A STUDY ON FUNDUS FINDINGS IN PREGNANCY INDUCED HYPERTENSION

Submitted to

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

in partial fulfilment of the requirements

for the award of degree of

M.S. (BRANCH III)

OPHTHALMOLOGY

GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL

THE TAMILNADU DR. M. G. R. UNIVERSITY,

CHENNAI, TAMILNADU

APRIL 2014

CERTIFICATE

This is to certify that the study entitled **"A STUDY ON FUNDUS FINDINGS IN PREGNANCY INDUCED HYPERTENSION**" is the result of original work carried out by **Dr.Shruthi Suresh**, under my supervision and guidance at **STANLEY MEDICAL COLLEGE, CHENNAI.** The thesis is submitted by the candidate in partial fulfilment of the requirements for the award of **M.S Degree in Ophthalmology**, course from May 2011 to April 2014 at Stanley Medical College and Hospital, Chennai – 600001.

Prof. Dr. K.Kanmani, M.S., D.O.
Professor and Unit Chief
Department of Ophthalmology
Stanley Medical College and Hospital

Prof. Dr. K.BASKER, M.S., D.O
Professor and Head of the department
Department of Ophthalmology
Stanley Medical College and Hospital

Prof. Dr.S.GEETHA LAKSHMI, M.D, Ph.D
Dean
Government Stanley Medical College,
Chennai – 600001

DECLARATION

I, Dr. Shruthi Suresh solemnly declare that the dissertation titled "**A STUDY ON FUNDUS FINDINGS IN PREGNANCY INDUCED HYPERTENSION**" has been prepared by me.

This is submitted to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, in partial fulfilment of the requirement for the award of M.S., (ophthalmology) Branch – III degree course from May 2011 to April 2014 from Stanley Medical College and Hospital, Chennai - 600001.

Place : Chennai

Date : **Dr. SHRUTHI SURESH**

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INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : A study on fundus findings in preganancy induced hypertension

Principal Investigator : Dr.Shruthi Suresh

Designation : PG in M.S.(Ophthal)

Department : Department of Ophthalmology
Government Stanley Medical College,
Chennai-10

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 08.04.2013 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

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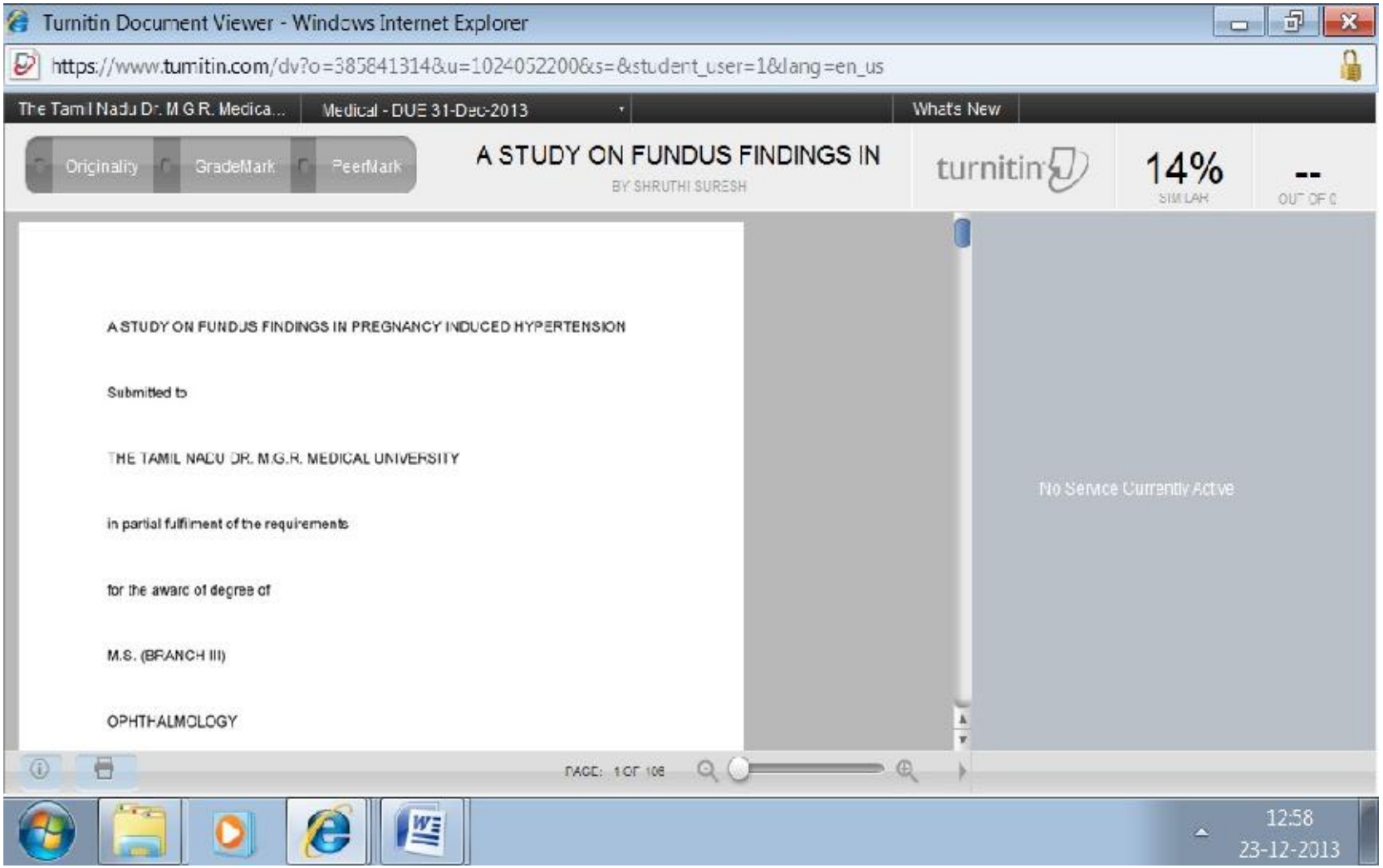
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A STUDY ON FUNDUS FINDINGS IN PREGNANCY INDUCED HYPERTENSION

ABSTRACT

INTRODUCTION : Hypertensive disorders in pregnancy are considered the major cause of maternal morbidity and mortality in developing as well as developed countries. It is the most common medical problem in pregnancy, complicating 7–10% of all pregnancies. The biggest limitation clinicians face is differentiating pregnancy induced hypertension from hypertension independent of pregnancy. Pregnancy can affect anywhere in the visual pathway from anterior segment to the visual cortex. Ocular sequelae of 30 – 100% is seen in patients with HELLP syndrome. Retinal and cerebral vessels share a lot of anatomical and embryological characteristics. Hence they may show similar patterns of damage from diseases like hypertension. This also suggests that examination of ocular fundus would provide a noninvasive view of intracranial vascular pathology. Fundus changes also plays an important role in determining the termination of pregnancy. This study has been done to understand if fundus findings correlate with the severity of hypertension, grades of proteinuria and levels of blood urea and serum uric acid.

AIM: The aim of this study is to determine the prevalence of retinal changes in pregnancy induced hypertension and to understand the association between retinal changes and severity of hypertension and proteinuria.

MATERIALS AND METHODS: A total of 100 patients admitted with pregnancy induced hypertension were included in this study. Patients with pre-existing hypertension, diabetes mellitus and renal disease and patients with raised blood sugar values were excluded from this study.

Their age and gravida were noted. Vision was checked and anterior segment examined. Fundus was examined. Blood pressure, grade of proteinuria, blood urea levels and serum uric acid levels were noted. Comparative study was done to find out if fundus findings had any correlation with the severity of hypertension, grades of proteinuria, blood urea and serum uric acid levels.

OBSERVATION : Maximum number of PIH cases were found in the age group of 21-25 years. 60% of the cases were seen in primigravidas. 54 patients had mild preeclampsia, 40 patients had severe preeclampsia and the rest 6 had eclampsia with seizures. Maximum number of patients(49) had grade 1+ proteinuria, 36 patients had grade 2+ proteinuria and only 15 patients had grade 3+ proteinuria. Maximum number of patients had either normal fundus (41%) or grade 1 hypertensive retinopathy (24%). 22% had grade 2, 6% had grade 3 and 2% of the cases had grade 4 hypertensive retinopathy. Another 2 % had macular edema. 3% of the cases studied showed central serous retinopathy. 54% of the cases studied had hypertensive retinopathy. This makes hypertensive retinopathy as the most frequently noted sign in PIH. Fisher's exact test was done between all the variables. There was no association of fundus findings with age or gravida of the patient. A significant positive correlation was found between fundus findings and severity of hypertension and proteinuria(P value < 0.001). Logistic regression analysis was also done, which gave similar results. In the present study, blood urea levels in mild preeclampsia group ranged from 9mg/dl to 40mg/dl with a mean value of 20.75mg%. In severe preeclampsia group, it ranged from 10 to 71mg/dl with a mean value of 27.67mg/dl. And in eclampsia group, blood urea levels ranged from 14 to 52mg/dl with a mean of 31.33mg/dl. Serum uric acid levels ranged from 2.6 to 11.2mg% in mild preeclampsia group with a mean of 4.98mg%. In severe preeclampsia group, it ranged from 3.1 to 9.2mg% with mean value of 5.82mg/dl. In eclampsia patients, the value ranged from

4.3 to 12.6mg% with a mean of 9.58mg%. This suggested a positive correlation between the severity of hypertension and blood urea and serum uric acid levels.

CONCLUSION : This study suggested a positive correlation of fundus findings with severity of hypertension and grade of proteinuria. The present study also suggested correlation of severity of hypertension with blood urea and serum uric acid levels. This study conveys the importance of routine fundus examination in all patients with pregnancy induced hypertension. Retinal changes is an important indicator in deciding the termination of pregnancy. Also, since there are anatomical and embryological similarities between the retinal and cerebral microcirculation, fundus changes may also suggest an underlying intracranial vascular pathology.

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PART – I

INTRODUCTION

Pregnancy is considered to be the only physiological state with most physiological parameters abnormal. There are profound anatomical, physiological and biochemical variations taking place in a woman during the short span of pregnancy.

Hypertensive disorders in pregnancy are considered the major cause of maternal morbidity and mortality in developing as well as developed countries. It is the most common medical problem in pregnancy, complicating 7 – 10% of all pregnancies. The biggest limitation clinicians face is differentiating pregnancy induced hypertension from hypertension independent of pregnancy.

DEFINITION

Pregnancy induced hypertension is defined as new onset hypertension with proteinuria or edema or both occurring after 20^{th} week of gestation and resolving shortly after delivery.

It was earlier called “toxaemia of pregnancy” since it was thought to be caused by the toxins present in blood during pregnancy. But this theory has been disproved and the term outdated.

REVIEW OF LITERATURE

HYPERTENSIVE RETINOPATHY

Retinopathy of pregnancy was first described in 1855 by von Graefe, just 4 years after the invention of ophthalmoscope by Von Helmholtz. In 1895, the first large series of retinopathy (35 cases) was described by Silex. He expressed the belief that retinopathy occurred once in about 3,000 pregnancies.

Miller was the first obstetrician to correlate fundus changes with pregnancy induced hypertension. Though he did his own ophthalmoscopy, he had an ophthalmologist to corroborate his findings. He suggested retinopathy not only as an indication for immediate termination of pregnancy, but also for sterilization to prevent future pregnancies.

In 1924, Chency did a study in a large number of PIH patients at the Boston Lying – In hospital. He found narrowing of retinal arterioles in most of the patients with marked hypertension. The degree of narrowing was dependent on the severity of hypertension and not on whether the condition was acute toxaemia or nephritis. He reasoned that

vasoconstriction in PIH is sudden and retina does not have the time to compensate for the diminished blood supply.

But in long standing arteriolar sclerosis, in spite of the more pronounced arteriolar constriction, the frequency of retinopathy is much less. This is because the change is slow developing and there is time for the retina to compensate.

In 1933, Masters did an ophthalmoscopic examination of 269 patients. He found a generalised uniform constriction of retinal arterioles in all the patients whose systolic blood pressure was more than 150mm of Hg.

Wagner in his study found constriction of arterioles in 70% of the women with PIH and considered to be usually the primary sign of retinal involvement. In 60% of these patients, these spastic lesions disappeared on termination of pregnancy and blood pressure returned to normal. In the rest 40%, elevated blood pressure persisted and organic lesions developed in the arterioles. At necropsy, the arterioles throughout the body were found to be permanently damaged in patients with retinopathy and he expressed the belief that majority of them would have persisted hypertension.

Sadowsky was the first one to correlate vascular changes with severity of PIH and foetal mortality and used progressive retinal arteriolar change as a guideline for termination of pregnancy.

In a study conducted by Schultz and O'Brien in 46 patients, they found normal fundus in 9, arteriolar spasm in 13, vascular sclerosis in 12 and retinopathy in 12 patients.

In 1960, Borras reported fundus findings in 150 patients and found narrowed arterioles in 77.4%, haemorrhages and exudates in 8% and disc edema in 4%.

In 1995, Capoor et al reported the presence of white centered haemorrhages in patients with PIH.

EXUDATIVE RETINAL DETACHMENT

First reported spontaneous retinal detachment in PIH was in 1855 by Von Graefe. Retinal detachment is a very uncommon finding in PIH, but its presence is now well recognised.

In a study done by Fry, he noted a 1.2% in preeclampsia and 10.4% in eclampsia.

Hallum reported 6 cases of exudative retinal detachment in a study done on 30 patients.

Mittelstrass and Wolghagen reported 1 case in the 973 cases studied.

Kronenberg reported 2 cases in 20,358 pregnancies and Bosco reported 1 case in 18,524 pregnancies.

Verderame in 1911 was the first to suggest pathological changes in the choroid to be the cause for the retinal detachment. Till then, it was believed that both choroid and retina plays a role in its pathogenesis. Kenny et al in 1972 was the first to prove the role of choroid in the aetiology of retinal detachment by doing colour fluorescein angiography. In 1996, Valluri et al performed diagnostic indocyanine green angiography in patients with PIH and established the role of choroidal vasculature in the pathogenesis of serous retinal detachment. Non perfusion was seen in the early phases of the angiogram. In the late phases of the angiogram, staining of the choroidal vasculature with subretinal leakage was seen along with multiple punctuate areas of blocked fluorescence.

Retinal detachments can exist independent of the presence of angiospasm or both of them can coexist.

In 1995, Menchini et al described a case of pigment epithelial tear following PIH in a 28 year old woman. She was found to have pigment epithelial tear in the macular region after abruption placenta and delivery. He presumed the tear to be an aftermath of RPE detachment.

According to ACOG (American College of Obstetricians and Gynaecologists), diagnosis of pregnancy induced hypertension is based on the following criteria:

Systolic BP of 140mm of Hg or more

Diastolic BP of 90mm of Hg or more

Increase of 30mm of Hg or more in systolic BP

Increase of 15mm of Hg or more in diastolic BP

This should be based on the tests done on 2 different occasions done at least 6hours apart.

DIFFERENT FORMS OF HYPERTENSIVE DISORDERS IN PREGNANCY

1. CHRONIC PERSISTING HYPERTENSION :: Hypertension (systolic BP >=140 mm Hg or diastolic BP >-90 mm Hg or both) that is present before 20weeks of gestation or prior to pregnancy. Elevated readings should be documented on more than one occasion.

2. GESTATIONAL HYPERTENSION : New onset hypertension (systolic BP >=140mm Hg or diastolic BP >=90mm Hg or both) presenting at or after 20 weeks of gestation without proteinuria or other features of preeclampsia.

3. PREECLAMPSIA :: Hypertension plus significant proteinuria(300mg/more of 24hour proteinuria) with or without edema.

4. SEVERE PREECLAMPSIA :: Severe hypertension with systemic disturbances like cerebral or visual disturbances, epigastric pain, oliguria, pulmonary edema, cyanosis etc.

5. ECLAMPSIA :: New onset Grand mal seizures in women with Preeclampsia.

But ACOG guidelines note that other causes may be more likely if seizures exist beyond 48 – 72 hours postpartum.

6. HELLP SYNDROME :: Serious systemic disorder associated with preeclampsia manifesting as haemolytic anemia, elevated liver enzymes and low platelet count. It is noted to occur in approximately 20% of women with preeclampsia.

ACOG GUIDELINES FOR DIAGNOSIS

1. Mild Preeclampsia :: Systolic BP 140 – 159 mm Hg or Diastolic BP 90 – 109 mm Hg or both recorded on 2 occassions recorded 4-6 hours apart. Proteinuria >= 300mg/24 hours or urine dipstick >= 1.

Starts after 20 weeks and normalises by 6 -12 weeks postpartum.

Edema is no longer considered criteria for diagnosis.

2. Severe Preeclampsia :: Systolic BP>=160 or diastolic BP >=110 while on bed rest.

Nephrotic range proteinuria

Sudden oliguria

CNS disturbances

Pulmonary edema / cyanosis

Epigastric / left upper quadrant pain

Liver dysfunction

Thrombocytopenia

Intrauterine growth retardation

Severe preeclampsia can be diagnosed even with mildly elevated BP if there is other evidence of significant end organ disease.

3. Eclampsia :: new onset of Grand mal seizures with no other identifiable cause.

Can occur with or without other prior symptoms like hypertension and proteinuria

4. HELLP Syndrome :: Related disorder of pregnancy with haemolytic anemia, elevated liver enzymes and lowered platelet count. It may or may not occur in conjunction with hypertension / preeclampsia, although majority will have preeclampsia symptoms.

Management is prompt delivery of foetus irrespective of presence or absence of hypertension or proteinuria.

RISK FACTORS

1. Extremes of maternal age (<20 or >35)
2. History of chronic hypertension
3. Previous history of Preeclampsia
4. Primigravida
5. Multiple gestation
6. Molar Pregnancy
7. Coexisting Diabetes Mellitus
8. Coexisting Renal Disease
9. Vascular disease
10. Women who are underweight/overweight
11. Preexisting connective tissue disorder
12. Thrombophilias
13. Female relative with history of PIH
14. Hydrops fetalis
15. Sickle cell disease

PATHOPHYSIOLOGY OF PIH

Pregnancy inducedchypertension is characterised by vasospasm (17), changes in coagulation system and disturbance in volume and BP control. It is considered a systemic vascular disorder where both hypertension and proteinuria implicate endothelium as the target of the disease. The hypertension of preeclampsia is characterized by peripheral vasoconstriction and decreased arterial compliance.

