATIONS						Diagnosis
														Anterior or segment	FUNDOSCOPY									Urine Protein	HB% (in gram %)	Platelet counts (in lakhs /cu. mm)	Blood urea (in mg%)	S. Creatinine (in mg%)	S. Urica acid (in mg%)	
															Glow	Media	Disc	C:D Ratio	Blood Vessels	A-V Crossing changes	A:V Ratio	Background	Macula							
58	ASHWINI	20	635137	36	NIL	LS	G1P1A0L0	-	+	86	150/100	140/90	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	10	1.86	17	0.5	7.9	GR I RT.
59	SMITA.G	24	635604	39	NIL	LS	G1P1A0L0	-	+	82	140/100	140/90	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	11.2	2.73	13	0.6	4.8	GR I RT.
60	RESHMA	18	224397	40	NIL	LS	G1P1A0L0	-	+	86	150/100	150/100	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	10	1.86	15	0.4	2.6	N FUN
61	MAHADEVI.H	28	226406	37	NIL	LS	G1P1A0L0	-	+	94	160/104	140/90	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	9	2.12	28	0.8	4.7	GR I RT.
62	MALAN.D	20	227277	38	NIL	CON, H, LS	G1P1A0L0	-	+	110	170/120	160/100	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+++	12	3.54	15	0.7	6.8	N FUN
63	SHANTA.B	35	230321	33	NIL	H, LS	G1P1A0L0	-	+	96	158/108	150/100	>6/60	NIL	P	C	N	0:3	GAN +	NIL	3:5	N	N	+	10.5	2.3	20	0.7	7.6	GR I RT.
64	KAMALLAWA.A.	19	229548	39	NIL	LS	G1P1A0L0	+	+	86	140/100	130/90	>6/60	NIL	P	C	N	0:3	N	NIL	2;3	N	N	+	9.2	1.64	10	0.4	2.8	N FUN
65	SULOCHANA.M.P	32	228958	39	NIL	LS	G2P1A0L1	-	+	94	140/100	140/100	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	12.8	1.92	10	0.4	5	N FUN
66	LAXMI.G	20	636378	37	NIL	BV, LS	G2P1A1L0	-	+	100	150/104	150/100	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	++++	13.5	1.88	11	0.9	6.8	GR I RT.
67	SAVITA.S	22	229533	39	NIL	LS	G1P1A0L0	-	+	80	130/100	130/100	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	11.5	1.67	12	0.5	3.6	GR I RT.
68	YALLAWA	22	229678	38	NIL	LS	G3P2AOL2	+	+	90	150/90	140/80	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	3.1	0.14	38	1.9	5.3	N FUN
69	SUVARNA	30	230122	39	NIL	CON, BV, H, LS	G1P1A0L0	-	+	110	160/110	160/100	>6/60	NIL	P	C	N	0:3	N	NIL	3:5	N	N	+	13.3	3.21	10	0.6	5	GR I RT.
70	VAISHALI	27	230649	38	NIL	H, LS	G1P1A0L0	+	+	94	152/110	150/100	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	++	8.7	0.6	36	1.1	8.3	N FUN
71	SHOBHA.B	20	230626	40	NIL	BV, H, LS	G1P1A0L0	-	+	92	160/100	160/100	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	8.6	1.85	15	0.6	4.6	N FUN
72	MAHANANDA.S.K	21	230773	39	NIL	H, LS	G1P1A0L0	-	+	88	156/90	156/90	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	10.4	2.06	14	0.4	7.2	N FUN
73	SHREYA.S	20	230653	37	NIL	LS	G2P0A1L0	-	+	120	142/90	150/100	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	2:3	N	N	+	9	1.88	11	0.4	2.9	GR I RT.

