A COMPARATIVE STUDY ON ASSOCIATION BETWEEN SYSTEMIC HYPERTENSION,PERFUSION PRESSURE AND GLAUCOMA IN AN ADULT POPULATION OF NORTH KARNATAKA

By

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In

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Under the guidance of

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LIST OF CONTENTS

LIST OF ABBREVIATIONS

OPP	Ocular Perfusion Pressure
POAG	Primary Open Angle Glaucoma
PACG	Primary Angle Closure Glaucoma
ONH	Optic Nerve Head
BCE	Before Common Era
IOP	Intraocular Pressure
OCT	Optical Coherence Tomography
RGC	Retinal Ganglion Cell
ER	Endoplasmic Reticulum
C/D ratio	Cup/Disc ratio
VHG	Van Herick Grading
AC	Anterior Chamber
CT	Corneal Thickness
Na-K ATPase	Sodium Potassium adenosine triphosphatase
TM	Trabecular Meshwork
CC	Collector Channel
CP	Ciliary Processes
GAT	Goldmann Applanation Tonometer
AGM	Anti Glaucoma Medication

LSD	Lysergic acid Diethylamide
EVP	Episcleral Venous Pressure
COAG	Chronic Open Angle Glaucoma
NCT	Non-Contact Tonometer
ECM	Extra Cellular Matrix
JCT	Juxtacanalicular Connective Tissue
SC	Schlemm's Canal
SBP	Systolic Blood Pressure
DBP	Diastolic Blood Pressure
CHD	Coronary Heart Disease
ARIC	Atherosclerosis Risk In Communities
MAP	Mean Arterial Pressure
CRV	Central Retinal Vein
CRA	Central Retinal Artery
MOPP	Mean Ocular Perfusion Pressure
ROS	Reactive Oxygen Species
FD-OCT	Fourier Domain Optical Coherence Tomography
TRBF	Total Retinal Blood Flow
SSADA	Split-Spectrum Amplitude Decorrelation Angiography

OBF	Ocular Blood Flow
NO	Nitric Oxide
ET-1	Endothelin-1
NOS	Nitric Oxide Synthase
CAI	Carbonic Anhydrase Inhibitor
RNFL	Retinal Nerve Fiber Layer
OHTS	Ocular Hypertension Treatment Study
EGPS	European Glaucoma Prevention Study
SD-OCT	Spectral Domain Optical Coherence Tomography
PGA	Prostaglandin Analogs
SLT	Selective Laser Trabeculoplasty
MIGS	Minimally Invasive Glaucoma Surgery
SD	Standard Deviation
IOPR	Intraocular pressure of the Right Eye
IOPL	Intraocular pressure of the Left Eye
OPPR	Ocular Perfusion Pressure of the Right Eye
OPPL	Ocular Perfusion Pressure of the Left Eye
MSPP	Mean Systolic Perfusion Pressure
MDPP	Mean Diastolic Perfusion Pressure
CCB	Calcium Channel Blocker

LIST OF TABLES

LIST OF GRAPHS

LIST OF FIGURES

Abstract

Purpose: Glaucoma, a leading cause of blindness, involves progressive retinal ganglion cell loss and optic nerve damage. It affects 3.54% of individuals aged 40-80, with a higher prevalence in Africa and Asia. Raised intraocular pressure is a known risk factor, but blood supply to the optic nerve also plays a role. Systemic hypertension and nocturnal blood pressure drops may influence glaucoma, though their exact relationship remains unclear. This study aims to examine the impact of blood pressure and perfusion pressures on glaucoma in North Karnataka's adult population.

Materials and Methods: This cross-sectional study on the association between systemic hypertension, perfusion pressure, and glaucoma was conducted in hypertensives and normotensives from May 2023 to December 2024. A comprehensive eye examination was conducted, including blood pressure and intraocular pressure measurements, from which mean perfusion pressures were calculated. Statistical analysis was performed using the Chi-square test, Mann Whitney U test, Spearman rho and Kruskal Wallis Test at $p<0.05$ significance.

Results: This study evaluated 168 participants (84 hypertensive cases and 84 normotensive controls). Hypertensive individuals showed significantly higher intraocular and ocular perfusion pressure than controls at all time intervals ($p<0.05$). Blood pressure was highest at 1 pm and 6 pm. OPP values were positively correlated with systolic, diastolic, and mean arterial pressures ($P<0.05$). Antihypertensive medications significantly influenced OPP, with patients on combined medications showing the highest OPP.

Conclusion: This study underscores the importance of screening hypertensive individuals for glaucoma, emphasizing the need to carefully evaluate blood pressure, intraocular pressure and ocular perfusion pressures. Managing these factors is crucial for preventing optic nerve damage and glaucoma progression.

Introduction

"Modern medicine can prevent glaucoma blindness, yet too many are left in the dark."

- Robert Brown

Glaucoma is a chronic, slowly progressive disease with loss of retinal ganglion cells and their neurons (1). Glaucoma is characterized by optic neuropathy with a characteristic cupping of the optic nerve head and a distinctive pattern of visual field loss (2). Glaucoma is the second leading cause of blindness globally, after cataracts (3). Globally, 3.54% of individuals aged 40-80 years are affected by glaucoma (4). The prevalence of POAG is highest in Africa at 4.20%, while Asia has the highest prevalence of PACG at 1.09%. In 2013, approximately 64.3 million people worldwide had glaucoma, which can rise to 111.8 million by 2040 (4). The pathophysiology of glaucoma is not fully understood owing to its multifactorial nature (3,5).

Among various risk factors for glaucoma, raised intraocular pressure is modifiable, as it causes a direct mechanical effect on the optic nerve head (6,7). The most common cause of elevated intraocular pressure is a reduction in the outflow capacity of aqueous humor, typically occurring at the anterior chamber angle and the trabecular meshwork (1). It is widely acknowledged that other additional factors, particularly those influencing the blood flow to the ONH, might play a crucial role (5).

Systemic hypertension might contribute to glaucoma risk by directly affecting the small vessels of the optic disc. However, having systemic hypertension alone does not appear to increase the chances of glaucoma or being suspected of having the condition (3). Nighttime reductions in blood pressure may be involved in glaucoma progression and are poorly understood (1).

Some research points to systemic hypertension as a potential risk factor for glaucoma, whereas other studies highlight low systemic pressure as a contributing factor to its

onset and progression. Despite this, the link between blood pressure levels and glaucoma remains unclear (7).

The present study is taken up to determine the relationship between systemic hypertension, perfusion pressure, and glaucoma in an adult populationof North Karnataka and to study the effect of blood pressure and nocturnal hypotension in the pathogenesis of glaucoma.

Aims and Objectives

- To determine the relationship between blood pressure, ocular perfusion pressure and glaucoma in an adult population of North Karnataka.
- To study the effect of blood pressure and nocturnal hypotension in the pathogenesis of glaucoma.

GLAUCOMA HISTORY

The word "glaucoma" comes from the ancient Greek word "glaukos", which means "blue," "green," or "light grey." Glaucoma does not imply anything regarding pupil color in the present day. Instead, glaucoma constitutes a range of conditions with an underlying optic neuropathy frequently linked to increased intraocular pressure (8–10). The understanding and management of glaucoma have evolved significantly over time, with contributions from various cultures and scientific advancements.

Ancient Period:

Hippocrates (460-370 BCE) described a condition resembling glaucoma as "glaykosis". This referred to a cloudy or bluish eye appearance (9).

Medieval period:

During the Middle Ages, Arab scholars, including Al-Razi and Ibn Sina, advanced the understanding of ocular diseases. They described conditions that resemble glaucoma and emphasized its irreversible blindness (11).

Renaissance period:

The term "glaucoma" became more defined during the Renaissance period. Physicians began associating it with the hardening of the eyeball and vision loss. Early efforts to measure intraocular pressure were made in the 17th Century using crude techniques (10).

18th and 19th Century:

The modern understanding of glaucoma began in the 18th century with the invention of the ophthalmoscope. The advent of the ophthalmoscope marked a period of significant advancements

in glaucoma management. A German ophthalmologist, Albrecht von Graefe (1828-1870), significantly contributed to the field by identifying the link between increased IOP and optic nerve damage. Graefe also pioneered surgical interventions like iridectomy for angle-closure glaucoma (10).

20th Century:

The invention of the Goldmann applanation tonometer in the mid-20th century revolutionized the measurement of IOP, making it a cornerstone of glaucoma diagnosis. The concept of primary open-angle glaucoma (POAG) and angle-closure glaucoma (ACG) was established, resulting in a formal classification system (10). Since the 19th century, pharmacologic treatments to reduce intraocular pressure have included eserine and pilocarpine, followed by epinephrine, adrenergic agonists, carbonic anhydrase inhibitors, beta-blockers, and prostaglandin analogs in the 20th century (12).

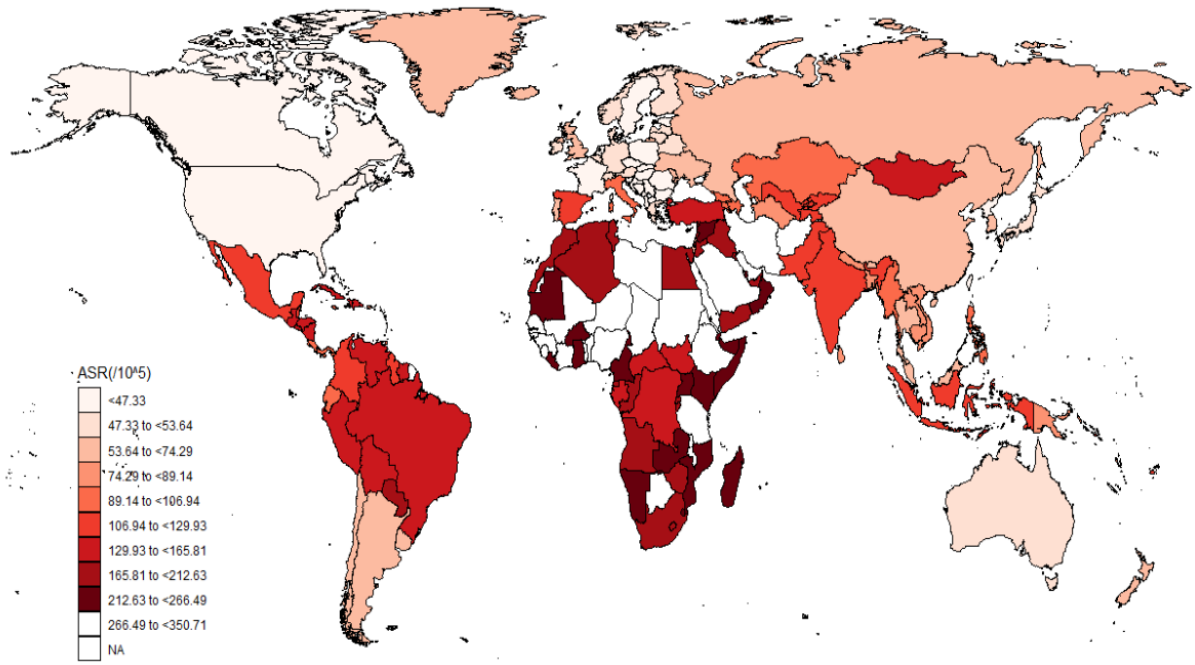
21st Century:

Advanced imaging techniques like optical coherence tomography (OCT) have enhanced early detection and monitoring of glaucoma(13). Minimally invasive glaucoma surgeries and new pharmacological agents have expanded treatment options, emphasizing individualized care.

EPIDEMIOLOGY OF GLAUCOMA

Glaucoma impacts over 67 million people globally, with approximately 6.6 million people believed to be blind. According to Bourne et al., glaucoma is the second leading cause of blindness globally, after cataracts (14). According to Thylefors et al., glaucoma causes 14% of all recorded cases of blindness(15). The number of people aged 40 to 80 years affected by glaucoma worldwide was estimated to be 64.3 million in 2013 and 76.0 million in 2020. Tham et al. predict this will rise to 111.8 million by 2040 (4).

Figure 1: The global impact of glaucoma on both genders in 2019, covering 204 countries and territories, according to a 2023 study (16)



Leske et al. stated that the risk of glaucoma increases with age, with prevalence peaked in individuals aged 70 years and older (17).

Cook et al. stated that POAG is more prevalent in Africa, and limited healthcare access exacerbates the condition's progression and associated blindness (18).

Liang et al. mentioned that PACG is a significant concern in Asia, particularly in China and India (19).

DEFINITION OF GLAUCOMA

Glaucoma is a chronic, progressive optic neuropathy encompassing various ocular conditions that cause damage to the optic nerve (retinal ganglion cell death by apoptosis) with loss of visual function and normal or raised intraocular pressure (20).

CLASSIFICATION OF GLAUCOMA

The European Glaucoma Society provides a detailed classification of glaucoma, as outlined here (21):

I) Primary Congenital Forms/Childhood Glaucoma

a) Primary Congenital Glaucoma

b) Late-onset childhood open-angle glaucoma/Early Juvenile glaucoma

c) Secondary Childhood Glaucoma

II) Primary Open-angle Glaucoma

a) Primary Open-Angle Glaucoma

- Primary Open-Angle Glaucoma / High-pressure Glaucoma
- Primary Open-Angle Glaucoma / Normal Pressure Glaucoma

b) Primary Juvenile Glaucoma

c) Primary Open-Angle Glaucoma Suspect

d) Ocular Hypertension

III) Secondary Open-Angle Glaucoma

IV) Primary Angle Closure

a) Primary Angle-Closure Suspect

b) Acute Angle Closure

c) Intermittent Angle Closure

d) Chronic Angle-Closure Glaucoma

e) Status Post-Acute Angle-Closure Suspect

V) Secondary Angle Closure

The two main categories of glaucoma are primary and secondary, with open-angle and angle-closure glaucoma being the major subtypes (22). Open-angle glaucoma is classified into primary

open-angle, normal tension and secondary open-angle, whereas primary angle closure and secondary closed-angle glaucoma are both included under closed-angle glaucoma (23).

ANATOMY AND PHYSIOLOGY

❖ OPTIC NERVE HEAD

The optic nerve plays a crucial role in the pathophysiology of glaucoma and is divided into four parts (24)

1. Intraocular part (Optic nerve head) (1 mm in length)

2. Intraorbital part (25–30 mm)

3. Intracanalicular part (4–10 mm)

4. Intracranial part (10 mm)

At the surface of the optic nerve head, which is the most susceptible part to increased IOP, retinal ganglion cell axons make a sharp bend as they pass through the fenestrated scleral canal, known as the lamina cribrosa. These axons are grouped into roughly 1,000 fascicles within the ONH and are aided by astrocytes (25).

Divisions of the ONH:

From anterior to posterior, it is divided into four distinct portions,

1. Surface Nerve Fiber Layer

Made up of nerve fibers and is the innermost layer. As these axonal bundles are traced posteriorly through the intraocular portion, they gradually acquire more interactional glial tissue (26).

2. Pre-Laminar area

The anterior segment of the lamina cribrosa comprises axons and astrocytes (25).

3. Lamina Cribrosa area

The lamina cribrosa comprises fenestrated sheets of scleral connective tissue and elastic fibers. Fenestrae are lined by the astrocytes, dividing the layers, with neuronal fascicles traversing these gaps (25).

4. Retro-laminar area

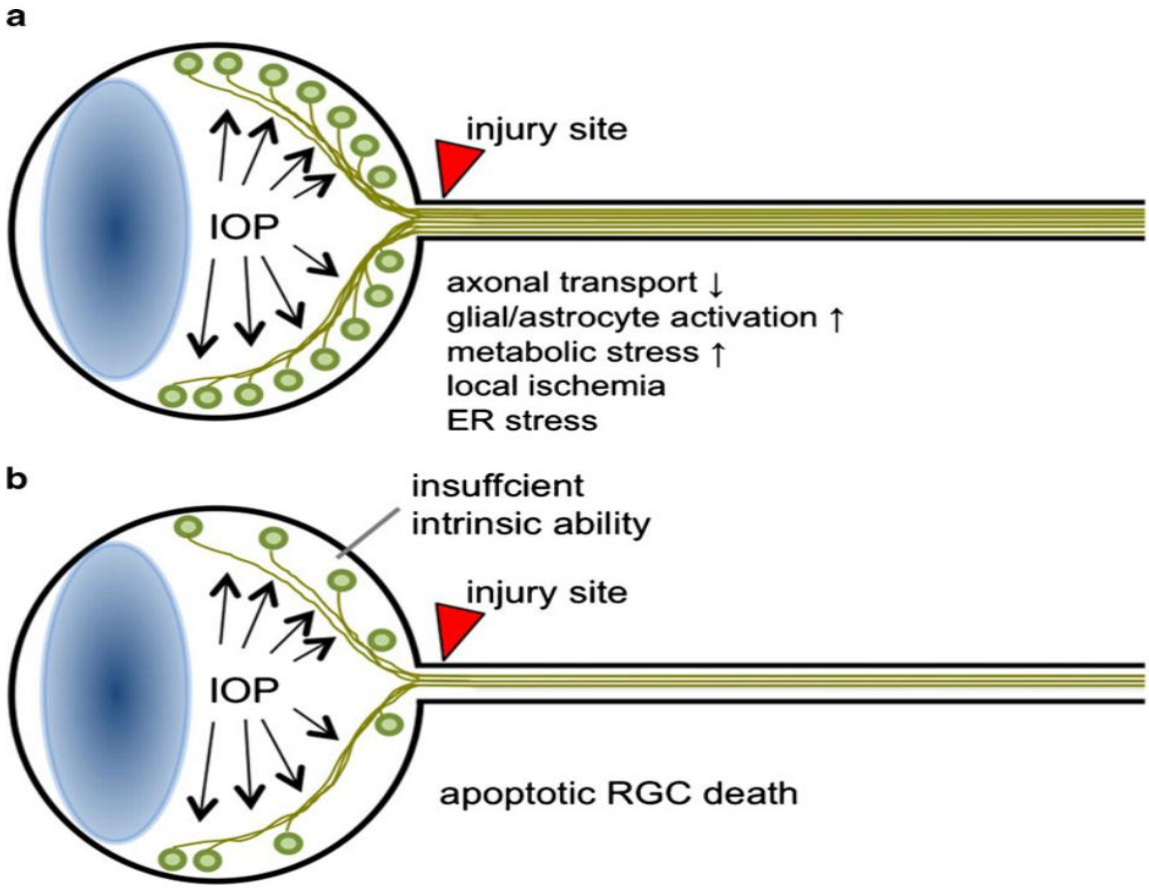
The key features of this are a reduction in astrocytes and the presence of myelin, which is synthesized by oligodendrocytes. Connective tissue septa enclose the axonal bundles.

The exact posterior limit of the retrolaminar region remains unclear. In an India ink experiment on monkey eyes, it was found that the ink did not reach 3 to 4 mm behind the lamina cribrosa under elevated intraocular pressure conditions (27).

Conversely, a comparable study by Author et al., using unlabeled microspheres, indicated enhanced blood flow in the retrolaminar region despite elevated IOP, causing retinal blood flow to cease (25).

Glaucomatous optic atrophy appears to stem from disturbances in axoplasmic flow. It remains uncertain whether this is directly caused by the mechanical effects of elevated intraocular pressure or if it results from vascular changes. The damage to the ONH and the nerve fiber layer, which contains retinal ganglion cell axons, is strongly linked with the vision loss seen in glaucoma (25).

Figure 2: Pathophysiology of glaucoma (28): a) Eye showing retinal RGC axons projecting to the optic nerve. Glaucoma, marked by raised IOP and localized changes at the ONH, results in axonal damage in the laminar region due to factors such as reduced axonal transport, increased glial activation and metabolic stress, local ischemia and ER stress b) Distally, the axons degenerate after injury, whereas proximally, the axons survive and do not regenerate. Ultimately, there is apoptotic death of RGCs in the retina, thus inducing partial visual field loss.



Aqueous humor dynamic factors—which are closely linked to IOP—are essential to our comprehension of glaucoma because, in addition to being the most prevalent and well-understood causative risk factor, they are currently modifiable factors that can prevent the progression of optic neuropathy(25). Other risk factors include family history, race, advancing age, myopia, hyperopia, central corneal thickness, C/D ratio, smoking, systemic hyper- and hypotension, vasospasm, and diabetes(29–33).

❖ INTRAOCULAR PRESSURE

It is the pressure exerted by the fluids within the eye. Pressure = Force/Area; therefore, intraocular pressure refers to the force the aqueous humor applies to the eye's internal surface. IOP results from a precise balance between aqueous humor production and drain(33).

Understanding the aqueous humor production and outflow on a fundamental level helps address intraocular pressure.

▪ Aqueous humour dynamics

Anatomy

Aqueous humor flows out mainly through the anterior chamber angle, containing the trabecular meshwork. In contrast, the ciliary body, where the aqueous humor is produced, is also critical to its overall regulation.

The ciliary body splits into pars plicata anteriorly and pars plana posteriorly, forming a part of the anterior chamber angle. The ciliary processes within the pars plicata, specifically in the corona ciliaris, produce aqueous humor. These include 70–80 major ciliary processes, more prominent ridges, and smaller minor and intermediate ones. The smooth muscle in the pars plicata also contributes to accommodation and uveoscleral outflow.

The components of the anterior chamber angle, from posterior to anterior, include the iris root, ciliary body band, scleral spur, trabecular meshwork and Schwalbe's line.

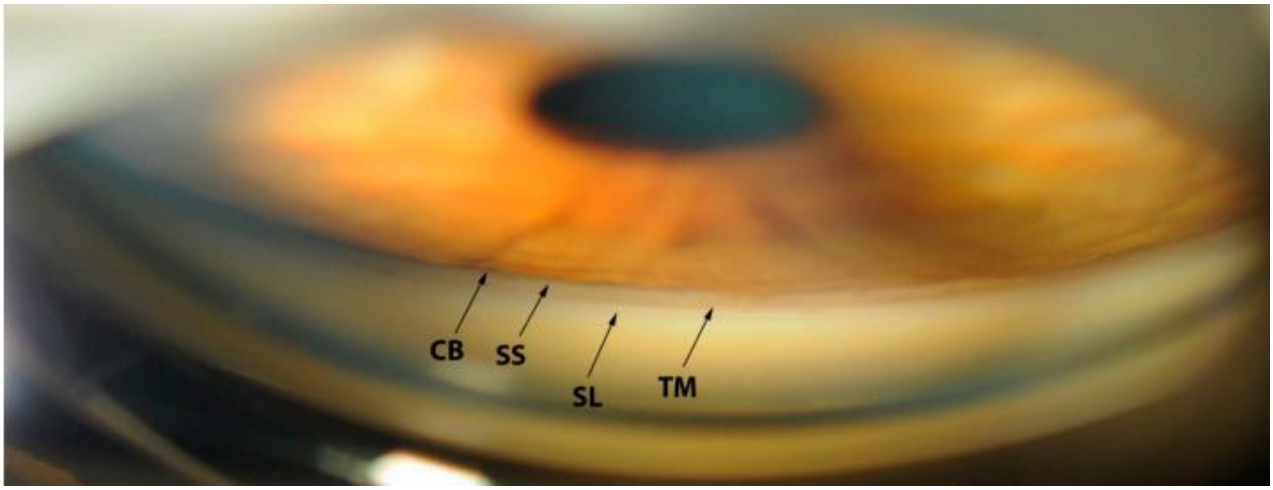


Figure 3: Gonioscopy view of anterior chamber angle (34)

As the iris extends into the anterior side of the ciliary body, it exposes the ciliary body band—a later structure that varies in width—between the iris root and the scleral spur.

The area where the cornea and the sclera meet is called the limbus. The scleral sulcus features a distinct posterior boundary, and the scleral spur and its anterior wall that reaches the peripheral cornea are the indentations on the inner side of the limbus (25).

The trabecular meshwork - resembles a sieve. It is categorized into 1) Uveal meshwork, 2) Corneoscleral meshwork, and 3) Juxta-canalicular meshwork (25).

Uveal meshwork- Forms the innermost part of the trabecular meshwork. It is organized into rope-like trabeculae that stretch towards the peripheral cornea (25).

Corneoscleral meshwork- Forms the middle layer, stretches to the scleral sulcus from the scleral spur and comprises trabecular sheets (25).

Juxta-canalicular meshwork- Forms the outermost part and consists of a connective tissue lined with endothelium (25).

A ridge, known as the Schwalbe line, is created where the trabecular meshwork inserts into the peripheral cornea. The Schlemm canal communicates with the episcleral veins through intrascleral channels (25).

The main route of aqueous humor outflow involves the trabecular meshwork, Schlemm canal, and intrascleral channels, known as the conventional flow. This understanding is fundamental in studying aqueous humor dynamics and its relevance to glaucoma treatment(25).

Grading of anterior chamber angle

1. Anterior chamber angle grading system using slit lamp bio-microscopy

    - Van Herick grading system uses a 4-level grading scheme, in which limbal ACD is graded ≤25% (VHG 1), 25% (VHG 2), >25% and ≤50% (VHG 3), and >100% of the corneal thickness (VHG 4)(34)

2. Anterior chamber angle grading system using gonioscopy (35)

a) Shaffer's grading

- Shaffer's grading is the most commonly used. It evaluates both the angle width and the angle structures to determine the classification of angle grades (35).
- Consideration of the angle structures: in grade 3, the structures up to the scleral spur; in grade 2, up to the trabecular meshwork; in grade 1, only the Schwalbe's line is visible; and in grade 0, none of the angle structures are visible (35).
- Considering the angle width, angles between 35 and 45 degrees are classified as grade 4, those between 20 and 35 as grade 3, those between 10 and 20 as grade 2 and those ≤10 as grade 1, a closed angle (zero degrees) classified as grade 0 (35).

b) **Scheie's grading**: Scheie's angle grading uses larger numbers to represent narrower angles and similarly classified angle pigmentation (35).

c) **Spaeth's grading**: Primarily involves evaluating the extent of iris insertion, angle width, and the structure of the peripheral iris (35).

- **Aqueous humor outflow dynamics**

Fluorophotometry is the best method to measure aqueous flow (36).

Comprehensive knowledge of aqueous humor dynamics is an academic pursuit and a crucial tool for assessing and treating glaucoma. This understanding is a cornerstone in the management of this sight-threatening condition.

The ciliary processes in the pars plicata part fill the sulcus and anterior chamber and enter the posterior chamber through three mechanisms.

1)In diffusion, a form of passive transport, lipid-soluble substances pass through the lipid layers of the cell membrane, with the movement being related to the concentration gradient.

2)In ultrafiltration, a passive process, water-soluble molecules flow through the cell membrane in response to an osmotic gradient in the ciliary processes. Capillary, as well as intraocular pressures, have a hydrostatic pressure difference that allows fluid to pass into the eye, while the oncotic gradient prevents it(25,37).

3)In active transport, which relies on the energy-dependent (Na-K ATPase), larger and charged water-soluble substances are moved across the cell membrane.

The aqueous humor inflow and outflow determine the IOP. An increase in IOP due to impaired aqueous flow may result in optic nerve damage and glaucoma(38). The outflow is pressure-dependent and pressure-independent (0.22 to 0.30 μL/min/mmHg). Outflow facility declines with age and can be influenced by surgery as well as trauma, medications and endocrine factors. Glaucoma patients with elevated IOP have a lower outflow facility (25,37).

The two pathways of aqueous humor drainage are trabecular (conventional) and uveoscleral (unconventional) outflow.

A) Conventional outflow

Aqueous in the anterior chamber (AC) enters multi-layered trabecular meshwork (TM) and then Schlemm's canal, collector channels (CCs), intrascleral venous plexus and finally into aqueous and episcleral veins, which join the venous circulation (38). Trabecular outflow accounts for about 80-90% of total aqueous humor outflow(25).

B) Unconventional outflow

Aqueous from the anterior chamber (AC) enters the ciliary body cleft through the suprachoroidal space, which finally enters venous circulation of the ciliary body, sclera and orbit (38).

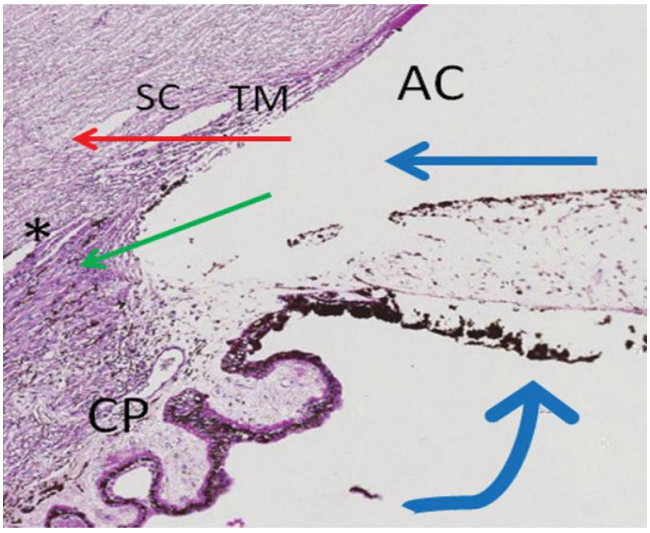


Figure 4: Aqueous Humor Outflow (38)

Aqueous from AC (anterior chamber) secreted by CP (ciliary processes) enters two pathways. The red arrow represents conventional outflow, and the green arrow represents unconventional outflow. TM is a trabecular meshwork, and SC is Schlemm's canal.

Uveoscleral outflow accounts for about 5-15% of total aqueous humor outflow (25).

The uveo-lymphatic pathway, a variation of the uveoscleral pathway, has been found to have an alternate lymphatic endpoint (39).

Contraction of the ciliary muscle (e.g., Parasympathomimetics) leads to increased trabecular outflow and decreased uveoscleral outflow. In contrast, vice-versa occurs during ciliary muscle relaxation (e.g., Cycloplegics) (40).

IOP is influenced by trabecular, uveoscleral outflow, episcleral venous pressure, and aqueous humor generation (measured as aqueous flow)(36).

"GOLDMANN'S EQUATION" (41),

$IOP = (F/C) + P$

F=Aqueous flow rate (Fin-Fout), Fin=Aqueous production and Fout= Unconventional aqueous outflow, C = conventional aqueous outflow, P = episcleral venous pressure

Changes in these variables will influence the IOP.