Vasospasm is due to increased sensitivity to Antithrombin III and imbalance between PGI2 and TXA1 activity. Arterial vasospasm may cause endothelial damage and contribute to increased permeability, leading onto edema. Endothelial damage further decreases intravascular volume, predisposing the woman with preeclampsia to pulmonary edema.

There is also an imbalance between proangiogenic and antiangiogenic factors during preeclampsia. The two important antiangiogenic factors implicated in preeclampsia are soluble vascular endothelial growth factor (VEGF) and soluble endoglin. Nitric oxide signalling is involved in vascular relaxation and is reduced in preeclampsia.

Immunologic factors may be playing an important role. Mother's immune system may perceive the placenta/fetus as a foreign protein(antigen). This leads to an abnormal immunologic response.

This theory is supported by the fact that there is increased incidence of preeclampsia/eclampsia in primigravidas/multiparous women pregnant by a new partner. Also there is increased incidence among women exposed to a large mass of trophoblastic tissue like in twin pregnancies and hydatidiform mole.

Another factor studied is genetic predisposition. Greater frequency of preeclampsia and eclampsia is seen among daughters and granddaughters of women with history of preeclampsia. This suggests an autosomal recessive gene controlling maternal immune response.

Diets lacking in nutrients especially proteins, calcium, sodium, magnesium, vitamins E and C can also be causative factor.

Preeclampsia can progress from mild disease to severe preeclampsia/ HELLP Syndrome/ eclampsia.

The main pathogenic factor is not increase in blood pressure, but poor perfusion because of vasospasm. Vasospasm impedes blood flow to all organs and raises blood pressure.

Function in organs like placenta, kidneys, liver and brain decreases by as much as 40 – 60%.

THEORIES IN RELATION TO PIH

Numerous theories have been proposed to explain the root cause of PIH.

Two leading theories are the immune theory and the genetic-conflict theory. The immune theory considers PIH as a maternal immune maladaptation to foreign foetal antigens derived from the paternal sperm. Exposure to paternal sperm for a long time enhances maternal immune tolerance. So previous gestations with a single partner increase the tolerance to subsequent gestations from the same partner. So there is an increased incidence of preeclampsia in teenage mothers and nulliparous mothers. Also there is an increased incidence of preeclampsia in multiparous mothers who change partners.

Genetic-conflict theory explains PIH as a consequence of the natural evolutionary conflict between the competing interests of fetal(paternal) genes and maternal genes during pregnancy. Evolution will select for fetal genes that maximise transfer of nutrients across the placenta, but the selection pressure for maternal genes is the limitation of transfer beyond an optimum. So, evolution will favour fetal genes that

would raise maternal blood pressure, and thereby placental perfusion. Thes genes will conflict against the maternal genes that act to limit the maternal blood pressure. Genomic imprinting happens as a result of this conflict. Certain genes are selectively expressed only from the maternally or paternally inherited chromosomes. This has a role in the development of preeclampsia. Oudejans et al. have identified a preeclampsia susceptibility locus on chromosome 10q22.1.

CLINICAL FEATURES

MILD PREECLAMPSIA (6)

1. Systolic BP of 140 – 159 mm Hg and diastolic BP of 90 – 109 mm Hg
2 1 + to 2+ Random proteinuria
3. Weight gain of 2lbs/week in second trimester and 1lbs/week in third trimester
4. Mild edema on face and upper extremities

SEVERE PREECLAMPSIA

1. Blood pressure >=160/100
2. 3+ - 4+ Random Proteinuria
3. Oliguria(<500ml/day)

4. Visual disturbances(typical scintillations and scotomata) thought to be due to cerebral vasospasm

5. Pulmonary Edema

6. Pedal Edema

7. Microangiopathic Hemolytic Anemia

8. Thrombocytopenia

9. Epigastric pain – Due to hepatic dysfunction with swelling and inflammation, leading to stretching of liver capsule. Pain is mostly constant and moderate to severe in intensity.

ECLAMPSIA

1. Seizures

2. patient may experience unconsciousness for a variable period of time.

3. Tonic clonic seizures

4. Prolongation of relaxation phase of Deep Tendon Reflexes

5. Brief periods of apnoea

6. Retinal changes may also occur

OCULAR COMPLICATIONS

The complications of preeclampsia extend to involve multiple organs and systems, the eye and visual system are no exception. Visual

symptoms concern up to 25% of patients with severe preeclampsia and 50% of patients with eclampsia.

Preeclampsia/eclampsia has various ocular manifestations. Blurred vision is the most common visual complaint. (3) The most common ocular finding is focal/generalized arteriolar narrowing. Other common symptoms are photopsia, visual field defects, sudden inability to focus, and in severe cases**,** complete blindness.

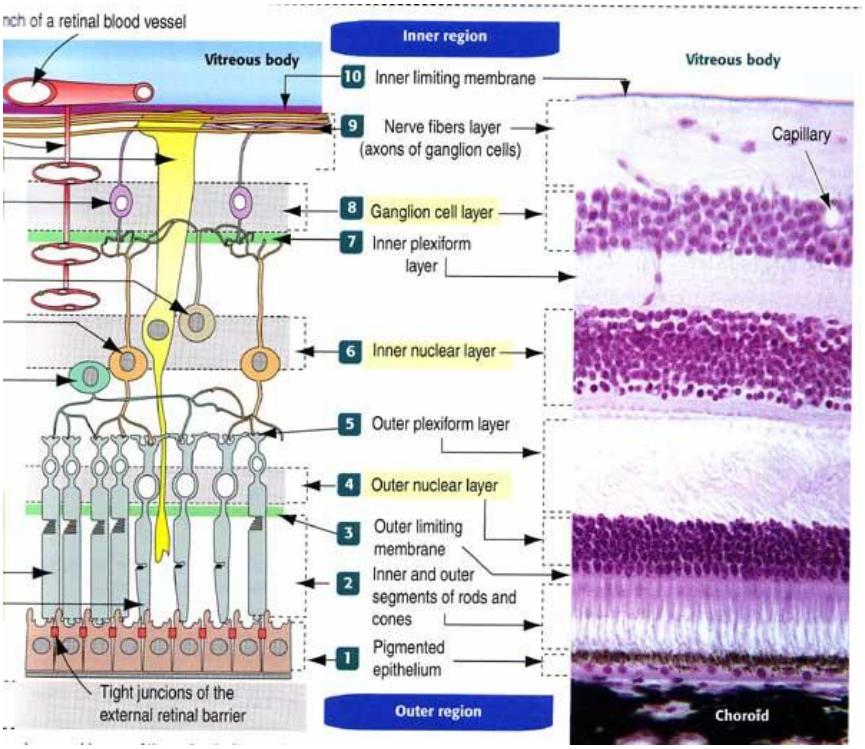
Complete blindness is rare, with an incidence of 1 – 3%. Blindness in preeclampsia/eclampsia syndrome can be due to the involvement of cortex, retina or optic nerve. Earlier, most cases of blindness were attributed to retinal pathology including vascular abnormalities, edema or retinal detachment and acute ischemic optic neuropathy as a result of decreased blood supply to the prelaminar portion of the optic nerve. But nowadays, more emphasis is being placed on cortical blindness.(4,5)

RETINAL AND CEREBRAL MICROCIRCULATION

Retinal and cerebral vessels share a lot of anatomical and embryological characteristics. Hence they may show similar patterns of damage from diseases like hypertension. This also suggests that examination of ocular fundus would provide a noninvasive view of intracranial vascular pathology.

The retinal vessel pathology is an important marker for stratification of patients' risk for having or developing cerebrovascular disease (27).

LAYERS OF RETINA



1. Retinal Pigment Epithelium
2. Layer of Rods and Cones
3. External limiting membrane
4. Outer nuclear layer
5. Outer plexiform layer
6. Inner nuclear layer
7. Inner plexiform layer
8. Ganglion cell layer
9. Nerve fibre layer
1o. External limiting membrane

ANATOMY OF RETINAL AND CHOROIDAL VASCULATURE

Retina receives its nutrition from 2 discrete circulatory systems – retinal blood vessels and choroidal blood vessels. Both are branches of Ophthalmic artery, which in turn is the first branch of Internal Carotid Artery. The major branches of Ophthalmic artery are Central Retinal Artery, Posterior Ciliary Arteries and Muscular branches.

There are 2 posterior ciliary artery in each retina –medial and lateral. Watershed area between both is vertically oriented zone situated between Optic disc and macula.

Anterior choriocapilaris is supplied by branches from long posterior ciliary arteries and anterior ciliary arteries. Posterior choriocapillaris supplied by short posterior ciliary arteries. Watershed zone is at the equator.

VENOUS drainage of choroid – Vortex veins are between 4 and 7 in number, one or two in each quadrant, located at equator. They drain into superior and inferior orbital veins, which drain into Cavernous sinus and Pterygoid plexus respectively.

Outer six layers of retina is supplied by Choriocapillaris and inner four layers by Central Retinal Artery. There is a small overlap at watershed zone at Outer Plexiform layer.

Central retinal artery and central retinal vein enter retina at Optic disc and seen in superficial nerve fibre layer. Central retinal artery is an end artery with no significant anastomoses. Central retinal artery divides into superior and inferior branches and later into superonasal, superotemporal, inferonasal and inferotemporal branches.

BLOOD SUPPLY OF THE RETINA

Outer four layers of the retina comprising the retinal pigment epithelium, layer of rods and cones, external limiting membrane and outer nuclear layer get their nutrition from choriocapillaris.

Inner six layers comprising outer plexiform layer, inner nuclear layer, inner plexiform layer, ganglion cell layer, nerve fibre layer and internal limiting membrane get blood supply from the central retinal artery.

The fovea is an avascular area supplied mainly by diffusion from the choriocapillaris.

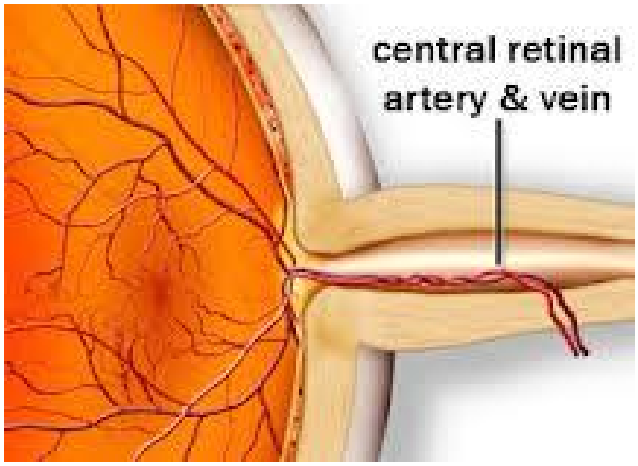
Macular region gets blood supply by small twigs from the superotemporal and inferotemporal branches of central retinal artery. Sometimes, cilioretinal artery is seen originating as a small hook within the temporal region of the optic disc. It runs towards the macula and supplies it, when present. It retains the central vision, even when central retinal artery occlusion is present.

Retinal vessels are end arteries. But anastomoses between the retinal vessels and ciliary system of vessels exist with the vessels which enter the optic nerve head from the arterial circle of Zinn or Haller.

Arterial circle of Zinn or Haller is formed by an anstomoses between 2 to 4 or more short posterior ciliary arteries and lies in the sclera around the optic nerve. From the arterial circle, branches run forward into the choroid, inward into the optic nerve and backward to the plial network.

CENTRAL RETINAL ARTERY

Central retinal artery arise from the ophthalmic artery near the optic foramen and courses ahead with 5 – 6 right angle bends.



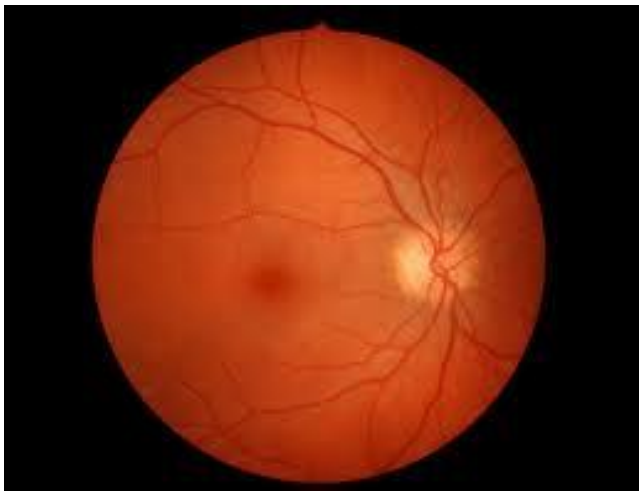
It runs forward below the optic nerve in a wavy course adherent to the dural sheath to about 10 -15mm behind the eyeball. At this point, the artery bends upward along the inferomedial part of the nerve and pierces the dura and arachnoid. It receives a covering from both during the process. In the subarachnoid space, it bends forward and after a short course, it again bends upward at nearly right angle and invaginates the pia to reach the centre of the nerve. The vessel is clothed by pia along with

the pial vessels. It is also surrounded by a sympathetic nerve plexus (nerve of Tiedemann).

In the centre of the optic nerve, the artery bends forward and then in company with the vein, it passes anteriorly and pierces the lamina cribrosa to appear inside the eye. Vein lies temporal to the artery.

In the optic nerve head, it lies superficially in the nasal part of the physiological cup, covered only by thin layer of glial tissue (connective tissue meniscus of Kuhnt) which closes the physiological cup. Here it divides into superior and inferior branches and further into nasal and temporal branches at or near the margin of the optic disc.

In the retina, all the four branches divide dichotomously as they proceed towards the ora serrata, where they end without anastomoses.



DISTRIBUTION OF RETINAL CAPILLARIES

CAPILLARY FREE ZONE – Around each of the layer of retinal arteries and veins, more prominent around arteries, measuring upto 100microns in diameter. Capillaries are absent in fovea(foveal avascular zone 400 – 500 microns in diameter) and far retinal periphery.

RETINAL CAPILLARY PLEXUS – Superficial in Superficial nerve fibre layer and Deep plexus between Outer nuclear and Inner plexiform layer. The deep capillary network is more dense and complex than the superficial. There are anastomotic capillaries which run from one to the other.

Three layered pattern of capillaries is seen particularly in macular region. The capillary network is especially well developed here.

Four layered pattern seen in and around the disc, where nerve fibre layer is thick. This is to support the extremely thick nerve fibre layer characteristic of this region.

Single layered pattern seen especially towards periphery of retina.

ARTERIOVENOUS CROSSINGS

Arteries are narrower than veins with arteriovenous ratio of 2:3. Arteries lie above the veins in 54 – 71 % of eyes.

At sites of AV crossings, there is thinning of ganglion cell, inner and outer nuclear layer. At points of AV crossings, arteries and veins share a common adventitious sheath. Lumen of vein at point of crossing is narrowed to approximately $2/3^{rd}$ the diameter of adjacent venous lumen. Retinal veins lose muscularis more peripherally in retina. So it is possible that veins at 2^{nd} and 3^{rd} crossings are more compressible.

In normal eyes, percentage of arteries being anterior to veins is 78% in superotemporal quadrant, 70% in inferotemporal quadrant and 60% in nasal quadrants.

BLOOD SUPPLY OF THE OPTIC NERVE

THE INTRAOCULAR PART

The surface nerve fibre layer is mainly supplied by capillaries from the retinal arterioles which anastomose with vessels of the prelaminar region. Sometimes this is supplemented by the cilioretinal artery.

The prelaminar region is supplied by vessels of the ciliary region. There is controversy regarding if these vessels are coming from

peripaillary choroidal vessels or from separate branches of short posterior ciliary arteries.

The lamina cribrosa region is supplied by the ciliary vessels derived from short posterior ciliary arteries and arterial circle of Zinn – Haller.

The retrolaminar region is supplied by both the ciliary nd retinal circulation with the former coming from recurrent pial vessels.

THE INTRAORBITAL PART

Supplied by periaxial and axial system of vessels.

The periaxial system is derived from six branches of internal carotid artery namely ophthalmic artery, long posterior ciliary arteries, short posterior ciliary arteries, lacrimal artery and central artery of retina.

The axial system supplies the axial part of optic nerve is derived from intraneural branches of central retinal artery, central collateral arteries which come from the central retinal artery before it pierces the nerve and the central artery of the optic nerve.

THE INTRACANALICULAR PART

It is supplied by the periaxial system of vessels fed mainly by branches from the ophthalmic artery.

THE INTRACRANIAL PART

This part is supplied exclusively from the periaxial system of vessels. Pial plexus comes from branches from internal carotid artery, branches from anterior cerebral artery, small recurrent branches from the ophthalmic artery and the twigs from the anterior communicating artery.

HISTOLOGY

Hypertension is mainly a disease of the arterial part of the capillaries. The outer layers differ for the artery, arteriole and capillaries.

Central Retinal Artery:

Tunica Intima composed of a single layer of endothelial cells, a subendothelial layer of circularly arranged elastic tissue and an internal elastic lamina composed of elastic fibrils.

Tunica Media composed of many layers of smooth muscle interspersed among elastic fibres.

Tunica Adventitia composed of mostly collagen with circular and longitudinal elastic fibres. It is the thickest layer.

Arterioles:

Tunica Intima with single layer of endothelium and sparse or absent elastic fibres.

Tunica Media composed of 2 – 4 layers of smooth muscles with sparse elastic fibres. It is ill defined.

Tunica Adventitia composed of loosely arranged collagen fibres.

Capillaries:

Composed of inner layer of endothelial cells, intramural pericytes and basement membrane.

BLOOD – RETINAL BARRIER

The endothelial cells of a normal retinal capillary are closely bound together about the lumen by intercellular junctions of the zonula occludens type. These junctions normally prohibit a free flow of fluids and solutes from the vascular lumen into the retinal interstitium and forms the blood-retinal barrier. There is absence of fluorescein leakage at these tight junctions.

The endothelial cells of retinal capillaries are encircled by a basement membrane around which is present the layer of pericytes(mural cells). The layer of pericytes is again surrounded by basement membrane. Normally the ratio of pericytes to endothelial cells is 1:1. But in certain diseases like diabetes mellitus, there is a relative decrease in the number of pericytes. With increasing age, there occurs a gradual decrease in the number of endothelial cells.