S.No.	Name	Age (in yrs)	I.P. No.	Duration of pregnancy (in weeks)	Positive history prior to pregnancy	Complaints during pregnancy	Obstretric history	Palor	Pedal Edema	Pulse (in beats/min)	B.P at Admn(in mm Hg)	BP at time of fundoscopy (in mm of Hg)	Vision	OCULAR EXAMINATION										INVESTIGATIONS						Diagnosis
														Anterior or segment	FUNDOSCOPY									Urine Protein	HB% (in gram %)	Platelet counts (in lakhs /cu. mm)	Blood urea (in mg%)	S. Creatinine (in mg%)	S. Urica acid (in mg%)	
															Glow	Media	Disc	C:D Ratio	Blood Vessels	A-V Crossing changes	A:V Ratio	Background	Macula							
74	SMITHA	21	231477	30	NIL	CON, BV, H, LS	G1P1A0L0	-	+	110	160/120	160/110	>6/60	NIL	P	C	N	0:3	GAN ++	GS	1:2	N	N	+	11.4	1.13	51	1.2	13.5	GR II RT.
75	SUNITA	27	231621	33	NIL	BV, H, LS	G3P1A1L1	-	+	88	150/80	150/80	>6/60	NIL	P	C	N	0:3	GAN +	NIL	3:5	N	N	++	7.8	2.43	24	0.8	9.1	GR I RT.
76	DRAKSHAYINI. A.H	30	232102	36	NIL	BV, H, LS	G1P1A0L0	-	+	86	160/110	150/100	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	++	10.9	2	17	0.5	5	GR I RT.
77	FAIROZA.M	28	232149	37	NIL	BV, H, LS	G2P1A1L0	+	+	90	200/120	160/110	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	++	9.3	2.11	16	0.6	5.8	GR I RT.
78	SUMA.B	26	232629	35	NIL	BV, H, LS	G2P1A0L1	-	+	88	150/96	140/90	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	15.1	1.92	15	0.4	5.8	N FUN
79	ASHWINI	26	232584	26	NIL	CON, LS	G1P1A0L0	-	+	106	180/90	160/80	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+++	8.4	3.14	44	1.4	9	N FUN
80	NANDINI	23	232957	38	NIL	LS	G1P1A0L0	-	+	86	140/94	140/90	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	9.4	2.11	21	0.6	6.4	N FUN
81	KAUSAR	24	233188	27	NIL	BV, H, LS	G4P2A1L2	-	+	96	200/130	170/120	>6/60	NIL	P	C	N	0:3	GAN ++	GS	1:2	N	N	++	10.2	2.71	26	0.6	6.1	GR II RT.
82	NEELOFER	23	233241	30	NIL	H, LS	G1P1A0L0	-	+	86	140/100	130/100	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	8.5	1.84	17	0.5	4.3	GR I RT.
83	BHARATI.I.K	24	233365	36	NIL	BV, H, LS	G2P1A1L0	-	+	82	160/90	160/90	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	+	11	1.82	16	0.5	6.2	GR I RT.
84	SHEETAL	19	233447	39	NIL	BV, H, LS	G1P1A0L0	-	+	107	160/110	152/110	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	+	11	2.25	19	0.7	6.5	GR I RT.
85	SWAPNA	24	233957	39	NIL	LS	G2P0A1L0	-	+	80	140/94	140/90	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	+	11.3	2.09	17	0.6	4.9	GR I RT.
86	ASHWINI	21	234167	41	NIL	BV, LS	G1P1A0L0	-	+	88	160/110	160/110	>6/60	NIL	P	C	N	0:3	GAN++	NIL	1:2	N	N	+	8.8	2.12	18	0.6	4.7	GR I RT.
87	CHAYA.N	19	234163	39	NIL	BV, LS	G1P1A0L0	-	+	90	156/104	150/100	>6/60	NIL	P	C	N	0:3	GAN +	NIL	3:5	N	N	+	9.8	2.31	15	0.4	4.6	GR I RT.
88	RAZIYA	23	234185	28	NIL	BV, LS	G1P1A0L0	-	+	86	150/114	150/100	>6/60	NIL	P	C	N	0:3	GAN +	NIL	3:5	N	N	+	13	1.45	19.6	0.6	5.9	GR I RT.