- ## IOP value:

In healthy adults, the average value is roughly 15 mmHg; its typical variation in the population is between 10 and 21 mmHg(42–44)

Goldmann applanation tonometer (GAT) gives the most accurate IOP values. With accuracy similar to GAT, the rebound tonometer is more dependable than the NCT and portable. Thus, this study used a rebound tonometer (iCare IC100) (45).

- ## Factors affecting IOP

1. Genetics

Studies have shown that heredity contributes to IOP (46–48). Recent studies have demonstrated several loci, or chromosomes, linked to IOP. "Beaver-Dam Eye Study" showed seven loci on

chromosomes “2, 5, 6, 7, 12, 15, and 19” associated with IOP (49). Nevertheless, these chromosomal regions have not been found to contain any “IOP genes.”

2. Environment

Environmental factors influencing intraocular pressure can be categorized into physical factors, cigarette smoking, dietary categories, and drug-related factors. Exposure to cold seems to lower IOP, likely because of lowered episcleral venous pressure (50). In contrast, low gravity triggers a significant spike in IOP because of upward fluid shifts within the body (51).

Smoking tobacco causes an immediate but temporary rise in the IOP, which may occur due to vasoconstriction and an increase in episcleral venous pressure (52).

The effects of different drugs, including AGMs, general anesthetics, recreational drugs, and systemic drugs, differ. General anesthesia generally reduces IOP, although some sedatives, such as ketamine, do not have this effect. Depolarizing muscle relaxants, like succinylcholine and suxamethonium, temporarily elevate IOP, possibly due to extraocular muscle contraction combined with intraocular vasodilation. Recreational drugs reduce IOP except LSD, which elevates the IOP. Clinically significant systemic medicines that may alter IOP include corticosteroids, anticholinergic agents, rare reactions to sulfonamides, anticholinergics, antihistamines, decongestants, and psychiatric drugs (25).

The influence of diet on IOP remains unexplored mainly (53). However, alcohol and omega-3 fatty acids lower IOP, but caffeine increases IOP (50).

2. Physiological

- Gender

In general, sex does not appear to significantly affect IOP in individuals between 20 to 40 years old. However, in older populations, the increase in IOP with age is more pronounced in women

than in men (25). The Barbados Eye Study involved diverse participants and showed higher IOP in women than in men (54).

- Age

With age, IOP tends to rise (25). Regarding the effects of aging on aqueous humor dynamics, research indicates a decline in the efficiency of both aqueous and uveoscleral outflow, along with a reduction in the production of aqueous humor. EVP appears to remain relatively stable with age (55).

-Ethnicity

As previously mentioned, there is a heightened risk for open-angle glaucoma among Black individuals and a risk for ACG in Asian populations (56). Black race has a thin cornea, larger C/D ratios, and elevated intraocular pressure, which raises the overall risk (57).

-Refractive error

Increased IOP leads to axial myopia in infants, as evidenced by buphthalmos. The connection between rising IOP and myopia has yielded mixed results in research, with some studies finding no link and others identifying it as a potential risk factor (25).

-Diurnal and postural variations

Like various biological measures, IOP is influenced by daily cyclical fluctuations. Knowing the daily changes in IOP and its absolute value could help assess the possibility of developing ocular pathology and modifying treatment plans for individuals with pre-existing disorders (58,59). The regulation of diurnal intraocular pressure variation involves complex physiological mechanisms. Increases in cortisol levels, shifts in blood and venous pressure circadian rhythms, aqueous humor formation, seasonal variations, and body position contribute to the diurnal variation in IOP (60,61).

A study by David et. showed that 40% of participants had peak IOP in the early morning, while 65% experienced it before noon (62). Recent investigations have focused on postural changes in IOP, showing a consistent increase in IOP at night, which is physiologically vital because individuals sleep supine (63,64). The main clinical benefit of assessing diurnal IOP variation is to prevent overlooking an elevation in pressure with single measurements. However, implementing diurnal measurements is often impractical, and the logistics of performing these measurements pose practical challenges. Many doctors measure IOP using a modified diurnal curve, taking readings every two hours in the office from early hours in the morning to late hours in the afternoon. Assessing IOP while the patient is supine during office visits is a better predictor of peak nocturnal IOP than sitting measurements (65).

Additionally, the IOP of the dependent eye increases when a person is in the lateral decubitus position. Gathering clinical history regarding sleep patterns and exercise types, especially yoga, may be significant for glaucoma patients (25).

-Exertional Influences

Activities that involve straining, such as the "Valsalva maneuver", electroconvulsive therapy, performing on high-resistance musical instruments, have shown to increase IOP. The mechanisms at play include elevated EVP, especially with the Valsalva maneuver, engorgement of the uveal tract, and potentially increased tone of the orbicularis (64,66,67).

-Eyelid and Eye movement

It has been shown that blinking can lead to an increase of 10 mm Hg in IOP, whereas vigorous squeezing of the eyelid can increase up to 90 mm Hg. In patients with thyroid ophthalmopathy, assessing IOP in an upgaze position during slit lamp examinations may be a useful clinical indicator of glaucoma risk (25).

-Systemic conditions

From a public health perspective, the two most frequently examined systemic diseases for their potential role in increasing the risk of glaucoma are hypertension and diabetes mellitus (25).

▪ Tonometry

Clinical tonometers determine IOP by relating the globe's deformation to the forces responsible for it.

Classification of tonometers:

They are categorized into 2 types:

i) Direct Tonometer – Manometry

ii) Indirect Tonometer

a) Indentation Tonometer (Truncated cone) - Schiotz Tonometer

b) Applanation Tonometer (Simple flattening)

- Contact Tonometer (Goldmann and Perkins)
- Non-Contact Tonometer (Air-Puff and Pulse Air)

c) Rebound Tonometer

IOP values depend on the consistency and accuracy of their measurements.

Schiotz indentation tonometer determines IOP by applying gravitational pressure to indent the eyeball, measuring the depth of corneal indentation with a metal plunger, and converting the reading to mmHg using a line scale (25).

Applanation tonometry is more accurate than indentation tonometry. The Goldmann applanation tonometer is the gold standard and gives the most accurate IOP values (45).

It relies on “Imbert Fick law” applied to thin-walled spheres (45):

$Pt = W/A \Rightarrow W = Pt \times A$

W = External force against the sphere, Pt = Pressure within the sphere, A = Area flattened by the external force

Non-contact tonometer (NCT) measures intraocular pressure by flattening the cornea with a focused puff of air. Since NCT does not involve direct eye contact, it is less uncomfortable for patients and a valuable screening tool (45).

The rebound tonometer uses a light probe that strikes the cornea and rebounds. Calculating the probe’s deceleration determines the IOP and displays the results after six rebounds (45). Due to its accuracy, similar to GAT, and its portability nature, the rebound tonometer (iCare IC100) was used in this study.

- **IOP homeostasis**

Changes in the aqueous humor outflow resistance are principally responsible for maintaining IOP; the extracellular matrix (ECM) is assumed to be the primary component of this resistance, and continuous ECM turnover is necessary to sustain it(68–70).

Numerous investigations examined the possibility of perfused human anterior segment organ cultures demonstrating IOP homeostatic behavior (71). When pressure is elevated in a perfusion organ culture, the anterior segment can perceive it and react by changing the outflow resistance (69,72,73).

ECM purportedly supplies a significant portion of the resistance; nevertheless, the pressure shift would stretch or distort the ECM, causing it to vary in composition, quantity, or organization and

altering the outflow resistance. As a result, the modified outflow resistance would cause the IOP to change(74).

There appears to be a direct cellular contribution that influences the outflow resistance in a way that is now only partially understood, maybe going beyond the ECM turnover involvement(70,75–77).

- **IOP homeostasis and glaucoma**

Undoubtedly, a common feature of most cases of glaucoma is the lack of IOP homeostatic function. IOP elevation is still a significant risk factor for glaucoma since it is primarily caused by the incapacity to keep the outflow resistance within allowable limits to prevent prolonged IOP elevation. Many cases of glaucoma result in a loss of efficient IOP homeostasis, which may be caused by (1) insufficient IOP sensing, (2) an inability to mount an effective outflow resistance adjustment, or (3) an irreversibly broken or disorganized resistance and restoration system(74).

On the other hand, glaucoma triggers and their aftermath may reduce TM JCT/SC cells' overall capacity to operate; as a result, the impact on the IOP homeostasis may be more indirect than a direct effect(78).

SYSTEMIC BLOOD PRESSURE

High blood pressure is the most critical risk factor for cardiac diseases. A "systolic blood pressure" (SBP) of ≥ 140 mmHg or a "diastolic blood pressure" (DBP) of ≥ 90 mmHg is considered "elevated blood pressure" (79).

A) Hypertensive retinopathy

Approximately one billion people worldwide suffer from hypertension, which is the most significant modifiable risk factor. Cardiovascular risk and systemic target organ damage are linked to hypertension. One of the markers of injury to the target organ is retinopathy(80,81).

A range of retinal vascular abnormalities, including both temporary and permanent microvascular damage from high blood pressure, are pathologically associated with hypertensive retinopathy(82).

I) Symptoms

The majority of people who have hypertensive retinopathy do not show any symptoms. Some people might experience clouded or diminished eyesight(83).

II) Signs

Signs include the development of arteriovenous crossing changes, retinal hemorrhages, retinal microaneurysms, soft exudates and, in extreme situations, optic disc and macular edema (84).

III) Classification of Hypertensive Retinopathy

Hypertensive retinopathy can be categorized as follows(80),

In the ***mild*** stage, there is generalized or focal narrowing of the arterioles, arteriovenous nicking, and increased arteriolar opacity.

In the ***moderate*** stage, retinal microaneurysm, retinal hemorrhages, soft and hard exudates are observed

In the ***malignant*** stage, moderate retinopathy is accompanied by papilloedema.

Table 3: The Keith, Wagener, and Barker classification of hypertensive retinopathy classification (81)

Grade	Classification	Symptoms
Grade I (mild hypertension)	Mild generalized retinal arteriolar narrowing or sclerosis	No symptoms
Grade II (more marked hypertension retinopathy)	Definite focal narrowing and arteriovenous crossings. Moderate to marked sclerosis of the retinal arterioles. Exaggerated arterial light reflex	Asymptomatic
Grade III (mild angiospastic retinopathy)	Retinal hemorrhages, exudates and cotton wool spots. Sclerosis and spastic lesions of retinal arterioles	Symptomatic
Grade IV	Severe grade III and Papilloedema	Reduced survival

The retina offers a glimpse into the human circulatory system. The anatomy and physiology of retinal arterioles are comparable to those of cerebral and coronary microcirculation, and they are readily and non-invasively visible. Accordingly, there is even more biological support in the case of hypertensive individuals for the link between hypertensive retinopathy and CHD(81,82).

According to the ARIC Study, those with soft exudates, retinal hemorrhages, and retinal microaneurysms had 2 to 3 times increased risk of experiencing a clinical stroke episode over three years compared to those without these abnormalities(80).

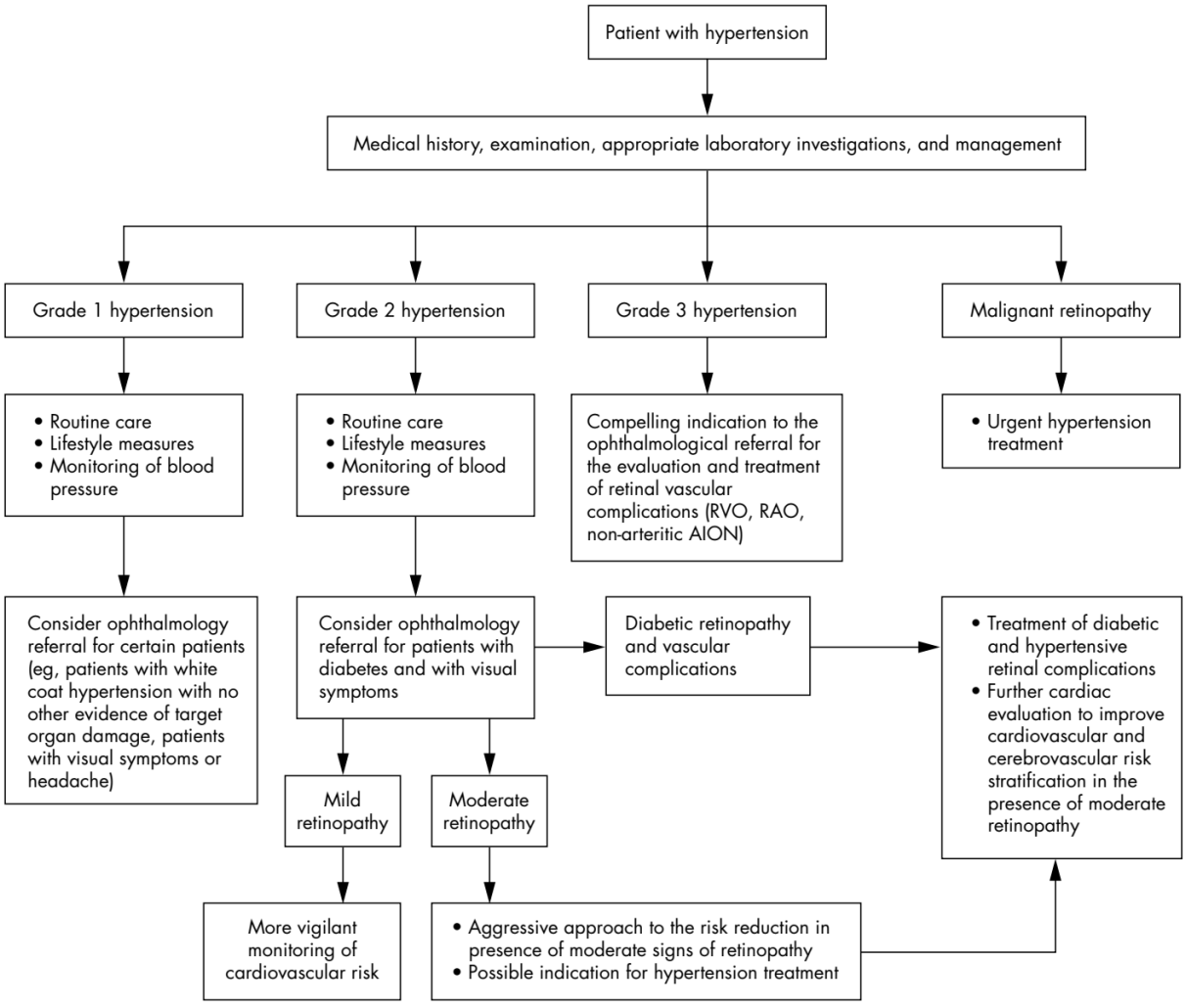
When both diabetes and hypertension are present, the risk of heart disease rises dramatically and results in a more rapid worsening and advancement of retinopathy (81).

The pathophysiological and morphological features of typical early retinopathy signs of diabetes and hypertension are identical(80).

The use of ophthalmoscopic examination for risk stratification is supported by retinopathy, which is an independent predictive indication of CHD risk in individuals with hypertension, even in cases of Stage 1 hypertension (82).

A significant practical influence on treatment decisions (e.g., antihypertensive and anti-platelet aggregation) and vigilant monitoring is the existence of retinopathy, which may indicate the need for a more intensive approach to associated cardiac risk factors and other co-morbidities(81).

Figure 5: Supplemental risk assessment by retinal examination in a flowchart (81)



Studies support both arterial hypertension and hypotension as protective and risk factors, although the precise relationship between the development of glaucoma and arterial blood pressure is complicated. One possible vascular risk factor for glaucoma is nocturnal hypotension. High blood pressure is thought to raise IOP, and for every 10 mm Hg increase in BP, there is an approximate 0.28 (0.08–0.48) mm Hg increase in IOP(85).

Gangwani et al. conducted a study and discovered that lower retinal nerve fiber layer thickness was correlated with greater SBP, DBP and mean arterial pressure (MAP), and a positive correlation between IOP and MAP was discovered (86).

Within the retinal circulation, the pressure differential between the central retinal vein (CRV) and central retinal artery (CRA) is equivalent to the arteriovenous pressure gradient, also known as mean ocular perfusion pressure or MOPP. Retinal vein pressure must be higher than intraocular pressure (IOP) to keep retinal veins from collapsing (this is known as the "Starling resistor effect")(87).

IOP closely resembles the pressure inside the CRV in physiological settings due to this homeostasis. Similarly, an ophthalmodynamometer can detect CRA pressure passively by calculating the external pressure required for the retinal arteries to collapse. An ophthalmodynamometer is inconvenient; the MAP is frequently used to estimate the CRA pressure, and the formula gives it **MAP =1/3(SBP) + 2/3(DBP)**(88,89).

B) Systemic blood pressure and intraocular pressure

Changes in systolic blood pressure are associated with alterations in IOP over time(90).

An association between a drop in IOP and lowered blood pressure might be attributed to specific antihypertensive agents that alter aqueous humor formation or drainage, such as ethacrynic acid, calcium channel blockers, beta-blockers, diuretics and selective alpha agonists (91).

Bill et al. showed that fluctuations in SBP led to minor alterations in aqueous humor production, likely due to heightened ciliary body capillary pressure, which may result in a rise in IOP(92).

Bill also showed that episcleral venous pressure, which plays a vital role in regulating aqueous humor outflow through the trabecular meshwork and into Sclemm's canal, may be affected by blood pressure(93).

According to Bengtsson et al., the age-related increase in intraocular pressure was influenced mainly by a simultaneous rise in SBP in the Swedish population (94).

Similarly, Klein et al. reported that in an American population, eye pressure variability was more strongly connected to SBP(95).

Leighton et al. showed a positive link between intraocular pressure and diastolic blood pressure(96).

With every heartbeat, the intraocular pressure increases and decreases by 1mm Hg.

Cullen et al. found that IOP increased by 6-7 mm Hg for each I00 mm Hg rise in SBP(97).

Systolic pressure showed a stronger correlation with intraocular pressure than diastolic blood pressure or mean arterial pressure and peak SBP boosts the filtration of aqueous humor, resulting in a slight yet continuous rise in IOP(98).

OCULAR PERFUSION PRESSURE

"Ocular blood flow" involves circulating oxygenated blood within the eye's vascular network. At the same time, OPP refers to the pressure that enables blood to flow through the intraocular vasculature, which is influenced by resistance from the caliber and tone of the vessels (99).

Of note is that there is no direct method for measuring absolute OPP, so arterial BP is a substitute. A sharp fall in the BP from antihypertensive treatment may lead to low DBP, lowering OPP(85).

The contrast in brachial and ocular blood pressure is caused by different flow resistance and hydrostatic pressure between the arm and the eye, causing discrepancies in brachial and ocular blood pressures, which have been explained by various correction factors depending on the body's posture.

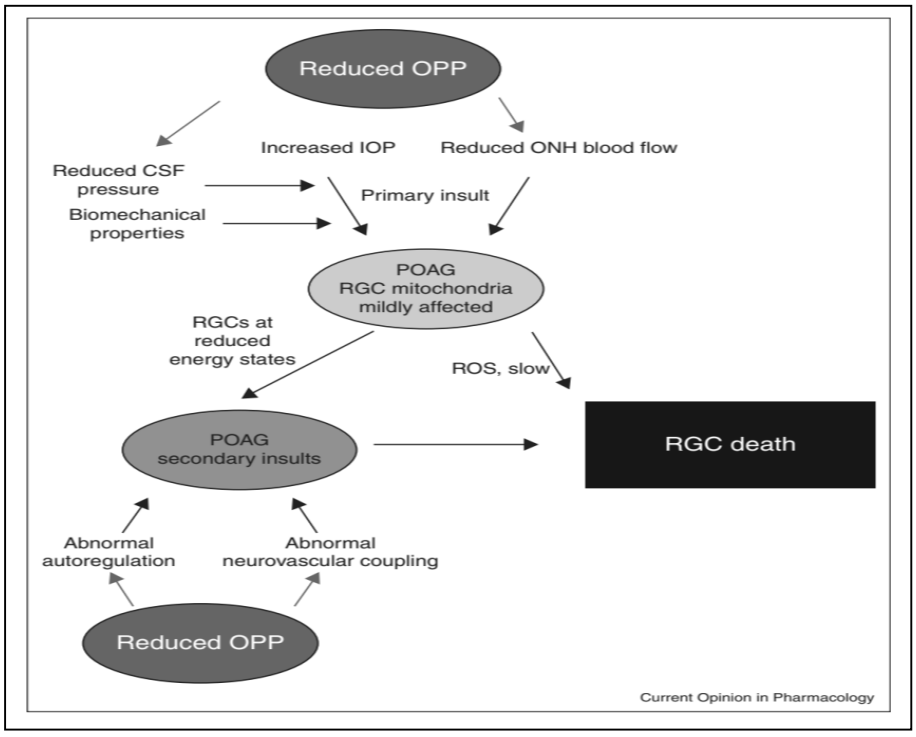
In humans in a sitting, the "two-thirds" adjustment factor is a recognized method for estimating CRA pressure from brachial blood pressure(88,100). As a result, MOPP can be determined as follows:

[MOPP = 2/3 MAP – IOP]

- ## Low OPP as a risk factor for glaucoma

Low mean OPP can hinder the blood flow to the optic nerve head, resulting in glaucomatous cupping and visual field defects(101). Long-term epidemiological research also demonstrated that low OPP contributes to a greater risk of glaucoma incidence, prevalence, and progression. This low OPP, along with dysregulations in MAP, can aggravate the progression of glaucoma(102).

Figure 6: Flowchart explaining potential OPP-dependent mechanisms that lead to glaucomatous damage (103).



As the flowchart above shows, RGC loss occurs due to primary and secondary insults. Elevated intraocular pressure (IOP) and ischemia at the optic nerve head (ONH) lead to the primary injury at this site. RGC mortality may result from secondary insults such as aberrant autoregulation of abnormal neurovascular coupling and oxidative stress brought on by reactive oxygen species (ROS) (103).

- **Blood flow autoregulation**

Autoregulation refers to the capacity of a vascular region to sustain blood flow even when there are variations in perfusion pressure(104).

Autoregulation strongly depends on myogenic and metabolic mechanisms in the retina and optic nerve head. Intrinsic choroidal neurons in choroids play a key role in blood flow regulation(103).

In healthy people, optic nerve autoregulation functions efficiently when IOP is below 27–30 mmHg, equating to a 40–50 percent decrease in OPP from its baseline, given a mean arterial pressure of 100 mmHg (105). Other research suggests that the upper limit of autoregulation may reach 34–60 percent higher than the baseline OPP (106,107).

A minor decrease in OPP does not necessarily lead to a deficiency in blood flow, as the retina actively maintains circulation, even in unfavorable conditions. In addition to OPP, ocular blood flow is also influenced by the resistance of blood vessels, which depends on factors such as blood viscosity (η) and vessel diameter (R). These elements together play a role in controlling blood flow (Q), as described by the Hagen–Poiseuille law (108,109):

$$\boldsymbol{Q = \frac{\pi R^4 \Delta P}{8 \eta L}}$$

ΔP - Pressure difference (between the two ends of a cylindrical pipe). It denotes OPP in the case of blood vessels in the eye (108,109)

L - length of the blood vessel (108,109).

A brief understanding of ocular blood flow is essential to grasp the concept of blood flow autoregulation.

▪ Ocular blood flow

Adjusting to changes in ocular activity and regulating ocular blood flow helps maintain a stable ocular temperature and retinal perfusion pressure (110).

A) Types of ocular blood flow

According to the responding rate, blood flow autoregulation can be categorized into static and dynamic (111). Static autoregulation includes myogenic, neurogenic, and metabolic factors (112) while dynamic autoregulation responds immediately to sudden changes in perfusion pressure. Extensive studies on the dynamic autoregulation of the outer ocular vascular system have revealed dense sympathetic innervation in the outer vessels (113).

B) Anatomy of blood flow

Nutrient supply to the retina comes from the choroidal and retinal blood flow, and their interaction may be key to maintaining optic nerve health (112). Though the retinal and brain circulations are comparable, the retina is unique because it lacks the autonomic nerve control in the brain's circulation. Tight junctions in endothelial form the blood-retinal barrier, akin to the blood-brain barrier (113).

Numerous studies have shown that effective blood flow autoregulation in the ONH may involve increased vascular capacitance. The extent of the reactive rise in vascular capacitance offsets the decrease in ONH vascular resistance caused by elevated IOP (114).

C) Evaluation of ocular blood flow

No single vascular parameter can fully assess the ocular blood flow. Every method assesses a distinct aspect of ocular circulation, each with its limitations, yet all contribute to a broader understanding of ocular hemodynamics (113,115):

1. **Pulsatile ocular blood flow** - Indicates choroidal blood flow (113).

2. **Colour Doppler imaging** - Used to examine the vascular circulation of retrobulbar area (116)

3. **Scanning laser Doppler flowmeter** – Provides a method to quantify the vascular circulation in the superficial layers of ONH and retina (116,117).

4. ***"*Doppler Fourier Domain" Optical Coherence Tomography (Doppler FD-OCT)**

- Provides "total retinal blood flow" (TRBF) (118).

5. **Angiography**

- Gold standard for in vivo evaluation of retinal circulation (113).

6. **Split-Spectrum "Amplitude-Decorrelation Angiography"- (SSADA-) OCT**

- The 3D algorithm used to picture ophthalmic microcirculation and measure optic disc perfusion might help the OBF evaluation (119,120).

D) Ocular Blood Flow Regulation: Mechanisms and Modulatory factors

Autoregulation ensures that the blood flow remains stable, only increasing when the eye's metabolic demands require it. However, when autoregulation fails, it may significantly impact the development of ocular vascular diseases.

"Classic autoregulation curve" characterizes the relationship between blood flow adjustments and changes in OPP over a specific range.

Impaired autoregulation occurs when blood flow deviates sharply in response to changes in pressure.

The regulation of blood flow over time is managed by static autoregulation, involving metabolic, myogenic, and neurogenic factors. Conversely, dynamic autoregulation reacts immediately to changes in perfusion pressure and is influenced by more contractile factors (121).

Local regulatory mechanisms primarily control the blood flow in the retinal and optic nerve head regions.

Endothelin-1, carbon dioxide, adenosine, angiotensin-II, oxygen, and nitric oxide are key mediators of these mechanisms. However, the contributions of angiotensin II and endothelin I to blood flow regulation in the retina and ONH are still debated (122).

Recent research revealed that the sympathetic nervous system mainly controls the choroid, while metabolic factors are autoregulated in response to changes in OPP (123),

1) Endothelin-1 (ET-1)

- A potent vasoconstrictor has been shown to influence interactions between the vascular endothelium and pericytes within the ocular microcirculation. Studies indicate that in healthy individuals, ET-1 plays a role in regulating the posterior segment of the eye, particularly in choroidal blood flow control. Glaucoma patients have shown higher levels of ET-1 in their glaucoma patients. Additionally, raised ET-1 concentrations can elevate IOP, reducing blood flow and proliferation of astrocytes and possibly contributing to RGC degeneration. Therefore, ET-1 is a significant risk factor. Because ET receptor antagonists can decrease retinal blood, endothelin antagonism is viewed as a potential treatment for glaucoma. Recently, CCBs have become widely used to enhance blood flow regulation and mitigate the vasoconstrictive effects of endothelin-1 (113).

2) Nitric Oxide

- NO is believed to play a key role in vascular relaxation and protecting endothelial cells. Nitric oxide synthase (NOS) affects the eye's pathological and physiological processes, regulating OBF and IOP. In conditions like glaucoma and diabetic retinopathy, dysfunction in NOS signaling is related to vascular abnormalities (113).

3) Estrogen

- Offers neuroprotection and has demonstrated improvements in retrobulbar circulation. Recent findings highlight a marked improvement in retinal blood flow and a reduced risk of glaucoma development (122,124)

4) Diurnal Variations

- Choroidal circulation is more susceptible to diurnal changes in the systemic environment than retinal and optic nerve head blood flow (125).

5) Adenosine

-There is strong evidence in healthy humans that adenosine influences retinal vasodilatation and IOP regulation. Through its action on A1, A2, and A3 receptors, adenosine is thought to stimulate adenylyl cyclase, leading to the modulation of ion channels, including decreasing calcium influx and activating chloride channels (126,127).

6) Carbonic Anhydrase

- Inhibiting this enzyme is believed to lower IOP by enhancing aqueous outflow. CAIs benefit OBF and are commonly employed to lower IOP in patients with OAG (128).

7) Myogenic Mechanisms

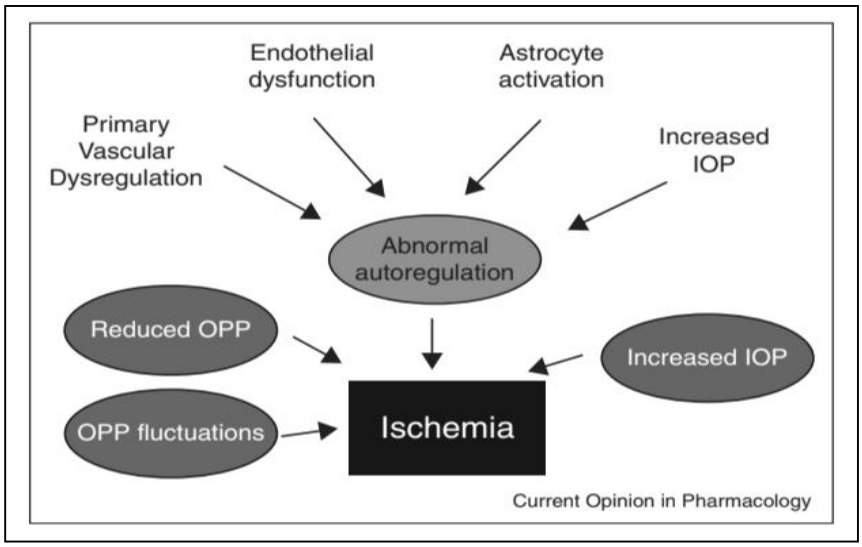
- The effect of this mechanism on ocular blood flow autoregulation is not substantial. When the vessel wall is stretched, calcium channels are activated, increasing calcium influx and causing the vessels to constrict. This rapid vasoconstrictive response is thought to help the system quickly adapt to rising perfusion pressure (129).