DIFFERENT FUNDUS CHANGES SEEN IN PREGNANCY INDUCED HYPERTENSION

Pregnancy can affect anywhere in the visual pathway from anterior segment to the visual cortex.

Incidence of preeclampsia in developed countries is almost 5% with a maternal mortality rate as high as 1.8%.

Ocular sequelae of 30 – 100% is seen in patients with HELLP syndrome.

1. Hypertensive Retinopathy and Choroidopathy
2. Cystoid Macular Edema
3. Serous Retinal Detachment
4. RPE lesions

5. Retinal Arterial Occlusions
6. Retinal venous occlusions (very rare)
7. Ischemic Optic Neuropathy
8. Ischemic Papillophlebitis
9. Optic Atrophy

HYPERTENSIVE RETINOPATHY

This is the most common fundus change(7) to be encountered in pregnancy. Hypertension has been ranked as the fourth largest mortality risk factor in the world. Hypertension affects precapillary arterioles and capillaries, anatomical loci of autoregulation and non perfusion. Elevation of systemic blood pressure leads to both focal and generalised arteriolar attenuation, presumably mediated by autoregulation. But the degree of attenuation depends on the amount of pre-existing sclerosis. So hypertensive retinopathy in its pure form is found only in young individuals.

Spasm and narrowing of retinal arterioles is reported in as many as 70% cases of toxaemia (18).

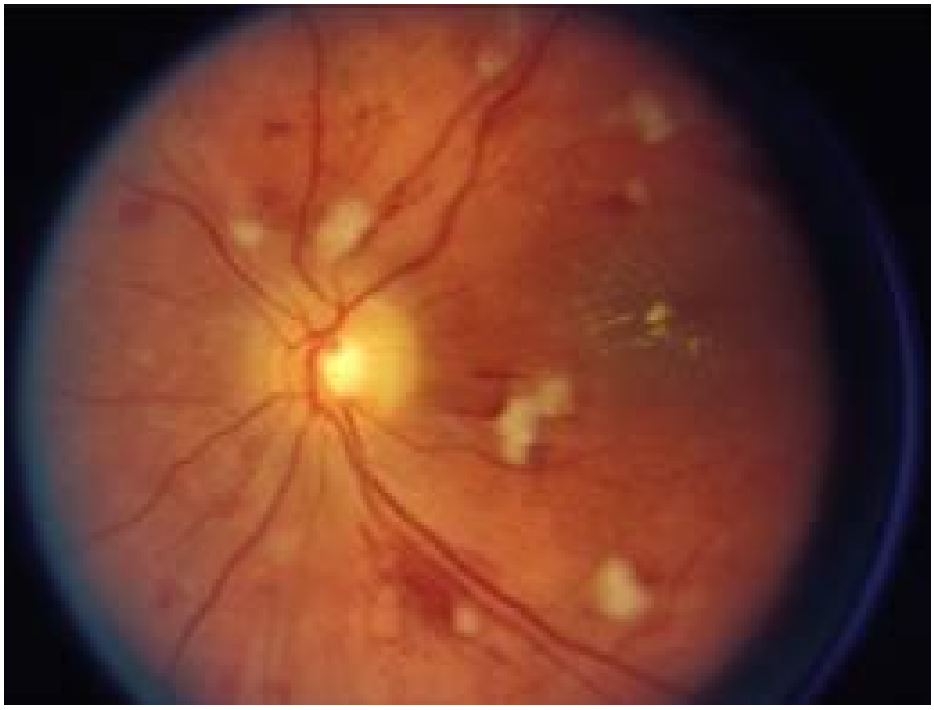
PATHOPHYSIOLOGY

When blood pressure becomes elevated, retinal arterioles tend to constrict to increase vascular resistance, to maintain steady perfusion to

the retinal tissue. Prolonged high blood pressure can lead to permanent arteriolar narrowing. There is no sympathetic innervations for retinal vessels. So this vasoconstriction is controlled by autoregulatory mechanisms (21). When the degree of hypertension exceeds the capacity of vessels to autoregulate, system fails and capillary blood succumb to elevated pressure. Prolonged exposure can lead to occlusion of terminal arterioles, capillary non perfusion, retinal ischemia, cotton wool spots, haemorrhages and retinal oedema (19).

Choroidal vessels have sympathetic innervations, which causes vasoconstriction in response to hypertension(8). If the blood pressure exceeds the capacity of sympathetic system to regulate perfusion, damage to choroidal vascular bed occurs. This leads on to choroidal occlusion and ischemia, ischemia of overlying retinal pigment epithelium and outer retina, exudative retinal detachments, and long term pigmentary changes (Elschnig's spots).

Signs



1. Arteriolar narrowing: may be focal or generalised. Focal narrowing is more of a diagnostic factor of raised blood pressure than generalised attenuation.

2. Supeficial haemorrhages: they are characteristically present in the superficial nerve fibre layer in the form of flame shaped haemorrhages. They are oriented in the direction of nerve fibre layer.their extent and degree indicate the severity of hypertension. They tend to absorb slowly and last even for 3months after the resolution of blood pressure. On fluorescein angiography, they

block out the background choroidal fluoroscein and obscure the capillary pattern.

3. Cotton wool spots: seen in severe hypertension (20). They appear as white fluffy opaque areas in the sensory retina. It results from an accumulation of axoplasm at a site adjacent to an area of vascular occlusion. Typical cotton wool spots are located in the superficial nerve fibre layer and arranged along the long axis. They are normally less than half a disc in diameter. They are almost always confined to the area adjacent to major vascular arcade. Cotton wool spots associated with arterial hypertension typically disappear after a period of 5 to 6 weeks. On fluorescein angiography, there will be capillary non perfusion. Adjacent to that there will be areas of capillary non perfusion. Surrounding the area of cotton wool spot, there will be uniform capillary dilatation,some atining of fluorescein or even leakage of fluorescein.

4. Vascular leakage: showing flame shaped haemorrhages and retinal edema. Chronic edema may result in deposition of hard exudates, rarely seen in PIH. Hard exudates represent an accumulation of lipid and/or protein within the sensory retina. They are formed in relation to leaking capillaries, very rarely seen in PIH. They are

normally deposited in the outer plexiform and inner nuclear layer but present in the macula superficially along the nerve fibre layer of Henle.

5. Disc edema: presence of disc edema is seen in grade 4 hypertensive retinopathy. Presence of accelerated hypertension with disc edema calls for malignant hypertension, a definite indicator for termination of pregnancy.

6. Arteriosclerosis: thickening of vessel wall characterized by intimal hyalinization, medial hypertrophy and endothelial hyperplasia. This leads on to changes at arteriovenous crossings. This sign is also indicative most probably of long standing hypertension.

Generalised narrowing of arterioles

This is most often the earliest sign seen in hypertensive retinopathy(12). The degree of narrowing can be assessed by comparing the calibre of the vessel with:

1) the average vessel calibre of individuals without hypertension

2) the calibre of the venule and expressed as a ratio. The normal AV ratio is 2:3

The former way of assessment is better because changes in the calibre of veins can affect the latter assessment.

Grades of generalised arterial narrowing

Grade 1 : calibre of the arteriole is 3/4th of the average calibre of normal arteriole or half the calibre of retinal vein.

Grade 2 : calibre of the arteriole is half the average calibre of normal arterioles or 1/3rd of the calibre of veins.

Grade 3 : calibre of the arteriole is 1/3rd the average calibre of normal arterioles or 1/4th the calibre of veins.

Grade 4 : thread like arteriole to even extreme levels of invisible arteriole.

Grades of focal constriction

Focal constriction of arterioles is seen when the diastolic pressure is more than 110mm of Hg. There will be focal decrease in the calibre of the arterioles. This change is temporary and disappears when blood pressure decreases.

Grade 1 : calibre of the narrowed part is 2/3rd of the calibre of the proximal segment.

Grade 2 : calibre of the narrowed part is ½ of the calibre of the proximal segment.

Grade 3 : calibre of the narrowed part is 1/3rd of the calibre of the proximal segment.

Grade 4 : artery is narrowed to the point that it is invisible beyond the point of constriction or it is visible as a fibrous cord.

Grades Of Arteriosclerosis

Grade 1 : subtle broadening of arteriolar light reflex (11), mild generalized arteriolar narrowing, particularly of small branches and vein concealment.

Grade 2 : obvious broadening of arteriolar light reflex and deflection of veins at arteriovenous crossings(**Salus sign**)

Grade 3 : 'copper-wiring' of arterioles, banking of veins distal to arteriovenous crossings(**Bonnet sign**), tapering of veins on both sides of the crossings(**Gunn sign**) and right angled deflection of veins

Grade 4 : 'siver-wiring' of arterioles associated with grade 3 changes.

Accelerated hypertension

This is characterized by fibrinoid necrosis of the arterioles with papilledema. Fibrinoid necrosis is not common in retinal arterioles and is usually observed in arteries and arterioles of choroid. Retinal haemorrhages, cottonwool spots and even capillary occlusions can occur.

CLASSIFICATIONS OF HYPERTENSIVE RETINOPATHY

THE KEITH – WAGNER – BARKER CLASSIFICATION (9)

Group 1 : minimal constriction of retinal arterioles with some tortuosity in patients with mild hypertension.

Group 2 : group 1 with definite focal narrowing and arteriovenous nicking.

Group 3 : group 2 with haemorrhages, exudates and vasospastic changes including arteriolar attenuation and cottonwool spots.

Group 4 : group 3 with optic disc edema.

SCHEIE CLASSIFICATION

Stage 0 : no visible retinal vascular abnormalities.

Stage 1 : diffuse arterial narrowing, especially in the smaller vessels with no focal constriction.

Stage 2 : more pronounced arteriolar narrowing, with focal areas of arteriolar constriction.

Stage 3 : severe focal and diffuse arterial narrowing retinal haemorrhages and/or exudates.

Stage 4 : stage 3 with disc swelling.

Dis edema starts resolving within few weeks after delivery, but haemorrhages takes time to disappear completely.

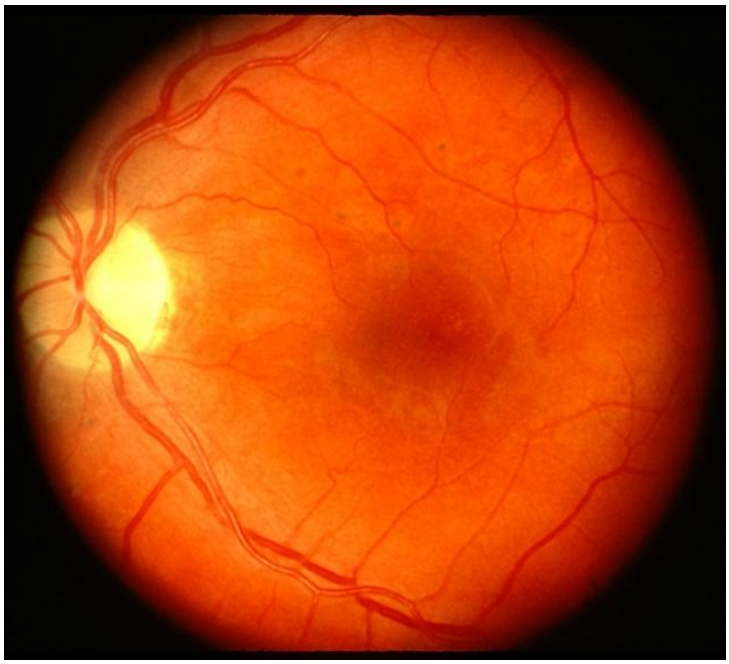
HYPERTENSIVE CHOROIDOPATHY

Typically occurs in young patients who experience an acute episode of hypertension associated with preeclampsia, eclampsia or renal hypertension. Fibrinoid necrosis of choroidal vessels can cause patchy non perfusion of areas of choriocapillaris. This is most easily seen on fundus fluorescein angiography. Lobular non perfusion of choriocapillaris results in a yellow patches of retinal pigment epithelium overlying it called Elschnig's spots. They profusely leak fluorescein. As they heal, they form hyperpigmented with a margin of hypopigmentation. They no

longer leak fluorescein, but transmission fluorescence through hypopigmented halo.

Siegrist’s streaks are linear patches of hyperpigmentation overlying the sclerotic choroidal arteries in chronic hypertension.

Localized bullous detachments of neurosensory retina or retinal pigment epithelium are sometimes seen. Most of this are considered to be due to fibrinoid necrosis of choroidal arteries with occlusion of choriocapillaris and pigment epithelial decompensation. It also happens because of break down of blood retinal barrier with endothelial cell decompensation.



STAGES OF HYPERTENSIVE CHOROIDOPATHY

Hypertensive choroidopathy can be divided into three phases.

During the acute ischemic phase, choroidal arterioles constrict, leading to necrosis of the choriocapillaris and retinal pigment epithelium and accumulation of subretinal exudates. Fundus shows white areas and focal serous detachment most often in the macula and peripapillary region. Fundus fluorescein angiography shows Patches of hypoperfused choriocapillaris, particularly in the central region of macula is seen.

The chronic occlusive phase is characterized by extreme narrowing or occlusion of choroidal capillaries. There is yellowing and leakage of retinal pigment epithelium overlying the occluded regions. Retinal detachment can occur in this phase. Pigment epithelial degenerations develop in the macula and peripheral retina and slowly becomes more extensive.

The last phase is the reparative phase, where occluded choroidal arteries, arterioles and choriocapillaris are recanalized. Retinal pigment epithelium heals and the retina attaches. Sometimes, elschnig’s spots with surrounding atrophy or nonspecific areas of mottling remain. Fundus fluorescein angiography shows underlying choroidal fluorescence.

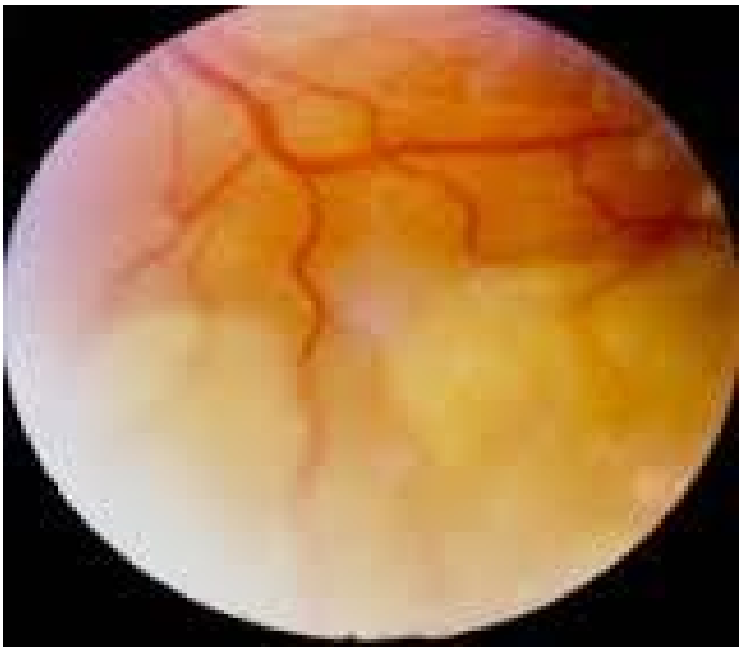
EXUDATIVE RETINAL DETACHMENT

Serous Retinal Detachment was first reported Von Graefe. An incidence of 1-2% is reported in preeclampsia and 10% association with eclampsia. Patients with HELLP syndrome has got a 7 fold rise in chance of developing serous Retinal Detachment when compared to patients with preeclampsia and eclampsia (22).

It is mostly bilateral, seen more in primigravida. It is diagnosed post partum in most of the cases. Serous Retinal Detachments resolve on its own postpartum. They are bilateral bullous, but often cystic lesions.

Serous retinal detachment is one of the main diagnosis to be kept in mind in case of sudden loss in vision in cases complicated with HELLP syndrome and toxaemia of pregnancy (4).

The main pathophysiology is choroidal Dysfunction with ischemia of the choriocapillaris.



The **causes of choroidal occlusion** are ::

1. Ocular Sympathetic Derangement

2. Fibrin platelet occlusion of choroidal arteries and choriocapillaris, which may occur as a part of Disseminated Intravascular Coagulation.

3. Embolic occlusion originating from the products of conception on an immunologic basis.

This leads on to :

A) Ischemia of the Retinal pigment Epithelium leading on to yellowish opacities.

B) fluid Pump Dysfunction leading on to Subretinal fluid accumulation.

Posterior ciliary artery blood flow velocity is increased in preeclampsia, suggesting vasospasm.

Retinopathy is associated with higher levels of blood pressure than serous retinal detachment.

Subretinal fluid can either :: 1)resolve or

2) remain mimicking macular dystrophy or tapetoretinal degeneration.

3) extensive chorioretinal atrophy can lead on to optic atrophy(23).

Fundus fluorescein angiography shows choroidal ischemia secondary to intense arteriolar spasm. Diagnosis can also be done using Indocyanine Green angiography.

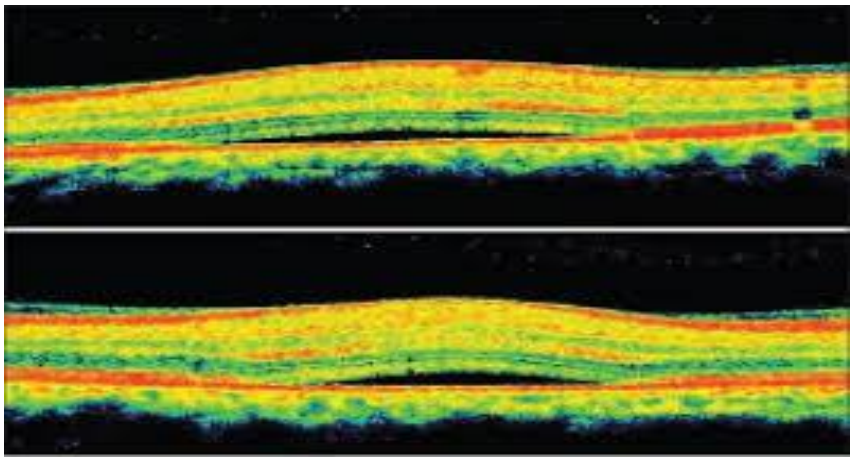
FEATURES ON FUNDUS FLUORESCEIN ANGIOGRAPHY

Early phase – choroidal non filling (30)

Mid phase – persistent choroidal non perfusion and hyperfluorescence

Late phase – extravasation of dye into the subpigment epithelial and subretinal spaces.