89	MANJULA	20	234366	35	NIL	BV, LS	G1P1A0L0	+	+	88	150/110	150/100	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	++	4.4	1.29	71	0.8	9.6	GR I RT.

S.No.	Name	Age (in yrs)	I.P. No.	Duration of pregnancy (in weeks)	Positive history prior to pregnancy	Complaints during pregnancy	Obstretric history	Palor	Pedal Edema	Pulse (in beats/min)	B.P at Admn(in mm Hg)	BP at time of fundoscopy (in mm of Hg)	Vision	OCULAR EXAMINATION										INVESTIGATIONS						Diagnosis
														Anterior or segment	FUNDOSCOPY									Urine Protein	HB% (in gram %)	Platelet counts (in lakhs /cu. mm)	Blood urea (in mg%)	S. Creatinine (in mg%)	S. Urica acid (in mg%)	
															Glow	Media	Disc	C:D Ratio	Blood Vessels	A-V Crossing changes	A:V Ratio	Background	Macula							
90	SAHISTA	22	234186	34	NIL	H,LS	G3P1A1L1	-	+	92	150/90	150/90	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	7.2	1.5	16	0.5	6.9	GR I RT.
91	SADIYA.S	28	234397	30	NIL	H, LS	G1P1A0L0	-	+	88	160/98	160/90	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	11.3	2.8	31	0.3	5.3	GR I RT.
92	RENU.C.S	30	234773	39	NIL	BV, H, LS	G2P0A1L0	-	+	100	170/110	160/100	>6/60	NIL	P	C	N	0:3	GAN++	NIL	1:2	N	N	+++	11.1	1.69	25	0.8	8.5	GR I RT.
93	UJWALA	30	234872	39	NIL	BV, H, LS	G1P1A0L0	-	+	100	200/120	180/110	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	++	11.2	2.1	18	0.6	6.6	GR I RT.
94	SAROJINI	25	235232	39	NIL	H, LS	G3P2AOL 2	-	+	84	150/110	140/100	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	10.2	2.54	20	0.7	4.6	N FUN
95	SARIKA.G	23	235541	39	NIL	H, LS	G2P0A1L0	-	+	92	150/100	150/100	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	+	9.6	1.8	21	0.8	7.6	GR I RT.
96	CHANNAMA.S.B	25	235679	40	NIL	BV, H, LS	G2P1AOL 1	-	+	90	160/100	160/100	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	11.3	1.97	16	0.6	4.8	GR I RT.
97	PUSHPLATA.N. D	27	235813	33	NIL	BV, H, LS	G1P1A0L0	-	+	86	210/120	170/110	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	GER	N	+	11	1.07	39	1	6.1	RD
98	KASHAWWA.V. D	20	235851	28	NIL	CON, BV, H, LS	G1P1A0L0	-	+	102	170/110	150/100	>6/60	NIL	P	C	N	0:3	GAN++	NIL	1:2	N	N	+	12	3.21	21	0.7	7.5	GR I RT.
99	LALITA.S	36	236330	39	NIL	BV, H, LS	G2P1A0L1	+	+	90	170/110	150/100	>6/60	NIL	P	C	N	0:3	FAN +	NIL	2:3	N	N	+	9.8	1.92	23	0.7	5	GR I RT.
100	REKHA.P	21	236498	27	NIL	BV, LS	G2P0A1L0	_	+	84	180/110	140/90	>6/60	NIL	P	C	N	0:3	FAN +	NIL	2:3	N	N	++	12.6	1.39	12	0.8	4.2	GR I RT.
101	SHANTAWWA.R .D	19	236539	30	NIL	BV, H, LS	G1P1A0L0	-	+	84	178/118	160/110	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	12.5	2.04	17	0.5	6.3	GR I RT.