▪ Blood flow dysregulation

For decades, it has been believed that any impairment of "vascular autoregulation" results in the onset and advancement of glaucoma. Any variation in "perfusion pressure" causes a fall in blood flow once the lower threshold of autoregulation is reached(130). Impairment in autoregulation is seen in some pathological conditions, such as diabetes mellitus and hypercholesterolemia and also arises from group correlations between ocular blood flow and ocular perfusion pressure(131–134).

Ocular perfusion pressure instability and a drop in nocturnal blood pressure are also risk factors for glaucoma(135).

Figure 7: Abnormal autoregulation in glaucoma and factors that may contribute: (103)



Flammer et al. have identified primary vascular dysregulation, where individuals without underlying health issues exhibit abnormal blood vessel responses to temperature fluctuations and mechanical or emotional stressors(110).

The underlying cause for this might be related to vascular endothelial dysfunction(136). During isometric exercise, endothelin and nitric oxide are crucial regulators of blood flow in the ONH and choroid (137,138). The autoregulatory mechanism is also maintained by glial cells in the ONH. Therefore, loss of glial cells in glaucoma can lead to altered autoregulatory mechanisms(139).

- **Neurovascular coupling**

Functional hyperemia is a response when neurons get active due to the blood flow of the brain and retina, which is called neurovascular coupling.

An irregular blood flow reaction to neuronal stimulation leads to cell death due to insufficient nutrient delivery. Astrocytes significantly regulate the vasodilatory response accompanying neural functions(140).

Nitric oxide contributes to the vasodilator response, and blocking the NO synthase lessens the retinal hyperemia induced by flicker stimulation in humans(141,142). Flicker stimulation is reduced in glaucoma (143–145).

Abnormal retinal neurovascular coupling appears to be related to primary vascular dysregulation since vasospastic individuals display a weaker response to flicker stimulation(146).

Irrespective of the causes behind decreased flicker responses in glaucoma, abnormal neurovascular coupling might influence secondary insults to retinal ganglion cells(103).

DIAGNOSIS OF GLAUCOMA

Diagnosing glaucoma requires assessing risk factors, checking vision, measuring IOP, measuring the central cornea's thickness, analyzing the nerve fiber layer and optic nerve head, and assessing visual fields (147).

- **Clinical presentation of glaucoma**

In glaucoma, Snellen's acuity remains intact until the condition progresses to an advanced stage. As the disease advances, the patient may read the chart more slowly or turn their head to compensate for visual field defects (148).

Ishihara plates show that red-green colour vision is generally preserved until glaucoma reaches an advanced stage. However, blue-yellow colour vision, usually only tested in clinical research, often declines early in the disease progression (149).

Optic nerve damage causes paradoxical dilatation of the pupil, as the afferent response is delayed. Assessing the relative pupillary light reflex is crucial during glaucoma evaluations (148).

- **Evaluation of Intraocular pressure**

To accurately diagnose the subtype of glaucoma, a crucial evaluation of the ocular anterior segment is required to identify pathological signs that are either caused by or account for increased IOP (148).

Elevated IOP can lead to pathological changes in the anterior segment, such as atrophy of crypts of the iris due to ischemia of the underlying longitudinal dilator muscle of the iris (148).

Table 4: Anterior segment manifestations related to glaucoma (148)

OCULAR EXAMINATION	SIGNS	GLAUCOMA DIAGNOSIS
Lids and adnexa	Nevus flameus	SturgeWeber Syndrome
Conjunctiva	Chemosis	Topiramate-induced, bilateral secondary angle closure glaucoma
Cornea	Habbs striae	Congenital glaucoma
Anterior chamber	Pigment release after pupil dilatation	Pigmentary glaucoma
Iris	Melt holes and stretch holes	Iridocorneal endotheliopathy
Lens	Subluxation into the anterior chamber	Pupillary block glaucoma

Regular monitoring of intraocular pressure is essential in high-risk glaucoma patients, often achieved through rebound tonometry (iCare ic100; iCare) or the "gold standard" GAT as discussed earlier (147).

IOP evaluation should consider potential optic nerve damage and /or increased central corneal values (148).

According to the OHTS and EGPS, CCT is an independent risk factor for developing POAG, underscoring the necessity of measuring it during glaucoma evaluations (148,150).

Weinreb et al. stated that approximately 50% of eyes affected by glaucoma have IOP readings within the normal range, reinforcing the need for complementary diagnostic imaging when necessary (151).

Definitions of target intraocular pressure given by different guidelines are as follows (152)

1. According to the European Glaucoma Society guidelines, target IOP is "the estimated average IOP achieved through treatment, which is expected to prevent further damage from glaucoma."

2. According to the American Academy of Ophthalmology, target IOP is “the IOP range sufficient to halt progressive damage caused by elevated pressure.”

3. According to the World Glaucoma Association, target IOP is “the estimated average IOP at which the risk of glaucoma-related vision loss and its impact on quality of life outweighs the risks of the treatment.”

Sihota et al., simplified target IOP as follows (152)

- Mild glaucoma – 15 to 17 mmHg
- Moderate glaucoma – 12 to 15 mmHg
- Severe glaucoma – 10 to 12 mmHg

- **Gonioscopy**

Gonioscopy is a crucial method for classifying glaucoma into open and closed-angle categories. The “angle” describes where the peripheral cornea and iris meet. The trabecular meshwork at this junction acts as the pathway for aqueous humor drainage from the eye (148).

Cohen et al. stated that trabecular meshwork cannot be directly observed because light from the meshwork is internally reflected (total internal reflection). A specialized prism uses the tear film to couple with the cornea, providing visualization of the angle structures under slit-lamp biomicroscopy (148).

As previously mentioned, Shaffer’s grading system is the most widely used method for assessing anterior chamber angles. A closed angle is typically associated with increased resistance to the flow of aqueous humor, a defining feature of primary angle closure glaucoma. An open angle with high IOP may be associated with findings such as excess pigment accumulation or trauma-related

angle deformities. However, elevated IOP can also occur with a normal-appearing trabecular meshwork, a hallmark of open-angle glaucoma, likely due to subtle biochemical or ultrastructural changes in the trabecular meshwork(148)

- **Evaluation of optic nerve head (ONH) and retinal nerve fiber layer (RNFL)**

I) Morphology of optic disc changes in glaucoma

Evaluating the optic nerve is essential in glaucoma assessment. The optic nerve generally displays only two observable responses to pathological damage: swelling and atrophy (148).

The optic disc alterations in glaucoma tend to be progressive and asymmetrical, exhibiting distinct clinical patterns. With the loss of axonal bundles, the neural rim undergoes thinning in characteristic ways.

A) Disc Patterns of Glaucomatous Optic Atrophy

- **Focal Atrophy**

Kitsos et al., in their study, said that the optic nerve atrophy caused by glaucoma is a unique form that can be detected by neuroimaging and results in the excavation or loss of neuroretinal rim tissue (153).

In glaucoma, the neural rim first thins in the inferotemporal region, with some loss in the superotemporal area, causing the optic cup to enlarge vertically or obliquely.

The loss of RGCs in glaucoma leads to an enlargement of the cup area, which results in a higher vertical cup-to-disc ratio (148).

Compared to a healthy optic nerve head, the glaucomatous eye exhibits a thinner inferior temporal rim relative to the superior temporal region with a reduced horizontal-to-vertical cup-to-disc ratio.

Compared to non-glaucomatous discs, the neural rim area in glaucomatous optic discs is generally smaller, providing a more accurate means of differentiating early glaucoma from normal eyes than the cup-to-disc ratio.

The first sign of neural rim atrophy is typically a minor, localized defect in the inferotemporal quadrant, known as polar notching, and the nasal quadrant is the last to degrade. When a retinal vessel traverses the thinned rim, it exhibits a sharp angulation at the disc margin, known as bayoneting.

- **Concentric Atrophy**

  Unlike focal atrophy, glaucomatous damage can sometimes cause cup enlargement in concentric circles, usually in the inferotemporal or superotemporal direction rather than horizontally.

  Since neural rim loss typically starts temporally and spreads circumferentially toward the poles, this process is known as temporal unfolding.

- **Progressive deepening of the Cup**

  Early glaucomatous optic atrophy primarily involves deepening of the optic cup, which occurs only when the lamina cribrosa is not initially exposed. As the cup deepens further, the gray fenestrations of the lamina cribrosa, termed the laminar dot sign, become apparent.

- **Pallor-Cup Differentiation**

  Cup enlargement often occurs before pallor in the early stages of glaucomatous optic atrophy, indicating a biphasic pattern, in contrast to other optic atrophy where pallor generally surpasses the cup.

  Both diffuse and focal cup enlargement can lead to pallor-cup discrepancy. Early glaucomatous changes known as saucerization involve diffuse and shallow cupping that extends to the disc margins, leaving central pallor, which indicates early glaucoma.

❖ **Advanced Glaucomatous Cupping**

When glaucomatous optic atrophy progresses without effective IOP-lowering intervention, it typically results in the total loss of neural rim tissue, leading to complete cupping, which is clinically observed as a wite disc with rim loss and bending vessels at the disc's margin.

In a study by Bianchi-Marzoli et al., it is said that while pathological cupping is a key indicator of glaucoma, it is not exclusive to it. Tumors that compress the anterior visual pathways, for example, can cause cupping without elevating IOP (154).

According to Hayreh SS et al., the blockage of vessels in the prelaminar region triggers tissue degeneration, resulting in disc cupping and visual field defects (155).

Hayreh SS et al. showed that there was a reduction in optic disc cupping in some cases a few days after significantly lowering the IOP to normal levels; this observation implies that the cupping may partly be due to the compression of loose prelaminar tissue by increased IOP. The decrease in pressure may contribute to cupping reversal, first by allowing the tissue to return to its original position and later through glial tissue regeneration, which could result in a paler disc appearance (155).

B) Vascular Signs of Glaucomatous Optic Atrophy

The prevalence of splinter hemorrhages is higher in patients with "normal-tension glaucoma" (35.3%) than in those with "COAG" (10.3%) or "suspected glaucoma" (10.4%).

C) Changes in Peripapillary Region Related to Glaucomatous Optic Atrophy

❖ **Nerve Fiber Bundle Abnormality**

Neural rim changes in glaucomatous optic atrophy, caused by the loss of axonal bundles, lead to RNFL defects.

A thinned peripapillary nerve fiber layer visualizes the underlying retinal pigment epithelium, revealing a choroid near the disc (148).

Initial glaucomatous damage may present as localized loss in specific areas or as a more widespread, diffuse loss.

- **Peripapillary Pigmentary Changes**

  They are often seen in glaucomatous optic atrophy but can also be present in other conditions, including myopia and age-related alterations.

  Peripapillary atrophy, encompassing both the alpha zone and beta zones, is more common and extensive in glaucomatous eyes compared to healthy eyes, with a noted progression in size over time.

  Studies show that individuals with ocular hypertension who do not exhibit peripapillary atrophy may have a reduced risk of developing glaucomatous damage.

  Optic disc edema is observed in acute congestive glaucoma, presumably caused by the abrupt compression of prelaminar vessels. This vascular compression induces anoxia, ultimately leading to edema (155).

SD-OCT appears to be a reliable tool for showing disease progression., often identifying changes before functional deficits are observed on visual field testing (156,157).

II) Significance of Optical Coherence Tomography (OCT) in diagnosing RNFL defects and optic disc changes:

Diagnosing glaucoma, especially in its early stages, can be challenging due to the lack of a standardized approach. Early glaucomatous structural changes can be identified with OCT imaging, while advanced glaucoma can be assessed by detecting functional changes via visual field testing (147).

Disc excavation and RNFL thinning are common signs of optic nerve head degeneration in all types of glaucoma. Thinning of the neuro-retinal rim, especially in the superior and inferior

sectors, is often seen in optic nerve head damage. At the same time, other areas of the ONH may retain their normal pink appearance with an intact neuro-retinal rim (158).

Glaucomatous damage triggers retinal ganglion cell apoptosis, evident on OCT as thinning between the ganglion cell layer and the internal limiting membrane (158).

- **Evaluation of visual field defects**

As glaucoma progresses, changes in the ONH and RNFL contribute to the development of visual field defects, which are not detectable in the early stages of glaucoma, as peripheral vision and visual acuity remain intact until considerable RNFL damage occurs (148).

In glaucoma, defects in the visual field reflect the arrangement of the nerve fiber layer bundles. Chiasmal lesions can produce cupping that appears similar to glaucomatous cupping, but the resulting field loss corresponds to the vertical meridian, whereas in glaucoma, the field loss typically aligns with the horizontal meridian (148).

The blood vessels in the prelaminar region of the optic disc are arranged segmentally, and their obliteration, or that of the adjacent peripapillary choroid from which they originate, may result in nerve fiber bundle defects. This vascular blockage can sometimes be acute and segmental, causing field defects secondary to sectoral cupping and ischemic optic neuropathy (155).

Posterior ciliary arteries with minimal intercommunication independently supply the ciliary circulation originating from the ophthalmic artery. Differences in perfusion pressure among those arteries may exist, making the area served by the artery with the lowest perfusion pressure more vulnerable to early glaucomatous damage. This can result in altitudinal or vertical hemianopia field defects (155).

A correlation between OCT imaging and visual field evaluations is evident, but no established standard exists for direct comparison (159).

The severity of glaucoma is classified as - mild, moderate, or severe. Severity grading systems mainly rely on functional field abnormalities (147).

According to the International Classification of Diseases (160),

- In mild glaucoma, no field defects are present.
- In moderate glaucoma, the field changes are limited to one hemifield (but outside the 5-degree of fixation).
- In Severe glaucoma, field defects affect both hemifields, with damage extending into the central 5 degrees of fixation.

APPROACHES TO GLAUCOMA TREATMENT

Glaucoma management aims to slow disease progression by IOP control (147).

A key approach is setting a target IOP, considering factors such as the patient's age, the level of IOP associated with retinal ganglion cell loss, and the severity of the disease. Since the target IOP is not fixed, it is essential to re-evaluate using both structural and functional optic nerve assessments. Controlling IOP to prevent disease progression primarily involves reducing IOP using ocular hypotensive medications and/ or surgical outflow procedures (148).

A) Medical Therapy in glaucoma

For decades, medications that lower intraocular pressure have been the cornerstone of therapy for most glaucoma patients, including topical prostaglandin analogs, beta-blockers, carbonic

anhydrase inhibitors (CAIs), and alpha agonists. They offer improved effectiveness and favorable safety profiles compared to older treatments, such as systemic oral CAI and topical pilocarpine (147,161).

Following standard pharmacotherapy guidelines, the goal is to achieve the target IOP with the least number of medications and minimal side effects. Corticosteroids should be used cautiously, as they may induce glaucoma (147).

PGAs are widely used for treating ocular hypertension and OAG. By enhancing uveoscleral outflow, PGAs compensate for reduced drainage through the trabecular meshwork (TM). These medications are typically administered once a day and are well tolerated (162).

Beta-blockers and CAIs reduce aqueous humor production in the ciliary body, lowering IOP. Beta-blockers reduce aqueous production by blocking sympathetic nerve endings in the ciliary body epithelium. Their benefits include a relatively low cost and the convenience of once-daily administration (147,151,163).

Brimonidine and iopidine, alpha-adrenergic agonists, lower IOP by reducing aqueous humor production and improving its outflow. They are usually applied twice to three times daily and prescribed as second-line treatments alongside other medications (147).

Rho kinase inhibitors are a novel class of medications that utilize a dual mechanism, promoting conventional outflow and reducing episcleral venous pressure (164).

Table 5: Summary of anti-glaucoma drugs by Wagner I et al. (147):

Group	Medications	Adverse reactions	Contraindication
Prostaglandin analogues	-Bimatoprost -Latanoprost -Tafluprost -Travoprost -Unoprostene -Latanoprostene Bunod	-Eyelash growth -Iris darkening -Keratitis -Conjunctival pigmentation -Uveitis	Hypersensitivity
Cholinergic agents	-Pilocarpine -Carbachol	-Myopia -Angle closure -Cataract -Retinal detachment	-Miosis -Bradycardia -Retinal detachment -Asthma -Inflammatory eye disease
Carbonic anhydrase inhibitors	1st generation (Systemic) -Acetazolamide -Methazolamide -Dichlorphenamide	-Renal calculi -Stevens-Johnson syndrome -Serum electrolyte imbalance	-Allergy to sulfa drugs -Sickle cell disease
	2nd generation (Topical) -Brinzolamide -Dorzolamide	-Corneal oedema -Metallic taste	
Beta adrenergic antagonist	Non-selective: -Carteolol -Levobunolol -Metipranolol -Timolol β1- selective: -Betaxolol	-Congestive heart failure -Exercise intolerance -Hypotension -Bronchospasm -Bradycardia	-Cardiovascular disease -Asthma -Diabetes mellitus -Chronic obstructive pulmonary disease
Alpha adrenergic agonist	-Apraclonidine -Brimonidine	-Hypotension -Fatigue -Allergic conjunctivitis	-Monoamine-oxidase inhibitor therapy
Rho-Kinase inhibitors	-Netarsudil	-Keratitis -Conjunctival haemorrhage -Corneal verticillate	-None
Hyperosmotic agents	-Glycerol -Mannitol -Isosorbide	-Congestive heart failure -Renal failure -Nausea -Vomiting -Headache	-Cardiovascular disease -Renal failure

Pharmacotherapy offers viable short-term solutions, but issues like high cost, adverse effects, and inadequate control of IOP hinder its long-term use. Nonadherence to the prescribed treatment

schedules also poses a significant problem, with fewer than 50% of glaucoma patients remaining compliant with their medication regimen after one year (163,165).

B) Laser Therapy in glaucoma

Laser and surgical treatments are considered when pharmacological therapy fails to control IOP and protect against vision loss (147).

Stein et al. concluded in their study that laser procedures effectively lower IOP and can significantly reduce the long-term expenses related to the ongoing use of multiple pressure-lowering drugs (163).

Laser trabeculoplasty and ab-interno excimer trabeculostomy (Glautec AG) are alternative procedures to lower intraocular pressure for patients unresponsive to medications. Laser trabeculoplasty (selective laser and argon laser) involves applying the laser spots to the TM, improving drainage of aqueous humor by inducing modifications (166).

SLT is favored due to its better safety record, effectiveness in lowering intraocular pressure (IOP), and capacity for repeat treatments. Laser trabeculoplasty is typically chosen over surgical procedures due to its less invasive nature and better safety profile (167).

Similar to laser trabeculoplasty, ab-interno excimer trabeculectomy is a MIGS technique utilizing a 308-nanometer excimer laser to generate micro-perforations in the trabecular meshwork and the Schlemm canal (168).

Lasers for ACG differ from those in OAG. In ACG, a laser peripheral iridotomy creates an opening in the peripheral iris to resolve the pupillary block. In contrast, laser peripheral iridoplasty applies low-power laser burns to shrink the peripheral iris and alleviate appositional angle closure when iridotomy fails (169,170).

C) Surgical Management in glaucoma

When medical and laser therapies fail to reduce IOP adequately, surgical intervention comes into play. These include traditional bleb-based procedures such as trabeculectomy, tube shunt implantation, and the more recent conjunctiva-sparing MIGSs (147).

In their study, Rolim De Moura et al. showed that patients in the non-surgical groups had a higher degree of visual field loss than those in the trabeculectomy group (171).

Boland et al. stated that adjunctive use of mitomycin C and 5-fluorouracil helped prevent scarring and significantly reduced IOP compared to trabeculectomy alone (172).

According to Ramulu et al., prosthetic devices designed to promote aqueous humor outflow became more commonly used in glaucoma filtration surgery starting in the early 1990s (173).

Goniotomy or trabeculectomy is the primary surgery for congenital/childhood glaucoma (174).

When glaucoma remains refractory to medical, surgical, and laser interventions, cyclo-destructive procedures may be considered a last resort, damaging the ciliary epithelium to reduce aqueous humor secretion and lower IOP (175).

FUTURE PATHWAYS

Future directions in glaucoma research concerning BP, IOP and OPP focus on refining our understanding of the complex interplay between these factors and their impact on disease progression. OPP, the difference between blood pressure and IOP, is crucial for ensuring proper blood circulation to the optic nerve. Imbalances between IOP and BP can lead to compromised blood flow to the optic nerve, increasing the risk of glaucoma. Researchers are interested in how low BP, especially nocturnal dips in BP, can reduce OPP and lead to optic nerve damage despite normal or controlled IOP. Advances in technology could allow continuous monitoring of IOP throughout the day, providing a more comprehensive picture of pressure fluctuations that traditional measurements may miss. More sophisticated BP management strategies, particularly for patients with hypertension, are being investigated to optimize OPP. Future therapies may combine IOP-lowering treatments with interventions to stabilize BP to ensure a balanced OPP, improving overall optic nerve health and reducing the risk of progression. This holistic approach, which considers mechanical and vascular health, leads to more personalized and effective treatments for glaucoma.

Study Materials and Methodology

Research Design:

This cross-sectional study on the association between systemic hypertension, perfusion pressure and glaucoma was conducted for one and a half years, from May 2023 to December 2024, at the Department of Ophthalmology, Shri B.M. Patil Medical College, Hospital, and Research Centre, Vijayapura.

A total of 168 participants who fulfilled the inclusion criteria were enrolled in the study. Patients with hypertension were taken in the experimental (study) group (n=84), and patients without hypertension were taken in the control group (n=84).

Inclusion Criteria:

1. Patients above the age of 40years
2. Patients with essential hypertension (SBP≥140mmHg, DBP ≥90mmHg)
3. Patients on anti-hypertensive medications since 1 year
4. Age and sex-matched patients without hypertension in the control group

Exclusion Criteria:

1. Patients diagnosed with secondary hypertension (endocrine, kidney disease, steroid-induced).
2. Patients in whom intraocular pressure cannot be performed.

- A full ophthalmic examination was conducted for all patients, incorporating detailed clinical history, best possible visual acuity with correction, blood pressure recording, slit lamp examination, Goldman 4 mirror gonioscopy, intraocular pressure, fundus examination and perimetry.

- The rebound tonometer (iCare IC100, Finland) was used to measure the intraocular pressure in both eyes. The device calculates the IOP based on the probe's deceleration and rebound time after impact. For accuracy, the device calculates an average of six readings and that average was used for analysis.
- Sphygmomanometer was used to measure blood pressure on the right arm while the patient was in a sitting position. Individuals with blood pressure ≥140/90 mmHg on two separate readings were included.
- Perfusion pressures were calculated as follows,

1. **Mean Ocular Perfusion Pressure (MOPP)**

   [MOPP=2/3(MAP)-IOP]

   [MAP=DBP+1/3(SBP-DBP)]

   Where, MAP=Mean Arterial Pressure, IOP=Intra Ocular Pressure, DBP=Diastolic Blood Pressure, SBP=Systolic Blood Pressure.

2. **Systolic Perfusion Pressure (SPP)**

   [SPP=SBP-IOP]

3. **Diastolic Perfusion Pressure (DPP)**

   [DPP=DBP-IOP]

Intraocular and blood pressures were recorded daily at 7 am, 1 pm, 6 pm and 12 am. Perfusion pressures were then calculated separately for each eye at all time intervals.

Statistical Analysis

To ensure 99% power for detecting a difference in means between two independent groups using a t-test with a 1% significance level (3), the sample size was calculated using "G*Power ver. 3.1.9.4 software". Based on this calculation, the study was assigned a sample size of 168.

The data obtained is entered in a Microsoft Excel sheet, and statistical analyses are performed using a statistical package for the social sciences (SPSS) (Version 20). Results are presented as Mean, SD, counts and percentages, and diagrams. Normally distributed continuous variables between the two groups will be compared using an independent sample t-test. For not normally distributed variables, the Mann-Whitney U test is used. Categorical variables between the two groups are compared using the Chi-square test/Fisher's exact test. If $p<0.05$, it was considered statistically significant. All statistics are performed two-tailed.

Ethical Perspective

The study received ethical approval from the committee responsible for overseeing research adherence to ethical guidelines. Their endorsement granted under Order number BLDE (DU)/IEC/865/2022-23, dated 1st April 2023, adhered strictly to the principles outlined in the Helsinki Declaration (176).

Results

The present study included 168 participants, with 84 classified as hypertensives (cases) and 84 as normotensives (controls). Cases consisted of individuals with a known history of hypertension for at least one year and on medication, as well as those without a prior diagnosis but with blood pressure ≥140/90 mmHg on two separate readings. Controls included participants without a history of hypertension and with blood pressure <140/90 mmHg.

Table 4: Comparison of age and gender between subjects with and without hypertension.

Parameters		Number of participants (n)		Chi-square test value	Significant value
		Hypertensives (n=84)	Normotensives (n=84)		
AGE (in years)	41-50	6	7	0.845	0.932
	51-60	23	26		
	61-70	39	35		
	71-80	14	15		
	>81	2	1		
GENDER	Male	27	27	0.001	1.00
	Female	57	57		

The Pearson Chi-Square test was used to compare age, gender between the hypertensives and normotensives and were not significant with p value > 0.05 (Table 4). A T-test was used to calculate the mean age. The mean age of participants in cases is 64.20 ± 8.659 (Mean ± SD) and in controls is 63.83 ± 8.713 (Mean ± SD). The male-to-female ratio was equal (M:F = 27:57) in both groups.

Table 5: Comparison of risk factors like diabetes, smoking, and alcohol between subjects with and without hypertension.

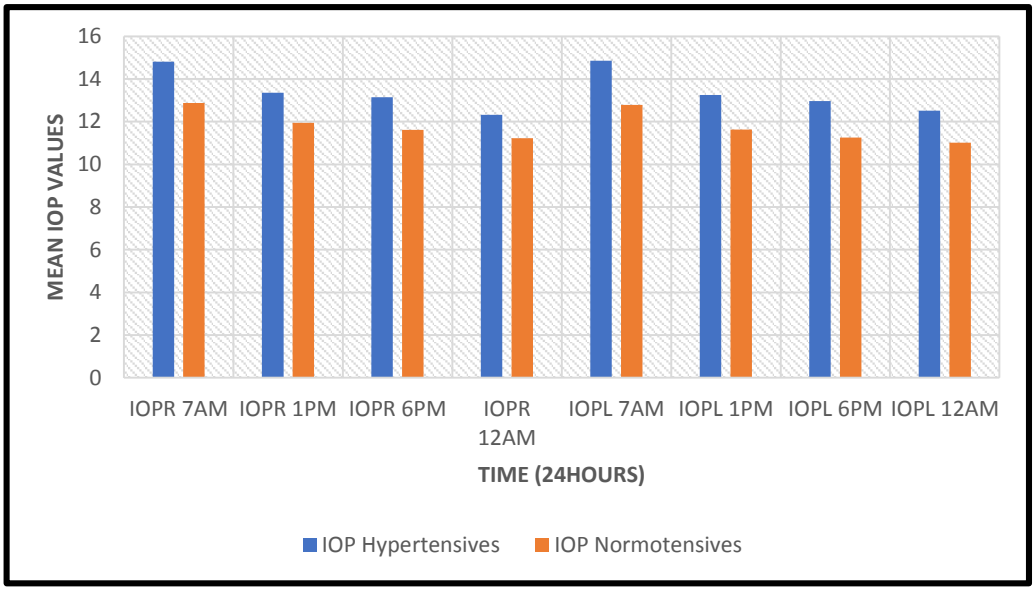
Parameters		Number of participants (n)		Chi-square test value	Significant value
		Hypertensives (n=84)	Normotensives (n=84)		
DIABETIC	Yes	11	0	11.77	0.001*
	No	73	84		
SMOKING	Yes	10	0	10.633	0.001*
	No	74	0		
ALCOHOL	Yes	10	0	10.633	0.001*
	No	74	0		
*P value<0.05 implies that it is significant statistically					

The Pearson Chi-Square test was used to compare diabetes, smoking and alcohol risk factors between the hypertensives and normotensives and these parameters showed significance with p value <0.05 (Table 5).

Table 6: Comparison of mean IOP values in both groups at different time intervals in 24 hours

IOP		Mean value (in mmHg)		Mann-Whitney U test	Significant value
		Hypertensives	Normotensives		
Right Eye	7 am	14.81 ± 3.287	12.88 ± 1.929	2074.00	0.001*
	1 pm	13.36 ± 3.033	11.94 ± 1.846	2485.00	0.001*
	6 pm	13.15 ± 3.262	11.62 ± 1.862	2546.50	0.002*
	12 am	12.32 ± 2.743	11.23 ± 1.623	2653.00	0.005*
Left Eye	7 am	14.86 ± 3.387	12.79 ± 1.994	2180.50	0.001*
	1 pm	13.25 ± 3.515	11.63 ± 1.925	2474.00	0.001*
	6 pm	12.96 ± 2.796	11.26 ± 1.876	2064.50	0.001*
	12 am	12.52 ± 2.852	11.02 ± 1.575	2265.00	0.001*
*P value<0.05 implies that it is significant statistically					

Graph 1: Bar graph representation of IOP in both groups

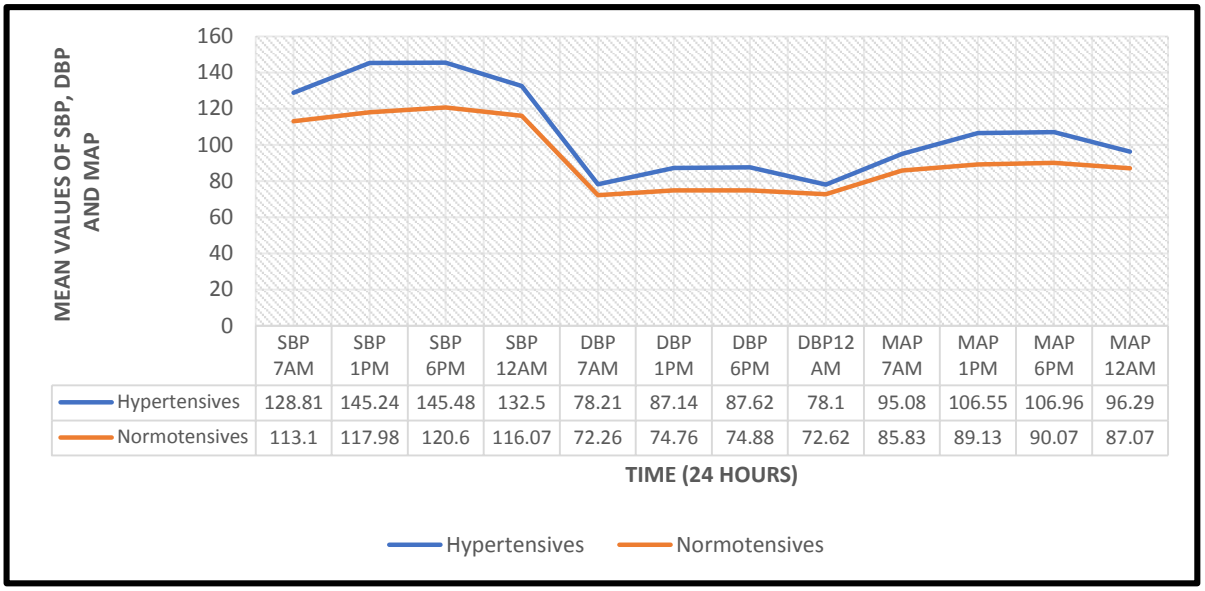


To compare the mean IOP values of both the right and left eye in both groups the Mann-Whitney U test was used (Table 6, Graph 1). Hypertensives showed higher values (RE=14.81mmHg at 7 am) than normotensives (RE=12.88mmHg at 7 am), but all the mean values were within the normal range of 11 to 15mmHg in both the eyes and showed significance with p value <0.05 at all the time intervals in 24 hours. At 7 am, the mean IOP was slightly higher, gradually decreasing as the day progressed, illustrating the typical diurnal variation in IOP.