FFA is to be avoided in pregnant women, until and unless it is absolutely necessary. No teratogenic effects has been reported till date, but it has been reported to get transmitted in breast milk of lactating women.



OCT shows fluid accumulation between the neurosensory retina and retinal pigment epithelium and detachment of the sensory retina from the pigment epithelium.

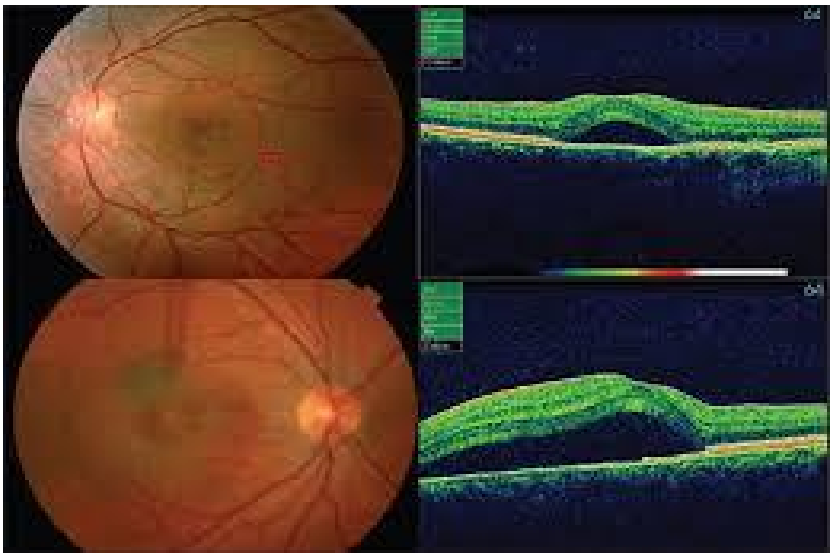
TREATMENT

Conflicting opinions exist as to whether maternal and fetal outcome is worse in patients with fundoscopic signs. Serous retinal detachment management is conservative and involves treating the

underlying condition. Spontaneous resolution usually occurs within few weeks and visual prognosis is excellent. After delivery, the subretinal fluid is reabsorbed by the retinal pigment epithelium and visual acuity return to pre-detachment levels within weeks. However, patients with severe preeclampsia may be left with permanent visual loss, despite resolution of subretinal fluid due to extensive RPE necrosis.

CENTRAL SEROUS CHORIORETINOPATHY

Central serous retinopathy is seen 10 times more commonly in men than women (14). In women, it has a strong association with pregnancy. It is seen especially late in pregnancy. Peak age of incidence is 20 – 50yrs.



Serous retinal detachment is mostly seen bilaterally whereas central serous retinopathy is mostly unilateral. It is seen mostly in the third trimester of pregnancy and resolves few months after delivery.

SYMPTOMS

Unilateral metamorphopsia, moderately decreased vision, micropsia, abnormal colour vision and scotomas (28).

It can recur in subsequent pregnancies. Recurrences always happen in the same eye. It is not determined yet if it is coincident with pregnancy or related to hypercoagulability or related to haemodynamic changes of pregnancy. It is defined as the accumulation of subretinal fluid with circumscribed neurosensory detachment in macula at level of retinal pigment epithelium.

PATHOPHYSIOLOGY

Increased levels of endogenous cortisol with increased permeability of blood retinal barrier, choriocapillaris and retinal pigment epithelium. White fibrinous exudates are found in 90% of pregnancy associated cases of central serous chorioretinopathy, compared with 20% of general cases.

Vision returns to normal within few months after delivery, but changes in central visual field, metamorphopsia and retinal pigment epithelial alterations persist (29).

Subretinal white exudates is seen more commonly in central serous chorioretinopathy associated with pregnancy than in males and in non pregnant women (approx. 10%). This difference is presumed to be due to increased deposition of fibrin.

DIAGNOSIS

Fundus examination shows a round well delineated, shallow, serous macular neurosensory detachment, surrounded by a halo of light. Foveal reflex is absent from the macula and a prominent yellow coloured spot is present in its place. This is due to retinal xanthophylls being visualized. Subretinal fibrin precipitates may be seen as multiple gray-white dots on the posterior surface of detached retina. Small round serous pigment epithelial detachments may be present. Serous pigment epithelial detachments are typically less than a quarter of disc diameter in size. They are usually located in the superior half of neurosensory detachment and may be surrounded by a pink halo. Sometimes the neurosensory detachment is located below the pigment epithelial detachment due to gravitational force acting on the subretinal fluid.

Optical Coherence Tomography aids in diagnosis. Serous detachment of neurosensory retina from the pigment epithelial layer is seen. SD-OCT demonstrates discrete changes in reflectivity within the outer nuclear and plexiform layers. Multiple small pigment epithelial detachments or intraretinal precipitates may be seen.

Fundus fluorescein angiography is not usually done. But if done, it shows the presence of one or several hyperfluorescent leaks in the level of RPE (15). It can either spread symmetrically to all sides giving the “ink blot” appearance. In 10% of the cases, the dye rises within the neurosensory detachment in a fashion. There are normally one or two leakage points but it can be as many as seven or more.

The leakage point is normally situated under the retinal detachment. If it is not visualised, the next most likely location is the superior portion of the affected area. In chronic CSC, atrophic RPE tracts appear as mottled hyperfluorescence.

VASCULAR OCCLUSIONS

There is increased levels of clotting factors and clotting activity during pregnancy. Several pathologic sources of thrombosis and embolic

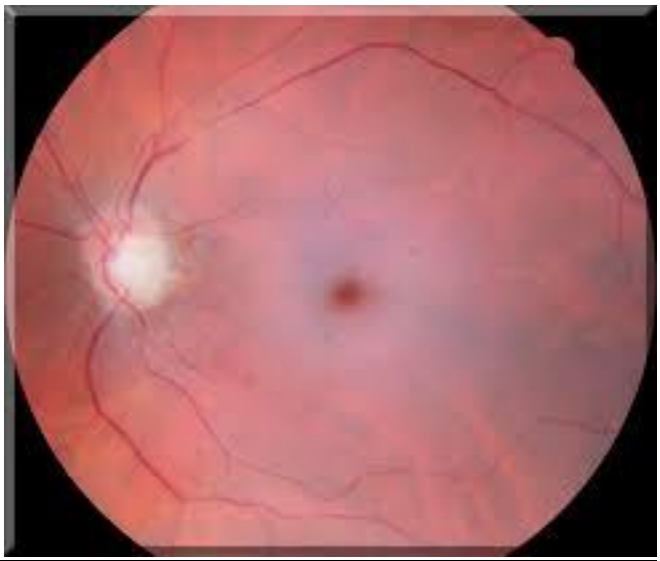
activity can also occur. This leads to increased chances of vascular occlusions in pregnancy(25).

In fact, there is a 13 fold increase in risk of cerebral infarction in pregnant women in comparison with non pregnant women.

Branch Retinal Artery Occlusion is the commonest followed by central artery Occlusion. Vein occlusions are rare in pregnancy. It can happen secondary to hypercoagulable state or amniotic fluid embolism.

RETINAL ARTERY OCCLUSION

Blodi reported that multiple arterial occlusions were seen within 24 hours after child birth in 4 women. Fundus shows Purtscher like retinopathy from arteriolar obstruction by complement mediated leucocyte aggregation.



This condition is characterized by bilateral visual loss, mostly diagnosed shortly after delivery with widespread cotton wool spots with or without intraretinal haemorrhage. Visual prognosis is guarded. Vision can vary from 20/20 to 20/400. Field defect is compatible with areas of occlusion (26).

Fundus shows retinal patches characterized by ischemia and intraretinal haemorrhage, similar to Purtscher's retinopathy.

Some cases may resolve spontaneously with visual recovery. Some cases end up with focal arteriolar narrowing and optic disc pallor.

RETINAL VEIN OCCLUSION

This is exceedingly rare. Only 5 cases of Central Retinal Vein occlusion has been reported till date. No case of Branch Retinal Vein occlusion has been reported.

HYPERTENSIVE OPTIC NEUROPATHY

Hypertensive optic neuropathy results from severely elevated blood pressure. It can be divided into 3 phases.

First phase is acute ischemic phase. Vasoconstriction in the relaminar optic nerve head leads to axonal hydropic swelling, axolemma disruption and glial swelling. This vasoconstriction is more severe in the

retrolaminar region and leads on to endothelial swelling and degeneration of pericytes. This ends up in vacuolated axons and glial swelling. Pathology is thought to be due to optic nerve ischemia and raised intracranial pressure.

Second phase is the resolution phase. Axonal swelling in the optic nerve is decreased. Disintegrated myelinated axons and lipid-laden microglial cells are present in the retrolaminar part. There is evident degeneration of endothelial cells and pericytes.

The third phase is the atrophic phase. Axons of prelaminar part are replaced by proliferated glial cells, and myelinated axons largely disappear from the retrolaminar optic nerve. Lipid laden microglia are absent. Chronic optic nerve swelling is progressively replaced by optic atrophy.

PART – II

AIM OF THE STUDY

The aim of this study is to determine the prevalence of retinal changes in pregnancy induced hypertension and to understand the association between retinal changes and severity of hypertension and proteinuria.

MATERIALS AND METHODS

Pregnant females admitted with pregnancy induced hypertension in Stanley Medical College and Hospital from Jan2013 – Dec2013 were included in this study.

A total of 100 patients were included in this study. All patients were explained about the nature and purpose of the study and an informed consent was taken.

Examination included

Age, gravida and para of the patients were noted

Gestational age was noted

Relevant ocular history was extracted

Visual acuity was checked using Snellen chart and for patients who could not be shifted, bedside vision was taken.

Slitlamp examination of the anterior segment was done, wherever possible.

Pupils were dilated using tropicamide eyedrops and fundus evaluation was done using indirect ophthalmoscope.

Fundus picture was taken, wherever possible.

Systemic examination was done to rule out other co-morbidities.

Blood pressure was recorded for all the patients.

Routine urine analysis for the presence of protein and sugar was done.

Protein was analysed using urine dipstick method.

Biochemical investigations including blood urea, serum creatinine, serum uric acid and total proteins were done and recorded.

Patients were followed up after delivery and reassessed for persistence of fundus changes.

INCLUSION CRITERIA

Pregnant females with new onset of hypertension 28^{th} week of gestation with proteinuria admitted in Stanley Medical College and Hospital.

EXCLUSION CRITERIA

Patients with pre-existing hypertension, diabetes mellitus and renal disease.

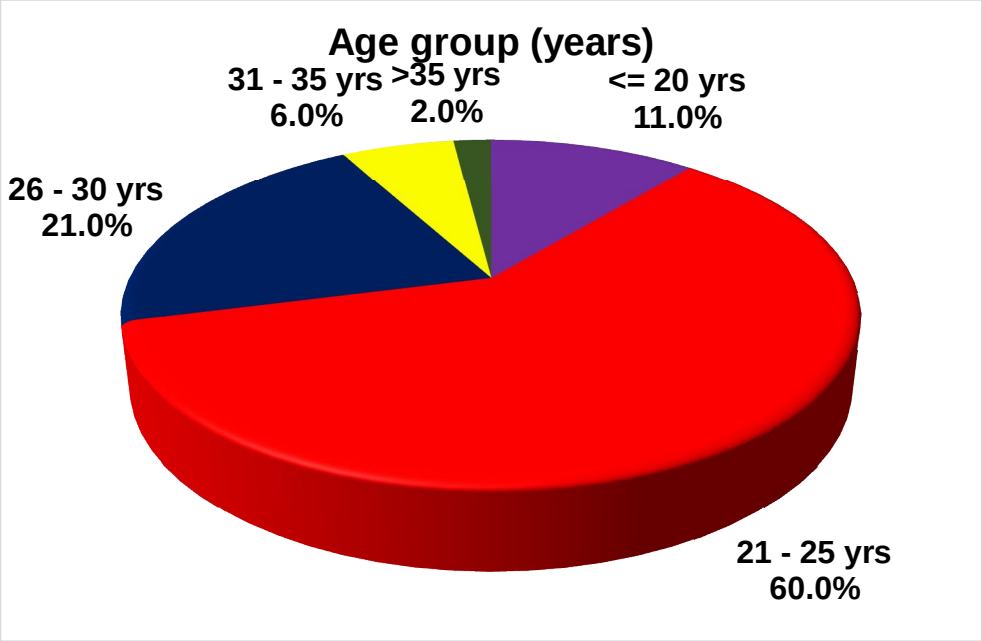
Patients with raised blood sugar values.

OBSERVATION

Table 1 : Agewise incidence of PIH

Age group (years)	N	%
<= 20 yrs	11	11.0
21 - 25 yrs	60	60.0
26 - 30 yrs	21	21.0
31 - 35 yrs	6	6.0
>35 yrs	2	2.0
Total	100	100.0

Graph 1 showing agewise incidence of PIH

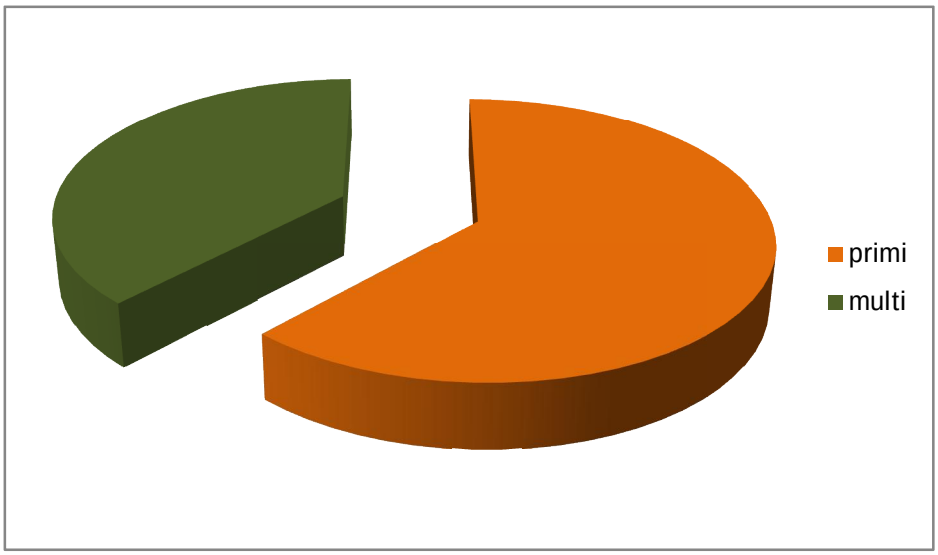


Maximum number of PIH cases were found in the age group of 21 – 25 years. This observation could also be because of the fact that more number of pregnant women tend to fall in to this group.

Table 2 : Incidence of PIH in relation to parity

Gravida	N	%
Primigravida	62	62.0
Multigravida	38	38.0
Total	100	100.0

Graph 2 showing incidence of PIH in relation to parity



60% of the cases of PIH was found in primigravida and the rest 40% in multigravida.

Table 3 : Grouping of patients based on gestational age

Period of gestation (weeks)	Number of patients	Percentage
20-24	3	3
25-28	7	7
29-32	23	23
>32	67	67

Maximum number of PIH patients(67%) had gestational age>32 weeks at the time of presentation. 23 patients had gestational age of 29-32 weeks, 7 patients between 25-28 weeks and only 3 patients <25 weeks.

Graph 3 : Period of gestation

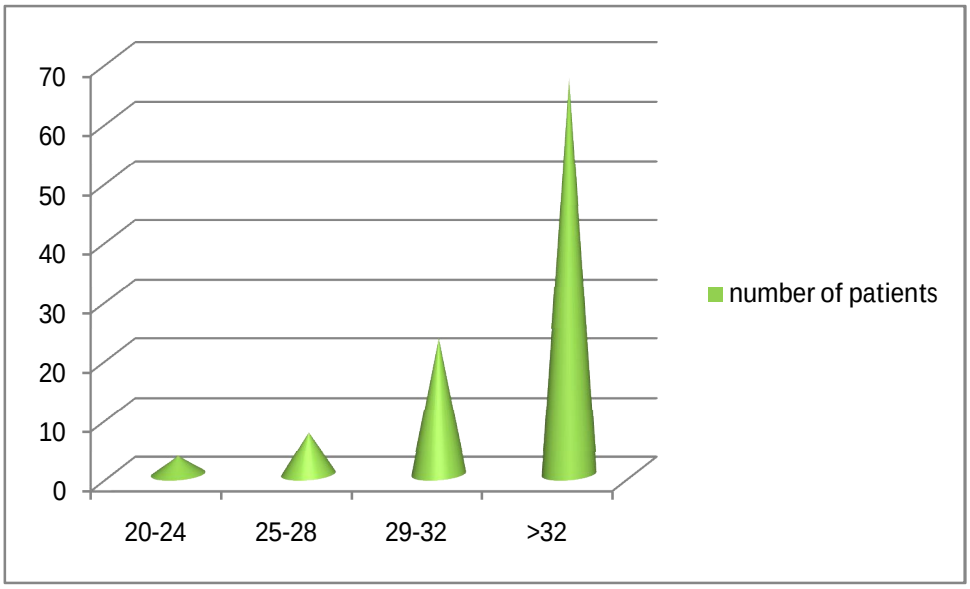


Table 4 : Grouping of patients according to severity of hypertension

Severity of Hypertension	Number of patients
Mild preeclampsia	54
Severe preeclampsia	40
Eclampsia	06

54 patients fell in the category of mild preeclampsia, 40 in the group of severe preeclampsia and 6 patients had hypertension with seizures.