102	FIROZA.J	28	236907	39	NIL	H, LS	G1P1A0L0	-	+	90	180/110	170/100	>6/60	NIL	P	C	N	0:4	N	NIL	2:3	N	N	+	10.2	2.12	10	0.5	6.7	N FUN
103	AYESHA.I	22	236942	35	NIL	H, LS	G1P1A0L0	+	+	82	170/100	170/100	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	8	1.88	20	0.6	4	N FUN
104	SHAKUNTALA. G	24	238319	37	NIL	H, LS	G3P1A2L0	-	+	88	150/90	140/90	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	+	9.4	1.91	16	0.5	5.5	GR I RT.
105	MANJULA.K.M	22	238268	39	NIL	H, LS	G1P1A0L0	+	+	86	156/108	150/100	>6/60	NIL	P	C	N	0:3	FAN +	NIL	2:3	N	N	++	10.5	3.08	13	0.4	2.7	GR I RT.

S.No.	Name	Age (in yrs)	I.P. No.	Duration of pregnancy (in weeks)	Positive history prior to pregnancy	Complaints during pregnancy	Obstretric history	Palor	Pedal Edema	Pulse (in beats/min)	B.P at Admn(in mm Hg)	BP at time of fundoscopy (in mm of Hg)	Vision	OCULAR EXAMINATION										INVESTIGATIONS						Diagnosis
														Anterior or segment	FUNDOSCOPY									Urine Protein	HB% (in gram %)	Platelet counts (in lakhs /cu. mm)	Blood urea (in mg%)	S. Creatinine (in mg%)	S. Urica acid (in mg%)	
															Glow	Media	Disc	C:D Ratio	Blood Vessels	A-V Crossing changes	A:V Ratio	Background	Macula							
106	GAURABI	22	238782	39	NIL	BV, H, LS	G1P1A0L0	+	+	92	200/110	140/100	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	+	9	2.12	17	0.6	4.4	GR I RT.
107	SHWETA.S	23	237918	37	NIL	H, LS	G1P1A0L0	-	+	86	150/90	150/90	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	10	1.76	16	0.5	4.3	GR I RT.
108	SAVITHA	20	235596	39	NIL	LS	G1P1A0L0	-	+	82	140/100	140/100	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	9	1.82	14	0.5	3.9	GR I RT.
109	SHIREEN	24	239756	39	NIL	LS	G1P1A0L0	-	+	96	140/96	140/90	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	11	2.06	14	0.5	3.7	GR I RT.
110	ROOPA.R	24	239827	32	NIL	H, LS	G1P1A0L0	-	+	86	160/110	150/100	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	+	9.5	1.72	16	0.6	3	GR I RT.
111	SAVITRI	30	239906	38	NIL	LS	G1P1A0L0	-	+	94	150/110	140/100	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	12.7	2.01	27	0.5	4.7	GR I RT.
112	SUREKHA	23	243764	38	NIL	LS	G1P1A0L0	-	+	92	140/90	140/90	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	8.1	1.73	7	0.2	4.3	N FUN
113	LAXMI.G	22	242853	35	NIL	BV, H, LS	G1P1A0L0	-	+	88	160/130	150/120	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	+++	10.5	2.25	34	0.5	5.9	GR I RT.
114	SHIVSHANKAR AMMA.S	32	243075	33	NIL	BV, H, LS	G3P1A1L1	-	+	96	160/110	150/100	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	12.6	1.94	6	0.6	5.6	GR I RT.
115	SHANTA.K	40	243439	32	NIL	H, LS	G3P0A2L0	-	+	94	160/100	150/90	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	10	1.88	19	0.9	5.9	GR I RT.
116	SANGEETA	27	243307	39	NIL	LS	G3P2AOL 2	-	+	96	140/100	130/90	>6/60	NIL	P	C	N	0:3	N	NIL	2:3	N	N	+	11	1.92	18	0.7	6	N FUN
117	SHEELADEVI	18	243514	26	NIL	BV,H, LS	G2P1A0L1	-	+	90	180/110	170/100	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	+	10.5	1.98	22	0.6	4.3	GR I RT.
118	NAGRATNA	30	243784	37	NIL	BV, LS	G1P1A0L0	-	+	78	140/90	140/90	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	+	10	2.05	29	1.2	9.3	GR I RT.
119	RADHIKA	23	243918	37	NIL	BV, H, LS	G1P1A0L0	-	+	92	180/130	170/110	>6/60	NIL	P	C	N	0:3	GAN ++	GS	1:2	SHG	N	++	14.3	1.77	27	1.1	8.4	GR III RT.
120	KAVITA.B	28	244893	35	NIL	CON, BV, H, LS	G1P1A0L0	+	+	86	190/110	160/100	>6/60	NIL	P	C	N	0:4	GAN ++	NIL	1:2	N	N	++	8	1.92	43	0.4	6.2	GR I RT.
121	DEEPA.G	20	245137	39	NIL	BV, H, LS	G2P0A1L0	-	+	88	160/104	140/90	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	8.5	2.48	20	0.5	5.1	GR I RT.

S.No.	Name	Age (in yrs)	I.P. No.	Duration of pregnancy (in weeks)	Positive history prior to pregnancy	Complaints during pregnancy	Obstretric history	Palor	Pedal Edema	Pulse (in beats/min)	B.P at Admn(in mm Hg)	BP at time of fundoscopy (in mm of Hg)	Vision	OCULAR EXAMINATION										INVESTIGATIONS						Diagnosis
														Anterior or segment	FUNDOSCOPY									Urine Protein	HB% (in gram %)	Platelet counts (in lakhs /cu. mm)	Blood urea (in mg%)	S. Creatinine (in mg%)	S. Urica acid (in mg%)	
															Glow	Media	Disc	C:D Ratio	Blood Vessels	A-V Crossing changes	A:V Ratio	Background	Macula							
122	GANGAWWA	35	345226	38	NIL	H, LS	G2P1A0L1	-	+	86	150/90	140/90	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	8.9	2.63	12	0.8	5.2	GR I RT.
123	IRRAVA.S	38	245301	38	NIL	BV, H, LS	G4P2A1L2	-	+	88	160/90	140/90	>6/60	NIL	P	C	N	0:4	GAN+	NIL	3:5	N	N	+	12	2.32	18	0.7	5.1	GR I RT.
124	SHOBHA.M	23	245444	37	NIL	LS	G2P1A1L0	-	+	84	140/90	140/90	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	9	1.98	15	0.5	3.8	GR I RT.
125	LAXMI.B	22	245595	38	NIL	LS	G1P1A0L0	-	+	88	140/90	140/90	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	+	11.5	2.64	13	0.5	4.1	GR I RT.
126	SUNITHA	34	247359	34	NIL	H, LS	G2P0A1L0	-	+	86	160/96	150/100	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	SHG	N	+	10	2.08	20	0.6	4.6	GR III RT.
127	SHANTA.N	30	234654	38	NIL	H, LS	G4P1A2L1	-	+	82	160/100	180/110	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	+	11.4	1.75	18	0.7	5.3	GR I RT.
128	MAHADEVI.S.K	20	247791	40	NIL	BV, H, LS	G1P1A0L0	-	+	92	170/120	150/100	>6/60	NIL	P	C	N	0:3	GAN ++	NIL	1:2	N	N	+++	8.5	3.45	22	0.6	7	GR I RT.
129	PREMA.M	18	266168	31	NIL	LS	G1P1A0L0	-	+	88	160/100	150/100	>6/60	NIL	P	C	N	0:4	GAN ++	NIL	1:2	N	N	++++	11.1	3	25	0.7	6.5	GR I RT.
130	GEETABAI.M.P	36	250010	36	NIL	H, LS	G1P1A0L0	-	+	90	160/100	160/100	>6/60	NIL	P	C	N	0:3	GAN+	NIL	3:5	N	N	++	9	2.53	25	1.3	6.2	GR I RT.