Table 7: Mean SBP, DBP and MAP values in both groups at different time intervals in 24 hours

SBP, DBP AND MAP		Mean value (in mmHg)		Mann-Whitney U test value	Significant value
		Hypertensives	Normotensives		
SBP	7 am	128.81 ± 16.531	113.10 ± 6.581	1368.50	0.001*
	1 pm	145.24 ± 20.563	117.98 ± 8.328	693.50	0.001*
	6 pm	145.48 ± 19.537	120.60 ± 7.816	784.0	0.001*
	12 am	132.50 ± 16.271	116.07 ± 6.589	1211.50	0.001*
DBP	7 am	78.21 ± 9.205	72.26 ± 5.672	2285.0	0.001*
	1 pm	87.14 ± 8.441	74.76 ± 5.906	927.50	0.001*
	6 pm	87.62 ± 9.394	74.89 ± 5.910	946.50	0.001*
	12 am	78.10 ± 7.835	72.62 ± 5.833	2192.0	0.001*
MAP	7 am	95.08 ± 10.71	85.83 ± 4.44	1575.50	0.001*
	1 pm	106.55 ± 11.29	89.13 ± 5.16	567.00	0.001*
	6 pm	106.96 ± 11.89	90.07 ± 4.93	729.00	0.001*
	12 am	96.29 ± 9.80	87.07 ± 4.49	1458.00	0.001*
*P value <0.05 implies that it is significant statistically					

Graph 2: Graphical representation of SBP, DBP and MAP in both groups

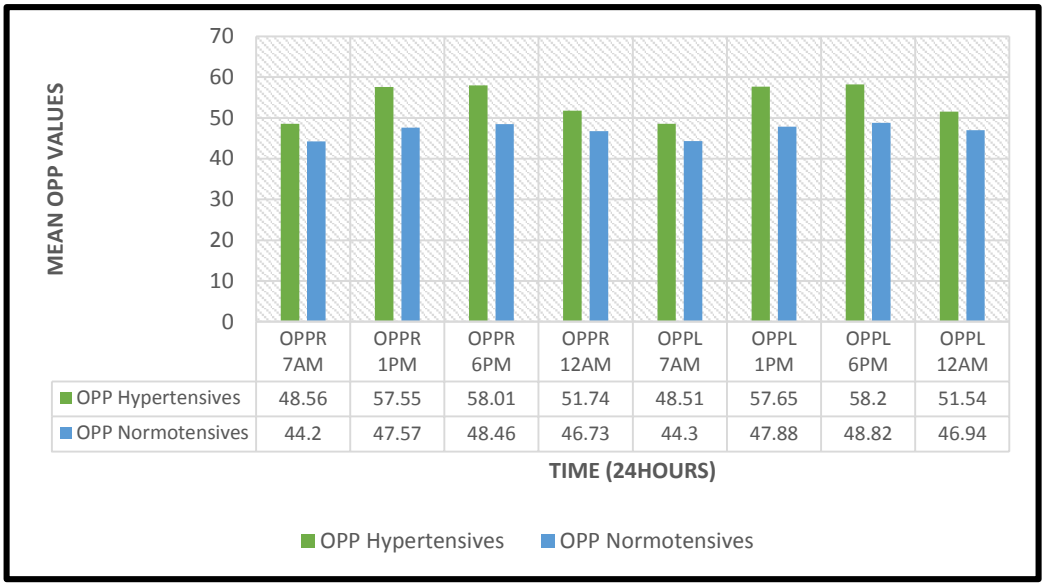


To compare the mean SBP, DBP and MAP in both groups, the Mann-Whitney U test was performed (Table 7, Graph 2). There was a significance with the p value<0.05 at all intervals in 24 hours. In hypertensives, the mean values were SBP=120 to 150mmHg, DBP=70 to 90mmHg and MAP=90 to 110mmHg and in normotensives, SBP=110 to 120mmHg, DBP=70 to 80mmHg and MAP=80 to 90mmHg. Among the cases, the highest blood pressure was recorded at 1 pm (SBP=145.24 ± 20.563, DBP=87.14 ± 8.441) and 6 pm (SBP=145.48 ± 19.537, DBP=87.62 ± 9.394). Mean arterial pressure, which is calculated using SBP and DBP, was also high at 1 pm (106.55 ± 11.29) and 6 pm (106.96 ± 11.89).

Table 8: Mean OPP values in both groups at different time intervals in 24 hours

OPP		Mean value (in mmHg)		Mann-Whitney U test	Significant value
		Hypertensives	Normotensives		
Right Eye	7 am	48.56 ± 8.02	44.20 ± 3.72	2368.50	0.001*
	1 pm	57.55 ± 7.56	47.57 ± 4.04	885.00	0.001*
	6 pm	58.01 ± 7.98	48.46 ± 4.22	1005.00	0.001*
	12 am	51.74 ± 6.54	46.73 ± 4.03	1887.50	0.001*
Left Eye	7 am	48.51 ± 8.19	44.30 ± 3.79	2454.00	0.001*
	1 pm	57.65 ± 7.13	47.88 ± 3.95	784.50	0.001*
	6 pm	58.20 ± 8.09	48.82 ± 4.16	999.00	0.001*
	12 am	51.54 ± 7.00	46.94 ± 4.07	2074.50	0.001*
*P value<0.05 implies that it is significant statistically					

Graph 3: Bar Graph representation of OPP in both groups



To compare the mean OPP of both eyes in both groups, the Mann-Whitney U test was used (Table 8, Graph 3). The values were significant, with p value<0.05 at all the time intervals in 24 hours. Though the OPP values were not within the normal range in a few participants, the overall mean OPP values were within normal limits of 40 to 60 mmHg. They were higher in the hypertensives compared to normotensives. The highest values were noted at 1 pm (RE=57.55 ± 7.56**,** LE=57.65 ± 7.13) and 6 pm (RE=58.01 ± 7.98, LE=58.20 ± 8.09) in the hypertensives, which corresponded with the blood pressure which was also highest at 1 pm and 6 pm.

Table 9: Mean SPP values in both groups at different time intervals in 24 hours

SPP		Mean value (in mmHg)		Mann-Whitney U test	Significant value
		Hypertensives	Normotensives		
Right Eye	7 am	114 ± 16.77	100 ± 6.98	1670.00	0.001*
	1 pm	131.9 ± 20.24	106.1 ± 8.53	778.00	0.001*
	6 pm	132.3 ± 19.3	109 ± 8.11	810.50	0.001*
	12 am	120.17 ± 15.9	104.8 ± 7.14	1401.50	0.001*
Left Eye	7 am	113.9 ± 16.9	100.2 ± 7.22	1665.00	0.001*
	1 pm	132.01 ± 19.7	106.5 ± 8.62	758.50	0.001*
	6 pm	132.51 ± 19.4	109.5 ± 8.07	861.00	0.001*
	12 am	119.9 ± 16.4	105.04±7.05	1519.50	0.001*
*P value<0.05 implies that it is significant statistically					

Table 10: Mean DPP values in both groups at different time intervals in 24 hours

DPP		Mean value (in mmHg)		Mann-Whitney U test	Significant value
		Hypertensives	Normotensives		
Right Eye	7 am	63.88 ± 10.18	59.66 ± 6.33	2795.00	0.02*
	1 pm	73.98 ± 8.6	63.22 ± 6.4	1188.00	0.001*
	6 pm	74.8 ± 9.4	63.6 ± 7.2	1149.00	0.001*
	12 am	66.01 ± 8.20	61.71 ± 6.66	2484.50	0.001*
Left Eye	7 am	63.6 ± 10.13	59.8 ± 6.3	2875.50	0.04*
	1 pm	74.10 ± 8.36	63.53 ± 6.25	1134.00	0.001*
	6 pm	74.97 ± 9.65	63.92 ± 7.18	1219.00	0.001*
	12 am	65.80 ± 8.42	61.95 ± 6.80	2710.00	0.01*
*P value<0.05 implies that it is significant statistically					

To compare the mean SPP and DPP values of both eyes in both groups (Table 9,10), the Mann-Whitney U test was used. The values were significant, with p value<0.05 at all the time intervals in 24 hours. The overall mean SPP and DPP values were within normal limits and were higher in hypertensives than normotensives. Similar to mean OPP values, the highest mean values of SPP and DPP were noted at 1 pm and 6 pm in the hypertensives, which corresponded with the blood pressure, which was also highest at 1 pm and 6 pm.

SPP at 1 pm and 6 pm is (RE =131.9 ± 20.24, LE =132.01 ± 19.7 and RE=132.3 ± 19.3, LE=132.51 ± 19.4) and DPP at 1 pm and 6 pm is (RE=73.98 ± 8.6, LE=74.10 ± 8.36 and RE=74.8 ± 9.4, LE=74.97 ± 9.65).

Table 11: Comparison of OPP with SBP, DBP and MAP at 7 am in hypertensives

Hypertensives		7 am					
		SBP		DBP		MAP	
		r value	P value	r value	P value	r value	P value
7 am	OPPR	0.789	0.001*	0.838	0.001*	0.910	0.001*
	OPPL	0.790	0.001*	0.844	0.001*	0.913	0.001*

Table 12: Comparison of OPP with SBP, DBP and MAP at 1 pm in hypertensives

Hypertensives		1 pm					
		SBP		DBP		MAP	
		r value	P value	r value	P value	r value	P value
1 pm	OPPR	0.801	0.001*	0.823	0.001*	0.908	0.001*
	OPPL	0.781	0.001*	0.790	0.001*	0.883	0.001*

Table 13: Comparison of OPP with SBP, DBP and MAP at 6 pm in hypertensives

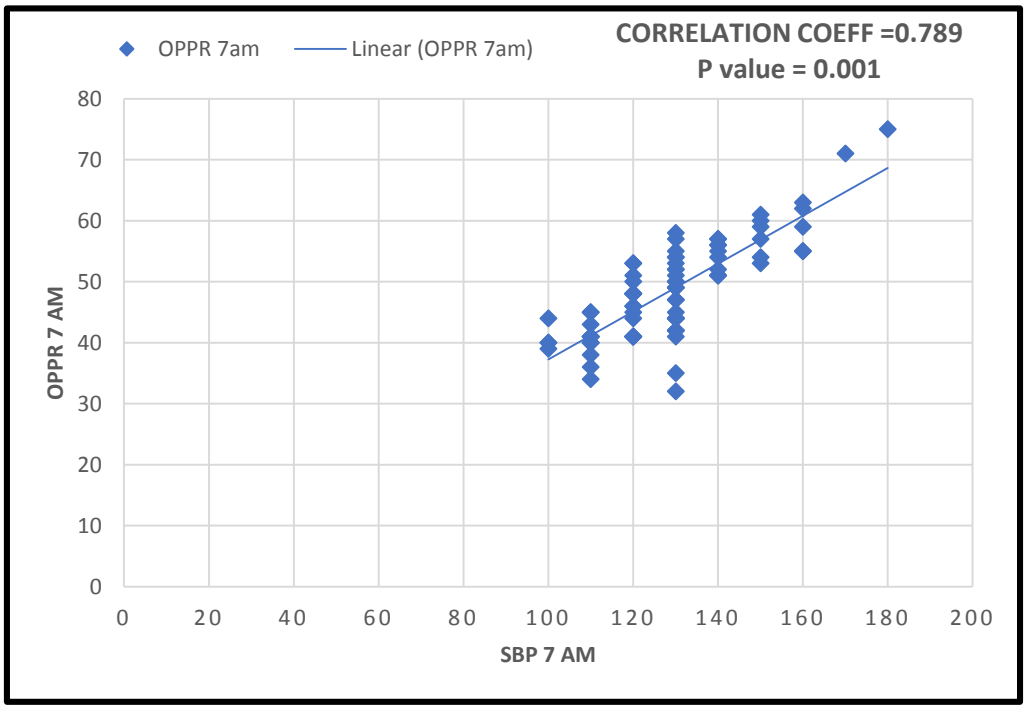
Hypertensives		6 pm					
		SBP		DBP		MAP	
		r value	P value	r value	P value	r value	P value
6 pm	OPPR	0.827	0.001*	0.761	0.001*	0.875	0.001*
	OPPL	0.841	0.001*	0.798	0.001*	0.898	0.001*

Table 14: Comparison of OPP with SBP, DBP and MAP at 12 am in hypertensives

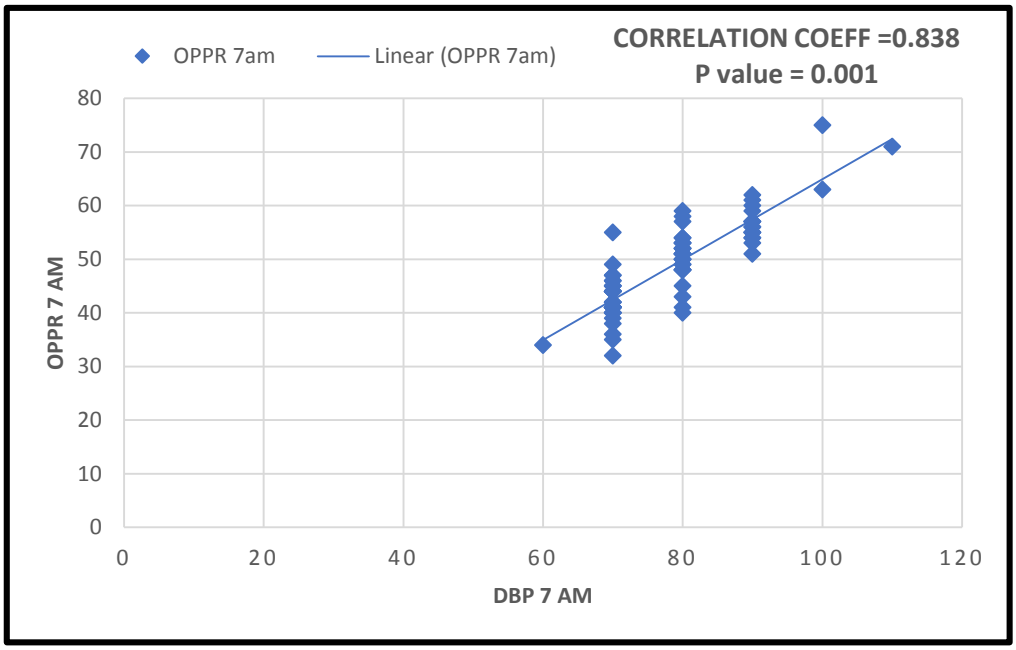
Hypertensives		12 am					
		SBP		DBP		MAP	
		r value	P value	r value	P value	r value	P value
12 am	OPPR	0.858	0.001*	0.797	0.001*	0.896	0.001*
	OPPL	0.856	0.001*	0.786	0.001*	0.894	0.001*

The strength of the relationship between OPP and SBP, OPP and DBP and also OPP and MAP at 7 am, 1 pm, 6 pm and 12 am in 24 hours was analyzed using Spearman's rho. All values were positively correlated with correlation coefficient ® value < 1 and p-value < 0.05 (Tables 11,12, 13 and 14).

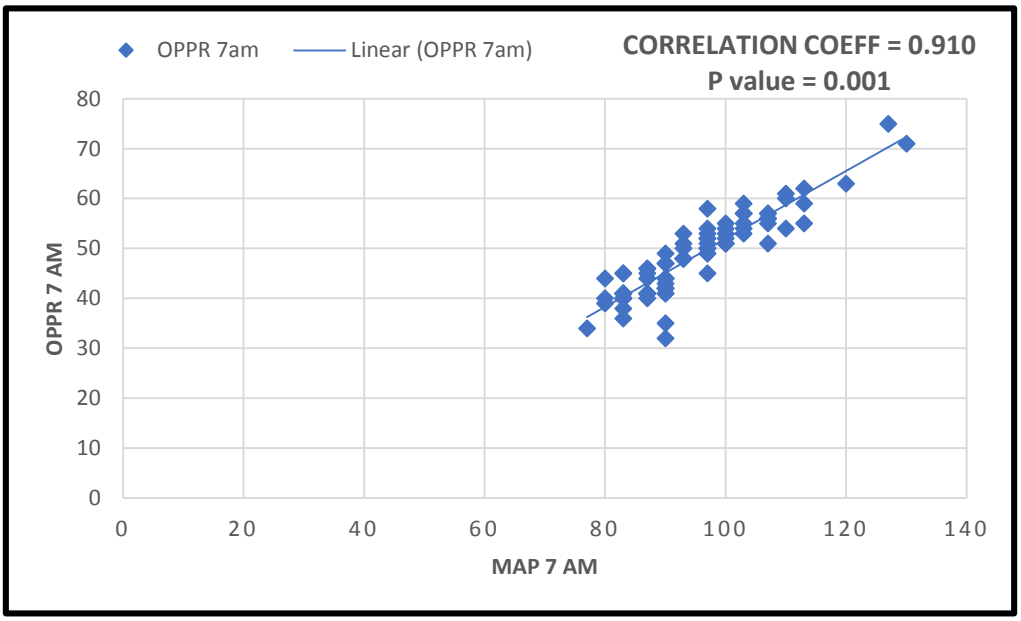
Graph 4: Scatter plot between OPPR and SBP at 7 am



Graph 5: Scatter plot between OPPR and DBP at 7 am



Graph 6: Scatter plot between OPPR and MAP at 7 am

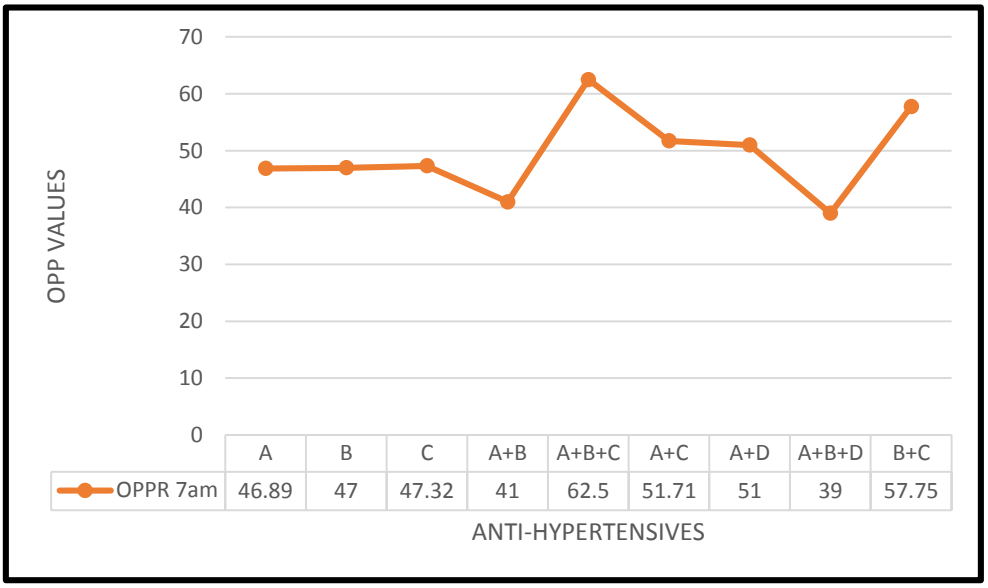


The strength of the relationship between OPP and SBP, OPP and DBP, and OPP and MAP is illustrated using scatter plots. OPP of the right eye was considered, and as the ideal time for blood pressure measurement is early in the morning before food, 7 am values of SBP, DBP and MAP were taken to plot the graph (Graphs 4,5 and 6).

Table 15: Comparing the effect of antihypertensive medications on ocular perfusion pressure of the right eye in 24 hours in hypertensives.

Medications	N	OPPR (MEAN ± SD)			
		7 am	1 pm	6 pm	12 am
Angiotensin receptor blockers (A)	19	46.89 ± 7.5	57.16 ± 5.8	57.58 ± 5.8	52.74 ± 6.1
Beta-blockers (B)	6	47 ± 7.64	54.83 ± 8.2	57 ± 9.8	51.33 ± 7.1
Calcium channel blockers (C)	22	47.32 ± 6.25	56.77 ± 8	56.55 ± 7.9	50.86 ± 5.92
A+B	1	41	47	45	49
A+B+C	2	62.5 ± 0.7	69 ± 8.4	79.5 ± 2.12	64.5 ± 4.9
A+C	7	51.71 ± 9.3	58 ± 2.6	59 ± 5.7	49.57 ± 5.9
A+Diuretics(D)	2	51 ± 5.65	57.5 ± 3.5	61.5 ± 4.9	49 ± 4.2
A+B+D	1	39	65	54	46
B+C	4	57.75 ± 12.1	58.25 ± 6.9	57 ± 9.05	49.75 ± 5.67
None	104	44.98 ± 4.9	49.63 ± 6.9	50.40 ± 6.5	47.83 ± 5.35
P value		0.01*	0.01*	0.01*	0.01*
*P value<0.05 implies that it is significant statistically					

Graph 7: Graphical representation comparing OPP in the right eye and the effect of antihypertensive medications in the cases group at 7 am.



To compare the effect of antihypertensives (A-Angiotensin receptor blockers, B-Beta blockers, C-Calcium channel blockers, D-Diuretics) on ocular perfusion pressure of the right eye in 24 hours (Table 15), Kruskal Wallis Test was used. All the values were significant, with a p-value <0.005. The overall lowest OPP (Mean=39) was seen in patients on A+B+D antihypertensives at 7 am, and the highest OPP (Mean=79.50±2.12) was seen in patients on A+B+C antihypertensives at 6 pm.

For easy understanding, a graphical representation (graph 7) comparing the effect of antihypertensives on OPP in the right eye was done using the values recorded at 7 am. Patients on angiotensin receptor blockers (Mean=46.89 ± 7.5) and beta blockers (Mean=47 ± 7.64) showed the lowest OPP value, and patients on A+B+C antihypertensives showed the highest OPP value (Mean=62.5±0.7).

Discussion

Glaucoma has long been associated with various risk factors, with IOP being a key indicator of disease progression. The IOP levels in glaucoma patients can vary, making it essential to assess the strength and consistency of the connection between ocular pressures influencing the eye and glaucoma (177).

The average age of patients in the hypertensive group was 64.20 ± 8.659 years, while in the normotensive group, it was 63.83 ± 8.713 years, a difference that was not statistically significant ($p>0.05$). This is similar to studies done by Onakoya et al. (hypertensives=56.7 ± 12.95, normotensives=54.7 ± 9.65) and Ekwufulem et al. (hypertensives=57.59 ± 9.28, normotensives=50.47 ± 9.83) where the hypertensive group had a higher mean age than the normotensive group and not significant($p>0.05$) (178,179). Though the comparison of age between the groups was not significant, results suggest that age could be a relevant factor to consider in studies related to glaucoma and hypertension.

The male-to-female ratio was equal (M:F = 27:57) in both groups but not significant. The comparison of other parameters like diabetes, smoking and alcohol was significant ($p<0.05$).

Intraocular pressure of both right and left eyes, blood pressure and perfusion pressures are recorded at four specific time points: 7 AM, 1 PM, 6 PM and 12 AM in both groups. The IOP measurements at 7 AM are taken for comparison considering the diurnal patterns, which are characteristically higher in the morning. The right eye's IOP and perfusion pressure values are presented and discussed in detail for simplicity and ease of understanding, given that no significant difference was observed in the mean values between both eyes. The ideal time for BP measurement is early morning before food, i.e., 7 am. Hence, the measurements taken at 7 am were used for the discussion.

When comparing the mean IOP values between hypertensives and normotensives, the mean IOP was significantly higher in hypertensives (14.81 mmHg) compared to normotensives (12.88 mmHg), with $p<0.05$. Deb AK et al. showed a similar significance, with $p<0.05$; the hypertensive group had a mean IOP of 15.37 mmHg, and the normotensive group had a mean IOP of 13.41 mmHg (3). Similar significance was also seen in studies done by Onakoya et al. (hypertensives=28.4 mmHg, normotensives=15.2 mmHg) and Ekwufulem et al. (hypertensives=14.71 mmHg, normotensives=12.07 mmHg) (3,178,179). This indicates that individuals with systemic hypertension are more at risk of developing elevated IOP than normotensives.

The mean SBP, DBP and MAP values were significantly higher in the hypertensive group (SBP=128 mmHg, DBP=78.21 mmHg, MAP=95.08 mmHg) compared to the normotensive group (SBP=113.10 mmHg, DBP=74.76 mmHg, MAP=85.83 mmHg) ($p<0.05$). These findings are consistent with the study by Ekwufulem et al., i.e., in hypertensives (SBP=128.21 mmHg, DBP=76.52 mmHg, MAP=93.63 mmHg) and normotensives (SBP=120.58 mmHg, DBP=77.38 mmHg, MAP=91.68 mmHg) (178).

Leske et al. showed that hypertension might provide some protection against glaucoma. They explain that elevated blood pressure could temporarily enhance perfusion to the ONH. However, this protection may diminish over time, and with prolonged hypertension, the ONH may experience compromised perfusion, ultimately leading to the development of glaucoma (180).

Lower SBP and DBP were found to be linked to a higher incidence of POAG. A potential reason for this could be that reduced SBP and low DBP may compromise the blood supply to the ONH, resulting in retinal ganglion cell death. When this factor is combined with elevated IOP, which can cause venous collapse, the impairment of blood flow is further exacerbated, worsening the risk of

glaucomatous damage (7,181). Our study observed no low systolic and diastolic blood pressure (SBP and DBP) values, indicating that none of the participants were hypotensive.

Pache and Flammer, in their study, reported that the nocturnal drop in BP is a predisposing factor for glaucoma onset (1). One possible reason could be that taking anti-hypertensive medications at bedtime causes a drop in nighttime blood pressure, leading to a decrease in ONH perfusion(3). In this study, we observed a decline in blood pressure during the early morning hours, precisely at 7 AM, in both hypertensives (Mean SBP=128.81 mmHg, Mean DBP=78.21 mmHg) and normotensives (Mean SBP=113.10 mmHg, Mean DBP=72.26 mmHg) compared to the measurements taken at midnight i.e., in hypertensives (Mean SBP=132.50 mmHg, Mean DBP=78.10 mmHg) and in normotensives (Mean SBP=116.07 mmHg, Mean DBP=72.62 mmHg). This phenomenon was likely influenced by the fact that many of the participants were administering antihypertensive medications in the late evening hours before sleep.

Mean ocular perfusion pressure was significantly higher in the hypertensives (Mean OPP = 48.56 mmHg) than in normotensives (Mean OPP = 44.20 mmHg) with $p<0.05$, suggesting a protective effect on the optic nerve head (ONH).

These findings contrast the study by Ekwufulem et al., which reported lower MOPP in hypertensives (Mean = 47.10 mmHg) compared to normotensives (Mean = 48.99 mmHg) with $p=0.012$. MOPP, which ensures adequate perfusion to the optic nerve head, is regulated by the balance between the blood and intraocular pressures. Therefore, in hypertensive individuals, these two parameters should be considered together rather than in isolation to assess the risk to optic nerve health (178).

The present study also showed a positive correlation between MOPP and SBP, DBP, and MAP using Spearman's rho test with correlation coefficient ® value < 1 and $p<0.05$ at 7 am, 1 pm, 6 pm and 12 am in 24 hours.

There's additional evidence for the vascular mechanism, where a lower OPP leads to reduced blood supply to the optic nerve, impairing autoregulation and ultimately contributing to glaucomatous optic nerve damage (177,182)

Autoregulation refers to the eye's ability to maintain stable blood flow despite fluctuations in perfusion pressures. When this autoregulatory mechanism fails, it can disrupt blood flow, leading to ischemia and eventual damage to the optic nerve (183).

A sharp drop in blood pressure results in low perfusion pressure, causing ischemic conditions that harm the optic nerve head (184). On the other hand, excessively high blood pressure leads to arteriosclerosis of the vessels, which raises resistance to blood flow and lowers perfusion pressure, preventing adequate nourishment to the optic nerve (3,183).

Like MOPP, mean SPP and DPP were significantly higher in the hypertensives than normotensives. A study by Gore V et al. showed that low MAP, SPP, and DPP increase POAG prevalence, which are the independent risk factors for OAG (7). This shows the importance of perfusion pressures in glaucoma development.

Ocular perfusion pressure was highest in patients taking calcium channel blockers (Mean = 47.32mmHg), followed by those on beta blockers (Mean = 47mmHg) and angiotensin receptor blockers (Mean = 46.89mmHg), suggesting a potential effect of CCB's on optic nerve health. Since all values were within normal limits for patients on all types of antihypertensives, these medications, when used appropriately, have minimal impact on ocular health.

These findings contrast the study by Muskens et al., which indicated that beta blockers have a protective effect on optic nerve health and concluded that using calcium channel blockers was associated with glaucoma (185). Several studies support the use of CCBs in patients with normal tension glaucoma. This recommendation is based on findings from both human and animal studies,

which demonstrate that blocking membrane-bound calcium channels can lead to the dilation of ocular blood vessels and enhance the perfusion of the optic nerve (186,187).