Graph 4 : Grouping patients according to severity of hypertension

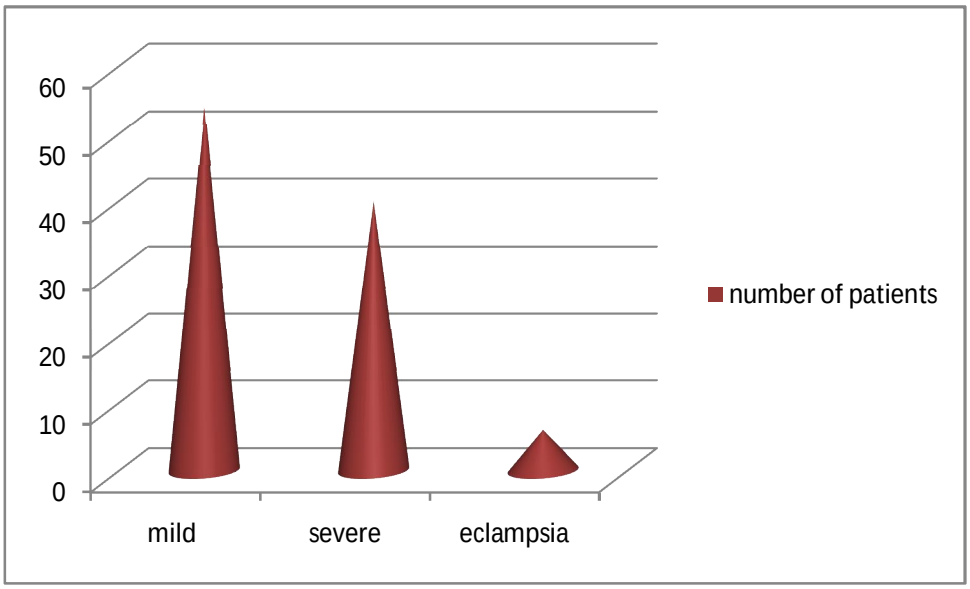


Table 5 : Grouping of patients according to grades of proteinuria

Grades of proteinuria	Number of patients
1+	49
2+	36
3+	15

Maximum number of patients(49) had grade 1+ proteinuria, 36 patients had grade 2+ proteinuria and only 15 patients had grade 3+ proteinuria.

Graph 5 : Grouping patients according to grades of proteinuria

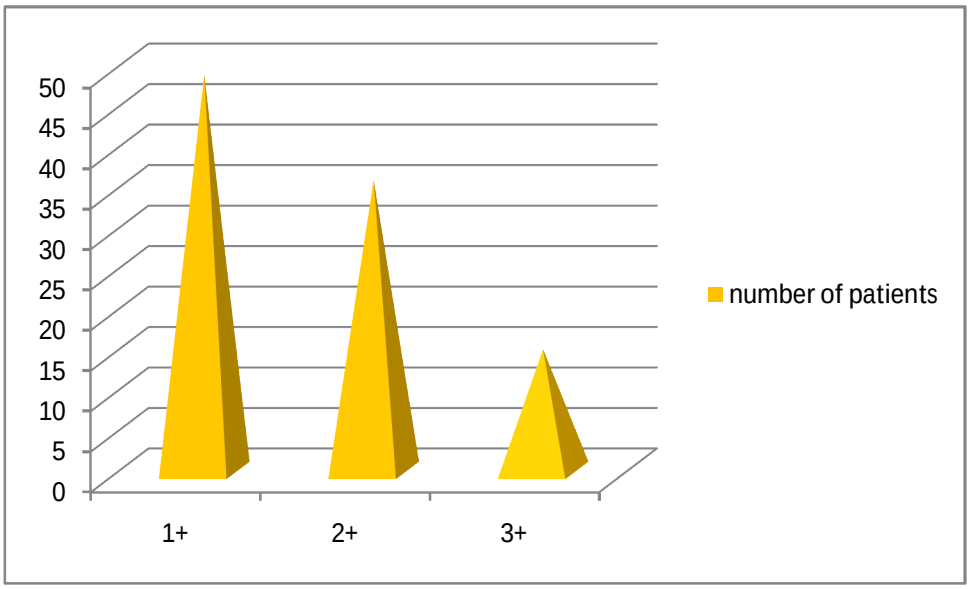
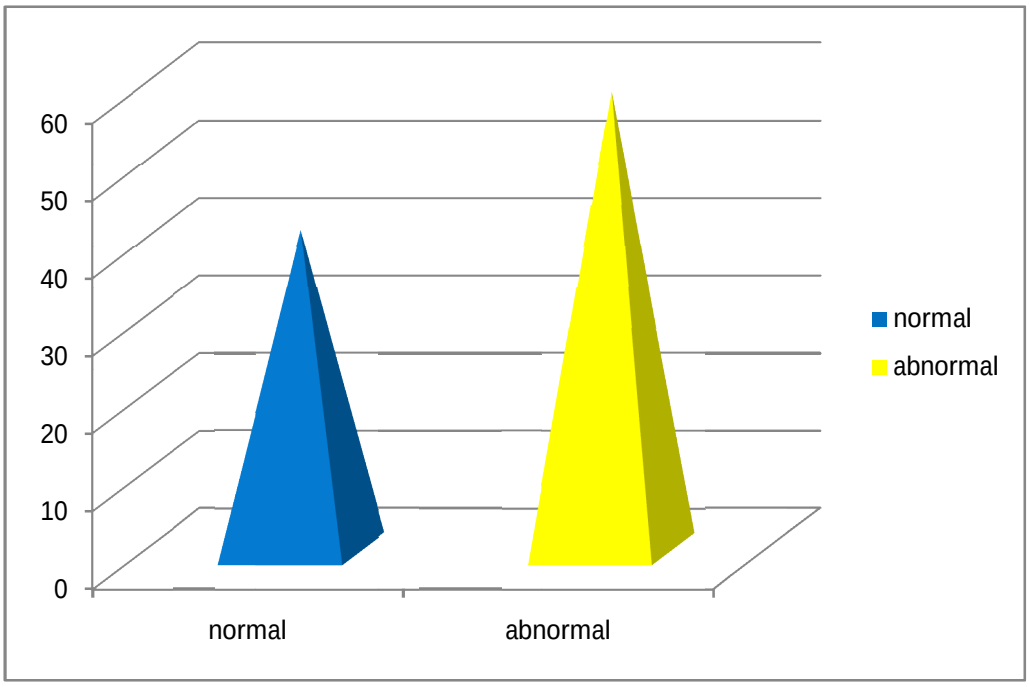


Table 6 : Fundus findings in PIH

Fundus Findings	N	%
Normal	41	41.0
Abnormal	59	59.0
Total	100	100.0

Graph 6 : Fundus findings in PIH

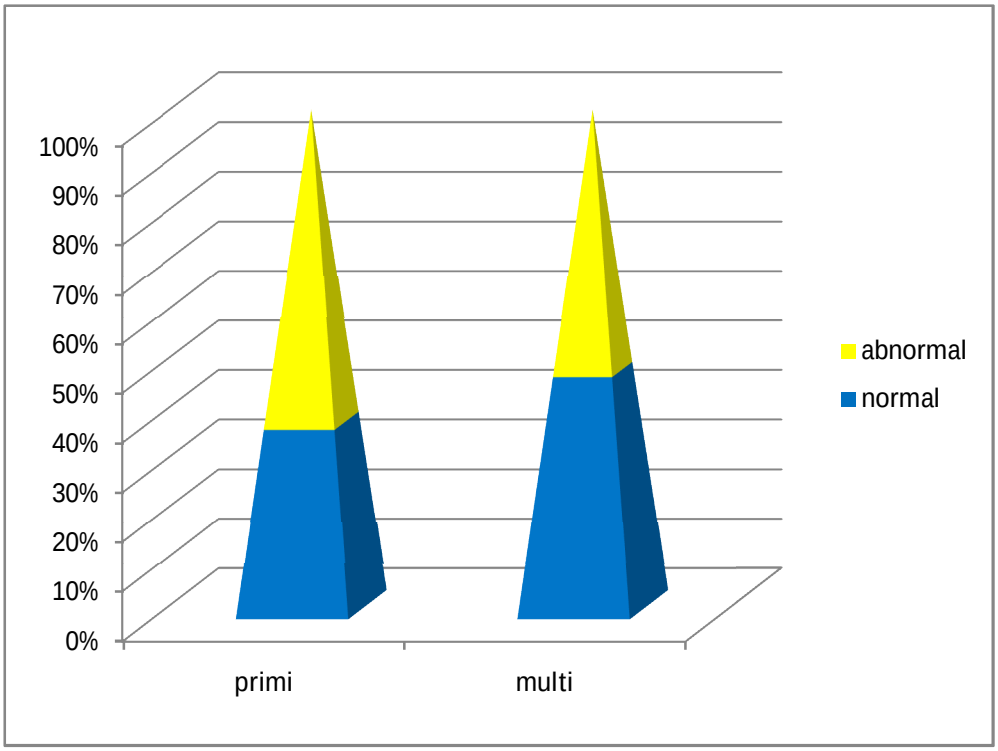


Out of the 100 PIH patients studied, 41 patients had normal fundus findings and the rest 59 patients had some abnormal findings in their fundus.

Table 7 : Comparing the parity and fundus findings

Fundus findings	Gravida					
	Primigravida		Multigravida		Total	
	N	%	N	%	N	%
Normal	23	37.1	18	47.4	41	62.0
Abnormal	39	62.9	20	52.6	59	38.0
Total	62	100.0	38	100.0	100	100.0

Graph 7 : Comparing parity and fundus findings



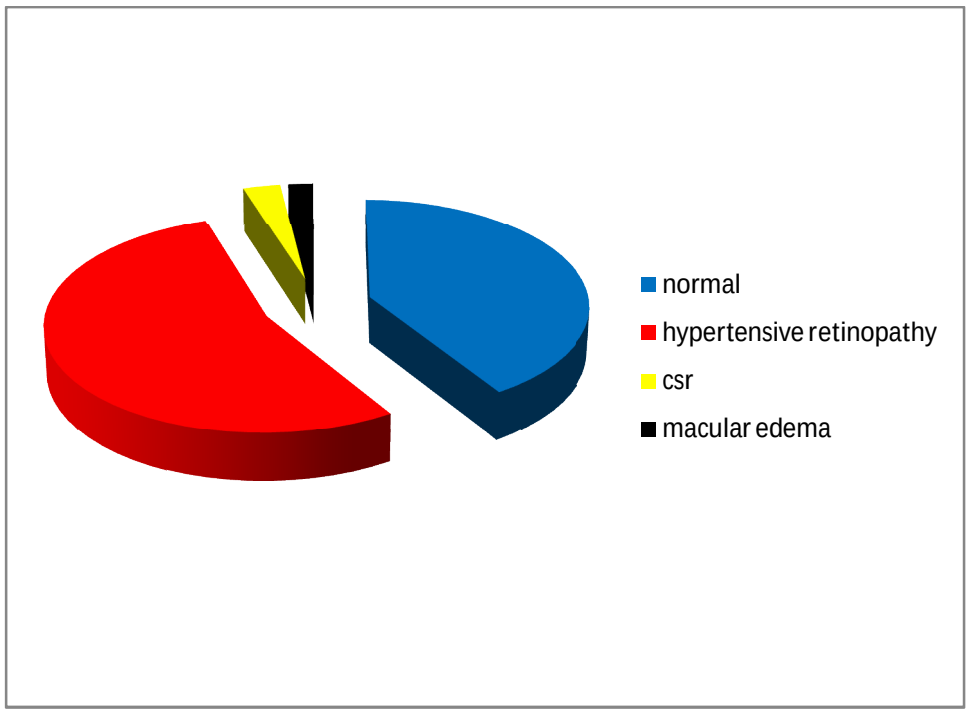
Of the 62 primigravidas, 23 (37.1%) had normal fundus findings and the rest 39(62.9%) had abnormal findings.

Of the 38 multigravidas, 18(47.4%) had normal fundus findings and the rest 20(52.6%) had abnormal findings on fundus examination.

Table 8 : Fundus changes observed

Fundus finding	N	%
Normal	41	41.0
Hypertensive retinopathy	54	54.0
Central serous retinopathy	3	3.0
Macular edema	2	2.0
Total	100	100.0

Graph 8 showing the different fundus changes

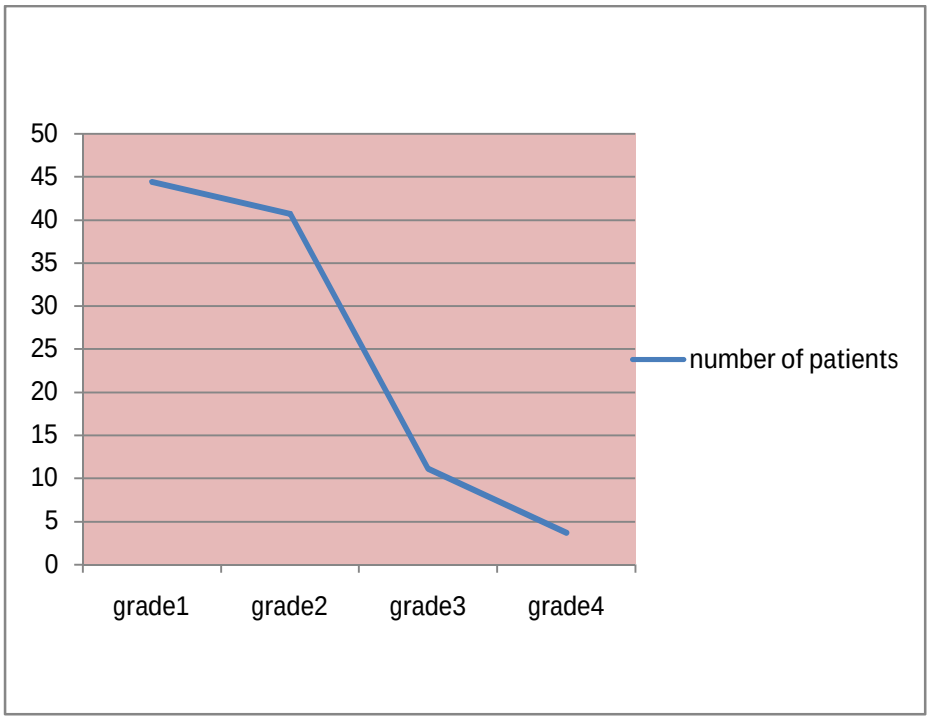


Maximum number of patients had either normal fundus (41%) or grade 1 hypertensive retinopathy (24%). 22% had grade 2, 6% had grade 3 and 2% of the cases had grade 4 hypertensive retinopathy. Another 2 % had macular edema. 3% of the cases studied showed central serous retina. 54% of the cases studied had hypertensive retinopathy. This makes hypertensive retinopathy as the most frequently noted sign in PIH

Table 9 : Comparing the different grades of retinopathy

GRADES OF RETINOPATHY	N	%
GRADE 1	24	44.4
GRADE2	22	40.7
GRADE 3	6	11.1
GRADE 4	2	3.7
TOTAL	54	100.0

Graph 9 comparing the different grades of retinopathy



A total of 54 patients had hypertensive retinopathy. Of all the PIH patients with hypertensive retinopathy changes, 44.4% had grade 1 hypertensive retinopathy changes with narrowing of the retinal arterioles. 40.7% had grade 2 changes, 11.1% had grade 3 and the rest 3.7% had grade 4 changes. This study shows that maximum number of patients with hypertensive retinopathy had grade 1 and 2 changes.

Table10: Correlating severity of PIH with blood urea values(mg%)

Severity of PIH	minimum	Maximum	Mean value
Mild preeclampsia	9.0	40.0	20.75
Severe preeclampsia	10.0	71.0	27.67
Eclampsia	14.0	52.0	31.33

In the present study,blood urea levels in mild preeclampsia group ranged from 9mg/dl to 40mg/dl with a mean value of 20.75mg%. in severe preeclampsia group, it ranged from 10 to 71mg/dl with a mean value of 27.67mg/dl. And in eclampsia group, blood urea levels ranged from 14 to 52mg/dl with a mean of 31.33mg/dl.

Graph10 : Correlating severity of PIH with mean blood urea levels

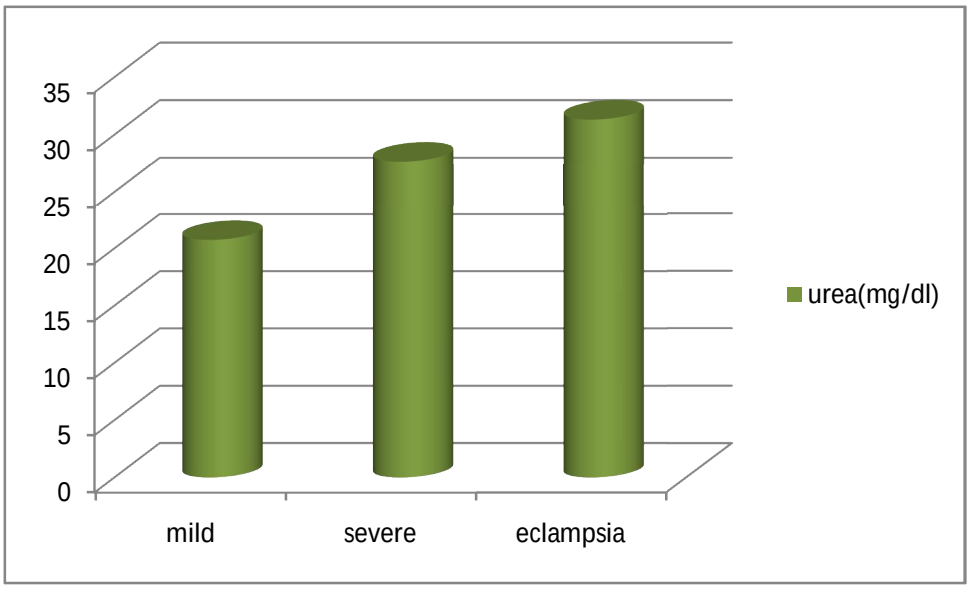


Table11 : Correlating severity of PIH with serum uric acid levels(mg/dl)

Severity of PIH	Minimum	Maximum	Mean
Mild preeclampsia	2.6	11.2	4.98
Severe preeclampsia	3.1	9.2	5.82
Eclampsia	4.3	12.6	9.58

Serum uric acid levels ranged from 2.6 to 11.2mg% in mild preeclampsia group with a mean of 4.98mg%. in severe preeclampsia group, it ranged from 3.1 to 9.2mg% with mean value of 5.82mg/dl. In eclampsia patients, the value ranged from 4.3 to 12.6mg% with a mean of 9.58mg%.

Graph 11: Correlating PIH with mean serum uric acid levels

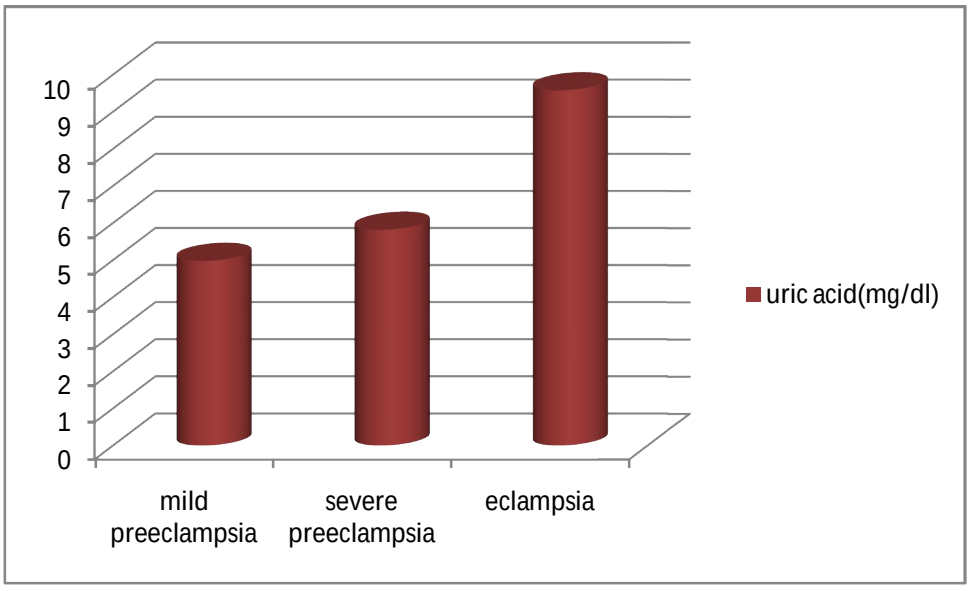


Table 12 : Grouping of fundus changes according to parity

Fundus finding	Gravida					
	Primigravida		Multi gravida		Total	
	N	%	N	%	N	%
Gr 1 HTN Retinopathy	14	22.6	10	26.3	24	24.0
Normal	23	37.1	18	47.4	41	41.0
Gr 2 HTN Retinopathy	16	25.8	6	15.8	22	22.0
Gr 3 HTN Retinopathy	3	4.8	3	7.9	6	6.0
Gr 4 HTN Retinopathy	2	3.2	0	.0	2	2.0
Central serous retinopathy	3	4.8	0	.0	3	3.0
Macular edema	1	1.6	1	2.6	2	2.0
Total	62	100.0	38	100.0	100	100.0

Chi-Square Test	**P-Value**
Fisher's Exact Test	**0.565**

Fundus findings and parity were compared and studied. P value from Fisher’s exact test was 0.565. PIH was found more in primigravidas in comparison to multigravidas. But the fundus findings had no correlation with parity. The difference between both was found to be statistically insignificant.

Graph 12 grouping the fundus findings in relation to parity

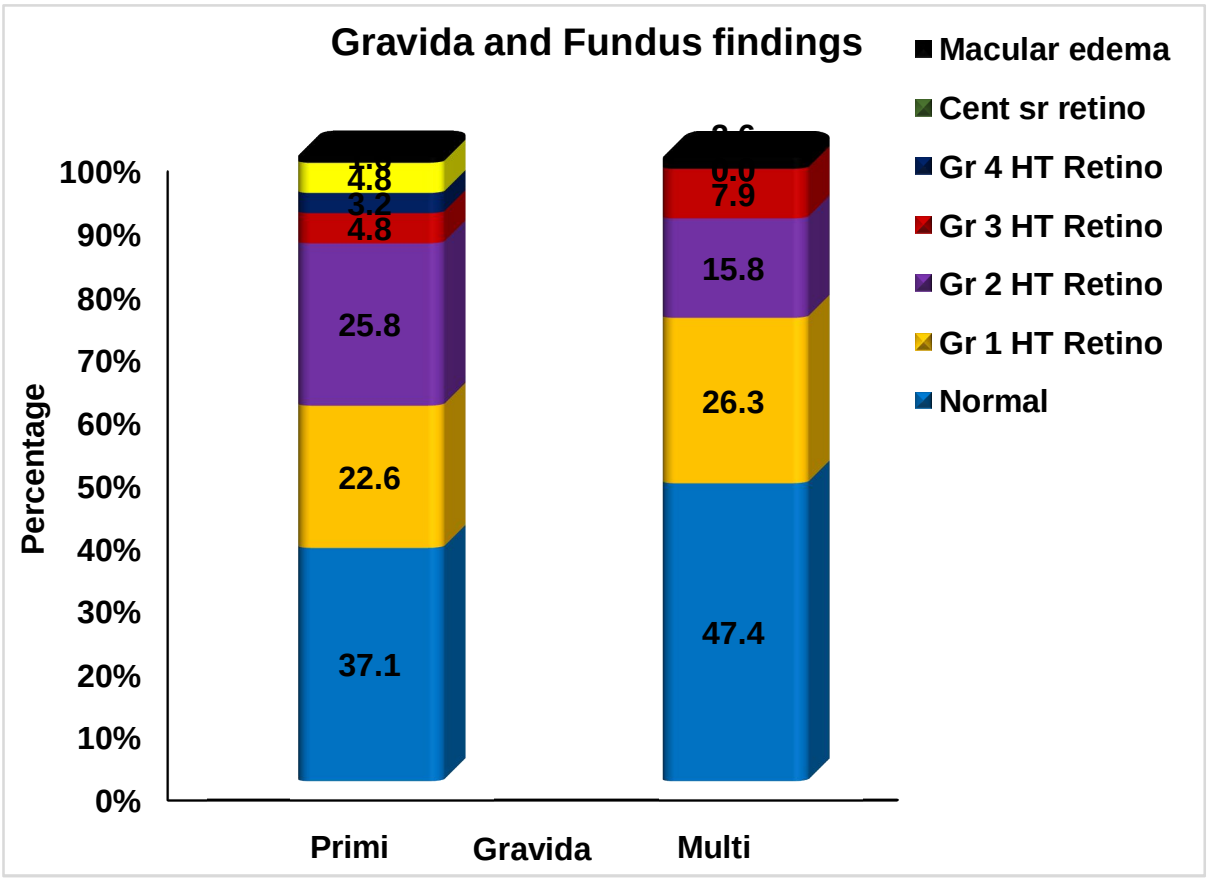
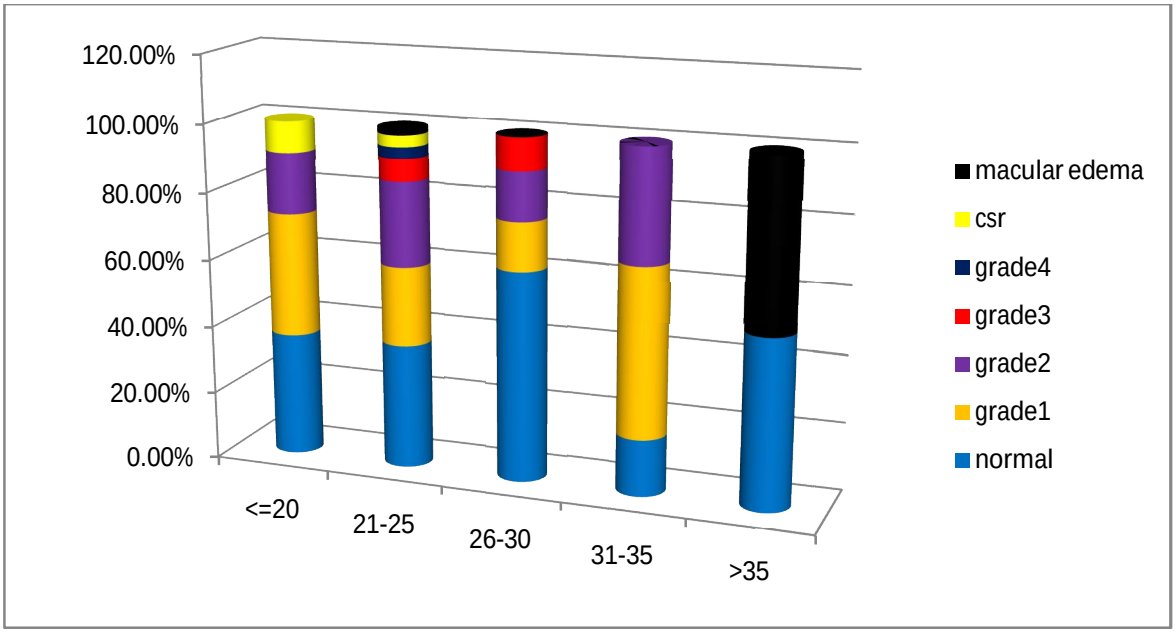


Table 13 : Grouping the fundus findings according to age group

Fundus finding	Age group (years)											
	<= 20 yrs		21 - 25 yrs		26 - 30 yrs		31 - 35 yrs		>35 yrs		Total	
	N	%	N	%	N	%	N	%	N	%	N	%
Normal	4	36.4	22	36.7	13	61.9	1	16.7	1	50.0	41	41.0
Gr 1 HTN Retinopathy	4	36.4	14	23.3	3	14.3	3	50.0	0	.0	24	24.0
Gr 2 HTN Retinopathy	2	18.2	15	25.0	3	14.3	2	33.3	0	.0	22	22.0
Gr 3 HTN Retinopathy	0	.0	4	6.7	2	9.5	0	.0	0	.0	6	6.0
Gr 4 HTN Retinopathy	0	.0	2	3.3	0	.0	0	.0	0	.0	2	2.0
Central serous retinopathy	1	9.1	2	3.3	0	.0	0	.0	0	.0	3	3.0
Macular edema	0	.0	1	1.7	0	.0	0	.0	1	50.0	2	2.0
Total	11	100.0	60	100.0	21	100.0	6	100.0	2	100.0	100	100.0

Graph 13 grouping the fundus findings according to age group



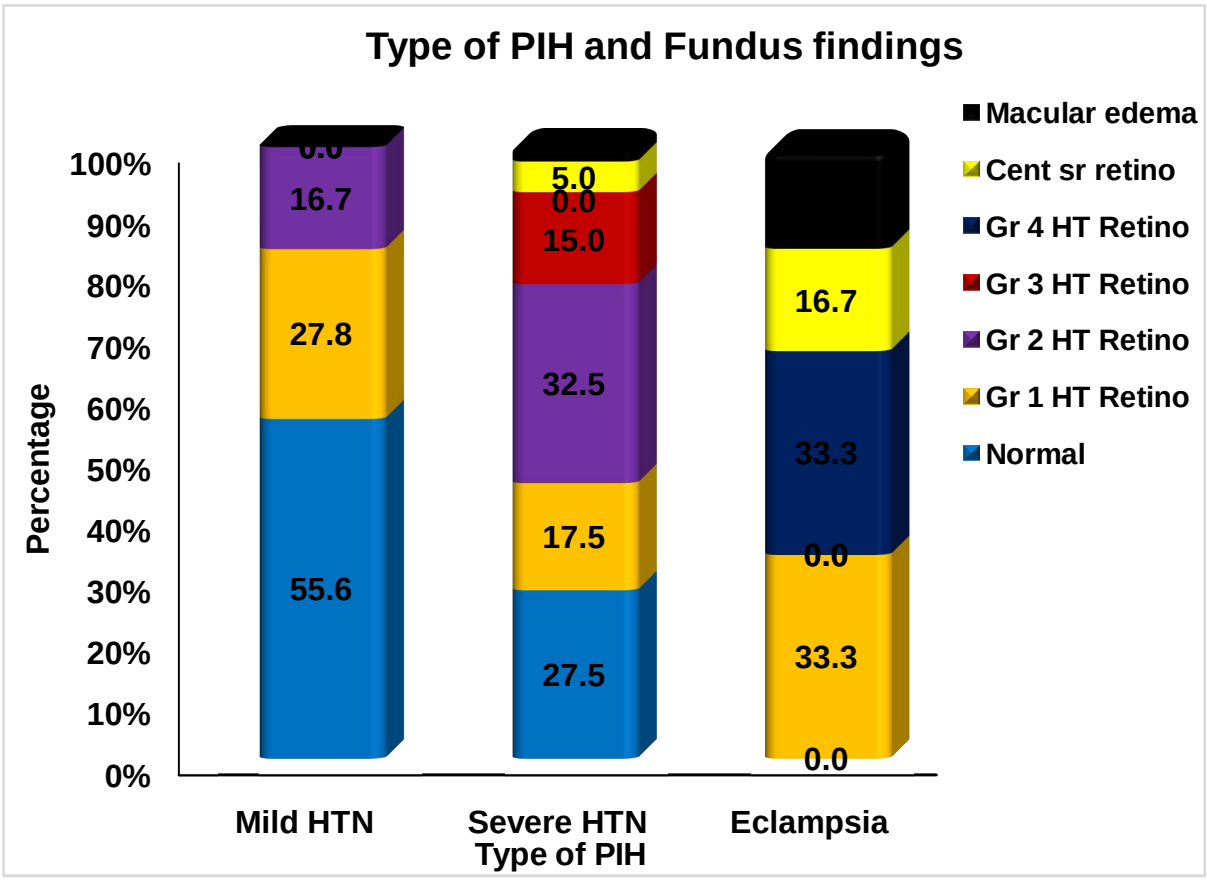
Chi-Square Test	P-Value
Fisher's Exact Test	0.502

The different fundus findings were grouped based on their age and the data was studied and compared. Fisher's Exact test was done on the data and P value was 0.502, which is statistically insignificant. This suggests that there is no association between the age of the patient and their fundus findings.

Table 14 : Correlating fundus findings with severity of hypertension

Fundus finding	type of PIH							
	Mild HTN (DBP <100)		Severe HTN (DBP >=100)		Eclampsia		Total	
	N	%	N	%	N	%	N	%
Normal	30	55.6	11	27.5	0	.0	41	41.0
Gr 1 HTN Retinopathy	15	27.8	7	17.5	2	33.3	24	24.0
Gr 2 HTN Retinopathy	9	16.7	13	32.5	0	.0	22	22.0
Gr 3 HTN Retinopathy	0	.0	6	15.0	0	.0	6	6.0
Gr 4 HTN Retinopathy	0	.0	0	.0	2	33.3	2	2.0
Central serous retinopathy	0	.0	2	5.0	1	16.7	3	3.0
Macular edema	0	.0	1	2.5	1	16.7	2	2.0
Total	54	100.0	40	100.0	6	100.0	100	100.0

Graph 14 correlating fundus findings with severity of hypertension



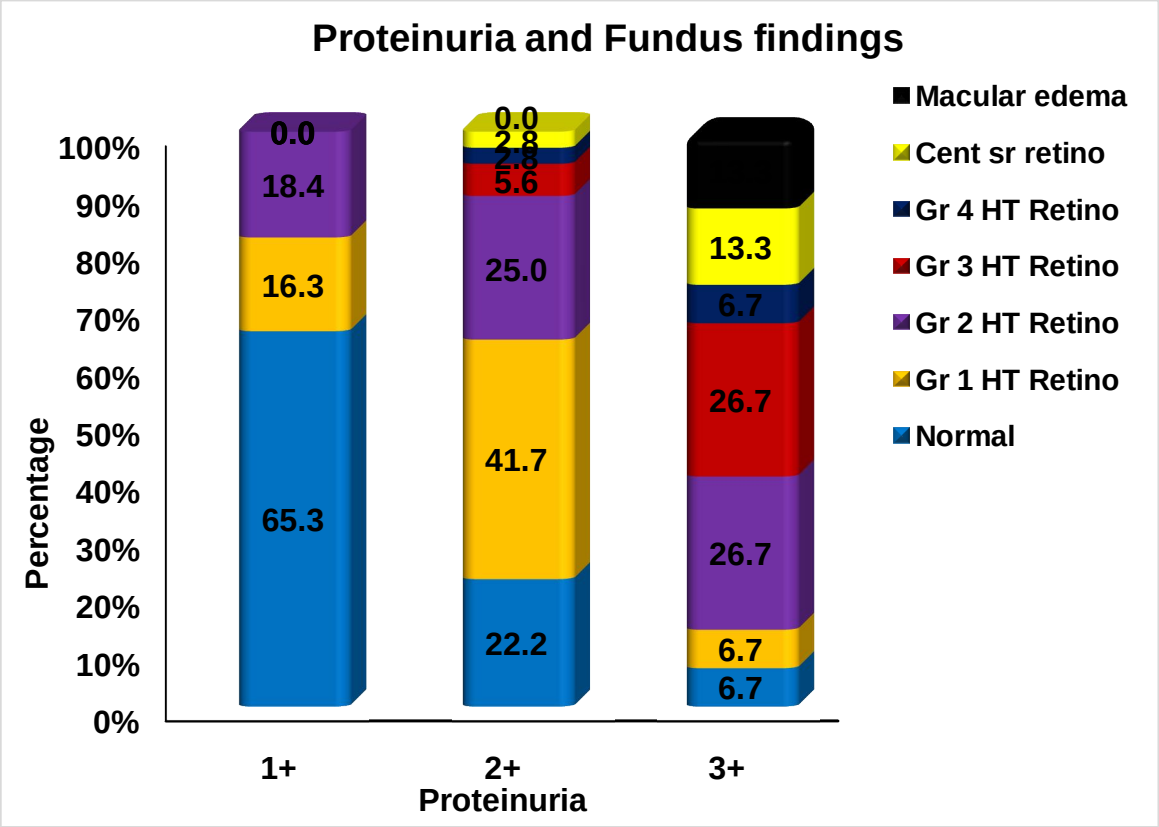
Chi-Square Test	P-Value
Fisher's Exact Test	<0.001

Fundus changes found in all the 100 cases was correlated with the severity of hypertension and Fisher's exact test was done on the same. P value for the test was found to be <0.001, showing that the two variables have a strong association. That means, as the severity of hypertension increases, there is more chance of the patient having abnormal fundus finding.