Thus, the study underscores the importance of thoroughly screening all hypertensive patients for glaucoma to prevent ocular complications associated with the disease.

Key Strengths and Limitations

The strength of the study lies in the 24-hour monitoring of blood pressure, intraocular pressure and perfusion pressures, which contrasts with many population-based studies that relied on single readings. However, the limitations include a small sample size, and a more appropriate approach would involve conducting follow-up assessments with patients to assess glaucoma progression. A longitudinal study tracking hypertensive and normotensive individuals, assessing visual fields and other relevant parameters, offers a more effective way of evaluating the glaucoma risk.

Summary

This cross-sectional study, conducted over one-and-a-half-years, compared an association between systemic hypertension, perfusion pressure and glaucoma.

- The study involved 168 participants, 84 hypertensive cases and 84 controls with equal male and female age over 40 years.
- The mean age of the hypertensive (64.20 ± 8.66 years) was slightly higher than the normotensive group (63.83 ± 8.71 years).
- Intraocular pressure was higher in the hypertensive group (Right eye =14.81 ± 3.287, Left eye = 14.86 ± 3.387 at 7 am, $p<0.05$), with significant differences observed at all time intervals. However, the values remained within the normal range.
- Systolic, diastolic and mean arterial pressures were higher in hypertensives ($p<0.05$), especially at 1 pm and 6 pm.
- Ocular perfusion pressure was also significantly higher in hypertensive individuals (Right eye = 48.56 ± 8.02, Left eye = 48.51 ± 8.19 at 7 am, $p<0.05$), particularly at peak blood pressure times, showing a positive correlation with blood pressure.
- Systolic and diastolic perfusion pressures were also higher in hypertensives than in the normotensives in 24 hours ($p<0.05$).
- The type of antihypertensive medication influenced OPP ($p<0.05$), suggesting that hypertension and its treatment are the risk factors for nocturnal hypotension and can affect ocular health, particularly the optic nerve head.
- This emphasizes the importance of considering both IOP and blood pressure in glaucoma risk assessment and the need for screening hypertensive individuals for glaucoma to prevent further ocular damage.

Conclusion

This study examines the relationship between BP, OPP and glaucoma. Hypertensive individuals showed higher IOP, potentially increasing the risk of glaucoma. The MOPP, MSPP, and MDPP were significantly higher in the hypertensives than in the normotensives, supporting the vascular role in glaucoma development. Low BP and nocturnal BP drops often influenced by antihypertensive medications reduce ONH blood supply, exacerbating ischemic damage when combined with elevated IOP. Autoregulatory failures in ONH blood flow further contribute to optic nerve injury. Comprehensive screening of hypertensive patients for glaucoma and careful evaluation of BP, IOP, and perfusion pressures like OPP, SPP and DPP should be prioritized by ophthalmologists to ensure effective prevention.

Appendix I

Consent form

STUDY SUBJECT CONSENT FORM

I confirm that Dr SNEHA L has explained the purpose of the research, the study procedure, the benefits, and the possible discomfort that I may experience in the language best understood by me. Therefore, I agree to participate as a subject in this research project and willfully consent.

_____ _____

(Participant) (Date)

_____ _____

(Witness to above signature) (Date)

ಅಧ್ಯಯನವಿಷಯಕಾನ್ಸೆಂಟ್ಾಾರ್ಮ್

ಡಾ. ಸ್ನೇಹಾ ನನಗ್ ಸೆಂಶ್ ೀಧ್ಯ್ ಉದ್ದೇಶ' ಅಧ್ಯಯನದ ವಿಧಾನ ಮತ್ತು ಸೆಂಭವನೇಯ ಅಸವಸಥತ್ಗಳು ಮತ್ತು ನನನ ಸವೆಂತ್ಾಷ್ಯಲ್ಲಿ ನಾನತ ಅನತಭವಿಸಬಹತದಾದ ಪ್ರಯೇಜನಗಳನತನ ವಿವರಿಸಿದ್ದೇನ್ ಎಂದತ ನಾನತ ಖಚಿತ್ ಪ್ಡಿಸತತ್ುೀನ್. ಮೇಲ್ಲನ ಎಲ್ಾೆ ವಿಷಯಗಳನತನ ನನನ ಸವೆಂತ್ ಭಾಷ್ಯಲ್ಲಿ ವಿವರವಾಗಿ ವಿವರಿಸಲ್ಾಗಿದ್ ಮತ್ತು ನಾನತ ಅದನತನ ಅರ್್ಮಾಡಿಕ್ ೆಂಡಿದ್ದೇನ್. ಆದದರಿೆಂದ' ಈ ಸೆಂಶ್ ೀಧ್ನಾಯೇಜನ್ಯಲ್ಲಿ ವಿಷಯವಾಗಿ ಭಾಗವಹಿಸಲತ ಒಪ್ಪಿಗ್ ನೇಡಲತ ನಾನತ ಒಪ್ಪಿತ್ುೀನ

(ಭಾಗವಹಿಸತವವರತ) (ದಿನಾೆಂಕ್

Appendix II

Case Proforma

APPENDIX X

DEPARTMENT OF OPHTHALMOLOGY
B.L.D.E UNIVERSITY'S SHRI B.M.PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA-586103

CASE PROFORMA

Case No: ☐☐

Name : ☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

Age: ☐☐ years Sex: ☐ (1-Male 2-Female) IP no: ☐☐☐☐☐☐☐

Address: ☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐
☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐
☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

Contact no: ☐☐☐☐☐☐☐☐☐☐ | ☐☐☐☐☐☐☐☐☐☐

Is the patient eligible for study? (1-Yes, 2-No): ☐

Has informed consent been given? (1-Yes, 2-No): ☐

Chief Complaints:

1. Diminution of vision: Right Eye ☐ Duration: ☐☐ days/months/years
   Left Eye ☐ Duration: ☐☐ days/months/year

2. Others (if any): ……....

……

History of Present Illness:

1. Diminution of vision: Insidious (1) or Sudden (2): ☐
   Progressive (1) or Non-progressive (2): ☐
   Painless (1) or Painful (2): ☐
   For distance (1) or for near (2): ☐
2. Diplopia / Polyopia: Present (1) or Absent (2): ☐
3. Coloured halos: Present (1) or Absent (2): ☐
4. Black spots / non seeing area before eye
   Present (1) or Absent (2): ☐
5. Redness: Present (1) or Absent (2): ☐
6. Watering: Present (1) or Absent (2): ☐
7. Discharge: Present (1) or Absent (2): ☐
8. Pain in eyes: Present (1) or Absent (2): ☐
9. Headache: Present (1) or Absent (2): ☐
10. H/O present trauma: Present (1) or Absent (2): ☐
11. H/O wearing glasses: Present (1) or Absent (2): ☐
    Near (1) or Far (2) or Both (3): ☐
    Duration: ☐

Past history:

1. H/O past trauma to eye: Present (1) or Absent (2): ☐
2. Ocular surgery: Present (1) or Absent (2): ☐

   Type of surgery:……

   When performed? : ……..…..

3. Diabetes: Present (1) or Absent (2): ☐

   Duration:……..

   Medication:……..

4. Hypertension: Present (1) or Absent (2): ☐

   Duration:……..

   Medication:……..

5. CAD: Present (1) or Absent (2): ☐
   Duration:……..
   Medication:……..

6. Any other medical disorder : ……

Personal History:

1. Smoking: Present (1) or Absent (2): ☐
   Duration:…….
2. Alcohol intake: Present (1) or Absent (2): ☐
   Duration:……..
3. Diet: Vegetarian(1) or Non Vegetarian (2) or Mixed (3): ☐

Family History:

Family history of glaucoma (1 – Present; 2 – Absent) : ☐

General Physical Examination:

1. Built: ☐
   (Well built – 1, Moderately built – 2, Poorly built – 3, Emaciated – 4)

2. P I C K E L Present (1) or Absent (2): ☐

3. Pulse : ……/minute

4. Temperature: .....degree Fahrenheit

5. Blood pressure: …….. / .....mmHg

6. Respiratory rate ..... cycles per minute

Systemic Examination:

1. CVS: [ ] Normal – 1, Abnormal – 2

   If 2, specify:……

2. CNS: [ ] Normal – 1, Abnormal – 2

   If 2, specify:……

3. Respiratory System [ ] Normal – 1, Abnormal – 2

   If 2, specify:……

4. Per abdomen: [ ] Normal – 1, Abnormal – 2

   If 2, specify:……

Current medication (if any) :

Visual Acuity:

	RE	LE
DISTANT		
PINHOLE		

NEAR		
AIDED		

Ocular Examination:

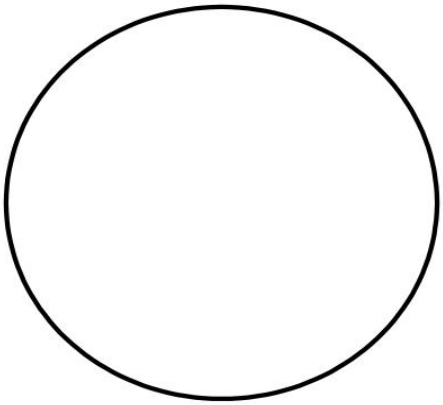
Adnexa: 1- Normal 2- Abnormal	☐	☐
Sclera: 1- Normal 2- Congested	☐	☐
Conjunctiva 1- Normal 2- Conjunctival Congestion 3- Ciliary congestion 4- Chemosis	☐	☐
Cornea 1- Normal 2- Opacity 3- Vascularization	☐	☐

Anterior Chamber 1- Normal depth 2- Shallow 3- Deep	☐	☐
Iris 1- Normal colour and pattern 2-Abnormal	☐	☐
Pupil **Shape:** 1-Round and regular; 2- Irregular **Reaction:** Direct: 1-Present; 2-Absent Indirect: 1-Present; 2-Absent Near reflex: 1-Present; 2-Absent **Pseudo exfoliation granules in margin** 1- Present 2- Absent	**Size**..... mm ☐ ☐ ☐ ☐ ☐	**Size**..... mm ☐ ☐ ☐ ☐ ☐

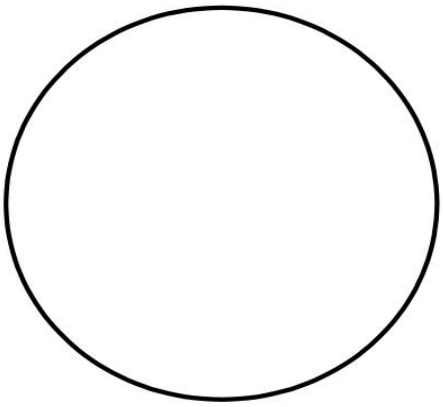
Lens **Clarity:** 1-Clear; 2-Opaque 1- Cataract; 2- PCIOL If cataract present: 1- Immature 2- Mature 3- Hyper mature A) Cortical cataract (1-Present;2-Absent) B) Nuclear sclerosis (1-Present;2-Absent) If present: GRADE: C) Posterior Sub capsular cataract (1-Present 2-absent)	☐ ☐ ☐ ☐ ☐ ☐ …… ☐	☐ ☐ ☐ ☐ ☐ ☐ …….. ☐
• **Lacrimal duct patency** (1-Patent, 2-Regurgitation, 2A-Clear fluid; 2B-Mucopurulent; 2C- Blocked)	☐	☐

FUNDUS EXAMINATION:

Fundus	**Right eye**	**Left eye**
Glow		
Media		
Disc		
CD ratio		
Blood vessels		
Background		
Macula		



RIGHT EYE



LEFT EYE

HYPERTENSIVE RETINOPATHY : ……..……...

DIABETIC RETINOPATHY : ……..……..

ANY OTHERS : ……..…….

DIAGNOSIS:

INVESTIGATIONS

1. BLOOD PRESSURE

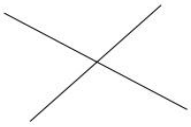
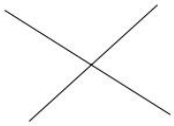
TIME	7:00am	1:00pm	6:00pm	12:00am
SYSTEMIC BLOOD PRESSURE (SBP)				
DIASTOLIC BLOOD PRESSURE(DBP)				
MEAN ARTERIAL BLOOD PRESSURE(MAP) [MAP=DBP+1/3(SBP-DBP)]				

2. INTRA OCULAR PRESSURE: (with……)

TIME	Right Eye IOP	Left Eye IOP
7:00am		
1:00pm		
6:00pm		
12:00am		

3.GONIOSCOPY:

Grading of angle by Shaffer's method

Right eye	Left eye
	

4.PERFUSION PRESSURE (Normal OPP – 40 to 60mmHg)

TIME	7:00am	1:00pm	6:00pm	12:00am
MEAN OCULAR PERFUSION PRESSURE (MOPP) [MOPP=2/3(MAP)-IOP]				
SYSTOLIC PERFUSION PRESSURE (SPP) [SPP=SBP-IOP]				
DIASTOLIC PERFUSION PRESSURE (DPP) [DPP=DBP-IOP]				

Dr. Sneha L
Investigator
PG Student
Department of Ophthalmology

Prof. (Dr.) Rekha Mudhol
Guide
Professor and HOD
Department of Ophthalmology

Appendix III

Institutional Ethical Clearance

BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 865/2022-23 1/4/2023

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinizes the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "A COMPARTIVE STUDY ON ASSOCIATION BETWEEN SYSTEMIC HYPERTENSION, PERFUSION PRESSURE AND GLAUCOMA IN AN ADULT POPULATION OF NORTH KARNATAKA.

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR SNEHA L

NAME OF THE GUIDE: PROF.(Dr). REKHA R. MUDHOL, Professor, Dept. of OPHTHALMOLOGY.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA

Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

Dr.Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA

MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103. Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

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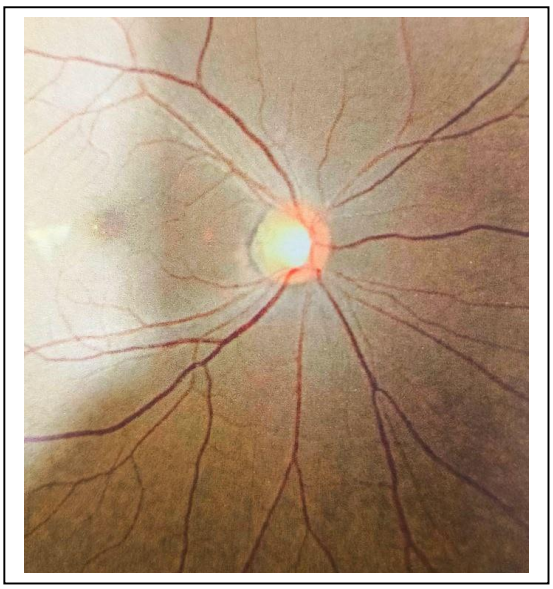
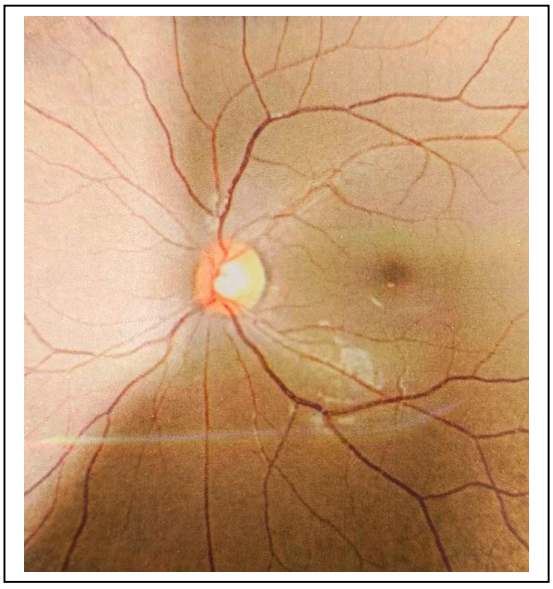
Appendix IV

Colour Plates



A



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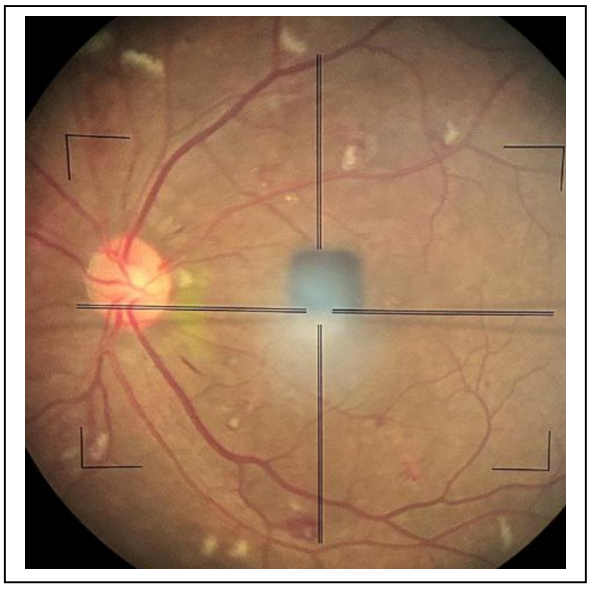
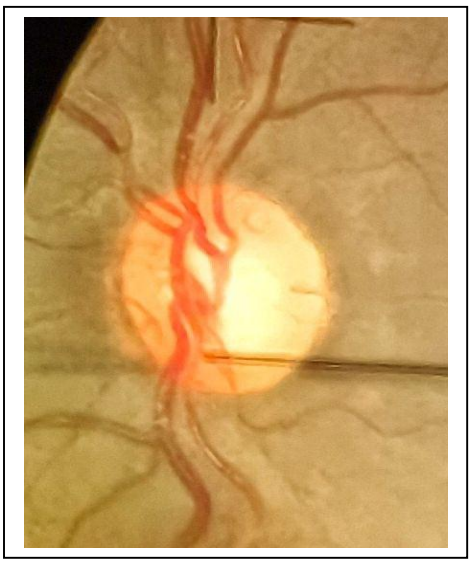


C

Figure 8: Fundus photographs (A) Right Eye and (B) Left Eye showing 0.5 cup: disc ratio, healthy NRR and bright foveal reflex; (C) Mixed retinopathy showing arteriolar attenuation, soft exudates, hard exudates and haemorrhages



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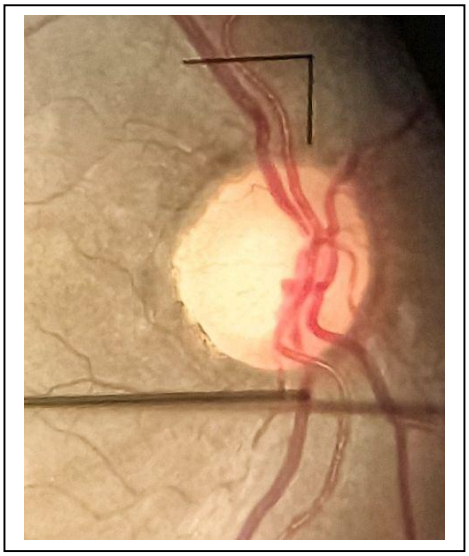

B

C

Figure 9: Optic disc photographs: (A) Optic disc with 0.4 cup: disc ratio; (B) Optic disc showing temporal pallor; (C) Hyperaemic optic disc

Figure 10: iCare IC100 Rebound Tonometer photograph

Appendix V

Master Chart

Key to Master Chart:

HTN	Hypertension
K/C	Known Case
YR	Year
MON	Month
DM	Diabetes Mellitus
MED	Medications
A	Angiotensin Receptor Blockers
B	Beta Blockers
C	Calcium Channel Blockers
D	Diuretics
N	None
RE	Right Eye
LE	Left Eye
HR	Hypertensive Retinopathy
GON	Glaucomatous Optic Neuropathy
AM	Ante Meridiem
PM	Post Meridiem
SBP	Systolic Blood Pressure
DBP	Diastolic Blood Pressure
IOPR, IOPL	Intraocular Pressure of Right and Left eyes
OPPR, OPPL	Ocular Perfusion Pressure of Right and Left eyes
MAP	Mean Arterial Pressure
SPPR, SPPL	Systolic Perfusion Pressure of Right and Left eyes
DPPR, DPPL	Diastolic Perfusion Pressure of Right and Left eyes

S.No	GROUP	NAME	AGE	SEX	HTN K/C	DURATION	DM K/C	MED	SMOKING	ALCOHOL	FUNDUS	SBP 7Aam
1	CASE 1	SHARADA DEELIP	53	FEMALE	YES	1YR	NO	B	NO	NO	NORMAL	130
2	CONTROL 1	MAHADEVI	50	FEMALE	NO		NO		NO	NO	NORMAL	110
3	CASE 2	MALLAMMA MADDIMANI	75	FEMALE	YES	1YR	NO	C	NO	NO	NORMAL	100
4	CONTROL 2	MADIVALAWWA AGASABAL	75	FEMALE	NO		NO		NO	NO	NORMAL	120
5	CASE 3	SAKKUBAI JADHAV	65	FEMALE	YES	YR	NO	B+C	NO	NO	NORMAL	180
6	CONTROL 3	RUDRAMMA BADIGER	65	FEMALE	NO		NO		NO	NO	NORMAL	110
7	CASE 4	DANAPPA KORI	55	MALE	YES	5YRS	YES(5YRS)	A	YES	YES	MILD DR	150
8	CONTROL 4	VIJAYENDRA KATTI	55	MALE	NO		NO		NO	NO	NORMAL	120
9	CASE 5	NINGAPPA TALWAR	64	MALE	YES	5YRS	NO	C	YES	YES	G-1 HR	140
10	CONTROL 5	LAXMAN BABLESHWAR	65	MALE	NO		NO		NO	NO	NORMAL	120
11	CASE 6	VIJAYSING LAMANI	62	MALE	NO		NO	A+C	NO	YES	NORMAL	170
12	CONTROL 6	BASAVARAJ HONALLI	60	MALE	NO		NO		NO	NO	NORMAL	120
13	CASE 7	BAPURAY SARAWAD	61	MALE	YES	4YRS	NO	A+B+D	YES	YES	NORMAL	100
14	CONTROL 7	RACHAPPA BILAGI	60	MALE	NO		NO		NO	NO	NORMAL	120
15	CASE 8	IRASANGAPPA BINJALABAVI	67	MALE	YES	10YRS	NO	C	NO	NO	NORMAL	140
16	CONTROL 8	MALLIKARJUN MADGYAL	66	MALE	NO		NO		NO	NO	NORMAL	110
17	CASE 9	CHANDRASHEKAR BAGALI	63	MALE	YES	7YRS	NO	A+C	NO	NO	LE COAT'S	140
18	CONTROL 9	AB CHIRALADINNI	63	MALE	NO		NO		NO	NO	NORMAL	100
19	CASE 10	LAXMIBAI DEVARHIPPARGI	50	FEMALE	YES	1MON	NO	A+D	NO	NO	G-3 HR	140
20	CONTROL 10	GANGAVVA TOGARI	50	FEMALE	NO		NO		NO	NO	NORMAL	100
21	CASE 11	LAKKAWWA AMBIGER	68	FEMALE	NO		NO	N	NO	NO	NORMAL	130
22	CONTROL 11	KAMALAVVA MANDOLI	66	FEMALE	NO		NO		NO	NO	NORMAL	120
23	CASE 12	MAHADEVI PUJARI	55	FEMALE	YES	1YR	NO	A	NO	NO	NORMAL	110
24	CONTROL 12	NAGAVVA BANDIWADDAR	55	FEMALE	NO		NO		NO	NO	NORMAL	120
25	CASE 13	LAXMIBAI MARAGUR	65	FEMALE	YES	6DAYS	NO	A	NO	NO	NORMAL	110
26	CONTROL 13	NILAVVA KURI	65	FEMALE	NO		NO		NO	NO	NORMAL	110
27	CASE 14	SARUPA CHAVAN	46	FEMALE	NO		NO	B	NO	NO	NORMAL	130
28	CONTROL 14	SARASWATI METI	44	FEMALE	NO		NO		NO	NO	NORMAL	110
29	CASE 15	CHANDRABAI LAMANI	62	FEMALE	YES	1YR	NO	A+B+C	NO	NO	NORMAL	160
30	CONTROL 15	NANDAMMA V	60	FEMALE	NO		NO		NO	NO	NORMAL	100
31	CASE 16	NINGAPPA PUJARI	68	MALE	YES	1YR	YES	N	YES	YES	NORMAL	130
32	CONTROL 16	DATTU KAMBALE	69	MALE	NO		NO		NO	NO	NORMAL	120

S.No	GROUP	NAME	AGE	SEX	HTN K/C	DURATION	DM K/C	MED	SMOKING	ALCOHOL	FUNDUS	SBP 7Aam
33	CASE 17	MAYAVVA HARUGERI	60	FEMALE	YES	6MON	YES	A+B+C	NO	NO	NORMAL	160
34	CONTROL 17	SUNADA BENAL	60	FEMALE	NO		NO		NO	NO	NORMAL	120
35	CASE 18	MALLAPPA KOLAKAR	60	MALE	NO		NO	N	YES	NO	NORMAL	120
36	CONTROL 18	RACHAPPA H	60	MALE	NO		NO		NO	NO	NORMAL	120
37	CASE 19	SURESH KUMATAGI	78	MALE	YES	6MON	NO	N	NO	NO	NORMAL	120
38	CONTROL 19	BASAPPA DAVALAGI	78	MALE	NO		NO		NO	NO	NORMAL	110
39	CASE 20	AMBAVVA KANURKAR	60	FEMALE	NO		YES (2MONTHS)	C	NO	NO	NORMAL	160
40	CONTROL 20	LAXMAWWA KAMBAGI	60	FEMALE	NO		NO		NO	NO	NORMAL	110
41	CASE 21	INDRAVVA DODAMANI	55	FEMALE	NO		YES(9YEARS)	N	NO	NO	NORMAL	110
42	CONTROL 21	BHARATI CHANDNERI	56	FEMALE	NO		NO		NO	NO	NORMAL	110
43	CASE 22	PARVATI MUNJI	41	FEMALE	YES	1YR	NO	A	NO	NO	NORMAL	130
44	CONTROL 22	SATAWWA HONNALLI	43	FEMALE	NO		NO		NO	NO	NORMAL	100
45	CASE 23	BEERAPPA MANNAGOL	55	MALE	YES	1.5MON	NO	C	NO	NO	NORMAL	140
46	CONTROL 23	KALLAPPA PUJARI	55	MALE	NO		NO		NO	NO	NORMAL	110
47	CASE 24	NAGAMMA KEMBHAVI	70	FEMALE	YES	6MON	YES(1YEAR)	C	NO	NO	NORMAL	150
48	CONTROL 24	IRAWWA MATHAPATI	70	FEMALE	NO		NO		NO	NO	NORMAL	120
49	CASE 25	DUNDAVVA SONAGAVI	60	FEMALE	NO		NO	C	NO	NO	NORMAL	130
50	CONTROL 25	INTAJABI MAKANDAR	59	FEMALE	NO		NO		NO	NO	NORMAL	110
51	CASE 26	BOURAMMA KODANGAL	82	FEMALE	YES	4YRS	NO	A	NO	NO	NORMAL	110
52	CONTROL 26	SITAVVA MADAR	80	FEMALE	NO		NO		NO	NO	NORMAL	110
53	CASE 27	JAIBUNI FOUJI	63	FEMALE	YES	20YRS	YES(3YRS)	A	NO	NO	NORMAL	130
54	CONTROL 27	MALLAWWA SITTIMANI	64	FEMALE	NO		NO		NO	NO	NORMAL	110
55	CASE 28	LAXMIBAI BIRADAR	62	FEMALE	YES	3YRS	NO	C	NO	NO	NORMAL	130
56	CONTROL 28	BOURAMMA MALI	60	FEMALE	NO		NO		NO	NO	NORMAL	110
57	CASE 29	ANNAPOORNA AGASAR	50	FEMALE	YES	10YRS	YES(10YRS)	A+C	NO	NO	NORMAL	110
58	CONTROL 29	KAMALA NAWADAGI	50	FEMALE	NO		NO		NO	NO	NORMAL	110
59	CASE 30	RAVATAPPA HANDI	80	MALE	YES	4MONTHS	NO	N	YES	YES	RE GON	110
60	CONTROL 30	SOMANNA PUJARI	80	MALE	NO		NO		NO	NO	NORMAL	110
61	CASE 31	BABASAB HOSAMANI	65	MALE	YES	1YR	NO	B	NO	NO	NORMAL	150
62	CONTROL 31	DASTAGIRSAB KORABU	65	MALE	NO		NO		NO	NO	NORMAL	120
63	CASE 32	RAMJAN MUJAWAR	61	MALE	YES	1MON	NO	A+B	NO	NO	NORMAL	120
64	CONTROL 32	SHARANAPPA PUJARI	62	MALE	NO		NO		NO	NO	NORMAL	130

S.No	GROUP	NAME	AGE	SEX	HTN K/C	DURATION	DM K/C	MED	SMOKING	ALCOHOL	FUNDUS	SBP 7Aam
65	CASE 33	SHIVAMMA KATTIMANI	48	FEMALE	YES	1YR	NO	A	NO	NO	NORMAL	130
66	CONTROL 33	RAJASHREE DAMANI	45	FEMALE	NO		NO		NO	NO	NORMAL	120
67	CASE 34	SAJANBI MULLA	74	FEMALE	YES	1YR	NO	A	NO	NO	NORMAL	130
68	CONTROL 34	YALLAVVA PUJARI	73	FEMALE	NO		NO		NO	NO	NORMAL	110
69	CASE 35	NINGAVVA TALWAR	60	FEMALE	YES	1MON	NO	C	NO	NO	NORMAL	120
70	CONTROL 35	GANGABAI BIRADAR	60	FEMALE	NO		NO		NO	NO	NORMAL	120
71	CASE 36	HULGAVVA MADAR	70	FEMALE	YES	1MON	NO	N	NO	NO	NORMAL	130
72	CONTROL 36	GURASHANTAVVA	70	FEMALE	NO		NO		NO	NO	NORMAL	120
73	CASE 37	MAMTAJ BEGAM	68	FEMALE	YES	8YR	YES(8YRS)	C	NO	NO	NORMAL	130
74	CONTROL 37	GUJJAWWA KODAHONNA	69	FEMALE	NO		NO		NO	NO	NORMAL	110
75	CASE 38	PADMAVATI NAIKODI	57	FEMALE	YES	1YR	NO	A	NO	NO	NORMAL	120
76	CONTROL 38	VALUBAI KARATI	55	FEMALE	NO		NO		NO	NO	NORMAL	100
77	CASE 39	SIDARAYA KOKATANUR	85	MALE	YES	1MON	NO	N	NO	NO	NORMAL	100
78	CONTROL 39	SHIVAPPA JAGAMSHETTY	85	MALE	NO		NO		NO	NO	NORMAL	100
79	CASE 40	RAVI PUJARI	53	MALE	NO		NO	N	NO	NO	NORMAL	130
80	CONTROL 40	SIDDARAM KOLI	51	MALE	NO		NO		NO	NO	NORMAL	110
81	CASE 41	BHIMBAI GENNUR	65	FEMALE	YES	1 YR	YES	N	NO	NO	NORMAL	110
82	CONTROL 41	CHAMPUBAI GODEKAR	65	FEMALE	NO		NO		NO	NO	NORMAL	110
83	CASE 42	SHIVAVVA BARADDI	78	FEMALE	NO		NO	N	NO	NO	NORMAL	130
84	CONTROL 42	TANIBAI MASHYAL	75	FEMALE	NO		NO		NO	NO	NORMAL	120
85	CASE 43	GOURABAI KODAHONNA	69	FEMALE	YES	4MONTHS	NO	A+C	NO	NO	NORMAL	120
86	CONTROL 43	DILSHAD DAKHANI	67	FEMALE	NO		NO		NO	NO	NORMAL	110
87	CASE 44	BORAMMA SAJJAN	65	FEMALE	YES	1 YEAR	NO	A	NO	NO	NORMAL	130
88	CONTROL 44	SHEELAVATHI RUDRAPPA	65	FEMALE	NO		NO		NO	NO	NORMAL	120
89	CASE 45	SARUBAI LAD	60	FEMALE	YES	1 YEAR	NO	C	NO	NO	NORMAL	110
90	CONTROL 45	KASHAWWA KAMBAR	60	FEMALE	NO		NO		NO	NO	NORMAL	110
91	CASE 46	FATHIMA MULLA	72	FEMALE	YES	1 YEAR	NO	B	NO	NO	NORMAL	140
92	CONTROL 46	LAXMIBAI N	72	FEMALE	NO		NO		NO	NO	NORMAL	110
93	CASE 47	SHANTABAI GAYAKWAD	60	FEMALE	YES	10YEARS	NO	C	NO	NO	NORMAL	110
94	CONTROL 47	GOURABAI SHIVASHARAN	60	FEMALE	NO		NO		NO	NO	NORMAL	120
95	CASE 48	SUBHADRABAI TAJAV	74	FEMALE	YES	5YEARS	NO	A+C	NO	NO	NORMAL	120
96	CONTROL 48	NAGAVVA WAGMORE	73	FEMALE	NO		NO		NO	NO	NORMAL	110

S.No	GROUP	NAME	AGE	SEX	HTN K/C	DURATION	DM K/C	MED	SMOKING	ALCOHOL	FUNDUS	SBP 7Aam
97	CASE 49	SHANTA BIDARI	65	FEMALE	YES	4MONTHS	NO	C	NO	NO	NORMAL	120
98	CONTROL 49	KASHIBAI POTADAR	62	FEMALE	NO		NO		NO	NO	NORMAL	110
99	CASE 50	KOUSHALABAI HAJERI	50	FEMALE	YES	5YEARS	NO	A+C	NO	NO	NORMAL	120
100	CONTROL 50	SONABAYI LAMANI	50	FEMALE	NO		NO		NO	NO	NORMAL	110
101	CASE 51	MODINBI KARNAL	67	FEMALE	YES	4MONTHS	NO	C	NO	NO	NORMAL	130
102	CONTROL 51	SHANKAREMMA BADIGER	66	FEMALE	NO		NO		NO	NO	NORMAL	110
103	CASE 52	KADUBAYI PAWAR	55	FEMALE	YES	1MONTH	NO	A	NO	NO	NORMAL	110
104	CONTROL 52	KASTURIBAI SHINDE	57	FEMALE	NO		NO		NO	NO	NORMAL	110
105	CASE 53	SIDDAVVA KATTIMANI	69	FEMALE	NO		NO	N	NO	NO	NORMAL	130
106	CONTROL 53	NEELAMMA PATIL	70	FEMALE	NO		NO		NO	NO	NORMAL	120
107	CASE 54	JAITUNABEE RAMAPUR	74	FEMALE	YES	1 YEAR	NO	C	NO	NO	NORMAL	120
108	CONTROL 54	PARAWATI HIREMATH	72	FEMALE	NO		NO		NO	NO	NORMAL	120
109	CASE 55	SIDDAMMA PATIL	62	FEMALE	YES	2YEARS	NO	C	NO	NO	NORMAL	130
110	CONTROL 55	BANGARAMMA BIRADAR	62	FEMALE	NO		NO		NO	NO	NORMAL	110
111	CASE 56	BHIMAPPA MADAGI	79	MALE	NO		NO	N	NO	NO	NORMAL	110
112	CONTROL 56	MALLAPPA BHIRAGOND	77	MALE	NO		NO		NO	NO	NORMAL	120
113	CASE 57	GOLLALAPPA PATIL	76	MALE	YES	1MONTH	NO	B	NO	NO	NORMAL	100
114	CONTROL 57	MALLAPPA TOTAD	75	MALE	NO		NO		NO	NO	NORMAL	120
115	CASE 58	LALABI BHAVIKATTI	60	FEMALE	YES	1YEAR	NO	A	NO	NO	NORMAL	120
116	CONTROL 58	SHANTABAI PATTAR	62	FEMALE	NO		NO		NO	NO	NORMAL	100
117	CASE 59	SHARANAPPA KUMBAR	65	MALE	NO		NO	N	NO	NO	NORMAL	140
118	CONTROL 59	HUSEENSAB PINJAR	65	MALE	NO		NO		NO	NO	NORMAL	120
119	CASE 60	BASAVARAJ DESAI	56	MALE	YES	3YEARS	NO	C	NO	NO	NORMAL	130
120	CONTROL 60	RAJASAB JAMABAGI	56	MALE	NO		NO		NO	NO	NORMAL	110
121	CASE 61	SHAMBAI RAJAPUT	72	FEMALE	NO		NO	N	NO	NO	G-1 HR	160
122	CONTROL 61	SURTABAI RATHOD	70	FEMALE	NO		NO		NO	NO	NORMAL	120
123	CASE 62	BASANNA LALASANGI	74	MALE	YES	2MONTHS	NO	N	NO	NO	NORMAL	120
124	CONTROL 62	MALLAPPA PUJARI	72	MALE	NO		NO		NO	NO	NORMAL	110
125	CASE 63	MADEVI JAMADAR	58	FEMALE	NO		NO	N	NO	NO	HAZY	160
126	CONTROL 63	MALLAMMA BHUDHYAL	58	FEMALE	NO		NO		NO	NO	NORMAL	110
127	CASE 64	IRAPPA NAVI	69	MALE	NO		NO	N	NO	NO	G-1 HR	140
128	CONTROL 64	YALLAPPA HOSAMANI	70	MALE	NO		NO		NO	NO	NORMAL	110

S.No	GROUP	NAME	AGE	SEX	HTN K/C	DURATION	DM K/C	MED	SMOKING	ALCOHOL	FUNDUS	SBP 7Aam
129	CASE 65	BUDDAMMA SOMPUR	58	FEMALE	YES	1 YEAR	NO	B+C	NO	NO	NORMAL	120
130	CONTROL 65	SANGAVVA KAMBAR	57	FEMALE	NO		NO		NO	NO	NORMAL	120
131	CASE 66	MANAPPA VISHWAKARMA	75	MALE	YES	4YEARS	NO	B	NO	NO	NORMAL	130
132	CONTROL 66	MALLAPPA YALAWAR	78	MALE	NO		NO		NO	NO	NORMAL	120
133	CASE 67	MAHADEVI GUDAMI	64	FEMALE	YES	10YEARS	NO	B+C	NO	NO	G-1 HR	130
134	CONTROL 67	BASAVVA HONAMURGI	65	FEMALE	NO		NO		NO	NO	NORMAL	110
135	CASE 68	PARASURAM GAJAKOSH	66	MALE	YES	6MONTHS	NO	C	YES	YES	NORMAL	110
136	CONTROL 68	APPASAHEB PATIL	67	MALE	NO		NO		NO	NO	NORMAL	100
137	CASE 69	LAXMIBAI MALA	70	FEMALE	NO		NO	N	NO	NO	NORMAL	150
138	CONTROL 69	MABUBBI MANAGULI	69	FEMALE	NO		NO		NO	NO	NORMAL	110
139	CASE 70	IRAMMA BASUPATTAD	68	FEMALE	YES	1 YEAR	NO	A	NO	NO	NORMAL	120
140	CONTROL 70	SHANTABAI RATHOD	65	FEMALE	NO		NO		NO	NO	NORMAL	120
141	CASE 71	KRISHNA BANDAGER	70	MALE	YES	10 YEARS	NO	C	YES	YES	NORMAL	130
142	CONTROL 71	VISHWANATH BALAKUNDRI	70	MALE	NO		NO		NO	NO	NORMAL	110
143	CASE 72	TIPPARAY HIREKORBAR	69	MALE	YES	2YEARS	NO	A	YES	YES	NORMAL	120
144	CONTROL 72	MALLAPPA HOSAMANI	67	MALE	NO		NO		NO	NO	NORMAL	110
145	CASE 73	SUMITRA SINDAGI	70	FEMALE	NO		NO	A	NO	NO	NORMAL	150
146	CONTROL 73	SARASWATI HIREMATH	72	FEMALE	NO		NO		NO	NO	NORMAL	120
147	CASE 74	SONAWWA BHANDARBATTI	65	FEMALE	YES	6MONTHS	NO	B+C	NO	NO	NORMAL	130
148	CONTROL 74	PRABHAVATI HIREMATH	63	FEMALE	NO		NO		NO	NO	NORMAL	110
149	CASE 75	MAINABHI YEDRAMI	55	FEMALE	YES	1YEAR	NO	A+C	NO	NO	NORMAL	110
150	CONTROL 75	RAJABI MAKANDAR	52	FEMALE	NO		NO		NO	NO	NORMAL	110
151	CASE 76	CHANDRASHEKAR JADHAV	61	MALE	YES	1YEAR	NO	A	NO	NO	NORMAL	130
152	CONTROL 76	SHANKAR LAMANI	60	MALE	NO		NO		NO	NO	NORMAL	110
153	CASE 77	SHANTAWWA DASAR	55	FEMALE	YES	4MONTHS	NO	A	NO	NO	NORMAL	140
154	CONTROL 77	SHANUBAYI LAMANI	52	FEMALE	NO		NO		NO	NO	NORMAL	110
155	CASE 78	MUKTABAI NARAYANAKAR	69	FEMALE	YES	1YEAR	NO	C	NO	NO	NORMAL	110
156	CONTROL 78	NINGAWWA GARASANGI	70	FEMALE	NO		NO		NO	NO	NORMAL	120
157	CASE 79	SUNDARABAI SIRINAL	59	FEMALE	YES	10MONTHS	NO	A	NO	NO	NORMAL	130
158	CONTROL 79	MANAMMA KORI	60	FEMALE	NO		NO		NO	NO	NORMAL	120
159	CASE 80	MUTTABAI RATHOD	52	FEMALE	YES	10YEARS	NO	C	NO	NO	G-1 HR	130
160	CONTROL 80	NAGAMMA BIRADAR	55	FEMALE	NO		NO		NO	NO	NORMAL	110

S.No	GROUP	NAME	AGE	SEX	HTN K/C	DURATION	DM K/C	MED	SMOKING	ALCOHOL	FUNDUS	SBP 7Aam
161	CASE 81	LAXMIBAI DANGE	79	FEMALE	YES	1YEAR	YES	A	NO	NO	NORMAL	140
162	CONTROL 81	SHANTABAI JIGAJANAGI	80	FEMALE	NO		NO		NO	NO	NORMAL	110
163	CASE 82	CHAMPABAYI BADIGER	65	FEMALE	YES	3MONTHS	NO	C	NO	NO	NORMAL	120
164	CONTROL 82	TEERTHABAI BALABATTI	65	FEMALE	NO		NO		NO	NO	NORMAL	110
165	CASE 83	BHIMASHANKAR BADIGER	68	MALE	NO		NO	N	YES	YES	NORMAL	150
166	CONTROL 83	BHAGAWANT WALIKAR	68	MALE	NO		NO		NO	NO	NORMAL	120
167	CASE 84	SHANTABAI KANNUR	65	FEMALE	YES	5YEARS	NO	A+D	NO	NO	NORMAL	130
168	CONTROL 84	GADEVVA BAJANTRI	68	FEMALE	NO		NO		NO	NO	NORMAL	120

S.No	GROUP	NAME	SBP 1pm	SBP 6pm	SBP 12am	DBP 7am	DBP 1pm	DBP 6pm	DBP 12am	IOPR 7am	IOPR 1pm	IOPR 6pm	IOPR 12am
1	CASE 1	SHARADA DEELIP	130	120	120	80	70	80	70	15	10	10	8
2	CONTROL 1	MAHADEVI	120	120	110	80	70	70	80	12	13	11	12
3	CASE 2	MALLAMMA MADDIMANI	100	120	110	70	80	70	70	9	9	11	10
4	CONTROL 2	MADIVALAWWA AGASABAL	130	120	110	70	70	80	80	11	12	13	12
5	CASE 3	SAKKUBAI JADHAV	150	130	110	100	90	90	70	10	8	9	9
6	CONTROL 3	RUDRAMMA BADIGER	120	130	120	70	80	80	70	14	12	13	10
7	CASE 4	DANAPPA KORI	150	140	130	90	90	90	80	13	10	13	9
8	CONTROL 4	VIJAYENDRA KATTI	130	120	120	80	90	90	70	11	10	10	12
9	CASE 5	NINGAPPA TALWAR	200	160	130	90	110	100	80	14	12	13	13
10	CONTROL 5	LAXMAN BABLESHWAR	130	130	120	80	70	80	70	10	13	12	11
11	CASE 6	VIJAYSING LAMANI	150	180	120	110	90	90	70	16	15	12	10
12	CONTROL 6	BASAVARAJ HONALLI	110	120	120	70	70	80	70	13	11	11	10
13	CASE 7	BAPURAY SARAWAD	170	130	120	70	90	80	70	14	13	11	12
14	CONTROL 7	RACHAPPA BILAGI	110	130	110	70	80	70	80	12	11	11	12
15	CASE 8	IRASANGAPPA BINJALABAVI	160	170	140	90	90	100	90	14	11	11	12
16	CONTROL 8	MALLIKARJUN MADGYAL	110	130	120	70	80	80	80	12	10	10	9
17	CASE 9	CHANDRASHEKAR BAGALI	140	150	130	80	90	90	70	13	12	12	9
18	CONTROL 9	AB CHIRALADINNI	120	120	110	70	80	70	70	13	11	10	10
19	CASE 10	LAXMIBAI DEVARHIPPARGI	150	140	120	90	90	90	70	16	18	13	12
20	CONTROL 10	GANGAVVA TOGARI	110	110	120	70	70	80	80	13	12	10	9
21	CASE 11	LAKKAWWA AMBIGER	160	150	120	80	90	90	80	20	18	26	17
22	CONTROL 11	KAMALAVVA MANDOLI	110	110	120	80	70	80	70	12	10	8	9
23	CASE 12	MAHADEVI PUJARI	140	140	130	70	100	90	80	14	13	12	11
24	CONTROL 12	NAGAVVA BANDIWADDAR	130	120	120	70	70	80	70	13	10	10	9
25	CASE 13	LAXMIBAI MARAGUR	140	140	120	70	80	90	70	15	12	14	13
26	CONTROL 13	NILAVVA KURI	110	130	120	80	70	70	80	14	11	12	11
27	CASE 14	SARUPA CHAVAN	190	160	120	70	110	100	80	25	24	19	16
28	CONTROL 14	SARASWATI METI	110	120	120	70	80	80	80	11	12	12	11
29	CASE 15	CHANDRABAI LAMANI	170	200	170	100	90	120	100	17	15	17	14
30	CONTROL 15	NANDAMMA V	120	120	110	70	70	80	70	12	12	11	9
31	CASE 16	NINGAPPA PUJARI	150	160	130	90	100	90	80	15	14	11	12
32	CONTROL 16	DATTU KAMBALE	130	130	120	70	80	90	80	16	14	11	10

S.No	GROUP	NAME	SBP 1pm	SBP 6pm	SBP 12am	DBP 7am	DBP 1pm	DBP 6pm	DBP 12am	IOPR 7am	IOPR 1pm	IOPR 6pm	IOPR 12am
33	CASE 17	MAYAVVA HARUGERI	210	200	160	90	100	110	90	13	16	15	14
34	CONTROL 17	SUNADA BENAL	110	110	120	70	80	70	60	9	12	10	11
35	CASE 18	MALLAPPA KOLAKAR	150	140	130	70	90	90	80	14	13	13	11
36	CONTROL 18	RACHAPPA H	130	130	120	80	80	70	80	13	12	11	9
37	CASE 19	SURESH KUMATAGI	130	140	150	70	80	90	90	17	16	13	14
38	CONTROL 19	BASAPPA DAVALAGI	110	130	120	80	70	70	80	15	16	15	12
39	CASE 20	AMBAVVA KANURKAR	170	180	170	90	90	100	90	20	18	19	17
40	CONTROL 20	LAXMAWWA KAMBAGI	110	120	120	80	70	70	80	13	12	10	11
41	CASE 21	INDRAVVA DODAMANI	140	160	120	80	90	90	70	19	15	19	14
42	CONTROL 21	BHARATI CHANDNERI	110	120	120	60	70	80	60	13	11	12	13
43	CASE 22	PARVATI MUNJI	150	160	130	70	90	90	80	18	14	14	12
44	CONTROL 22	SATAWWA HONNALLI	110	110	120	70	80	70	80	14	13	11	12
45	CASE 23	BEERAPPA MANNAGOL	140	160	140	90	100	90	90	20	18	18	15
46	CONTROL 23	KALLAPPA PUJARI	110	120	110	60	60	70	70	18	14	13	12
47	CASE 24	NAGAMMA KEMBHAVI	180	170	170	80	90	80	80	16	12	10	18
48	CONTROL 24	IRAWWA MATHAPATI	130	130	120	80	70	80	80	12	14	11	14
49	CASE 25	DUNDAVVA SONAGAVI	130	160	150	70	80	80	90	16	18	15	14
50	CONTROL 25	INTAJABI MAKANDAR	120	120	110	80	80	70	70	16	14	15	14
51	CASE 26	BOURAMMA KODANGAL	140	130	130	70	90	80	90	15	14	9	11
52	CONTROL 26	SITAVVA MADAR	120	120	110	80	80	70	70	14	12	12	11
53	CASE 27	JAIBUNI FOUJI	130	140	150	70	80	90	90	19	15	16	18
54	CONTROL 27	MALLAWWA SITTIMANI	130	120	120	80	70	80	70	12	10	11	12
55	CASE 28	LAXMIBAI BIRADAR	160	170	150	80	90	90	80	16	12	11	14
56	CONTROL 28	BOURAMMA MALI	110	110	120	70	80	70	70	14	11	12	10
57	CASE 29	ANNAPOORNA AGASAR	150	140	130	80	90	90	80	17	15	15	16
58	CONTROL 29	KAMALA NAWADAGI	110	120	120	80	80	70	80	14	12	12	13
59	CASE 30	RAVATAPPA HANDI	120	140	120	70	80	90	70	19	21	22	18
60	CONTROL 30	SOMANNA PUJARI	120	130	120	80	80	70	70	13	10	11	9
61	CASE 31	BABASAB HOSAMANI	130	140	140	90	80	90	80	19	18	17	15
62	CONTROL 31	DASTAGIRSAB KORABU	120	120	120	70	70	80	80	18	14	14	15
63	CASE 32	RAMJAN MUJAWAR	130	130	120	70	80	70	80	17	18	15	13
64	CONTROL 32	SHARANAPPA PUJARI	130	130	120	70	80	70	70	19	18	18	17

S.No	GROUP	NAME	SBP 1pm	SBP 6pm	SBP 12am	DBP 7am	DBP 1pm	DBP 6pm	DBP 12am	IOPR 7am	IOPR 1pm	IOPR 6pm	IOPR 12am
65	CASE 33	SHIVAMMA KATTIMANI	130	140	140	70	80	90	80	18	14	19	16
66	CONTROL 33	RAJASHREE DAMANI	130	130	120	70	70	80	80	13	11	9	9
67	CASE 34	SAJANBI MULLA	130	140	130	70	80	80	70	13	10	9	8
68	CONTROL 34	YALLAVVA PUJARI	130	130	120	70	80	70	70	13	11	13	12
69	CASE 35	NINGAVVA TALWAR	130	130	120	70	90	90	80	13	11	15	14
70	CONTROL 35	GANGABAI BIRADAR	130	130	130	70	70	80	70	13	11	10	12
71	CASE 36	HULGAVVA MADAR	140	160	150	70	90	90	80	16	14	15	13
72	CONTROL 36	GURASHANTAVVA	130	130	120	70	70	80	80	9	10	12	10
73	CASE 37	MAMTAJ BEGAM	150	160	130	70	80	90	80	28	17	18	23
74	CONTROL 37	GUJJAWWA KODAHONNA	120	130	130	70	80	80	70	11	9	8	8
75	CASE 38	PADMAVATI NAIKODI	160	150	130	70	90	90	80	17	15	12	12
76	CONTROL 38	VALUBAI KARATI	110	110	100	80	70	70	70	12	11	16	14
77	CASE 39	SIDARAYA KOKATANUR	130	120	100	80	80	70	70	18	16	14	12
78	CONTROL 39	SHIVAPPA JAGAMSHETTY	110	120	110	60	70	70	80	13	12	10	12
79	CASE 40	RAVI PUJARI	170	150	130	70	100	90	80	18	17	16	15
80	CONTROL 40	SIDDARAM KOLI	120	120	120	70	70	80	70	10	10	9	12
81	CASE 41	BHIMBAI GENNUR	110	120	110	70	70	80	70	14	15	15	14
82	CONTROL 41	CHAMPUBAI GODEKAR	110	100	100	70	80	70	80	13	12	11	11
83	CASE 42	SHIVAVVA BARADDI	150	150	130	80	100	90	80	7	8	7	7
84	CONTROL 42	TANIBAI MASHYAL	120	130	120	80	80	70	70	12	12	10	9
85	CASE 43	GOURABAI KODAHONNA	130	150	140	80	80	90	80	9	8	9	8
86	CONTROL 43	DILSHAD DAKHANI	110	110	110	70	70	80	70	14	11	14	12
87	CASE 44	BORAMMA SAJJAN	150	140	140	70	90	90	80	16	16	15	13
88	CONTROL 44	SHEELAVATHI RUDRAPPA	120	120	120	70	80	70	70	11	11	9	9
89	CASE 45	SARUBAI LAD	120	110	110	70	80	70	70	15	14	14	14
90	CONTROL 45	KASHAWWA KAMBAR	120	120	110	70	80	70	80	11	11	14	12
91	CASE 46	FATHIMA MULLA	160	170	160	80	90	100	90	16	14	12	13
92	CONTROL 46	LAXMIBAI N	120	120	120	70	80	80	70	13	13	14	12
93	CASE 47	SHANTABAI GAYAKWAD	140	140	120	70	90	80	70	17	16	14	14
94	CONTROL 47	GOURABAI SHIVASHARAN	130	130	120	80	80	70	80	16	16	12	12
95	CASE 48	SUBHADRABAI TAJAV	140	130	120	80	100	90	80	14	12	11	11
96	CONTROL 48	NAGAVVA WAGMORE	110	110	110	70	70	80	70	12	11	11	11

S.No	GROUP	NAME	SBP 1pm	SBP 6pm	SBP 12am	DBP 7am	DBP 1pm	DBP 6pm	DBP 12am	IOPR 7am	IOPR 1pm	IOPR 6pm	IOPR 12am
97	CASE 49	SHANTA BIDARI	120	120	120	70	80	80	70	12	10	11	10
98	CONTROL 49	KASHIBAI POTADAR	110	110	100	80	70	80	70	11	12	13	11
99	CASE 50	KOUSHALABAI HAJERI	150	140	130	80	90	90	80	14	18	17	15
100	CONTROL 50	SONABAYI LAMANI	120	120	110	70	80	70	70	16	16	16	15
101	CASE 51	MODINBI KARNAL	160	140	130	70	80	90	80	16	15	13	13
102	CONTROL 51	SHANKAREMMA BADIGER	120	120	110	70	70	80	70	15	15	13	12
103	CASE 52	KADUBAYI PAWAR	110	110	110	60	70	70	70	17	14	15	12
104	CONTROL 52	KASTURIBAI SHINDE	110	110	110	70	80	80	70	14	13	12	11
105	CASE 53	SIDDAVVA KATTIMANI	150	160	140	90	90	100	90	14	11	10	11
106	CONTROL 53	NEELAMMA PATIL	130	130	120	70	80	70	60	12	11	11	11
107	CASE 54	JAITUNABEE RAMAPUR	130	140	120	80	90	90	70	14	12	13	11
108	CONTROL 54	PARAWATI HIREMATH	110	110	120	70	60	70	70	14	12	11	12
109	CASE 55	SIDDAMMA PATIL	140	150	130	80	90	90	70	16	14	13	11
110	CONTROL 55	BANGARAMMA BIRADAR	110	120	110	70	80	80	70	14	14	13	12
111	CASE 56	BHIMAPPA MADAGI	120	120	110	70	70	80	70	14	12	12	10
112	CONTROL 56	MALLAPPA BHIRAGOND	130	120	120	60	70	80	70	12	11	10	9
113	CASE 57	GOLLALAPPA PATIL	110	100	100	70	80	70	70	13	13	11	11
114	CONTROL 57	MALLAPPA TOTAD	120	120	120	70	70	80	80	12	11	10	10
115	CASE 58	LALABI BHAVIKATTI	160	150	130	90	90	90	80	14	11	11	12
116	CONTROL 58	SHANTABAI PATTAR	100	110	110	70	70	70	70	12	11	13	12
117	CASE 59	SHARANAPPA KUMBAR	140	140	130	80	100	90	80	15	11	11	12
118	CONTROL 59	HUSEENSAB PINJAR	130	130	120	70	80	70	70	15	15	14	13
119	CASE 60	BASAVARAJ DESAI	140	140	130	80	90	80	70	11	8	10	9
120	CONTROL 60	RAJASAB JAMABAGI	120	120	120	70	70	80	70	11	11	12	11
121	CASE 61	SHAMBAI RAJAPUT	200	190	170	90	100	90	90	16	14	15	14
122	CONTROL 61	SURTABAI RATHOD	110	110	110	70	80	80	70	11	12	10	10
123	CASE 62	BASANNA LALASANGI	130	130	120	70	90	80	80	12	11	10	11
124	CONTROL 62	MALLAPPA PUJARI	100	110	100	70	70	70	80	13	14	12	11
125	CASE 63	MADEVI JAMADAR	160	170	160	70	90	90	80	12	10	11	12
126	CONTROL 63	MALLAMMA BHUDHYAL	120	130	120	70	70	80	70	12	10	10	11
127	CASE 64	IRAPPA NAVI	160	160	150	80	80	90	80	16	15	13	14
128	CONTROL 64	YALLAPPA HOSAMANI	110	110	110	70	70	60	60	15	15	13	12

S.No	GROUP	NAME	SBP 1pm	SBP 6pm	SBP 12am	DBP 7am	DBP 1pm	DBP 6pm	DBP 12am	IOPR 7am	IOPR 1pm	IOPR 6pm	IOPR 12am
129	CASE 65	BUDDAMMA SOMPUR	140	130	130	80	80	80	70	14	12	12	11
130	CONTROL 65	SANGAVVA KAMBAR	120	130	120	70	80	70	80	12	11	13	11
131	CASE 66	MANAPPA VISHWAKARMA	160	160	140	80	80	90	80	13	12	12	11
132	CONTROL 66	MALLAPPA YALAWAR	130	130	120	80	80	70	80	12	13	12	14
133	CASE 67	MAHADEVI GUDAMI	150	150	140	90	90	100	80	12	10	10	9
134	CONTROL 67	BASAVVA HONAMURGI	120	120	120	70	80	70	80	13	12	14	12
135	CASE 68	PARASURAM GAJAKOSH	120	120	120	70	80	70	80	10	9	10	10
136	CONTROL 68	APPASAHEB PATIL	100	110	110	70	80	80	80	14	11	10	11
137	CASE 69	LAXMIBAI MALA	170	170	150	90	90	90	80	12	11	9	10
138	CONTROL 69	MABUBBI MANAGULI	120	120	110	80	70	80	70	11	11	12	10
139	CASE 70	IRAMMA BASUPATTAD	140	140	130	70	80	90	70	17	16	16	14
140	CONTROL 70	SHANTABAI RATHOD	120	130	120	80	80	70	70	14	12	10	12
141	CASE 71	KRISHNA BANDAGER	150	140	140	80	90	90	80	15	14	14	13
142	CONTROL 71	VISHWANATH BALAKUNDRI	110	110	110	70	70	80	70	12	11	11	11
143	CASE 72	TIPPARAY HIREKORBAR	150	140	130	80	90	90	80	11	12	13	10
144	CONTROL 72	MALLAPPA HOSAMANI	120	120	120	80	80	70	70	11	9	9	10
145	CASE 73	SUMITRA SINDAGI	170	170	160	80	80	90	90	12	12	9	10
146	CONTROL 73	SARASWATI HIREMATH	120	120	110	70	80	80	70	13	12	12	11
147	CASE 74	SONAWWA BHANDARBATTI	140	140	130	80	70	70	70	14	12	15	14
148	CONTROL 74	PRABHAVATI HIREMATH	110	120	120	60	70	70	70	12	11	12	11
149	CASE 75	MAINABHI YEDRAMI	120	120	110	70	90	80	60	10	11	10	12
150	CONTROL 75	RAJABI MAKANDAR	120	110	110	80	80	70	60	15	15	12	12
151	CASE 76	CHANDRASHEKAR JADHAV	140	140	130	80	90	90	90	12	14	16	12
152	CONTROL 76	SHANKAR LAMANI	120	120	110	70	70	80	80	13	10	11	12
153	CASE 77	SHANTAWWA DASAR	170	150	140	90	90	100	90	15	13	15	13
154	CONTROL 77	SHANUBAYI LAMANI	120	110	110	80	70	70	70	11	11	10	11
155	CASE 78	MUKTABAI NARAYANAKAR	120	110	110	70	70	60	60	14	13	11	10
156	CONTROL 78	NINGAWWA GARASANGI	120	130	130	70	70	80	70	10	8	9	9
157	CASE 79	SUNDARABAI SIRINAL	140	140	130	80	90	90	70	12	12	15	13
158	CONTROL 79	MANAMMA KORI	120	120	110	60	60	60	60	12	12	10	11
159	CASE 80	MUTTABAI RATHOD	130	140	130	70	80	90	80	11	10	9	8
160	CONTROL 80	NAGAMMA BIRADAR	120	120	110	70	70	80	70	13	11	12	10

S.No	GROUP	NAME	SBP 1pm	SBP 6pm	SBP 12am	DBP 7am	DBP 1pm	DBP 6pm	DBP 12am	IOPR 7am	IOPR 1pm	IOPR 6pm	IOPR 12am
161	CASE 81	LAXMIBAI DANGE	140	160	160	90	90	90	80	15	13	12	11
162	CONTROL 81	SHANTABAI JIGAJANAGI	110	110	110	70	80	70	70	15	14	13	12
163	CASE 82	CHAMPABAYI BADIGER	120	130	130	80	80	80	70	12	12	11	9
164	CONTROL 82	TEERTHABAI BALABATTI	110	120	120	80	80	70	70	16	15	15	13
165	CASE 83	BHIMASHANKAR BADIGER	160	160	160	80	90	90	80	10	10	10	9
166	CONTROL 83	BHAGAWANT WALIKAR	120	130	120	70	80	80	70	13	12	11	11
167	CASE 84	SHANTABAI KANNUR	140	140	140	70	90	100	70	13	11	10	10
168	CONTROL 84	GADEVVA BAJANTRI	130	130	130	70	80	70	70	9	8	10	8

S.No	GROUP	NAME	IOPL 7am	IOPL 1pm	IOPL 6pm	IOPL 12am	OPPR 7am	OPPR 1pm	OPPR 6pm	OPPR 12am	OPPL 7am	OPPL 1pm	OPPL 6pm
1	CASE 1	SHARADA DEELIP	15	8	9	12	50	50	52	50	50	52	53
2	CONTROL 1	MAHADEVI	13	11	11	10	48	45	47	48	47	47	47
3	CASE 2	MALLAMMA MADDIMANI	13	10	10	9	44	49	47	45	40	48	48
4	CONTROL 2	MADIVALAWWA AGASABAL	11	11	12	10	47	48	49	48	47	49	50
5	CASE 3	SAKKUBAI JADHAV	10	9	11	10	75	65	60	46	75	64	58
6	CONTROL 3	RUDRAMMA BADIGER	13	13	11	11	41	50	52	48	42	49	54
7	CASE 4	DANAPPA KORI	11	9	14	8	60	63	58	56	62	64	57
8	CONTROL 4	VIJAYENDRA KATTI	12	10	9	11	51	59	57	46	50	59	58
9	CASE 5	NINGAPPA TALWAR	15	13	12	13	57	81	67	52	56	80	68
10	CONTROL 5	LAXMAN BABLESHWAR	9	10	11	11	52	47	53	47	53	50	54
11	CASE 6	VIJAYSING LAMANI	18	19	13	10	71	58	68	48	69	54	67
12	CONTROL 6	BASAVARAJ HONALLI	14	11	10	10	45	44	51	48	44	44	52
13	CASE 7	BAPURAY SARAWAD	16	13	13	17	39	65	54	46	37	65	52
14	CONTROL 7	RACHAPPA BILAGI	13	11	13	10	46	49	49	48	45	49	47
15	CASE 8	IRASANGAPPA BINJALABAVI	18	9	13	17	57	64	71	59	53	66	69
16	CONTROL 8	MALLIKARJUN MADGYAL	14	11	10	10	43	50	55	53	41	49	55
17	CASE 9	CHANDRASHEKAR BAGALI	13	10	14	13	54	59	61	51	54	61	59
18	CONTROL 9	AB CHIRALADINNI	12	11	11	9	40	51	48	45	41	51	47
19	CASE 10	LAXMIBAI DEVARHIPPARGI	14	15	12	12	55	55	58	46	57	58	59
20	CONTROL 10	GANGAVVA TOGARI	15	12	9	10	40	43	50	53	38	43	51
21	CASE 11	LAKKAWWA AMBIGER	23	26	23	20	45	57	47	45	42	49	50
22	CONTROL 11	KAMALAVVA MANDOLI	12	8	7	9	50	45	52	49	50	47	53
23	CASE 12	MAHADEVI PUJARI	14	11	13	11	41	62	59	54	41	64	58
24	CONTROL 12	NAGAVVA BANDIWADDAR	12	10	12	9	45	50	52	38	46	50	50
25	CASE 13	LAXMIBAI MARAGUR	13	14	15	13	40	55	57	45	42	53	56
26	CONTROL 13	NILAVVA KURI	16	10	11	11	46	44	48	51	44	45	49
27	CASE 14	SARUPA CHAVAN	27	29	19	16	35	67	61	46	33	62	61
28	CONTROL 14	SARASWATI METI	10	10	12	11	44	48	50	61	45	50	50
29	CASE 15	CHANDRABAI LAMANI	17	13	15	14	63	63	81	68	63	65	83
30	CONTROL 15	NANDAMMA V	13	12	12	9	41	46	51	46	40	46	50
31	CASE 16	NINGAPPA PUJARI	13	12	11	13	54	64	64	53	56	66	64
32	CONTROL 16	DATTU KAMBALE	14	12	11	11	42	51	58	52	44	53	58

S.No	GROUP	NAME	IOPL 7am	IOPL 1pm	IOPL 6pm	IOPL 12am	OPPR 7am	OPPR 1pm	OPPR 6pm	OPPR 12am	OPPL 7am	OPPL 1pm	OPPL 6pm
33	CASE 17	MAYAVVA HARUGERI	12	18	14	14	62	75	78	61	63	73	79
34	CONTROL 17	SUNADA BENAL	9	11	12	11	49	41	48	42	49	42	46
35	CASE 18	MALLAPPA KOLAKAR	19	15	13	14	44	60	58	54	39	58	58
36	CONTROL 18	RACHAPPA H	15	11	11	10	49	53	49	53	47	54	49
37	CASE 19	SURESH KUMATAGI	14	12	13	13	41	49	58	59	44	53	58
38	CONTROL 19	BASAPPA DAVALAGI	14	16	13	11	45	57	45	50	46	57	47
39	CASE 20	AMBAVVA KANURKAR	17	18	14	15	55	60	66	61	58	60	71
40	CONTROL 20	LAXMAWWA KAMBAGI	13	11	11	10	47	43	48	51	47	44	47
41	CASE 21	INDRAVVA DODAMANI	18	16	15	13	41	56	56	44	42	55	60
42	CONTROL 21	BHARATI CHANDNERI	13	11	15	14	38	44	50	40	38	44	47
43	CASE 22	PARVATI MUNJI	17	14	13	15	42	59	61	41	43	59	62
44	CONTROL 22	SATAWWA HONNALLI	15	12	11	11	39	47	44	50	38	48	44
45	CASE 23	BEERAPPA MANNAGOL	19	18	17	14	51	57	57	56	52	57	58
46	CONTROL 23	KALLAPPA PUJARI	16	14	14	11	33	37	45	43	35	37	44
47	CASE 24	NAGAMMA KEMBHAVI	15	14	12	10	53	68	63	55	54	66	61
48	CONTROL 24	IRAWWA MATHAPATI	12	13	13	12	50	46	54	48	50	47	52
49	CASE 25	DUNDAVVA SONAGAVI	18	14	16	14	44	47	56	59	42	51	55
50	CONTROL 25	INTAJABI MAKANDAR	15	14	14	13	44	48	43	43	45	48	44
51	CASE 26	BOURAMMA KODANGAL	16	13	10	11	40	57	56	58	39	58	55
52	CONTROL 26	SITAVVA MADAR	14	13	12	12	46	50	46	44	46	49	46
53	CASE 27	JAIBUNI FOUJI	19	16	16	20	41	50	55	55	41	49	55
54	CONTROL 27	MALLAWWA SITTIMANI	10	10	12	12	48	50	51	46	50	50	50
55	CASE 28	LAXMIBAI BIRADAR	15	11	13	14	49	63	67	55	50	64	65
56	CONTROL 28	BOURAMMA MALI	12	12	12	9	41	49	43	48	43	48	43
57	CASE 29	ANNAPOORNA AGASAR	18	15	12	13	43	58	56	49	42	58	59
58	CONTROL 29	KAMALA NAWADAGI	14	13	11	12	46	48	46	49	46	47	47
59	CASE 30	RAVATAPPA HANDI	16	11	14	10	36	41	49	40	39	51	57
60	CONTROL 30	SOMANNA PUJARI	12	10	9	9	47	52	49	49	48	52	51
61	CASE 31	BABASAB HOSAMANI	19	17	17	15	54	47	54	52	54	48	54
62	CONTROL 31	DASTAGIRSAB KORABU	15	11	14	13	40	44	48	47	43	47	48
63	CASE 32	RAMJAN MUJAWAR	17	16	14	14	41	47	45	49	41	49	46
64	CONTROL 32	SHARANAPPA PUJARI	19	17	18	18	41	47	42	41	41	48	42

S.No	GROUP	NAME	IOPL 7am	IOPL 1pm	IOPL 6pm	IOPL 12am	OPPR 7am	OPPR 1pm	OPPR 6pm	OPPR 12am	OPPL 7am	OPPL 1pm	OPPL 6pm
65	CASE 33	SHIVAMMA KATTIMANI	19	15	18	16	42	51	52	51	41	50	53
66	CONTROL 33	RAJASHREE DAMANI	14	10	9	10	45	49	56	53	44	50	56
67	CASE 34	SAJANBI MULLA	11	9	8	8	47	55	58	52	49	56	59
68	CONTROL 34	YALLAVVA PUJARI	14	10	13	11	42	54	47	46	41	55	47
69	CASE 35	NINGAVVA TALWAR	12	11	14	14	45	58	54	48	46	58	55
70	CONTROL 35	GANGABAI BIRADAR	14	11	11	12	45	49	55	48	44	49	54
71	CASE 36	HULGAVVA MADAR	15	14	12	12	44	57	60	56	45	57	63
72	CONTROL 36	GURASHANTAVVA	9	8	11	10	49	50	53	52	49	52	54
73	CASE 37	MAMTAJ BEGAM	29	19	18	25	32	52	57	42	31	50	57
74	CONTROL 37	GUJJAWWA KODAHONNA	11	10	9	8	44	53	57	52	44	52	56
75	CASE 38	PADMAVATI NAIKODI	17	16	11	12	41	60	61	53	41	59	62
76	CONTROL 38	VALUBAI KARATI	11	11	13	14	46	44	39	39	47	44	42
77	CASE 39	SIDARAYA KOKATANUR	19	16	18	12	40	49	44	41	39	49	40
78	CONTROL 39	SHIVAPPA JAGAMSHETTY	15	12	10	13	36	43	48	48	34	43	48
79	CASE 40	RAVI PUJARI	18	15	16	14	42	65	57	50	42	67	57
80	CONTROL 40	SIDDARAM KOLI	10	8	8	10	45	48	53	46	45	50	54
81	CASE 41	BHIMBAI GENNUR	14	13	15	15	41	40	47	41	41	42	47
82	CONTROL 41	CHAMPUBAI GODEKAR	14	12	12	11	42	48	42	47	41	48	41
83	CASE 42	SHIVAVVA BARADDI	9	7	7	8	58	70	66	58	56	71	66
84	CONTROL 42	TANIBAI MASHYAL	11	12	9	9	50	50	50	49	51	50	51
85	CASE 43	GOURABAI KODAHONNA	9	9	8	8	53	57	64	59	53	56	65
86	CONTROL 43	DILSHAD DAKHANI	15	9	11	12	41	44	46	43	40	46	49
87	CASE 44	BORAMMA SAJJAN	16	15	14	14	44	57	56	54	44	58	57
88	CONTROL 44	SHEELAVATHI RUDRAPPA	11	9	8	9	47	51	49	49	47	53	50
89	CASE 45	SARUBAI LAD	14	12	15	14	40	48	41	41	41	50	40
90	CONTROL 45	KASHAWWA KAMBAR	13	11	10	12	44	51	44	48	42	51	48
91	CASE 46	FATHIMA MULLA	16	15	14	12	51	61	70	62	51	60	68
92	CONTROL 46	LAXMIBAI N	13	15	12	12	42	49	48	46	42	47	50
93	CASE 47	SHANTABAI GAYAKWAD	17	18	15	14	38	55	53	44	38	53	52
94	CONTROL 47	GOURABAI SHIVASHARAN	14	15	14	12	46	49	48	50	48	50	46
95	CASE 48	SUBHADRABAI TAJAV	13	12	12	10	48	63	58	51	49	63	57
96	CONTROL 48	NAGAVVA WAGMORE	12	12	11	10	43	44	49	44	43	43	49

S.No	GROUP	NAME	IOPL 7am	IOPL 1pm	IOPL 6pm	IOPL 12am	OPPR 7am	OPPR 1pm	OPPR 6pm	OPPR 12am	OPPL 7am	OPPL 1pm	OPPL 6pm
97	CASE 49	SHANTA BIDARI	12	11	12	10	46	52	51	48	46	51	50
98	CONTROL 49	KASHIBAI POTADAR	11	13	12	12	49	43	47	42	49	42	48
99	CASE 50	KOUSHALABAI HAJERI	14	16	16	15	48	55	54	50	48	57	55
100	CONTROL 50	SONABAYI LAMANI	16	18	15	15	39	46	42	40	39	44	43
101	CASE 51	MODINBI KARNAL	17	14	14	13	44	56	58	52	43	57	57
102	CONTROL 51	SHANKAREMMA BADIGER	15	13	12	12	40	43	49	43	40	45	50
103	CASE 52	KADUBAYI PAWAR	15	14	13	13	34	41	40	43	36	41	42
104	CONTROL 52	KASTURIBAI SHINDE	14	12	12	12	41	47	48	44	41	48	48
105	CASE 53	SIDDAVVA KATTIMANI	14	10	8	10	55	62	70	60	55	63	72
106	CONTROL 53	NEELAMMA PATIL	12	11	10	11	46	54	49	42	46	54	50
107	CASE 54	JAITUNABEE RAMAPUR	14	12	14	13	48	57	58	47	48	57	57
108	CONTROL 54	PARAWATI HIREMATH	14	14	12	12	44	39	44	46	44	37	43
109	CASE 55	SIDDAMMA PATIL	14	14	15	11	49	57	60	49	51	57	58
110	CONTROL 55	BANGARAMMA BIRADAR	14	15	12	12	41	46	49	43	41	45	50
111	CASE 56	BHIMAPPA MADAGI	12	14	12	12	41	46	50	45	43	44	50
112	CONTROL 56	MALLAPPA BHIRAGOND	13	11	9	9	41	49	52	49	40	49	53
113	CASE 57	GOLLALAPPA PATIL	13	11	12	11	40	47	42	42	40	49	41
114	CONTROL 57	MALLAPPA TOTAD	11	11	9	10	46	47	52	52	47	47	53
115	CASE 58	LALABI BHAVIKATTI	13	13	12	12	53	64	62	53	54	62	61
116	CONTROL 58	SHANTABAI PATTAR	12	12	12	12	41	42	42	43	41	41	43
117	CASE 59	SHARANAPPA KUMBAR	12	11	9	13	52	64	60	53	55	64	62
118	CONTROL 59	HUSEENSAB PINJAR	14	15	13	12	43	50	46	45	44	50	47
119	CASE 60	BASAVARAJ DESAI	12	9	10	10	54	63	57	51	53	62	57
120	CONTROL 60	RAJASAB JAMABAGI	11	12	12	11	44	47	50	47	44	46	50
121	CASE 61	SHAMBAI RAJAPUT	16	16	15	14	59	75	67	64	59	73	67
122	CONTROL 61	SURTABAI RATHOD	11	10	8	9	47	48	50	45	47	50	52
123	CASE 62	BASANNA LALASANGI	14	12	10	13	46	58	55	51	44	57	55
124	CONTROL 62	MALLAPPA PUJARI	15	12	12	11	42	39	43	49	40	41	43
125	CASE 63	MADEVI JAMADAR	13	11	11	10	55	65	67	59	54	64	67
126	CONTROL 63	MALLAMMA BHUDHYAL	11	10	9	11	43	48	55	47	44	48	56
127	CASE 64	IRAPPA NAVI	16	16	14	14	51	56	62	55	51	55	61
128	CONTROL 64	YALLAPPA HOSAMANI	15	13	12	12	40	40	38	39	40	42	39

S.No	GROUP	NAME	IOPL 7am	IOPL 1pm	IOPL 6pm	IOPL 12am	OPPR 7am	OPPR 1pm	OPPR 6pm	OPPR 12am	OPPL 7am	OPPL 1pm	OPPL 6pm
129	CASE 65	BUDDAMMA SOMPUR	13	10	12	10	48	55	53	49	49	57	53
130	CONTROL 65	SANGAVVA KAMBAR	12	12	12	11	46	51	47	51	46	50	48
131	CASE 66	MANAPPA VISHWAKARMA	12	10	12	10	52	57	63	56	53	59	63
132	CONTROL 66	MALLAPPA YALAWAR	12	14	12	14	50	52	48	48	50	51	48
133	CASE 67	MAHADEVI GUDAMI	11	10	8	9	57	63	68	58	58	63	70
134	CONTROL 67	BASAVVA HONAMURGI	13	11	11	12	42	50	44	50	42	51	47
135	CASE 68	PARASURAM GAJAKOSH	11	9	12	10	45	53	48	52	44	53	46
136	CONTROL 68	APPASAHEB PATIL	12	12	10	10	39	47	50	49	41	46	50
137	CASE 69	LAXMIBAI MALA	11	11	10	10	61	67	69	59	62	67	68
138	CONTROL 69	MABUBBI MANAGULI	11	10	12	10	49	47	50	45	49	48	50
139	CASE 70	IRAMMA BASUPATTAD	17	14	15	16	41	51	55	46	41	53	56
140	CONTROL 70	SHANTABAI RATHOD	14	14	13	12	48	50	50	46	48	48	47
141	CASE 71	KRISHNA BANDAGER	15	12	14	14	50	59	57	54	50	61	57
142	CONTROL 71	VISHWANATH BALAKUNDRI	13	11	12	10	43	44	49	44	42	44	48
143	CASE 72	TIPPARAY HIREKORBAR	10	12	12	10	51	61	58	55	52	61	59
144	CONTROL 72	MALLAPPA HOSAMANI	12	12	10	10	49	53	49	48	48	50	48
145	CASE 73	SUMITRA SINDAGI	13	12	13	11	57	61	69	65	56	61	65
146	CONTROL 73	SARASWATI HIREMATH	13	10	10	11	45	50	50	44	45	52	52
147	CASE 74	SONAWWA BHANDARBATTI	15	10	15	14	51	50	47	46	50	52	47
148	CONTROL 74	PRABHAVATI HIREMATH	11	11	12	10	39	44	46	47	40	44	46
149	CASE 75	MAINABHI YEDRAMI	13	11	9	11	45	56	52	39	42	56	53
150	CONTROL 75	RAJABI MAKANDAR	16	15	13	12	45	47	43	39	44	47	42
151	CASE 76	CHANDRASHEKAR JADHAV	13	14	13	12	52	57	55	56	51	57	58
152	CONTROL 76	SHANKAR LAMANI	12	10	10	12	42	48	51	48	43	48	52
153	CASE 77	SHANTAWWA DASAR	15	14	13	12	56	65	63	58	56	64	65
154	CONTROL 77	SHANUBAYI LAMANI	13	11	10	10	49	47	43	42	47	47	43
155	CASE 78	MUKTABAI NARAYANAKAR	15	14	11	13	41	45	40	41	40	44	40
156	CONTROL 78	NINGAWWA GARASANGI	8	9	8	9	48	50	56	51	50	49	57
157	CASE 79	SUNDARABAI SIRINAL	13	14	14	13	53	59	56	47	52	57	57
158	CONTROL 79	MANAMMA KORI	8	10	8	11	41	41	43	40	45	43	45
159	CASE 80	MUTTABAI RATHOD	12	10	8	8	49	55	62	57	48	55	63
160	CONTROL 80	NAGAMMA BIRADAR	14	10	11	10	42	47	50	45	41	48	51

S.No	GROUP	NAME	IOPL 7am	IOPL 1pm	IOPL 6pm	IOPL 12am	OPPR 7am	OPPR 1pm	OPPR 6pm	OPPR 12am	OPPL 7am	OPPL 1pm	OPPL 6pm
161	CASE 81	LAXMIBAI DANGE	14	13	14	11	56	58	63	60	57	58	61
162	CONTROL 81	SHANTABAI JIGAJANAGI	14	13	13	11	40	46	42	43	41	47	42
163	CASE 82	CHAMPABAYI BADIGER	13	12	12	9	50	50	54	51	49	50	53
164	CONTROL 82	TEERTHABAI BALABATTI	13	12	12	13	44	45	43	45	47	48	46
165	CASE 83	BHIMASHANKAR BADIGER	11	10	8	9	59	65	65	62	58	65	67
166	CONTROL 83	BHAGAWANT WALIKAR	13	11	13	11	45	50	54	47	45	51	52
167	CASE 84	SHANTABAI KANNUR	13	13	12	11	47	60	65	52	47	58	63
168	CONTROL 84	GADEVVA BAJANTRI	9	10	8	9	49	57	50	52	49	55	52

S.No	GROUP	NAME	OPPL 12am	MAP 7am	MAP 1pm	MAP 6pm	MAP 12am	SPPR 7am	SPPR 1pm	SPPR 6pm	SPPR 12am	SPPL 7am	SPPL 1pm
1	CASE 1	SHARADA DEELIP	46	97	90	93	87	115	120	110	112	115	122
2	CONTROL 1	MAHADEVI	50	90	87	87	90	98	107	109	98	97	109
3	CASE 2	MALLAMMA MADDIMANI	46	80	87	87	83	91	91	109	100	87	90
4	CONTROL 2	MADIVALAWWA AGASABAL	50	87	90	93	90	109	118	117	98	109	119
5	CASE 3	SAKKUBAI JADHAV	45	127	110	103	83	170	142	121	101	170	141
6	CONTROL 3	RUDRAMMA BADIGER	47	83	93	97	87	96	108	117	110	97	107
7	CASE 4	DANAPPA KORI	57	110	110	107	97	137	140	127	121	139	141
8	CONTROL 4	VIJAYENDRA KATTI	47	93	103	100	87	109	120	110	108	108	120
9	CASE 5	NINGAPPA TALWAR	52	107	140	120	97	126	188	147	117	125	187
10	CONTROL 5	LAXMAN BABLESHWAR	47	93	90	97	87	110	117	118	109	111	120
11	CASE 6	VIJAYSING LAMANI	48	130	110	120	87	154	135	168	110	152	131
12	CONTROL 6	BASAVARAJ HONALLI	48	87	83	93	87	107	99	109	110	106	99
13	CASE 7	BAPURAY SARAWAD	41	80	117	97	87	86	157	119	108	84	157
14	CONTROL 7	RACHAPPA BILAGI	50	87	90	90	90	108	99	119	98	107	99
15	CASE 8	IRASANGAPPA BINJALABAVI	54	107	113	123	107	126	149	159	128	122	151
16	CONTROL 8	MALLIKARJUN MADGYAL	52	83	90	97	93	98	100	120	111	96	99
17	CASE 9	CHANDRASHEKAR BAGALI	47	100	107	110	90	127	128	138	121	127	130
18	CONTROL 9	AB CHIRALADINNI	46	80	93	87	83	87	109	110	100	88	109
19	CASE 10	LAXMIBAI DEVARHIPPARGI	46	107	110	107	87	124	132	127	108	126	135
20	CONTROL 10	GANGAVVA TOGARI	52	80	83	90	93	87	98	100	111	85	98
21	CASE 11	LAKKAWWA AMBIGER	42	97	113	110	93	110	142	124	103	107	134
22	CONTROL 11	KAMALAVVA MANDOLI	49	93	83	90	87	108	100	102	111	108	102
23	CASE 12	MAHADEVI PUJARI	54	83	113	107	97	96	127	128	119	96	129
24	CONTROL 12	NAGAVVA BANDIWADDAR	38	87	90	93	87	107	120	110	111	108	120
25	CASE 13	LAXMIBAI MARAGUR	45	83	100	107	87	95	128	126	107	97	126
26	CONTROL 13	NILAVVA KURI	52	90	83	90	93	96	99	118	109	94	100
27	CASE 14	SARUPA CHAVAN	46	90	137	120	93	105	166	141	104	103	161
28	CONTROL 14	SARASWATI METI	61	83	90	93	93	99	98	108	109	100	100
29	CASE 15	CHANDRABAI LAMANI	68	120	117	147	123	143	155	183	156	143	157
30	CONTROL 15	NANDAMMA V	46	80	87	93	83	86	108	109	101	87	108
31	CASE 16	NINGAPPA PUJARI	52	103	117	113	97	115	136	149	118	117	138
32	CONTROL 16	DATTU KAMBALE	51	87	97	103	93	104	116	119	110	106	118

S.No	GROUP	NAME	OPPL 12am	MAP 7am	MAP 1pm	MAP 6pm	MAP 12am	SPPR 7am	SPPR 1pm	SPPR 6pm	SPPR 12am	SPPL 7am	SPPL 1pm
33	CASE 17	MAYAVVA HARUGERI	61	113	137	140	113	147	194	185	146	148	192
34	CONTROL 17	SUNADA BENAL	42	87	90	83	80	111	109	100	109	111	99
35	CASE 18	MALLAPPA KOLAKAR	51	87	110	107	97	106	137	127	119	101	135
36	CONTROL 18	RACHAPPA H	52	93	97	90	93	107	118	118	111	105	119
37	CASE 19	SURESH KUMATAGI	60	87	97	107	110	103	114	127	136	106	118
38	CONTROL 19	BASAPPA DAVALAGI	51	90	83	90	93	95	94	115	108	96	94
39	CASE 20	AMBAVVA KANURKAR	63	113	117	127	117	140	152	161	153	143	152
40	CONTROL 20	LAXMAWWA KAMBAGI	52	90	83	87	93	97	98	110	109	97	109
41	CASE 21	INDRAVVA DODAMANI	45	90	107	113	87	91	125	141	106	92	124
42	CONTROL 21	BHARATI CHANDNERI	39	77	83	93	80	97	99	108	107	97	99
43	CASE 22	PARVATI MUNJI	38	90	110	113	97	112	136	146	118	113	136
44	CONTROL 22	SATAWWA HONNALLI	51	80	90	83	93	86	97	99	108	85	98
45	CASE 23	BEERAPPA MANNAGOL	57	107	113	113	107	120	122	142	125	121	122
46	CONTROL 23	KALLAPPA PUJARI	44	77	77	87	83	92	96	107	98	94	96
47	CASE 24	NAGAMMA KEMBHAVI	63	103	120	110	110	134	168	160	152	135	166
48	CONTROL 24	IRAWWA MATHAPATI	50	93	90	97	93	108	116	119	106	102	117
49	CASE 25	DUNDAVVA SONAGAVI	59	90	97	107	110	114	112	145	136	112	116
50	CONTROL 25	INTAJABI MAKANDAR	44	90	93	87	83	94	106	105	96	95	106
51	CASE 26	BOURAMMA KODANGAL	58	83	107	97	103	95	126	121	119	94	127
52	CONTROL 26	SITAVVA MADAR	43	90	93	87	83	96	108	108	99	96	107
53	CASE 27	JAIBUNI FOUJI	53	90	97	107	110	111	115	124	132	111	114
54	CONTROL 27	MALLAWWA SITTIMANI	46	90	90	93	87	98	120	109	108	100	120
55	CASE 28	LAXMIBAI BIRADAR	55	97	113	117	103	114	148	159	136	115	149
56	CONTROL 28	BOURAMMA MALI	49	83	90	83	87	96	99	98	110	98	98
57	CASE 29	ANNAPOORNA AGASAR	52	90	110	107	97	93	135	125	114	92	135
58	CONTROL 29	KAMALA NAWADAGI	50	90	90	87	93	96	98	108	107	96	97
59	CASE 30	RAVATAPPA HANDI	48	83	93	107	87	91	99	118	102	94	109
60	CONTROL 30	SOMANNA PUJARI	49	90	93	90	87	97	110	119	111	98	110
61	CASE 31	BABASAB HOSAMANI	52	110	97	107	100	131	112	123	125	131	113
62	CONTROL 31	DASTAGIRSAB KORABU	49	87	87	93	93	102	106	106	105	105	109
63	CASE 32	RAMJAN MUJAWAR	48	87	97	90	93	103	112	115	107	103	114
64	CONTROL 32	SHARANAPPA PUJARI	40	90	97	90	87	111	112	112	103	111	113

S.No	GROUP	NAME	OPPL 12am	MAP 7am	MAP 1pm	MAP 6pm	MAP 12am	SPPR 7am	SPPR 1pm	SPPR 6pm	SPPR 12am	SPPL 7am	SPPL 1pm
65	CASE 33	SHIVAMMA KATTIMANI	51	90	97	107	100	112	116	121	124	111	115
66	CONTROL 33	RAJASHREE DAMANI	52	87	90	97	93	107	119	121	111	106	120
67	CASE 34	SAJANBI MULLA	52	90	97	100	90	117	120	131	122	119	121
68	CONTROL 34	YALLAVVA PUJARI	47	83	97	90	87	97	119	117	108	96	120
69	CASE 35	NINGAVVA TALWAR	48	87	103	103	93	107	119	115	106	108	119
70	CONTROL 35	GANGABAI BIRADAR	48	87	90	97	90	107	119	120	118	106	119
71	CASE 36	HULGAVVA MADAR	57	90	107	113	103	114	126	145	137	115	126
72	CONTROL 36	GURASHANTAVVA	52	87	90	97	93	111	120	118	110	111	122
73	CASE 37	MAMTAJ BEGAM	40	90	103	113	97	102	133	142	107	101	131
74	CONTROL 37	GUJJAWWA KODAHONNA	52	83	93	97	90	99	111	122	122	99	110
75	CASE 38	PADMAVATI NAIKODI	53	87	113	110	97	103	145	138	118	103	144
76	CONTROL 38	VALUBAI KARATI	39	87	83	83	80	88	99	94	86	83	99
77	CASE 39	SIDARAYA KOKATANUR	41	87	97	87	80	82	114	106	88	81	114
78	CONTROL 39	SHIVAPPA JAGAMSHETTY	47	73	83	87	90	87	98	110	98	85	98
79	CASE 40	RAVI PUJARI	51	90	123	110	97	112	153	134	115	112	155
80	CONTROL 40	SIDDARAM KOLI	48	83	87	93	87	100	110	111	108	100	112
81	CASE 41	BHIMBAI GENNUR	40	83	83	93	83	96	95	105	96	96	97
82	CONTROL 41	CHAMPUBAI GODEKAR	47	83	90	80	87	97	98	89	89	96	98
83	CASE 42	SHIVAVVA BARADDI	57	97	117	110	97	123	142	143	123	121	143
84	CONTROL 42	TANIBAI MASHYAL	49	93	93	90	87	108	108	120	111	109	108
85	CASE 43	GOURABAI KODAHONNA	59	93	97	110	100	111	122	141	132	111	121
86	CONTROL 43	DILSHAD DAKHANI	43	83	83	90	83	96	99	96	98	95	101
87	CASE 44	BORAMMA SAJJAN	53	90	110	107	100	114	134	125	127	114	135
88	CONTROL 44	SHEELAVATHI RUDRAPPA	49	87	93	87	87	109	109	111	111	109	111
89	CASE 45	SARUBAI LAD	41	83	93	83	83	95	106	96	96	96	108
90	CONTROL 45	KASHAWWA KAMBAR	48	83	93	87	90	99	109	106	98	97	109
91	CASE 46	FATHIMA MULLA	63	100	113	123	113	124	146	158	147	124	145
92	CONTROL 46	LAXMIBAI N	46	83	93	93	87	97	107	106	108	97	105
93	CASE 47	SHANTABAI GAYAKWAD	44	83	107	100	87	93	124	126	106	93	122
94	CONTROL 47	GOURABAI SHIVASHARAN	50	93	97	90	93	104	114	118	108	106	115
95	CASE 48	SUBHADRABAI TAJAV	52	93	113	103	93	106	128	119	109	107	128
96	CONTROL 48	NAGAVVA WAGMORE	45	83	83	90	83	98	99	99	99	98	98

S.No	GROUP	NAME	OPPL 12am	MAP 7am	MAP 1pm	MAP 6pm	MAP 12am	SPPR 7am	SPPR 1pm	SPPR 6pm	SPPR 12am	SPPL 7am	SPPL 1pm
97	CASE 49	SHANTA BIDARI	48	87	93	93	87	108	110	109	110	108	109
98	CONTROL 49	KASHIBAI POTADAR	41	90	83	90	80	99	98	97	89	99	97
99	CASE 50	KOUSHALABAI HAJERI	50	93	110	107	97	106	132	123	115	106	134
100	CONTROL 50	SONABAYI LAMANI	40	83	93	87	83	94	104	104	95	94	102
101	CASE 51	MODINBI KARNAL	52	90	107	107	97	114	145	127	117	113	146
102	CONTROL 51	SHANKAREMMA BADIGER	43	83	87	93	83	95	105	107	98	95	107
103	CASE 52	KADUBAYI PAWAR	42	77	83	83	83	93	96	95	98	95	96
104	CONTROL 52	KASTURIBAI SHINDE	43	83	90	90	83	96	97	98	99	96	98
105	CASE 53	SIDDAVVA KATTIMANI	61	103	110	120	107	116	139	150	129	116	140
106	CONTROL 53	NEELAMMA PATIL	42	87	97	90	80	108	119	119	109	108	119
107	CASE 54	JAITUNABEE RAMAPUR	45	93	103	107	87	106	118	127	109	106	118
108	CONTROL 54	PARAWATI HIREMATH	46	87	77	83	87	106	98	99	108	106	96
109	CASE 55	SIDDAMMA PATIL	49	97	107	110	90	114	126	137	119	116	126
110	CONTROL 55	BANGARAMMA BIRADAR	43	83	90	93	83	96	96	107	98	96	95
111	CASE 56	BHIMAPPA MADAGI	43	83	87	93	83	96	108	108	100	98	106
112	CONTROL 56	MALLAPPA BHIRAGOND	49	80	90	93	87	108	119	110	111	107	119
113	CASE 57	GOLLALAPPA PATIL	42	80	90	80	80	87	97	89	89	87	99
114	CONTROL 57	MALLAPPA TOTAD	52	87	87	93	93	108	109	110	110	109	109
115	CASE 58	LALABI BHAVIKATTI	53	100	113	110	97	106	149	139	118	107	147
116	CONTROL 58	SHANTABAI PATTAR	43	80	80	83	83	88	89	97	98	88	88
117	CASE 59	SHARANAPPA KUMBAR	52	100	113	107	97	125	129	129	118	128	129
118	CONTROL 59	HUSEENSAB PINJAR	46	87	97	90	87	105	115	116	107	106	115
119	CASE 60	BASAVARAJ DESAI	50	97	107	100	90	119	132	130	121	118	131
120	CONTROL 60	RAJASAB JAMABAGI	47	83	87	93	87	99	109	108	109	99	108
121	CASE 61	SHAMBAI RAJAPUT	64	113	133	123	117	144	186	175	156	144	184
122	CONTROL 61	SURTABAI RATHOD	46	87	90	90	83	109	98	100	100	109	100
123	CASE 62	BASANNA LALASANGI	49	87	103	97	93	108	119	120	109	106	118
124	CONTROL 62	MALLAPPA PUJARI	49	83	80	83	90	97	86	98	89	95	88
125	CASE 63	MADEVI JAMADAR	61	100	113	117	107	148	150	159	148	147	149
126	CONTROL 63	MALLAMMA BHUDHYAL	47	83	87	97	87	98	110	120	109	99	110
127	CASE 64	IRAPPA NAVI	55	100	107	113	103	124	145	147	136	124	144
128	CONTROL 64	YALLAPPA HOSAMANI	39	83	83	77	77	95	95	97	98	95	97

S.No	GROUP	NAME	OPPL 12am	MAP 7am	MAP 1pm	MAP 6pm	MAP 12am	SPPR 7am	SPPR 1pm	SPPR 6pm	SPPR 12am	SPPL 7am	SPPL 1pm
129	CASE 65	BUDDAMMA SOMPUR	50	93	100	97	90	106	128	118	119	107	130
130	CONTROL 65	SANGAVVA KAMBAR	51	87	93	90	93	108	109	117	109	108	108
131	CASE 66	MANAPPA VISHWAKARMA	57	97	107	113	100	117	148	148	129	118	150
132	CONTROL 66	MALLAPPA YALAWAR	48	93	97	90	93	108	117	118	106	108	116
133	CASE 67	MAHADEVI GUDAMI	58	103	110	117	100	118	140	140	131	119	140
134	CONTROL 67	BASAVVA HONAMURGI	50	83	93	87	93	97	108	106	108	97	109
135	CASE 68	PARASURAM GAJAKOSH	52	83	93	87	93	100	111	110	110	99	111
136	CONTROL 68	APPASAHEB PATIL	50	80	87	90	90	86	89	100	99	88	88
137	CASE 69	LAXMIBAI MALA	59	110	117	117	103	138	159	161	140	139	159
138	CONTROL 69	MABUBBI MANAGULI	45	90	87	93	83	99	109	108	100	99	110
139	CASE 70	IRAMMA BASUPATTAD	44	87	100	107	90	103	124	124	116	103	126
140	CONTROL 70	SHANTABAI RATHOD	46	93	93	90	87	106	108	120	108	106	106
141	CASE 71	KRISHNA BANDAGER	53	97	110	107	100	115	136	126	127	115	138
142	CONTROL 71	VISHWANATH BALAKUNDRI	45	83	83	90	83	98	99	99	99	97	99
143	CASE 72	TIPPARAY HIREKORBAR	55	93	110	107	97	109	138	127	120	110	138
144	CONTROL 72	MALLAPPA HOSAMANI	48	90	93	87	87	99	111	111	110	98	108
145	CASE 73	SUMITRA SINDAGI	64	103	110	117	113	138	158	161	150	137	158
146	CONTROL 73	SARASWATI HIREMATH	44	87	93	93	83	107	108	108	99	107	110
147	CASE 74	SONAWWA BHANDARBATTI	46	97	93	93	90	116	128	125	116	115	130
148	CONTROL 74	PRABHAVATI HIREMATH	48	77	83	87	87	98	99	108	109	99	99
149	CASE 75	MAINABHI YEDRAMI	40	83	100	93	77	100	109	110	98	97	109
150	CONTROL 75	RAJABI MAKANDAR	39	90	93	83	77	95	105	98	98	94	105
151	CASE 76	CHANDRASHEKAR JADHAV	56	97	107	107	103	118	126	124	118	117	126
152	CONTROL 76	SHANKAR LAMANI	48	83	87	93	90	97	110	109	98	98	110
153	CASE 77	SHANTAWWA DASAR	59	107	117	117	107	125	157	135	127	125	156
154	CONTROL 77	SHANUBAYI LAMANI	43	90	87	80	80	99	109	100	99	97	109
155	CASE 78	MUKTABAI NARAYANAKAR	38	83	87	77	77	96	107	99	100	95	106
156	CONTROL 78	NINGAWWA GARASANGI	51	87	87	97	90	110	112	121	121	112	111
157	CASE 79	SUNDARABAI SIRINAL	47	97	107	107	90	118	128	125	117	117	126
158	CONTROL 79	MANAMMA KORI	40	80	80	80	77	108	108	110	99	112	110
159	CASE 80	MUTTABAI RATHOD	57	90	97	107	97	119	121	131	122	118	122
160	CONTROL 80	NAGAMMA BIRADAR	45	83	87	93	83	97	109	108	100	96	110

S.No	GROUP	NAME	OPPL 12am	MAP 7am	MAP 1pm	MAP 6pm	MAP 12am	SPPR 7am	SPPR 1pm	SPPR 6pm	SPPR 12am	SPPL 7am	SPPL 1pm
161	CASE 81	LAXMIBAI DANGE	60	107	107	113	107	125	127	148	149	126	127
162	CONTROL 81	SHANTABAI JIGAJANAGI	44	83	90	83	83	95	96	97	98	96	97
163	CASE 82	CHAMPABAYI BADIGER	51	93	93	97	90	108	108	119	121	107	108
164	CONTROL 82	TEERTHABAI BALABATTI	45	90	90	87	87	94	95	105	107	97	98
165	CASE 83	BHIMASHANKAR BADIGER	62	103	113	113	107	140	150	150	151	139	150
166	CONTROL 83	BHAGAWANT WALIKAR	47	87	93	97	87	107	108	119	109	107	109
167	CASE 84	SHANTABAI KANNUR	51	90	107	113	93	117	129	130	130	117	127
168	CONTROL 84	GADEVVA BAJANTRI	51	87	97	90	90	111	122	120	122	111	120

S.No	GROUP	NAME	SPPL 6pm	SPPL 12am	DPPR 7am	DPPR 1pm	DPPR 6pm	DPPR 12am	DPPL 7am	DPPL 1pm	DPPL 6pm	DPPL 12am
1	CASE 1	SHARADA DEELIP	111	108	65	60	70	62	65	62	71	58
2	CONTROL 1	MAHADEVI	109	100	68	57	59	68	67	59	59	70
3	CASE 2	MALLAMMA MADDIMANI	110	101	81	71	59	60	57	70	60	61
4	CONTROL 2	MADIVALAWWA AGASABAL	118	100	59	58	67	68	59	59	68	70
5	CASE 3	SAKKUBAI JADHAV	119	100	90	82	81	61	90	81	79	60
6	CONTROL 3	RUDRAMMA BADIGER	119	109	56	68	67	60	57	67	69	59
7	CASE 4	DANAPPA KORI	126	122	77	80	77	71	79	81	76	72
8	CONTROL 4	VIJAYENDRA KATTI	111	109	69	80	80	58	68	80	81	59
9	CASE 5	NINGAPPA TALWAR	148	117	76	98	87	67	75	97	88	67
10	CONTROL 5	LAXMAN BABLESHWAR	119	109	70	57	68	59	71	60	69	59
11	CASE 6	VIJAYSING LAMANI	167	110	94	75	78	60	92	71	77	60
12	CONTROL 6	BASAVARAJ HONALLI	110	110	57	59	69	60	56	59	70	60
13	CASE 7	BAPURAY SARAWAD	117	103	56	77	69	58	54	77	67	53
14	CONTROL 7	RACHAPPA BILAGI	117	100	58	69	59	68	57	69	57	70
15	CASE 8	IRASANGAPPA BINJALABAVI	157	123	76	79	89	78	72	81	87	73
16	CONTROL 8	MALLIKARJUN MADGYAL	120	110	58	70	70	71	56	69	70	70
17	CASE 9	CHANDRASHEKAR BAGALI	136	117	67	78	78	61	67	80	76	57
18	CONTROL 9	AB CHIRALADINNI	109	101	57	69	60	60	58	69	59	61
19	CASE 10	LAXMIBAI DEVARHIPPARGI	128	108	74	72	77	58	76	75	78	58
20	CONTROL 10	GANGAVVA TOGARI	101	110	57	58	70	71	55	58	71	70
21	CASE 11	LAKKAWWA AMBIGER	127	100	60	72	64	63	57	64	67	60
22	CONTROL 11	KAMALAVVA MANDOLI	103	111	68	60	72	61	68	62	73	61
23	CASE 12	MAHADEVI PUJARI	127	119	56	87	78	69	56	89	77	69
24	CONTROL 12	NAGAVVA BANDIWADDAR	108	111	57	60	70	61	58	60	68	61
25	CASE 13	LAXMIBAI MARAGUR	125	107	55	68	76	57	57	66	75	57
26	CONTROL 13	NILAVVA KURI	119	109	66	59	58	69	64	60	59	69
27	CASE 14	SARUPA CHAVAN	141	104	45	86	81	64	43	81	81	64
28	CONTROL 14	SARASWATI METI	108	109	59	68	68	69	60	70	68	69
29	CASE 15	CHANDRABAI LAMANI	185	156	83	75	103	86	83	77	105	86
30	CONTROL 15	NANDAMMA V	108	101	56	58	69	61	57	58	68	61
31	CASE 16	NINGAPPA PUJARI	149	117	75	86	79	68	77	88	79	67
32	CONTROL 16	DATTU KAMBALE	119	109	54	66	79	70	56	68	79	69

S.No	GROUP	NAME	SPPL 6pm	SPPL 12am	DPPR 7am	DPPR 1pm	DPPR 6pm	DPPR 12am	DPPL 7am	DPPL 1pm	DPPL 6pm	DPPL 12am
33	CASE 17	MAYAVVA HARUGERI	186	146	77	84	95	76	78	82	96	76
34	CONTROL 17	SUNADA BENAL	98	109	61	68	60	49	61	69	58	49
35	CASE 18	MALLAPPA KOLAKAR	127	116	56	77	77	69	51	75	77	66
36	CONTROL 18	RACHAPPA H	119	110	67	68	59	71	65	69	59	70
37	CASE 19	SURESH KUMATAGI	127	137	53	64	77	76	56	68	77	77
38	CONTROL 19	BASAPPA DAVALAGI	117	109	65	54	55	65	66	54	57	69
39	CASE 20	AMBAVVA KANURKAR	166	155	70	72	81	73	73	73	86	75
40	CONTROL 20	LAXMAWWA KAMBAGI	109	110	67	58	60	59	67	59	59	60
41	CASE 21	INDRAVVA DODAMANI	145	107	61	75	71	56	62	74	75	57
42	CONTROL 21	BHARATI CHANDNERI	105	106	47	59	68	47	47	59	65	46
43	CASE 22	PARVATI MUNJI	147	115	52	76	76	68	53	76	77	65
44	CONTROL 22	SATAWWA HONNALLI	99	109	56	67	59	68	55	68	59	69
45	CASE 23	BEERAPPA MANNAGOL	143	126	70	82	72	75	71	82	73	76
46	CONTROL 23	KALLAPPA PUJARI	108	99	42	46	57	58	44	46	56	59
47	CASE 24	NAGAMMA KEMBHAVI	158	160	64	78	70	62	65	76	68	70
48	CONTROL 24	IRAWWA MATHAPATI	117	108	68	56	69	66	68	57	67	68
49	CASE 25	DUNDAVVA SONAGAVI	144	136	74	79	92	96	72	83	91	96
50	CONTROL 25	INTAJABI MAKANDAR	106	97	64	66	55	56	65	66	56	57
51	CASE 26	BOURAMMA KODANGAL	120	119	55	76	71	79	54	77	70	79
52	CONTROL 26	SITAVVA MADAR	108	98	66	68	58	59	66	67	58	58
53	CASE 27	JAIBUNI FOUJI	124	130	51	65	74	72	51	64	74	70
54	CONTROL 27	MALLAWWA SITTIMANI	108	108	68	60	69	58	70	60	68	58
55	CASE 28	LAXMIBAI BIRADAR	157	136	64	78	79	66	65	79	77	66
56	CONTROL 28	BOURAMMA MALI	98	111	56	69	58	60	58	68	58	61
57	CASE 29	ANNAPOORNA AGASAR	128	117	63	75	75	64	62	75	78	67
58	CONTROL 29	KAMALA NAWADAGI	109	108	66	68	58	67	66	67	59	68
59	CASE 30	RAVATAPPA HANDI	126	110	51	59	68	52	54	69	76	60
60	CONTROL 30	SOMANNA PUJARI	121	111	67	70	59	61	68	70	61	61
61	CASE 31	BABASAB HOSAMANI	123	125	71	62	73	65	71	63	73	65
62	CONTROL 31	DASTAGIRSAB KORABU	106	107	52	56	66	65	55	59	66	67
63	CASE 32	RAMJAN MUJAWAR	116	106	53	62	55	67	53	64	56	66
64	CONTROL 32	SHARANAPPA PUJARI	112	102	51	62	52	53	51	63	52	52

S.No	GROUP	NAME	SPPL 6pm	SPPL 12am	DPPR 7am	DPPR 1pm	DPPR 6pm	DPPR 12am	DPPL 7am	DPPL 1pm	DPPL 6pm	DPPL 12am
65	CASE 33	SHIVAMMA KATTIMANI	122	124	52	66	71	64	51	65	72	64
66	CONTROL 33	RAJASHREE DAMANI	121	110	57	59	71	71	56	60	71	70
67	CASE 34	SAJANBI MULLA	132	122	57	70	71	62	59	71	72	62
68	CONTROL 34	YALLAVVA PUJARI	117	109	57	69	57	58	56	70	57	59
69	CASE 35	NINGAVVA TALWAR	116	106	57	79	75	66	58	79	76	66
70	CONTROL 35	GANGABAI BIRADAR	119	118	57	59	70	58	56	59	69	58
71	CASE 36	HULGAVVA MADAR	148	138	54	76	75	67	55	76	78	68
72	CONTROL 36	GURASHANTAVVA	119	110	61	60	68	70	61	62	69	70
73	CASE 37	MAMTAJ BEGAM	142	105	42	63	72	57	41	61	72	55
74	CONTROL 37	GUJJAWWA KODAHONNA	121	122	59	71	72	62	59	70	71	62
75	CASE 38	PADMAVATI NAIKODI	139	118	53	75	78	68	53	74	79	68
76	CONTROL 38	VALUBAI KARATI	97	86	68	59	54	56	69	59	57	56
77	CASE 39	SIDARAYA KOKATANUR	102	88	62	64	56	58	61	64	52	58
78	CONTROL 39	SHIVAPPA JAGAMSHETTY	110	97	47	58	60	68	45	58	60	67
79	CASE 40	RAVI PUJARI	134	116	52	83	74	65	52	85	74	66
80	CONTROL 40	SIDDARAM KOLI	112	110	73	77	84	75	73	79	85	77
81	CASE 41	BHIMBAI GENNUR	105	95	56	55	65	56	56	57	65	55
82	CONTROL 41	CHAMPUBAI GODEKAR	88	89	57	68	59	69	56	68	58	69
83	CASE 42	SHIVAVVA BARADDI	143	122	73	92	83	73	71	93	83	72
84	CONTROL 42	TANIBAI MASHYAL	121	111	68	68	60	61	69	68	61	61
85	CASE 43	GOURABAI KODAHONNA	142	132	71	72	81	72	71	71	82	72
86	CONTROL 43	DILSHAD DAKHANI	99	98	56	59	66	58	55	61	69	58
87	CASE 44	BORAMMA SAJJAN	126	126	54	74	75	67	54	75	76	66
88	CONTROL 44	SHEELAVATHI RUDRAPPA	112	111	59	69	61	61	59	71	62	61
89	CASE 45	SARUBAI LAD	95	96	55	66	56	56	56	68	55	56
90	CONTROL 45	KASHAWWA KAMBAR	110	98	59	69	56	68	57	69	60	68
91	CASE 46	FATHIMA MULLA	156	148	64	76	88	77	64	75	86	78
92	CONTROL 46	LAXMIBAI N	108	108	57	67	66	58	57	65	68	58
93	CASE 47	SHANTABAI GAYAKWAD	125	106	53	74	66	56	53	72	65	56
94	CONTROL 47	GOURABAI SHIVASHARAN	116	108	64	64	58	68	66	65	56	68
95	CASE 48	SUBHADRABAI TAJAV	118	110	66	88	79	69	67	88	78	70
96	CONTROL 48	NAGAVVA WAGMORE	99	100	58	59	69	59	58	58	69	60

S.No	GROUP	NAME	SPPL 6pm	SPPL 12am	DPPR 7am	DPPR 1pm	DPPR 6pm	DPPR 12am	DPPL 7am	DPPL 1pm	DPPL 6pm	DPPL 12am
97	CASE 49	SHANTA BIDARI	108	110	58	70	69	60	58	69	68	60
98	CONTROL 49	KASHIBAI POTADAR	98	88	69	58	67	58	69	57	68	59
99	CASE 50	KOUSHALABAI HAJERI	124	115	66	72	73	65	66	74	74	65
100	CONTROL 50	SONABAYI LAMANI	105	95	54	64	54	55	54	62	55	55
101	CASE 51	MODINBI KARNAL	126	117	54	65	77	67	53	66	76	67
102	CONTROL 51	SHANKAREMMA BADIGER	108	98	55	55	67	58	55	57	68	58
103	CASE 52	KADUBAYI PAWAR	97	97	43	56	55	58	45	56	57	57
104	CONTROL 52	KASTURIBAI SHINDE	98	98	56	67	68	59	56	68	68	58
105	CASE 53	SIDDAVVA KATTIMANI	152	130	76	79	90	79	76	80	92	80
106	CONTROL 53	NEELAMMA PATIL	120	109	58	69	59	49	58	69	60	49
107	CASE 54	JAITUNABEE RAMAPUR	126	107	66	78	77	59	66	78	76	57
108	CONTROL 54	PARAWATI HIREMATH	98	108	56	48	59	58	56	46	58	58
109	CASE 55	SIDDAMMA PATIL	135	119	64	76	77	59	66	76	75	59
110	CONTROL 55	BANGARAMMA BIRADAR	108	98	56	66	67	58	56	65	68	58
111	CASE 56	BHIMAPPA MADAGI	108	98	56	58	68	60	58	56	68	58
112	CONTROL 56	MALLAPPA BHIRAGOND	111	111	48	59	70	61	47	59	71	61
113	CASE 57	GOLLALAPPA PATIL	88	89	57	67	59	59	57	69	58	59
114	CONTROL 57	MALLAPPA TOTAD	111	110	58	59	70	70	59	59	71	70
115	CASE 58	LALABI BHAVIKATTI	138	118	76	79	79	68	77	77	78	68
116	CONTROL 58	SHANTABAI PATTAR	98	98	58	59	57	58	58	58	58	58
117	CASE 59	SHARANAPPA KUMBAR	131	117	65	89	79	68	68	89	81	67
118	CONTROL 59	HUSEENSAB PINJAR	117	108	55	65	56	57	56	65	57	58
119	CASE 60	BASAVARAJ DESAI	130	120	69	82	70	61	68	81	70	60
120	CONTROL 60	RAJASAB JAMABAGI	108	109	59	59	68	59	59	58	68	59
121	CASE 61	SHAMBAI RAJAPUT	175	156	74	86	75	76	74	84	75	76
122	CONTROL 61	SURTABAI RATHOD	102	101	59	68	70	60	59	70	72	61
123	CASE 62	BASANNA LALASANGI	120	107	58	79	70	69	56	78	70	67
124	CONTROL 62	MALLAPPA PUJARI	98	89	57	56	58	69	55	58	58	69
125	CASE 63	MADEVI JAMADAR	159	148	58	80	79	68	57	79	79	70
126	CONTROL 63	MALLAMMA BHUDHYAL	121	109	58	60	70	59	59	60	71	59
127	CASE 64	IRAPPA NAVI	146	136	64	65	77	66	64	64	76	66
128	CONTROL 64	YALLAPPA HOSAMANI	98	98	55	55	47	48	55	57	48	48

S.No	GROUP	NAME	SPPL 6pm	SPPL 12am	DPPR 7am	DPPR 1pm	DPPR 6pm	DPPR 12am	DPPL 7am	DPPL 1pm	DPPL 6pm	DPPL 12am
129	CASE 65	BUDDAMMA SOMPUR	118	120	66	68	68	59	67	70	68	60
130	CONTROL 65	SANGAVVA KAMBAR	118	109	58	69	57	82	58	68	58	82
131	CASE 66	MANAPPA VISHWAKARMA	148	130	67	68	78	69	68	70	78	70
132	CONTROL 66	MALLAPPA YALAWAR	118	106	68	67	58	66	68	66	58	66
133	CASE 67	MAHADEVI GUDAMI	142	131	78	80	90	71	79	80	92	71
134	CONTROL 67	BASAVVA HONAMURGI	109	108	57	68	56	68	57	69	59	68
135	CASE 68	PARASURAM GAJAKOSH	108	110	60	71	60	70	59	71	58	70
136	CONTROL 68	APPASAHEB PATIL	100	100	56	69	70	69	58	68	70	70
137	CASE 69	LAXMIBAI MALA	160	140	78	79	81	70	79	79	80	70
138	CONTROL 69	MABUBBI MANAGULI	108	100	69	59	68	60	69	60	68	60
139	CASE 70	IRAMMA BASUPATTAD	125	114	53	64	74	56	53	66	75	54
140	CONTROL 70	SHANTABAI RATHOD	117	108	66	68	60	58	66	66	57	58
141	CASE 71	KRISHNA BANDAGER	126	126	65	76	76	67	65	78	76	66
142	CONTROL 71	VISHWANATH BALAKUNDRI	98	100	58	59	69	59	57	59	68	60
143	CASE 72	TIPPARAY HIREKORBAR	128	120	69	78	77	70	70	78	78	70
144	CONTROL 72	MALLAPPA HOSAMANI	110	110	69	71	61	60	68	68	60	60
145	CASE 73	SUMITRA SINDAGI	157	149	68	68	81	80	67	68	77	79
146	CONTROL 73	SARASWATI HIREMATH	110	99	57	68	68	59	57	70	70	59
147	CASE 74	SONAWWA BHANDARBATTI	125	116	66	58	55	56	65	60	55	56
148	CONTROL 74	PRABHAVATI HIREMATH	108	110	48	59	58	59	49	59	58	60
149	CASE 75	MAINABHI YEDRAMI	111	99	60	79	70	48	57	79	71	49
150	CONTROL 75	RAJABI MAKANDAR	97	98	65	65	58	48	64	65	57	48
151	CASE 76	CHANDRASHEKAR JADHAV	127	118	68	76	74	78	67	76	77	78
152	CONTROL 76	SHANKAR LAMANI	110	98	70	77	82	78	71	77	83	78
153	CASE 77	SHANTAWWA DASAR	137	128	75	77	85	77	75	76	87	78
154	CONTROL 77	SHANUBAYI LAMANI	100	100	69	59	60	60	67	59	60	59
155	CASE 78	MUKTABAI NARAYANAKAR	99	97	56	57	49	50	55	56	49	47
156	CONTROL 78	NINGAWWA GARASANGI	122	121	60	62	71	61	62	61	72	61
157	CASE 79	SUNDARABAI SIRINAL	126	117	68	78	75	57	67	76	76	57
158	CONTROL 79	MANAMMA KORI	112	99	48	48	50	49	52	50	52	49
159	CASE 80	MUTTABAI RATHOD	132	122	59	70	81	72	58	70	82	72
160	CONTROL 80	NAGAMMA BIRADAR	109	100	57	59	68	60	56	60	69	60

S.No	GROUP	NAME	SPPL 6pm	SPPL 12am	DPPR 7am	DPPR 1pm	DPPR 6pm	DPPR 12am	DPPL 7am	DPPL 1pm	DPPL 6pm	DPPL 12am
161	CASE 81	LAXMIBAI DANGE	146	149	75	77	78	69	76	77	76	69
162	CONTROL 81	SHANTABAI JIGAJANAGI	97	99	55	66	57	58	56	67	57	59
163	CASE 82	CHAMPABAYI BADIGER	118	121	68	68	69	61	67	68	68	61
164	CONTROL 82	TEERTHABAI BALABATTI	108	107	64	65	55	57	67	68	58	57
165	CASE 83	BHIMASHANKAR BADIGER	152	151	70	80	80	71	69	80	82	71
166	CONTROL 83	BHAGAWANT WALIKAR	117	109	57	68	69	59	57	69	67	59
167	CASE 84	SHANTABAI KANNUR	128	129	57	79	90	60	57	77	88	59
168	CONTROL 84	GADEVVA BAJANTRI	122	121	61	72	60	62	61	70	62	61

Appendix VI

Plagiarism Report

8% Overall Similarity

The combined total of all matches, including overlapping sources, for each database.

Filtered from the Report

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Match Groups

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