Table 15 : Correlating fundus findings with proteinuria

Fundus finding	Proteinuria							
	1+		2+		3+		Total	
	N	%	N	%	N	%	N	%
Normal	32	65.3	8	22.2	1	6.7	41	41.0
Gr 1 HTN Retinopathy	8	16.3	15	41.7	1	6.7	24	24.0
Gr 2 HTN Retinopathy	9	18.4	9	25.0	4	26.7	22	22.0
Gr 3 HTN Retinopathy	0	.0	2	5.6	4	26.7	6	6.0
Gr 4 HTN Retinopathy	0	.0	1	2.8	1	6.7	2	2.0
Central serous retinopathy	0	.0	1	2.8	2	13.3	3	3.0
Macular edema	0	.0	0	.0	2	13.3	2	2.0
Total	49	100.0	36	100.0	15	100.0	100	100.0

Graph 15 correlating fundus findings with proteinuria

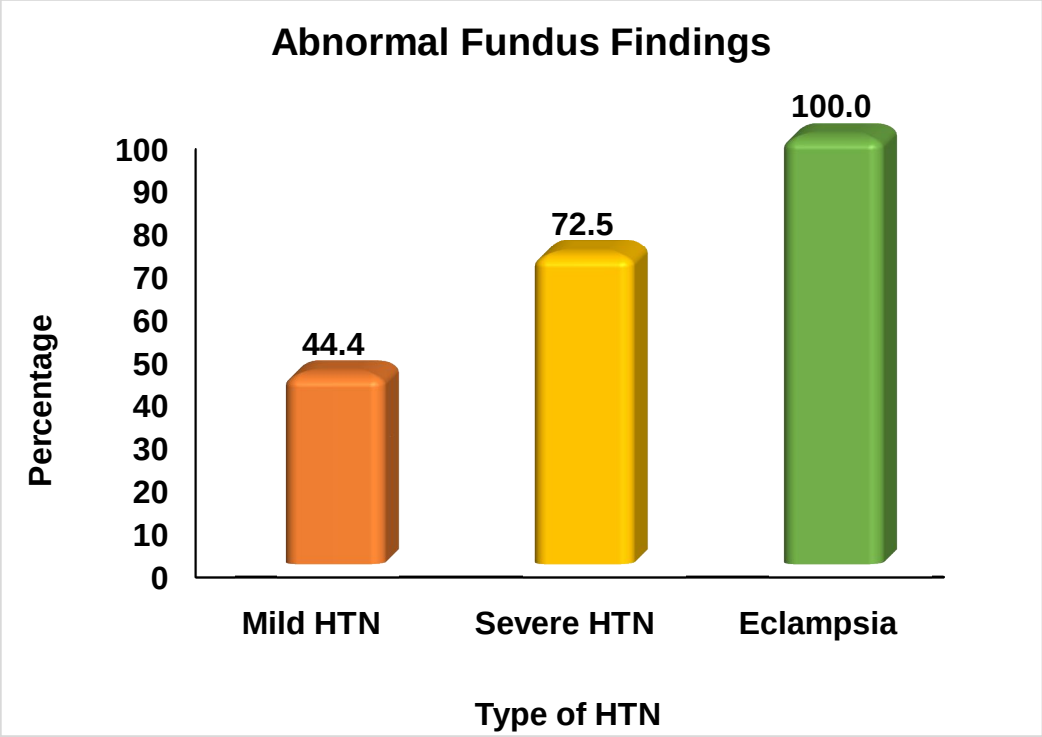


Chi-Square Test	P-Value
Fisher's Exact Test	<0.001

Fundus changes found in all the 100 cases were correlated with the degree of proteinuria. Fisher's exact test was done and the data compared. P value was found to be 0.001, which suggested positive correlation between the two variables. With increasing degrees of proteinuria, there is more chance of abnormalities in the fundus.

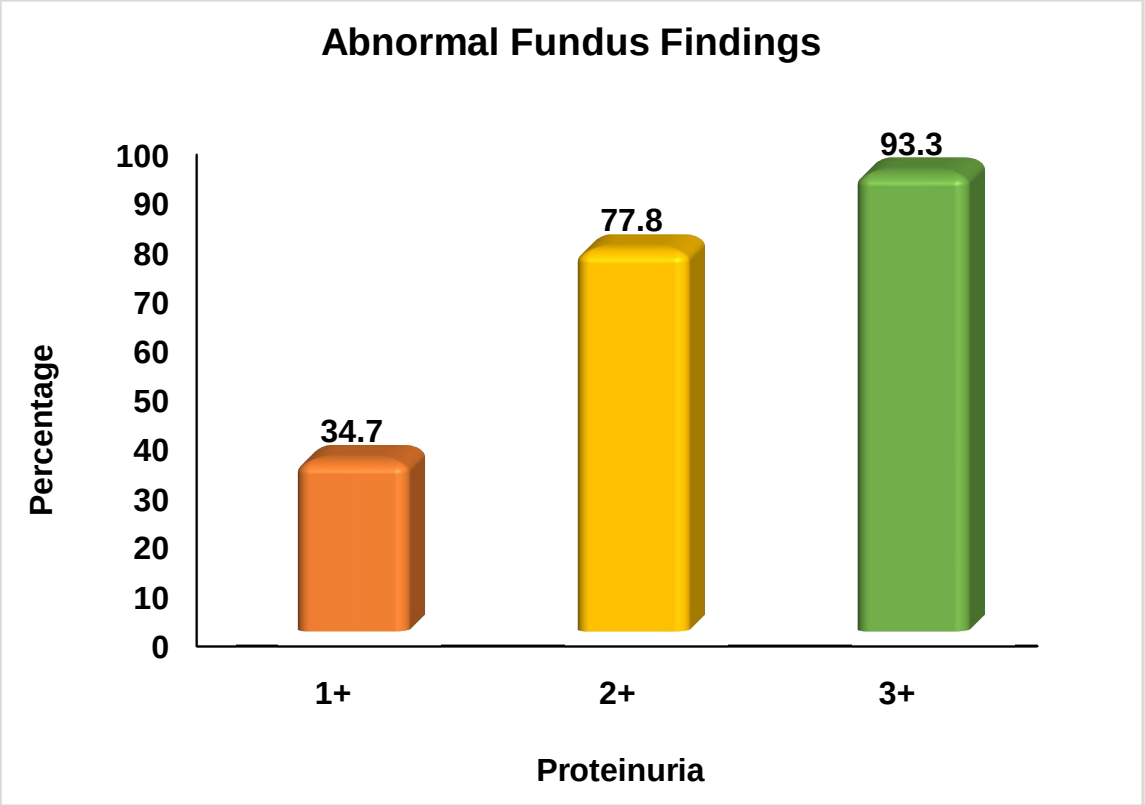
Multivariate Logistic regression analysis to compare fundus findings with hypertension and proteinuria

Graph 16 showing percentage of abnormal fundus findings with severity of hypertension



44% of patients with mild hypertension and 72.5% of patients with severe hypertension had abnormal fundus findings. There were only 6 patients with eclampsia and all 6 of them had abnormalities in their fundus. Because of this, it was not possible to do a multivariate analysis using both severity of hypertension and grade of proteinuria together as the variables. Hence a simple logistic regression was done using proteinuria as the variable.

Simple logistic regression analysis comparing fundus findings and grade of proteinuria



Factor	Fundus Findings Abnormal		Odds Ratio (95% CI)	P-Value
	N	%		
Proteinuria				
1+ (Ref)	17	34.7	1.00	
2+	28	77.8	5.59 (2.47 – 17.58)	<0.001
3+	14	93.3	26.35 (3.19 – 217.9)	0.002

With 95% confidence interval, Chances of patients with proteinuria 2+ having abnormal fundus findings is 5.59 times those with proteinuria 1+.

Chances of patients with proteinuria 3+ having abnormal fundus findings is 26.35 times those with proteinuria 1+.

DISCUSSION

Age grouping of PIH (Table 1)

A total of 100 cases were included in this study, of which 60 were in the age group of 21-26 years, 21 in the age group of 26-30 years, 11 cases had age <= 20 years and the rest >30 years.

Parity and PIH (Tables 2 and 7)

62% of the cases were primigravidas and the rest 38 cases were multigravidas. Though PIH as found more in primigravidas, there was no correlation between the parity and fundus findings observed. In a study done by Reddy et al from India(1), 43.5% of the 40 cases with PIH were primigravidas. Another study done by Ayush Singhal et al on 130 PIH patients noted that 56.15% of the patients were primigravidas.

Number of patients	Present study	Reddy et al	Ayush Singhal et al
Total	100	78	130
primigravidas	62%	43.5%	56.15%
multigravidas	38%	56.5%	43.85%

Severity of hypertension (Table 4)

In the present study, 54% of the cases studied had mild hypertension with blood pressure< 110 mm Hg. 40% had severe hypertension with blood pressure >=110 mm Hg and the rest 6% had hypertension with seizures (eclampsia).

Severity of hypertension	Present study	Reddy et al	Tadin et al	Ayush et al
Mild	54%	38.4%	55%	59.2%
Severe	40%	59%	25%	33.1%
eclampsia	6%	2.5%	20%	7.7%

All the patients studied had proteinuria. 49% had grade 1 proteinuria, 36% had grade 2 proteinuria and the rest 15% had grade 3 proteinuria.

Fundus changes in PIH (Tables 6 and 8)

Retinal changes were found in 59% of the patients studied. This is comparable to study done by Tadin et al(2) from Croatia. He reported 45% of retinal changes in their study on 40 patients with PIH.

	Present study	Reddy et al	Tadin et al	Ayush et al
Hypertensive retinopathy	54%	59%	45%	64%
Central serous retinopathy	3%	-	-	-
Macular edema	2%	-	-	-
Exudative RD	-	-	-	3.1%

Hypertensive retinopathy was the most frequently noticed finding seen in 54% of the patients. 3 cases of central serous retinopathy and 2 cases of macular edema were also noticed. Topical anti inflammatory medication was started for the patients with macular edema and central serous retinopathy.

Grades of hypertensive retinopathy (Table 9)

In a study done on 275 cases of preeclampsia and 125 cases of eclampsia by Reddy from India, he reported retinal changes in 53.4%

preeclampsia and in 71.2% eclampsia patients. The most common fundus finding they noticed was narrowing of arterioles(45.7%).

In a study done on 130 PIH patients by Ayush Singal, 57.69% patients were reported to have grade 1 changes, 3 patients each had grade 2 and 3 hypertensive retinopathy changes, 2 patients had grade 4 retinopathy changes. He reported 4 cases of exudative retinal detachment .

Hypertensive retinopathy	Present study	Reddy et al	Tadin et al	Ayush et al
Grade 1	24%	52.6%	25%	57.7%
Grade 2	22%	6.4%	15%	2.4%
Grade 3	6%	-	5%	2.4%
Grade 4	2%	-	-	1.5%

In the present study, 24% had grade 1 hypertensive retinopathy changes and 22% had grade 2 hypertensive retinopathy changes. 2 cases of grade 4 retinopathy was seen. There were no cases of exudative retinal detachment. Both the cases with grade 4 retinopathy were noted in patients with eclampsia and blood pressure beyond 160/110mm Hg. Termination of pregnancy was advised. 2 weeks after the termination of pregnancy, fundus picture showed no disc edema. Superficial haemorrhages and cotton wool spots were present, but resolving.

Correlation of fundus findings with severity of hypertension and proteinuria (Tables 14 and 15 with logistic regression analysis)

The present study showed a positive correlation between fundus findings, severity of hypertension and grades of proteinuria. This is similar to the study done by Tadin et al where he reported that the degree of retinopathy was directly proportional to severity of preeclampsia and proteinuria. Their study stated that hypertensive retinopathy is a valid and reliable prognostic factor in determining the severity of preeclampsia and that examination of fundus is a valuable and necessary diagnostic procedure in pregnant women with preeclampsia. They also noticed that hypertensive retinopathy is closely related to the appearance of edema.

Correlation of severity of hypertension with blood urea (Table10)

In a study done by Tandon & Kishore(14), Mean blood urea level in mild preeclampsia was noted to be 24.6 mg%, while in eclampsia it was 43.4 mg%. This corelates with the present study which shows a positive core;ation between blood urea levels and severity of hypertension.

Correlation of serum uric acid levels with severity of PIH (Table11)

In a study done by Tandon & Kishore, mean serum uric acid level in mild preeclampsia was 5.2mg% and in severe preeclampsia, it was 5.63mg%. In Eclampsia, the average value was 7.29mg%. The result of present study matches with the previous studies, showing a definite correlation between serum uric acid levels and severity of hypertension.

CONCLUSION

Pregnancy is a physiological state affecting all the physiological and biochemical parameters in the body.

Pregnancy induced hypertension is one of the leading causes of maternal and foetal morbidity and mortality, even in developed countries.

It is difficult to differentiate pregnancy induced hypertension from chronic hypertension complicating pregnancy.

Pregnancy induced hypertension is most commonly found in primigravidas or multigravidas pregnant by a new partner.

Retinal changes is a very good indicator of the severity of hypertension and proteinuria. Retinal changes help in differentiating chronic hypertension from pregnancy induced hypertension to a great extent.

Grade 1 and 2 hypertensive retinopathy is the most common fundus change observed in PIH patients. Less commonly, grade 3 and 4 hypertensive retinopathy and serous retinal detachment is noted. The presence of these changes carries a grave visual and systemic prognosis, until and unless the pregnancy is terminated.

Premature delivery of the foetus carries a better prognosis than continuing the pregnancy in these cases. The presence of disc edema, macular edema or serous retinal detachment is a clear indicator for termination of pregnancy.

Resolution of the retinal changes is observed shortly after termination of pregnancy. If retinal changes persist, chronic pre-existing hypertension has to be ruled out. In case of persisting fundus changes and elevated blood pressure, renal function tests has to be carried out.

The present study suggested a positive correlation with the severity of hypertension and grade of proteinuria.

Though PIH was more common in primigravidas, no correlation was found between parity and fundus changes. Similarly, no correlation was found between the age of the patient and fundus changes.

Examination of the retina is mandatory in all cases of pregnancy induced hypertension. It is an indirect indicator of the severity of hypertension.

A careful history taking is equally important as there is high chance of recurrence of retinal changes in PIH patients with fundus findings.

Persistence of retinal changes even after termination of pregnancy throws light over the presence of chronic persisting hypertension and renal complicatons in hypertension. Regular follow up of the patients is very essential in all cases of PIH all throughout the pregnancy and even after termination of pregnancy till the resolution of retinal changes.

LIMITATION OF THE STUDY

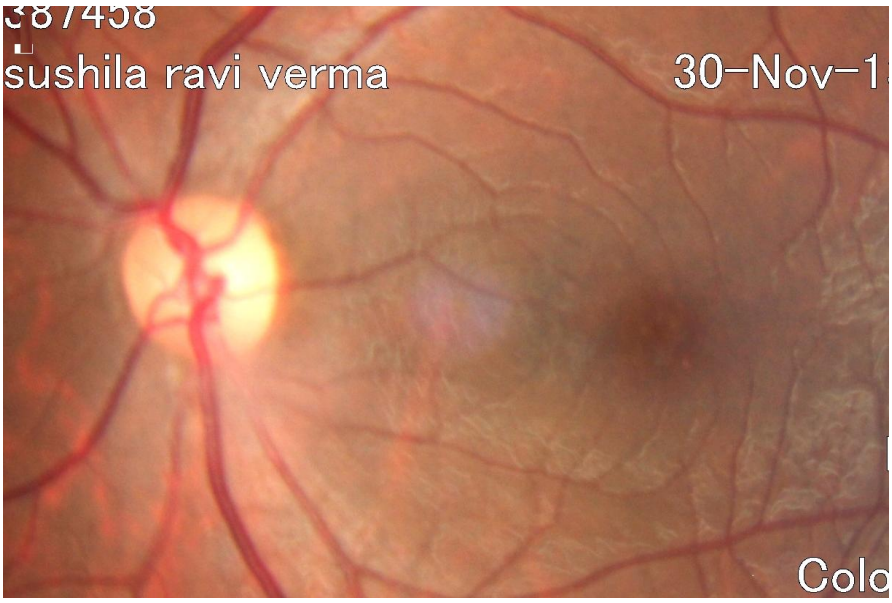
Small sample size

Limited number of patients in the eclampsia group.

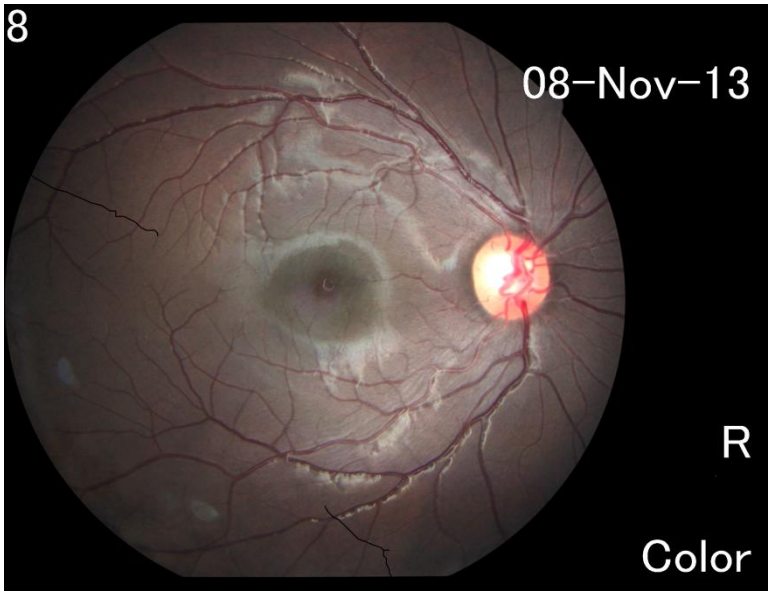
ANNEXURES

FUNDUS PHOTOGRAPHS

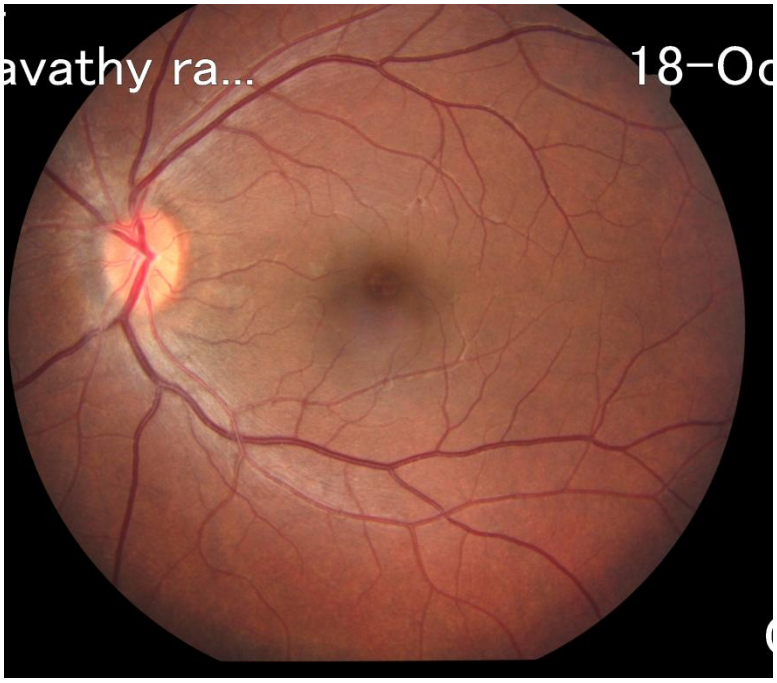
1. CYSTOID MACULAR EDEMA



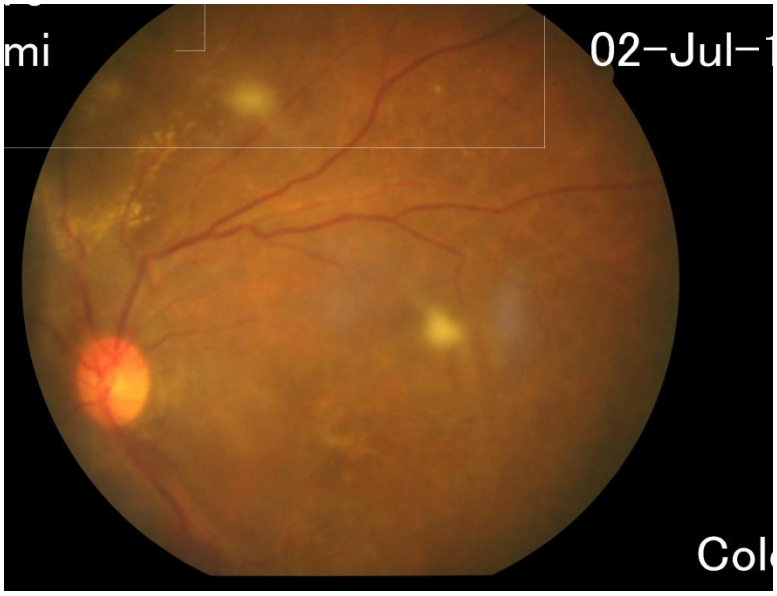
2. CENTRAL SEROUS RETINOPATHY



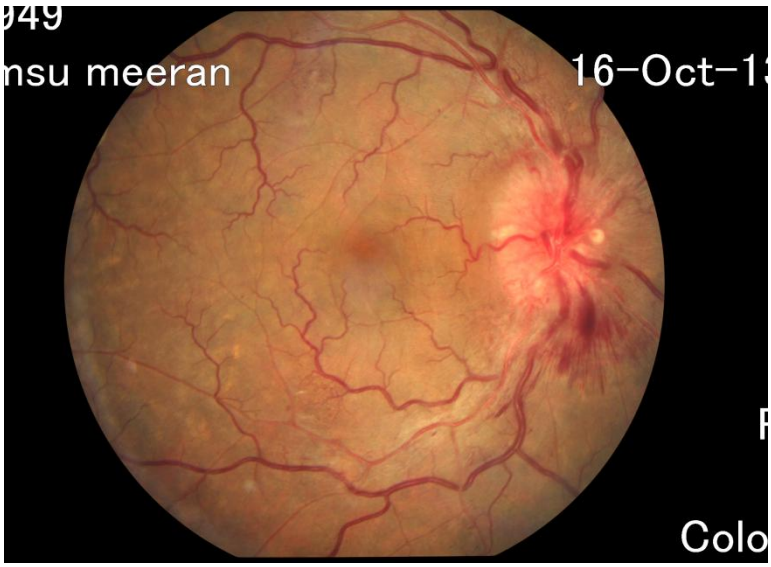
3. GRADE 1 HYPERTENSIVE RETINOPATHY SHOWING GENERALISED ARTERIOLAR ATTENUATION



4. GRADE 3 HYPERTENSIVE RETINOPATHY



5. GRADE 4 HYPERTENSIVE RETINOPATHY



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PROFORMA

NAME OF THE PATIENT : **AGE:** **IP NO:**

ADDRESS:

GRAVIDA:

GESTATIONAL AGE:

PRESENTING COMPLAINTS:

Headache

Blurred vision

Convulsions

Diplopia

Transient loss of vision

PAST HISTORY:

Hypertension

Diabetes mellitus

Coronary artery disease

Collagen vascular disease

EXAMINATION:

Pallor **Pedal edema** **B.P** **Pulse**

OCULAR EXAMINATION:

Lids

Conjunctiva

Cornea

Anterior chamber

Iris

Pupil

Lens

FUNDUS EXAMINATION:

Direct ophthalmoscopy

Indirect ophthalmoscopy

FUNDUS PHOTOGRAPH:

DIAGNOSIS:

INVESTIGATIONS:

MASTER CHART

S. No	Patient Name	Age	Gravida	BP	Symptom	vision	fundus	b(mg%)	proteinuria	BUN (mg/dl)	sr.uric acid (mg/dl)
1	Vijayalakshmi	17	Primi	160/110 with seizures	BV, CS	>6/60	CSR	12	3+	50	10
2	Lalitha	22	Primi	144/94	NIL	>6/60	Grade 1 HTNR	7	1+	12	3.2
3	Bagyalakshmi	18	Primi	150/110	H	>6/60	Normal	9	2+	20	3.1
4	Tamilselvi	23	Multi	146/92	NIL	>6/60	Grade 1 HTNR	10	1+	20	2.6
5	Gayathri	24	Multi	148/94	H	>6/60	Grade 2 HTNR	12	1+	22	4.3
6	Hamsaveli	21	Primi	152/114	NIL	>6/60	Normal	11	2+	42	3.3
7	Sundari	22	Primi	150/110	NIL	>6/60	Grade 2 HTNR	12	2+	28	9.2
8	Lakshmi	22	Primi	140/90	NIL	>6/60	Normal	10	1+	24	3.2
9	Saraswathi	27	Multi	144/92	NIL	>6/60	Normal	9	1+	20	5.2
10	Bhagyavathi	19	Primi	144/90	H	>6/60	Grade 1 HTNR	11	1+	12	2.9
11	Subhalakshmi	23	Multi	142/92	NIL	>6/60	Normal	9	1+	9	4.2
12	Malarvehzi	22	Primi	160/120 with seizures	BV, H	>6/60	Grade 4 HTNR	8	3+	42	12.6
13	Selva rani	21	Primi	148/90	NIL	>6/60	Normal	10	1+	10	5
14	Uma	23	Primi	140/90	H	>6/60	Grade 1 HTNR	10	1+	20	6.1
15	Maheshwari	28	Multi	160/110	NIL	>6/60	Grade 2 HTNR	11	2+	20	4.5
16	Kumari	20	Primi	140/90	NIL	>6/60	Grade 1 HTNR	12	1+	30	6.4

S. No	Patient Name	Age	Gravida	BP	Symptom	vision	fundus	b(mg%)	proteinuria	BUN (mg/dl)	sr.uric acid (mg/dl)
17	Saranya	24	Multi	150/112	NIL	>6/60	Grade 1 HTNR	9	2+	32	4.1
18	Suganya	22	Primi	142/92	NIL	>6/60	Normal	10	1+	22	3.5
19	Indhumathi	23	Multi	146/94	NIL	>6/60	Grade 1 HTNR	8	1+	32	4.9
20	Nandhini	28	Multi	144/90	NIL	>6/60	Normal	9	1+	24	3.3
21	Pallavi	19	Primi	150/118	NIL	>6/60	Grade 1 HTNR	11	2+	30	3.6
22	Ashawathi	22	Primi	144/92	H	>6/60	Normal	8	1+	22	6.3
23	Jayalakshmi	21	Primi	140/90	NIL	>6/60	Grade 1 HTNR	10	2+	28	11.2
24	Anandhavali	23	Multi	150/112	BV, H	>6/60	Grade 3 HTNR	7	3+	71	4.8
25	Anadhi	22	Primi	140/90	NIL	>6/60	Normal	11	1+	12	10.2
26	Kumadha	27	Multi	142/92	NIL	>6/60	Grade 1 HTNR	9	2+	20	4.5
27	Devi	21	Primi	150/100 with seizures	NIL	>6/60	Grade 1 HTNR	12	2+	14	7.2
28	Leela	26	Multi	144/92	NIL	>6/60	Normal	10	1+	24	3.4
29	Latha	23	Primi	146/94	H	>6/60	Grade 2 HTNR	10	1+	40	9.8
30	Swarna	27	Multi	140/90	NIL	>6/60	Normal	9	1+	12	4.6
31	Rani	18	Primi	150/116	NIL	>6/60	Grade 1 HTNR	6	2+	56	5.2
32	Meronizam	21	Primi	152/112	BV, CS, H	>6/60	CSR	8	3+	40	6.7
33	Jhotsna	25	Multi	142/90	NIL	>6/60	Normal	9	1+	28	7.2
34	Asthalakshmi	26	Primi	140/90	H	>6/60	Grade 2	10	1+	20	3.3

S. No	Patient Name	Age	Gravida	BP	Symptom	vision	fundus	b(mg%)	proteinuria	BUN (mg/dl)	sr.uric acid (mg/dl)
							HTNR				
35	Seena	21	Primi	160/112	H	>6/60	Grade 2 HTNR	11	2+	38	6.9
36	Meera	22	Primi	140/90	NIL	>6/60	Normal	9	1+	22	3.1
37	Anuja	25	Multi	140/90	NIL	>6/60	Grade 2 HTNR	12	2+	22	5.1
38	Lakshmi	27	Primi	160/110	H	>6/60	Grade 3 HTNR	11	3+	36	4.6
39	Meena	24	Primi	146/92	H	>6/60	Grade 1 HTNR	8	1+	18	5.3
40	Anitha	21	Primi	150/110	NIL	>6/60	Grade 2 HTNR	7	2+	28	5.2
41	Mumtaj	24	Primi	150/112	H	>6/60	Grade 1 HTNR	11	2+	14	5.4
42	Lalitha	23	Primi	148/92	H	>6/60	Normal	10	1+	20	4.2
43	Parvathi	26	Multi	144/90	H	>6/60	Grade 1 HTNR	9	2+	30	6.5
44	Subhalakshi	22	Primi	140/90	H	>6/60	Normal	12	1+	20	5.3
45	shamsu meeran	22	Primi	160/120 with seizures	BV,H	>6/60	Grade 4 HTNR	9	2+	22	11.8
46	Saranya	27	Multi	140/90	NIL	>6/60	Normal	8	1+	22	6.2
47	Usha	19	Primi	150/114	NIL	>6/60	Grade 2 HTNR	9	2+	26	6.7
48	Mallika	24	Primi	150/112	NIL	>6/60	Normal	10	2+	24	7.2
49	Valli	24	Primi	140/90	NIL	>6/60	Grade 2 HTNR	12	1+	24	2.9
50	Selvi	28	Multi	144/92	NIL	>6/60	Normal	8	1+	12	7.4

S. No	Patient Name	Age	Gravida	BP	Symptom	vision	fundus	b(mg%)	proteinuria	BUN (mg/dl)	sr.uric acid (mg/dl)
51	Thangamaal	22	Primi	150/116	H	>6/60	Grade 3 HTNR	9	3+	52	4.8
52	Kavya	28	Primi	146/94	NIL	>6/60	Normal	12	1+	18	6.1
53	Roja	17	Primi	154/112	NIL	>6/60	Normal	10	3+	28	5.9
54	Shailaja	22	Primi	150/110	H	>6/60	Grade 2 HTNR	9	2+	10	6.8
55	Kumudha	22	Primi	154/102 with seizures	NIL	>6/60	Grade 1 HTNR	10	3+	18	4.3
56	Saroja	24	Primi	148/94	H	>6/60	Grade 1 HTNR	11	1+	20	3.4
57	Savithri	27	Multi	140/90	NIL	>6/60	Normal	7	1+	20	3.6
58	Chellamaal	27	Primi	150/114	NIL	>6/60	Grade 1 HTNR	8	2+	20	7.2
59	Swarna	23	Primi	158/110	NIL	>6/60	Grade 3 HTNR	10	3+	38	4.5
60	Lakshmi	21	Primi	140/90	NIL	>6/60	Normal	12	1+	18	4.9
61	Anitha	26	Multi	140/90	NIL	>6/60	Normal	12	1+	22	5.2
62	Meera	28	Multi	140/90	NIL	>6/60	Normal	11	1+	18	6.1
63	Rajathi	22	Primi	150/116	NIL	>6/60	Normal	10	2+	10	5.2
64	Sridevi	27	Multi	144/92	NIL	>6/60	Normal	9	1+	20	2.4
65	Manorama	23	Primi	146/92	H	>6/60	Grade 2 HTNR	11	1+	30	6.7
66	Kasthuri	36	Multi	144/90	NIL	>6/60	Normal	10	1+	12	4.4
67	Priyanka	22	Primi	150/112	H	>6/60	Grade 2 HTNR	9	3+	32	4
68	Pallavi	28	Multi	148/92	NIL	>6/60	Grade 2 HTNR	8	1+	20	3.2

S. No	Patient Name	Age	Gravida	BP	Symptom	vision	fundus	b(mg%)	proteinuria	BUN (mg/dl)	sr.uric acid (mg/dl)
69	Indhuja	23	Primi	140/90	NIL	>6/60	Grade 1 HTNR	10	2+	24	2.9
70	Bindu	26	Multi	140/90	NIL	>6/60	Normal	11	1+	20	3.9
71	Lalitha	24	Primi	150/110	H	>6/60	Grade 2 HTNR	12	2+	24	4.5
72	Vasuki	25	Multi	140/90	NIL	>6/60	Normal	9	1+	18	3.4
73	Chellama	19	Primi	158/116	NIL	>6/60	Grade 2 HTNR	10	3+	36	5.3
74	Madhuri	27	Multi	140/90	NIL	>6/60	Normal	11	1+	16	4.4
75	Molly	21	Primi	150/114	H	>6/60	Grade 2 HTNR	12	2+	22	4.2
76	Vijaya	27	Multi	150/110	NIL	>6/60	Grade 3 HTNR	9	2+	26	6.8
77	Koushalya	20	Primi	144/90	H	>6/60	Normal	10	1+	20	5.6
78	Meenakshi	36	Multi	156/120	BV	>6/60	Macular Edema	8	3+	40	6.9
79	Pankajam	31	Multi	146/90	NIL	>6/60	Grade 1 HTNR	11	2+	22	2.8
80	Shyamala	24	Primi	152/114	BV, CS	>6/60	CSR	9	2+	66	8.7
81	Mercy	23	Primi	140/90	NIL	>6/60	Grade 2 HTNR	11	1+	18	6.7
82	Dharani	22	Primi	140/90	NIL	>6/60	Normal	8	1+	20	6.4
83	Phuspha	31	Multi	152/112	H	>6/60	Grade 2 HTNR	12	1+	24	8
84	Kaveri	24	Primi	144/92	NIL	>6/60	Normal	10	1+	20	5.7
85	Dhanya	25	Multi	160/120	NIL	>6/60	Grade 3 HTNR	9	2+	10	5.8
86	Anitha	24	Primi	142/90	NIL	>6/60	Normal	10	1+	22	6.2
87	Thenmozhi	32	Multi	150/116	NIL	>6/60	Grade 1	8	2+	12	6.6

S. No	Patient Name	Age	Gravida	BP	Symptom	vision	fundus	b(mg%)	proteinuria	BUN (mg/dl)	sr.uric acid (mg/dl)
							HTNR				
88	Rukmani	32	Multi	150/112	H	>6/60	Grade 1 HTNR	10	2+	20	7
89	Krishnaveni	19	Primi	150/114	NIL	>6/60	Normal	7	2+	22	6.9
90	Sharadha	23	Multi	140/90	NIL	>6/60	Grade 1 HTNR	12	2+	26	3.7
91	Vani	22	Primi	154/112	H	>6/60	Grade 2 HTNR	11	3+	28	4.5
92	Aruna	22	Primi	150/116	NIL	>6/60	Normal	10	1+	10	8.2
93	Visalakshi	24	Multi	142/90	NIL	>6/60	Grade 1 HTNR	8	2+	20	5.6
94	Shravanthi	31	Multi	150/110	NIL	>6/60	Normal	12	1+	12	4.3
95	Veena	21	Primi	150/112	H	>6/60	Grade 2 HTNR	10	3+	20	6.8
96	sushila varma	22	Primi	156/112 with seizures	BV,CS, H	>6/60	Macular Edema	11	3+	42	11.6
97	Sharmila	32	Multi	142/92	NIL	>6/60	Grade 2 HTNR	8	1+	24	4.5
98	Soundarya	23	Multi	154/110	NIL	>6/60	Normal	12	2+	18	6.9
99	Latha	21	Primi	152/114	H	>6/60	Normal	9	2+	10	6
100	Raaji	22	Primi	150/112	NIL	>6/60	Normal	10	2+	12	6.2

PROFORMA

NAME OF THE PATIENT : **AGE:** **IP NO:**

ADDRESS:

GRAVIDA:

GESTATIONAL AGE:

PRESENTING COMPLAINTS:

Headache

Blurred vision

Convulsions

Diplopia

Transient loss of vision

PAST HISTORY:

Hypertension

Diabetes mellitus

Coronary artery disease

Collagen vascular disease

EXAMINATION:

Pallor **Pedal edema** **B.P** **Pulse**

OCULAR EXAMINATION:

Lids

Conjunctiva

Cornea

Anterior chamber

Iris

Pupil

Lens

FUNDUS EXAMINATION:

Direct ophthalmoscopy

Indirect ophthalmoscopy

FUNDUS PHOTOGRAPH:

DIAGNOSIS:

INVESTIGATIONS: