"A CASE – CONTROL STUDY OF RETINAL VENOUS OCCLUSION IN RESPECT TO PREVALENCE OF COMMON RISK FACTORS AT KLES DR PRABHAKAR KORE HOSPITAL & MEDICAL RESEARCH CENTRE, BELAGAVI"

Submitted by:

Dr ARCHIT SUDHIR GUPTA

REG NO: BK0119004

Dissertation

Submitted to the KLE Academy of Higher Education and Research, Belagavi, Karnataka
In Partial Fulfilment
of the Requirements for the Degree of

MASTER OF SURGERY
IN
OPHTHALMOLOGY

Under the Guidance of

Dr ARVIND LAXMAN TENAGI MBBS, MS, FGO

Professor

DEPARTMENT OF OPHTHALMOLOGY,

JAWAHARLAL NEHRU MEDICAL COLLEGE,

BELAGAVI, KARNATAKA.

APRIL - 2022

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH, BELAGAVI, KARNATAKA

Declaration by the Candidate

I, hereby declare that this dissertation entitled **"A CASE – CONTROL STUDY OF RETINAL VENOUS OCCLUSION IN RESPECT TO PREVALENCE OF COMMON RISK FACTORS AT KLES DR PRABHAKAR KORE HOSPITAL & MEDICAL RESEARCH CENTRE, BELAGAVI"** is a bonafide and genuine research work carried out by me under the guidance of **Dr ARVIND LAXMAN TENAGI** MBBS, MS, FGO Professor, Department of Ophthalmology, Jawaharlal Nehru Medical College, Belagavi and co – guided by **Dr VIVEK B WANI** MBBS, MS, FRCSED Assistant Professor, Department of Ophthalmology, Jawaharlal Nehru Medical College; and Consultant, Vitreo – Retinal Department of Ophthalmology, KLES Dr Prabhakar Kore Hospital & Medical Research Centre, Belagavi.

Date:

Place:

Dr ARCHIT SUDHIR GUPTA

(REG. NO. BK0119004)

Department of Ophthalmology,
Jawaharlal Nehru Medical College,
Belagavi – 590010.

Certificate by the Guide

This is to certify that the dissertation entitled **"A CASE – CONTROL STUDY OF RETINAL VENOUS OCCLUSION IN RESPECT TO PREVALENCE OF COMMON RISK FACTORS AT KLES DR PRABHAKAR KORE HOSPITAL & MEDICAL RESEARCH CENTRE, BELAGAVI"** is a bonafide research work done by **Dr ARCHIT SUDHIR GUPTA** in partial fulfilment of the requirements for the degree of **Master of Surgery in Ophthalmology.**

Date:

Place:

Dr ARVIND LAXMAN TENAGI MBBS, MS, FGO
Professor,
Department of Ophthalmology,
Jawaharlal Nehru Medical College,
Belagavi – 590010.

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH, BELAGAVI, KARNATAKA

Certificate by the Co – Guide

This is to certify that the dissertation entitled **"A CASE – CONTROL STUDY OF RETINAL VENOUS OCCLUSION IN RESPECT TO PREVALENCE OF COMMON RISK FACTORS AT KLES DR PRABHAKAR KORE HOSPITAL & MEDICAL RESEARCH CENTRE, BELAGAVI"** is a bonafide research work done by **Dr ARCHIT SUDHIR GUPTA** in partial fulfilment of the requirements for the degree of **Master of Surgery in Ophthalmology.**

Date:

Place:

Dr VIVEK B WANI MBBS, MS, FRCSED
Assistant Professor,
Department of Ophthalmology,
Jawaharlal Nehru Medical College;
Consultant, Vitreo – Retinal Department,
KLES Dr PKH & MRC,
Belagavi – 590010.

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH, BELAGAVI, KARNATAKA

Endorsement by the Head of the Department / Principal / Head of the Institution

This is to certify that the dissertation entitled **"A CASE – CONTROL STUDY OF RETINAL VENOUS OCCLUSION IN RESPECT TO PREVALENCE OF COMMON RISK FACTORS AT KLES DR PRABHAKAR KORE HOSPITAL & MEDICAL RESEARCH CENTRE, BELAGAVI"** is a bonafide research work done by **Dr ARCHIT SUDHIR GUPTA** under the guidance of **Dr ARVIND LAXMAN TENAGI** MBBS, MS, FGO Professor, Department of Ophthalmology, Jawaharlal Nehru Medical College, Belagavi and Co – guided by **Dr VIVEK B WANI** MBBS, MS, FRCSED Assistant Professor, Department of Ophthalmology, Jawaharlal Nehru Medical College; and Consultant, Vitreo – Retinal Department of Ophthalmology, KLES Dr Prabhakar Kore Hospital & Medical Research Centre, Belagavi.

Seal & Signature of the HOD

Dr Shivanand C Bubanale MS, FGO

Professor and Head,

Department of Ophthalmology,

Jawaharlal Nehru Medical College,

Belagavi – 590010, Karnataka, India.

Date:

Place: Belagavi

Seal & Signature of the Principal

Dr (Mrs) N. S. Mahantashetti MD (Paeds)

Principal,

Jawaharlal Nehru Medical College,

Belagavi – 590010, Karnataka, India.

Date:

Place: Belagavi

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH, BELAGAVI, KARNATAKA

Copyright

Declaration by the Candidate

I hereby declare that the KLE Academy of Higher Education and Research, Belagavi, Karnataka, shall have the right to preserve, use and disseminate this dissertation in print or electronic format for academic / research purposes.

Date:

Place:

Dr ARCHIT SUDHIR GUPTA

(REG. NO. BK0119004)

Department of Ophthalmology,

Jawaharlal Nehru Medical College,

Belagavi – 590010.

PLAGIARISM ACCEPTED LETTER

JAWAHARLAL NEHRU MEDICAL COLLEGE

(Recognized by Medical Council of India, New Delhi)

Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)

Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

0831 - 2471350 0831 - 2470759 www.jnmc.edu principal@jnmc.edu

Ref No: MDC/PG/ Date: 10-12-2021.

ACCEPTANCE LETTER

The softcopy of thesis entitled: **"A CASE – CONTROL STUDY OF RETINAL VENOUS OCCLUSION IN RESPECT TO PREVALENCE OF COMMON RISK FACTORS AT KLES DR. PRABHAKAR KORE HOSPITAL & MEDICAL RESEARCH CENTRE, BELAGAVI "** has been submitted for Anti-Plagiarism check through Turnitin software. The scan has been carried out and the scanned output reveals a match percentage of **05%** which is within the acceptable limits of **10%** as per the guidelines given by UGC.

Guide.

KAHER BELAGAVI J.N. MEDICAL COLLEGE

Dr. (Mrs.) N.S. Mahantashetti.
Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BK0119004.
Postgraduate Student,
2019-20 Batch,
Department of Ophthalmology,
J. N. Medical College, Belagavi.

ACKNOWLEDGEMENT

It is with utmost respect and pleasure; I take this auspicious opportunity to express my heartfelt and humble gratitude to one and all.

I convey my heartfelt gratitude to my beloved teacher, mentor, and guide ***Dr ARVIND LAXMAN TENAGI*** ***MBBS, MS, FGO*** *Professor, Department of Ophthalmology, Jawaharlal Nehru Medical College, Nehru Nagar, Belagavi, for his relentless guidance, ideas, inspirations, encouragement, and his reassuring presence. His enthusiasm, integral view on research, and his mission to provide me with high – quality work has had a deep impact on me. He has been the constant guiding star for me and with utmost patience and care, he has very joyfully shared this journey of research conduction and dissertation completion with me. Without his support and patience, this endeavour would not have been possible at all.*

It was indeed a great opportunity to work under my co - guide ***Dr VIVEK B WANI*** ***MBBS, MS, FRCSED*** *Assistant Professor, Department of Ophthalmology, Jawaharlal Nehru Medical College; and Consultant, Vitreo – Retinal Department of Ophthalmology, KLES Dr Prabhakar Kore Hospital & Medical Research Centre, Belagavi, who despite his hectic schedule guided me throughout my journey. His vision, enthusiasm, and unique approach has also had an integral impression on me. He was a major resource and inspiration for getting my scientific questions answered and was instrumental in helping me complete the dissertation. I am forever grateful to him for his patience and total dedication.*

I would like to make a special mention to my beloved teacher and local guardian, ***Dr NAGBHUSHAN SHREYANS CHOUGULE*** ***MS (Ophthal)*** *Assistant*

Professor, Department of Ophthalmology, Jawaharlal Nehru Medical College, Belagavi, without whom I would have been an aimless pupil and my Ophthalmology residency would have been a lost cause. I thank him a lot for accepting me as his disciple, mentee and making me ready in exploring the paths of Ophthalmology and life beyond.

My heartfelt gratitude and appreciation towards my dear teacher ***Dr SHIVANAND C BUBANALE*** ***MS (Ophthal)*** *Professor and Head, Department of Ophthalmology, Jawaharlal Nehru Medical College, Belagavi, who believed in me and has been a constant source of support for me with his encouragement and provision of facilities which were needed for my study.*

I owe an immense debt of gratitude to all my Professors in the Department of Ophthalmology, ***Dr SHIVANAND B PATIL, Dr ARVIND Y YAKKUNDI, Dr MAHESH I MAGDUM, Dr REKHA R MUDHOL, Dr UMESH HARAKUNI, Dr SMITHA PRABHU, Dr BHAGYAJYOTHI KURBET, Dr CHETHANA WARAD, Dr PRAGYA PORWAL, Dr SHARVANI PAI BASTAWADI, Dr ROHINI D K, Dr FARHEEN MANIYAR, Dr APOORVA DESHPANDE JOSHI****, Jawaharlal Nehru Medical College, Belagavi, for their invaluable suggestions and support throughout the study, my residency tenure and otherwise.*

My sincere thanks to ***Dr (Mrs) N S MAHANTASHETTI*** ***MD (Paeds)*** *Principal, Jawaharlal Nehru Medical College, Belagavi, for her support and permission to undertake this study.*

I thank the Department of Ophthalmology, Hospital, and Department Staff, namely ***MAHANTESH DEYANNAVAR, JABEEN DONKAR, SADASHIV***

KAMBLE, PUNDLIK PATIL, *and* ***BALABAI PATIL****, and all the assistants, technicians collectively for their support in the selection and collection of the data.*

*No number of words can measure up to the deepest sense of gratitude and thankfulness that I owe to my Parents (****Dr SUDHIR BHAGCHAND GUPTA*** *and* ***Dr ALKA SUDHIR GUPTA****) and my little sister (****Dr ESHA SUDHIR GUPTA****) whose cherished blessings, countless sacrifices, and their hard work day in and day out; for my future have been behind all the success that I have achieved in my life today.*

I would also like to thank ***Dr PRAMOD S BHENDE****, Director and Chief Vitreo – Retinal Surgeon, Shri Bhagwan Mahavir Vitreo – Retinal Services and Medical Research Foundation, Sankara Nethralaya, Chennai, for his unwavering support, patient listening, and constant guidance despite his very hectic schedule and time – table clashes throughout my study and residency tenure.*

I would like to offer a special thanks to my fellow hostel friends for their insightful comments and suggestions, who supported me at every twist and turn in my journey, and without whom it would have practically been an impossible task to accomplish.

I would also like to express my deepest sense of gratitude, to all my other post – graduate colleagues in the Department and my friends who helped me in my study. I would like to thank my contemporaries, seniors, and juniors for their kind and valuable advice, support, and immense help and patience throughout my tenure in JNMC, KLE, BELAGAVI.

I would like to thank my trustworthy fellow contemporaries for their invaluable support and I am deeply thankful to all the people especially ***Dr JAYARAJ R MHETRI*** *who was instrumental in the indexing, statistical analysis, formatting, and completion of my dissertation; and* ***Mr ARUN HULLOLI*** *who helped me in the printing, and binding of my dissertation. And as always, it is impossible to mention everybody who has had an impact on this work; however, there are always those whose moral support is even more important. This acknowledgment would be incomplete if I fail to thank all the patients who took part and give their valuable time to be a part of my study.*

And last but not least I would thank GOD for showering the choicest blessings on me.

Date: ***Dr Archit Sudhir Gupta***

Place: ***Reg. No. BK0119004***

LIST OF ABBREVIATIONS

CRVO	-	Central retinal venous occlusion
BRVO	-	Branch retinal venous occlusion
HRVO	-	Hemi retinal venous occlusion
ST BRVO	-	Supero – temporal branch retinal venous occlusion
IT BRVO	-	Infero – temporal branch retinal venous occlusion
HTN	-	Hypertension
DM	-	Diabetes mellitus (Type 2)
HLD	-	Hyper – lipidaemia
PAD	-	Peripheral artery disease
DVT	-	Deep venous thrombosis
CVA	-	Cerebro – vascular accident
CAD	-	Coronary artery disease
OCT	-	Optical coherence tomography
FFA	-	Fundus fluorescein angiography
CVOS	-	Central vein occlusion study
BVOS	-	Branch vein occlusion study
RAPD	-	Relative afferent pupillary defect
PRP	-	Pan – retinal photo – coagulation
CME	-	Cystoid macular oedema
VEGF	-	Vascular endothelial growth factor
NVG	-	Neo – vascular glaucoma

ABSTRACT

Retinal venous occlusion is one of the important retinal vascular diseases that are sight – threatening, it is also one of the most common causes of loss of vision. The possible causes are an athero – sclerotic artery compressing the vein and intra – luminal thrombosis. It is usually seen in elderly patients. Retinal venous occlusions are classified as CRVO, HRVO, and BRVO. Retinal venous occlusion approximately affects about 13.4 million adult world population, 3.5 million affected by CRVO, and 12.9 million involved by BRVO. Multiple studies have shown the incidence of RVO to be about 1.6 % – 3.0 % in the elderly population, BRVO incidence is about 1.8 % and CRVO incidence is 0.2 %. The BRVO prevalence is about 0.5 % to 2 % and CRVO prevalence is 0.1 % to 0.2 %. The occurrence of occlusion is usually unilateral in both the cases of BRVO and CRVO. The assessment of risk factors associated with RVO is important, as it gives the health providers the area on which they can work and improve the management of RVO. Therefore, this assessment was done to know the risk factors associated with the cases when compared with the controls, who were age and sex – matched.

The present study aimed to study the prevalence of systemic risk factors, present in cases presenting with CRVO or BRVO or HRVO, and compare them with age and sex – matched controls. The study design of the present study was a One Year Case – Control; prospective, comparative, non – interventional observation study, where 31 cases and 31 controls were compared.

In the present study majority of the patients belonged to the age group of 61 – 70, i.e., 19, 61.4 %, further 9 patients i.e., 29 % of the study subjects belonged to the age group of 51 – 60 years and 9.6 % of the patients belonged to the age group of 40 – 50 years. 58 % i.e., 18 patients in each group were males, and 42 %, i.e., 13 patients in

each group were females. 42 % i.e., 13 patients' left eyes were affected and 58 %, i.e., 18 patients' right eyes were affected in cases. In controls, 48 % i.e., 15 patients' left eyes were affected, right eyes were affected in 16 patients i.e., 52 % of the patients. In our study, no significance was noted in the laterality of the affected eye. There was no significant age and sex – wise distribution noted. overall, the cases had more co – morbidities, 23 (74 %) of the study participants in cases were hypertensive, followed by 22 (71 %) patients who were obese, followed by 21 (61.2 %) of patients who were diabetics. 6 (19.3 %) of the study subjects of cases were having glaucoma, 16 (51.6 %) of the study subjects in cases were having hyper – lipidaemia, 4 (13 %) of the study subjects were having a history of heart disease. The controls had a lower number of co – morbidities where 11 (35.4 %) of the participants were hypertensive, 12 (38.7 %) had diabetes, 10 (32.2 %) had obesity, only 2 (7%) of the patients in the control group had a history of heart disease. No patient in the control group had glaucoma, 6 (19.3 %) of the patients had hyper – lipidaemia.

In the present study, 19 (61.3 %) of the study subjects in cases were smokers and 12 (38.7 %) were non – smokers, whereas in the control group only 8 (25.8 %) study subjects were smokers and 23 (74.2 %) were non – smokers. The association was statistically significant with chi – square value of 7.939 and p – value of 0.002. Ischemic CRVO was seen in 5 (16.5 %) of the cases, non – ischemic CRVO was seen in 4 (13.5 %) of the cases, superior HRVO was seen in only 1 (3 %) of the cases, inferior HRVO was seen in 4 (13.5 %) of the patients. ST BRVO was seen in maximum number with 8 (26.2 %) patients, IT BRVO was seen in 5 (16.5 %) patients and MT BRVO was seen in 4 (10.8 %) of the cases. The mean systolic blood pressure in the cases was 138.48 ± 14.51, whereas the mean systolic blood pressure in the controls was 122.47 ± 10.66, this mean difference was statistically significant with a t

– value of 4.95 and p – value of < 0.001. The mean diastolic blood pressure in the cases was 89.44 ± 10.54, whereas the mean diastolic blood pressure in the controls was 78.94 ± 11.45, this mean difference was statistically significant with a t – value of 3.77 and p – value of 0.003. The mean HbA1c in the cases was 7.95 ± 1.22, whereas the mean HbA1c value in the controls was 6.14 ± 0.69, this mean difference was statistically significant with a t – value of 7.74 and p – value of 0.001. The mean serum creatinine levels in the cases were 1.02 ± 0.24, whereas the mean creatinine levels in the controls were 6.14 ± 0.69, this difference in the mean was statistically not significant with a t – value of 1.38 and p – value of 0.170. The mean blood urea in the cases was 18.45 ± 3.45, whereas the mean blood urea in the controls was 16.74 ± 4.58, this mean difference was statistically not significant with a t – value of 1.66 and p – value of 0. 102. The mean BMI in the cases was 28.45 ± 4.66, whereas the mean BMI in the controls was 22.48 ± 3.14, this mean difference was statistically significant with a t – value of 5.91 and p – value of 0.0001.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF FIGURES

INTRODUCTION

Retinal venous occlusion is one of the important retinal vascular diseases that is sight threatening[1], and it is one of the most common causes of loss of vision. The possible causes are an athero – sclerotic artery compressing the vein[2] and intra – luminal thrombosis[3]. It is usually seen in elderly patients. Retinal venous occlusions are classified as CRVO, HRVO, and BRVO.

CRVO or Central retinal venous occlusion refers to the occlusion of the central retinal vein. Occlusion at the superior or inferior trunk involving either half of the retina is known as HRVO or Hemi retinal venous occlusion. BRVO or Branch retinal venous occlusion is when occlusion occurs at the level of any distal branches[4].

EPIDEMIOLOGY

Retinal venous occlusion approximately affects about 13.4 million adult world population, 3.5 million affected by CRVO, and 12.9 million involved by BRVO[5]. Multiple studies have shown the incidence of RVO to be about 1.6 % – 3.0 % in the elderly population, BRVO incidence is about 1.8 % and CRVOs incidence is 0.2 %[6]. The BRVO prevalence is about 0.5 % to 2 % and CRVO prevalence is 0.1 % to 0.2 %[6]. The occurrence of occlusion is usually unilateral in both the cases of BRVO and CRVO.

RVO is more commonly seen in the elderly and almost equally prevalent in males and females.

The elderly aged more than 70 years are three times more likely to develop RVO than the younger population. Ischemic CRVO is associated with poor visual acuity than

non – ischemic CRVO. Macular oedema is present in 30 % of non – ischemic CRVO, and 73 % of ischemic CRVO[3].

It is a known fact that some of the common vascular disorders namely, HTN, arterio – sclerosis, and DM are being associated with RVO. Findings from scientific studies help guide decisions regarding the management of patients with RVO and in evaluating the efficacy and risk of new treatments in the context of the natural history of the disease.

The assessment of risk factors associated with RVO is important, as it gives the health providers the area on which they can work and improve the management of RVO. Therefore, this assessment was done to know the risk factors associated with the cases when compared with the controls, who were age and sex – matched. The results from this study would help in establishing the association of risk factors with the RVO. Further, the health care providers can screen the high – risk patients and possibly take measures in the early stages of the disease or prevent the occurrence of RVO and thereby bring down the morbidity due to RVOs.

AIMS AND OBJECTIVES

To study the prevalence of systemic risk factors, present in cases presenting with RVO (CRVO or BRVO or HRVO) and compare them with age and sex – matched controls.

REVIEW OF LITERATURE

The present study aims to study the prevalence of systemic risk factors present in cases presenting with CRVO or HRVO or BRVO and compare them with age and sex – matched controls. Retinal venous occlusion is associated with multiple risk factors. Systemic vascular diseases are commonly associated which include HTN, diabetes vasculitis, athero – sclerosis, etc. Multiple studies done worldwide have shown an increased risk of RVO with systemic diseases.

Based on the site of occlusion of the vein, the retinal venous occlusion is dived into 6 types. CRVO is more frequently occurring, clinically BRVO is also more common and relevant. Few studies have shown homo – cysteine and plasminogen activator inhibitor type 1 playing a significant role in pathogenesis. Treatment of CRVO primarily consists of managing the underlying systemic disease and treating complications due to CRVO like neo – vascularization and macular oedema[7].

In the study by Mirko D C et al,117 patients were evaluated, with cardio – genic abnormalities and thrombophilic abnormalities, 61 were male, 56 were female, mean age was 51 ±13 years. In this study, 62 had CRVO, 48 had BRVO, and 7 had both. HTN was significantly more in cases when compared to controls, DM was in 17.9 % cases and 7.9 % in cases. BRVO was more in DM, HTN, obese patients. The study concluded that in BRVO, cardio – vascular factors played a major role, especially in older patients[8].

S S Hayreh et al, the atrial HTN prevalence in BRVO was significantly more than CRVO ($p < 0.001$) and HRVO ($p = 0.028$). BRVO had a significantly higher prevalence of peripheral vascular disease with a p – value of < 0.001, venous disease

(p – value of < 0.002), BRVO was significantly associated with cardio – vascular risk factors and cerebro – vascular diseases (p = 0.001)[9].

M L Shahsuvaryan et al, found that there is a higher risk of CRVO in patients with higher and un – controlled HTN, older patients had greater odds. As the age increased, the risk of CRVO also increased. CRVO risk increases with DM, kidney disease, and glaucoma. The authors concluded that the risk relation is present between CRVO and systemic illnesses such as HTN, DM, etc[10].

According to the study by Tony Yuan – Ting Chen et al in the United States, a total of 110 patients in the younger group (age 40 and under) and 795 patients in the older group (age 41 and above) were identified to have primary admission diagnosis of CRVO. In the older CRVO group, the most commonly associated systemic diseases included HTN (83.6 %), hyper – lipidaemia (29.8 %), DM (19.8 %), tobacco use (19.2 %), glaucoma (12.4 %), and obesity (11.1 %). On the other hand, most common systemic conditions associated with CRVO in younger patients included systemic venous thrombosis (14.3 %), DM (14.3 %), tobacco use (14.0 %), HTN (13.9 %), hyper – coagulable states (13.6 %), glaucoma (9.6 %), and rheumatoid arthritis / collagen vascular diseases (9.6 %). Among the risk factors studied, systemic venous thrombosis, hyper – coagulable states, rheumatoid arthritis / collagen vascular disease, migraine, pseudo – tumour cerebri, and retinal vasculitis were significantly more common in younger patients compared to older adults[11].

Shrestha R K et al studied the association between systemic illness and RVOs, a total of 100 patients were studied, 64 had BRVO and 34 had CRVO. HTN and DM were found in 84 % of the cases. 54 % was Isolated HTN and 8 % was DM. BRVO occurred twice as many times as CRVO[12].

In a study by Bum Joo Choo et al, 417 CRVO patients were included and 1,511 patients had BRVO. HTN was most commonly found in this study, with 17.3 % in CRVO and in BRVO it was 15.2 %; this was followed by DM. CRVO had a higher prevalence of DM and chronic kidney disease than the BRVO group, this was statistically significant with a p – value of 0.004, there was no difference in prevalence of other co – morbid diseases that were studied[13].

Suthasinee Sinawat et al studied systemic ab – normalities associated with RVO in young patients, the authors found that the most common ab – normalities associated with RVO were seen in 55.1 % of the patients. HTN was seen in 27.55 %, DM in 16.33 % and 5.1 % of dys – lipidaemia was seen. Although only 5.1 % of thrombophilic disorders were seen. Oral contraceptives were used by some patients[14].

A Korean study by Jin Y B et al found that the BRVO group was associated more with HTN and heart disease than CRVO. CRVO group had higher levels of CRP, VDRL, PTT. In CRVO, HTN was higher in older patients, these findings were statistically significant[15].

In another study by Robert D Sperduto et al, the results showed that increased risk of HRVO was significantly associated with HTN and DM. There was an increased risk of CRVO with a history of HTN and DM, risk of CRVO was reduced by physical activity and BMI and systemic HTN were associated with BRVO. Glaucoma history was associated with CRVO, BRVO, and HRVO[16].

According to a study done in National Eye Institute, Bethesda, increased risk of CRVO was associated with glaucoma, DM, HTN, risk of CRVO decreased with physical activity, the risk of occlusion decreased with post – menopausal oestrogens.

Other factors associated were Electro – cardiogram ab – normalities and history of HTN, hyper – lipidaemia, increased alfa – globulin level. Systemic HTN was associated with ischemic and non – Ischemic CRVO[17].

DIABETES MELLITUS

According to a study by J G Santigo et al, a total of 19,648 patients (13,571 diabetics; 6,077 non – diabetics) were considered, out of which, the prevalence of CRVO in DM was 72 (0.5 %), non-diabetics was 27 (0.4 %); disc neo – vascularization and pan – retinal photo – coagulation were significant in DM. The authors concluded that CRVO was more severe in type 1 DM which substantially increased the risk of neo – vascularization. Many other studies had similar findings[18].

In a meta – analysis by Yun wang et al, a total of 148,654 cases and controls 23,768,820 were studied. 37 study data were pooled and the results showed significant associations between DM and RVO with an odds ratio of 1.68, the study concluded that DM was a very important risk factor for RVO[19].

In a study by Karolina Kazimierz which was done in the CRVO and BRVO group, out of 23 patients' HTN was noted in 7 (58.3 %) and 9 (81.8 %) patients, respectively. Hyper – lipidaemia was observed in 5 (21.7 %), 2 (16.7 %), and 3 (27.3 %) subjects in the overall RVO group, CRVO group, and BRVO group, respectively. 2 (8.7 %) patients reported a past stroke incident. Neither was diagnosed with DM, and one of them was treated on account of HTN. Only 1 (4.3 %) participant from the CRVO group reported smoking. Out of all the patients, glaucoma was detected in 5 (21.7 %) eyes, 4 (33.3 %) of which were in the CRVO group and only 1 (9.1 %) of which was in the BRVO group. Regarding the patients' metabolic outcomes, the mean

fasting glucose level in the group with DM and the group without DM was 116 (range: 79 – 240 mg / dL) and 91.6 (range: 77 – 101) mg / dL), respectively. The mean Hb1Ac was 6.1 % (range: 5.4 – 9.9) in the group with DM and 5.2 % (range: 4.8 – 6.0) in the group without DM. The mean total cholesterol was 171.9 (range: 129 – 193 mg / dL) and 183.4 (range: 125 – 273 mg / dL) in group with the DM and without DM, respectively[20].

HYPER – LIPIDAEMIA

A meta – analysis by Paul R An O'Mahoney considered a total of 21 studies, which had 2,916 cases and 28,646 controls. HTN and hyper – lipidaemia were significantly associated with RVO with an odds ratio of 3.5 and 2.5 respectively. However, the association for dm was a little lower with an odds ratio of 1.5, both BRVO and CRVO had similar findings. The study concluded that HTN and hyper – lipidaemia are common risk factors for RVO in adults, and DM is less so. It remains to be determined whether lowering blood pressure and / or serum lipid levels can improve visual acuity or the complications of RVO[21].

In another study by Salaun N et al, the most frequent factors associated with CRVO were hyper – homo – cysteinemia (33 %), arterial HTN (23 %), hyper – cholesterolemia (20 %), open – angle glaucoma or intraocular HTN (13 %), and DM (10 %). As for BRVO, the only notable risk factors were arterial HTN and hyper – cholesterolemia (46.2 % and 38.5 % respectively)[22].

In a study by Jane Zea Chin Kuo et al, 22 patients were studied, 65 % had hypo – cholesteraemia, 64 % had hyper – tri – glyceridemia, and 42 % had homo – cysteinemia, 28 % of these patients had macular oedema and 16 % had optic atrophy and secondary glaucoma was seen in 12 %[23].

OBESITY

A study by Dong – Won Paik et al, which studied 23,061,531 patients who were 20 years or older, multi – variate cox regression was used for analysis for studying BMI (body mass index) and its association with RVO. The study found that the presence or absence of DM affects the RVO. There was a significant correlation between BMI and RVO[24].

According to the study conducted by The Eye Disease Case – Control Study Group at The University of Illinois, Chicago[25]**.** Several risk factors were significantly associated with BRVO in the screening analyses. Factors associated with increased risk of BRVO included higher BMI, a history of DM, higher blood glucose levels, a history of cardio – vascular disease, higher levels of systolic or diastolic BP, a history of HTN, electro – cardiogram ab – normalities, a history of glaucoma, higher levels of intra – ocular pressure, and higher serum levels of alfa – 2 globulin and tri – glycerides. The deficiencies of this study include that this study has included cases randomly and the controls have been chosen from the same locality leading to a mild bias geographically which in turn may alter the finding of associations of risk factors to RVO[26].

According to the Korea National Health and Nutrition Examination Survey (KNHANES), the overall RVO prevalence was 0.6 %, no gender predilection was observed. Patients with un – controlled HTN had an odds ratio of 3.46, un – medicated HTN had an odds ratio of 4.2, therefore HTN was significantly associated with RVO[27].

According to the study conducted at Attikon Hospital from January 2007 to September 2011, there were 51 cases and controls each who were age and sex – matched. Among them, Arterial HTN was present in 44 cases and 19 controls. 5 patients and 4 controls were current smokers, whereas 13 patients and 3 controls had DM. In this study, the results seemed conflicting with some arguing for and others against a role of the a – fore – mentioned factors in RVO development such as pro – thrombotic risk factors FV G1691A and FII G20210A. There were no other conflicts of interest[28].

A study in Thailand found that athero – sclerotic ab – normalities were the most common disease associated with RVO and made up 55.1 % of the patients. 27.55 % of patients had HTN and 16.33 % of patients had DM. However, older individuals were excluded from this study which was the major limitation as to the risk for RVO increases with age[29].

According to the study conducted at the University of Valencia, the BP in the RVO population was highly significant when compared with the general population. Also, the RVO group had a large proportion of un – diagnosed HTN, there; was also, a large portion of the population with sub – clinical organ damage. In this study, the cases and controls were not age and sex – matched but the controls were chosen according to the risk factors being evaluated such as different controls for venous factors[30].

According to the study conducted by S S Hayreh et al in Iowa, there was a significantly higher prevalence of arterial HTN in BRVO compared with CRVO ($P < 0.0001$) and HRVO ($P = 0.028$). A significantly greater prevalence of arterial HTN ($P = 0.025$) and DM ($P = 0.011$) was present in the ischemic CRVO compared with the

non – ischemic CRVO. Similarly, arterial HTN (P = 0.0002) and ischemic heart disease (P = 0.048) were more prevalent in major BRVOs than in macular BRVO. There was no significant difference in the prevalence of any systemic disease between CRVO and HRVO. The patients with BRVO showed a greater prevalence of arterial HTN (P < 0.005), DM (in young only, P = 0.0005) compared with the control population. This study was conducted in 2001 making it quite an older study to go by today's standards[31].

According to a study conducted in Taiwan, 904 End – Stage Renal Disease (ESRD) patients and 410 controls had RVO (P < 0.0001) during the follow – up period, leading to a significantly elevated risk of RVO in the ESRD patients compared with controls (incidence rate ratio = 3.05, 95 % CI = 2.72 – 3.43). After adjustment for potential confounders including DM, HTN, hyper – lipidaemia, congestive heart failure, and coronary artery disease, ESRD patients were 3.05 times more likely to develop RVOs. The deficiencies in the study included the fact that there was no confirmation if the controls had no ESRD history before January 1996, therefore the findings could have been compromised. More importantly, several confounding factors including BMI, smoking history, and alcohol consumption could not be assessed. Finally, the diagnosis of the ESRD, RVO and other co – morbidity disorders relied upon ICD – 9 codes which may lead to disease mis – classification[32].

According to the study conducted at Gutenberg, there was a risk of association of smoking in 21.1 % of men and 18.2 % of women with RVO. There was a risk of association of 9.3 % men and 5.5 % women with RVO. The deficiencies in this study include cases of RVO that could have been missed on fundus photographs, especially in cases of reperfusion in non – ischemic RVO. A selection bias might have been

present, as persons with severe RVOs might not be willing to participate in the study since they are severely hampered by deteriorated visual acuity / blindness and / or neo – vascularization[33].

SYSTEMIC RISK FACTORS ASSOCIATED WITH RVO:

Multiple studies have found that various systemic disorders such as type 2 DM, dys – lipidaemia and HTN, are strongly associated with the development of RVOs[5].

Studies have noted that 49 % of RVOs are associated with HTN, 21 % with dys – lipidaemia, and 5 % with Type 2 DM[4].

Peripheral artery disease elevated serum homo – cysteine, myocardial infarction, deep venous thrombosis, pulmonary embolism, oral contraceptive use in women, and other hyper – coagulable states are also associated with RVO[1-3].

In HTN patients, the risk of CRVO is higher by 66 %[1]. Patients with only DM had minimal risk of CRVO whereas patients with all 3 components of metabolic syndrome have a 56 % increased risk of developing CRVO[1]. Ischemic CRVO is generally seen in Metabolic syndrome[1].

RISK FACTORS FOR CRVO – OCULAR RISK FACTORS:

Open – angle glaucoma is one of the major ocular risk factors causing CRVO[2]. Lowered ocular pressure is also associated with RVO, as this results in venous stasis, it is a component of Virchow's triad. High levels of type 1 plasminogen activator inhibitor (PAI – 1) and hyper – homo – cysteinemia also appear to play a significant role in the pathogenesis of this disease. Congenital thrombophilic diseases like factor

V Leiden mutation, hyper – homo – cysteinemia, and anti – cardiolipin antibodies increase the risk of RVO. Cigarette smoking also increases the risk of RVO. Systemic inflammatory conditions like vasculitis and Behcet's disease increase the risk. Ophthalmic risk factors for RVO are ocular HTN and glaucoma, lower ocular perfusion pressure, and congenital and acquired changes in retinal arteries. Important thrombophilic disorders include, anti – phospholipid syndrome (APS), hyper – homo – cysteinemia, resistance to activated protein C (factor V Leiden mutation), proteins C and S deficiencies, deficiency of anti – thrombin, pro – thrombin gene mutation (G20210A)[8].

PATHOGENESIS

CENTRAL RETINAL VENOUS OCCLUSION:

The occlusion in the central retinal vein usually occurs due to a thrombus in the central retinal vein. There is athero – sclerosis in the retinal artery which leads to degenerative changes in the vessel wall which leads to endothelial damage and thus thrombosis is caused. The site of thrombosis occurrence is usually posterior to the lamina cribrosa. A major role is played by the proliferation of endothelial cells. Optic nerve problems, structural changes in the lamina cribrosa, or degenerative changes in vessels contribute to the thrombosis of the central retinal vein[7].

The interplay of three systemic changes known as Virchow's triad cause the thrombosis[6]:

1. Hemodynamic changes (Stasis, Turbulence)
2. Endothelial injury
3. Hyper – coagulability state

BRANCH RETINAL VENOUS OCCLUSION:

Figure 1: Branch Retinal Venous Occlusion

The incidence of BRVO is about 3 times more common than CRVOs[2]. One of the important pathologies which cause the BRVO are the diseases that affect the arterioles, an important finding in this disease is the retinal artery is rigid and compressed, also the artery is arteriosclerotic.

The most common site of BRVO is at an A – V crossing (arterio – venous crossing), at this site, the adventitial sheath is shared by both artery and vein. At A – V crossing, the artery is usually anterior to the vein[34,35].

Local inflammatory ocular diseases rarely could lead to secondary BRVO.

In diseases such as toxo – plasmosis, Behçet's syndrome, Eales' disease, and ocular sarcoidosis, the secondary BRVOs have been found. BRVOs are also seen in

conditions such as macro – aneurysms, optic disc drusen, retinal capillary haemangiomas, Coats' disease, etc. The inflammatory condition should be evaluated if the occlusion doesn't lie at A – V crossing, in such cases, retino – choroiditis, retinal vasculitis, should be suspected.

OCULAR MANIFESTATIONS

OCULAR MANIFESTATIONS OF CRVO:

SYMPTOMS:

1. The defective distant vision of sudden onset
2. Visual loss can be sudden or gradual, over days to weeks Visual loss ranges from mild to severe
3. Photophobia
4. Painful blind eye – is a late manifestation after the patient develops NVG without ever realizing that he / she had CRVO
5. Redness of eye
6. Can also be Asymptomatic

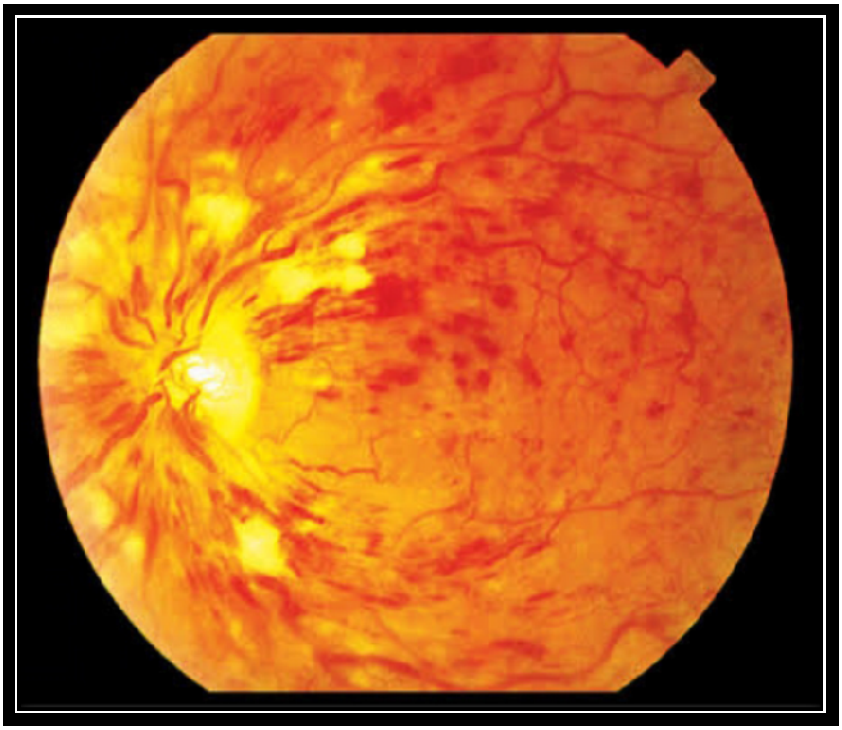


Figure 2: Ischemic Central Retinal Venous Occlusion

SIGNS:

The relative afferent pupillary defect (RAPD) is very helpful in separating the ischemic type from the non – ischemic type, during both the early and the late stages of the disease. There is a positive RAPD in haemorrhagic retinopathy.

In CRVO, characteristic retinal changes seen are:

1. In all 4 quadrants, the retinal veins are dilated and tortuous and dark
2. Intra – retinal haemorrhages
3. Macular oedema
4. Sub – retinal fluid
5. Cotton – wool spots
6. Intra – vitreal haemorrhage and rare sub – hyaloid haemorrhage
7. Disc oedema

Signs of HTN retinopathy[9] may also be associated. Macular oedema and ischemic maculopathy are the commonest causes leading to visual loss in RVO. Intra – retinal haemorrhages and hard exudates in the fovea have poor visual outcomes. Combined retinal vein and artery occlusion, usually results in poor visual prognosis[14,15]. The severity of CRVO depends on areas of retinal non – perfusion, fundus fluorescein angiography (FFA) is used to detect these. Minimally non – perfused CRVOs have mild to moderate loss of vision. Severe loss of visual acuity is seen in significantly non – perfused CRVOs.

DIFFERENT TYPES OF CRVO:

1. Ischemic CRVO
2. Non – ischemic CRVO

A Standard seven field fundus fluorescein angiography is used to diagnose these conditions. Capillary non – perfusion areas more than 10 – disc areas in FFA are ischemic CRVOs, these make – up about 20 % – 25 % of all the cases whereas Capillary non – perfusion areas less than 10 – disc areas in FFA are non – ischemic CRVOs, which make up the majority ranging from 75 % to 80 %.

There are many tests to differentiate between ischemic and non – ischemic CRVO.

They include functional tests like an assessment of visual acuity, visual fields, RAPD, ERG electro – retino – graphy can be done for diagnosis, other tests which are morphological test include, ophthalmoscopy and fluorescein fundus angiography. However, none of these tests have sensitivity and specificity 100 %. They can be done in a series of tests to improve diagnostic accuracy. Functional tests are superior to the

other morphological tests, which help in differentiating the ischemic and non – ischemic CRVO. If there is uni – ocular CRVO, RAPD is most reliable. Combined RAPD and ERG will diagnose 97 % of the cases, the next most reliable method is perimetry, and lastly, its visual acuity.

The fluorescein angiography sometimes provides limited information on retinal capillary non – perfusion (in at least one – third of the eyes during the early, acute phase) or it may sometimes provide mis – leading information. Ophthalmoscopic appearance is the least reliable[36].

One of the studies titled The Central Vein Occlusion Study (CVOS)[35] found that 34 % of non – ischemic CRVOs progressed to ischemic CRVO.

In 90 – DAY GLAUCOMA in ischemic CRVO, there is a higher rate of angle and iris neo – vascularization[37,38] which occur within 3 months of the onset of the disease.

In ischemic CRVOs the rate of neo – vascular glaucoma[39] (NVG) development ranges from 20 % – 63 %. However, the rate of NVG development in non – ischemic CRVO is negligible[18]. Optic nerve pallor and collateral vessels may develop during the phase of resolution; however, the following macular changes may occur:

1. Retinal pigmentary changes
2. ERM (epi – retinal membrane) formation
3. Sub – retinal fibrosis
4. Macular ischemia
5. Persistent macular oedema

HEMI RETINAL VENOUS OCCLUSION:

1. Superior HRVO
2. Inferior HRVO

The central retinal vein enters with two trunks in the optic nerve in 20 % of the population. They enter behind the lamina cribrosa, unite as a single trunk. In such cases, if there is occlusion of one of the trunks within the optic nerve head, it leads to the development of HRVO. The pathogenesis of HRVO is similar to CRVO. HRVOs neo – vascularization and response to therapy are also similar to CRVO. In patients younger than 50 years, papillophlebitis can occur[39].

BRANCH RETINAL VENOUS OCCLUSION:

Following are the different types of BRVO[6]:

1. Superotemporal BRVO – 52 %
2. Inferotemporal BRVO – 38 %
3. MT BRVO – 1 %
4. SN, IN BRVO – 9 %

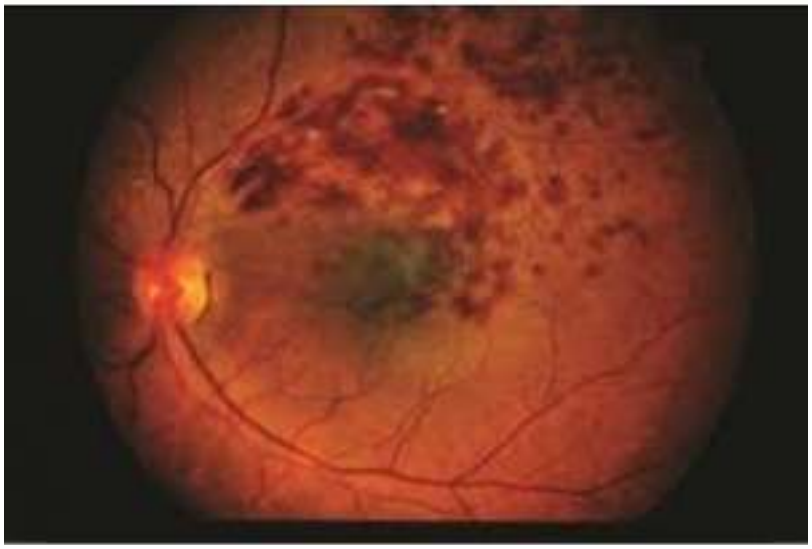


Figure 3: Supero – Temporal Branch Retinal Venous Occlusion

SYMPTOMS:

1. Sudden onset of defective vision which is painless
2. Visual field defects
3. The patient may be Asymptomatic

Oedema of the macula, ischemia, or neo – vascularization may lead to loss of visual acuity. Sometimes, the patient is asymptomatic and is detected in the routine eye examination

SIGNS:

1. Presence of flame – shaped haemorrhages
2. Conical shaped distribution of haemorrhages, the apex is at the site of occlusion
3. Cotton wool spots[18]
4. Veins are dilated and tortuous and dark
5. Haemorrhages may also be confluent
6. Sometimes there can sub – hyaloid haemorrhages or vitreal haemorrhages

INFERO – TEMPORAL BRVO:

Visual acuity may be from 20 / 20 (6 / 6) to CFCF. Asymptomatic BRVO can be seen if the macula is not involved. BRVO of the nasal quadrant is very rare, as most of these are not symptomatic, patients may not approach health care providers. In 20 % of them, neo – vascularization of the retina can occur, the occurrence of retinal neo – vascularization increases with increasing area of capillary non – perfusion[39].

PHASE OF RESOLUTION:

1. Haemorrhages resolve and fundus may appear normal
2. Collateral vessels can develop
3. Another feature of CRVO is collateral vessels crossing the horizontal raphe
4. Sclerosis of the retinal vein may occur
5. ERM and epithelial changes of macular retinal pigment may occur

INVESTIGATIONS IN RVO:

Fundus Fluorescein angiography (FFA) is the most useful diagnostic technique in RVO. In this technique, intravenous fluorescein dye is used for the visualisation of blood flow. Another technique is Fundus Auto Fluorescence (FAF), which is a non – invasive technique; in this retinal imaging method, the lipofuscin density is detected in retinal pigment epithelium[39].

FLUORESCEIN ANGIOGRAPHY works on the principle of a certain special property of sodium fluorescein, the property being its ability of molecules to emit

longer wavelength light when stimulated by shorter wavelength light. After stimulation by short wavelength light, electrons get back to their base energy due to the release of electro – magnetic waves which produce visible light[40].

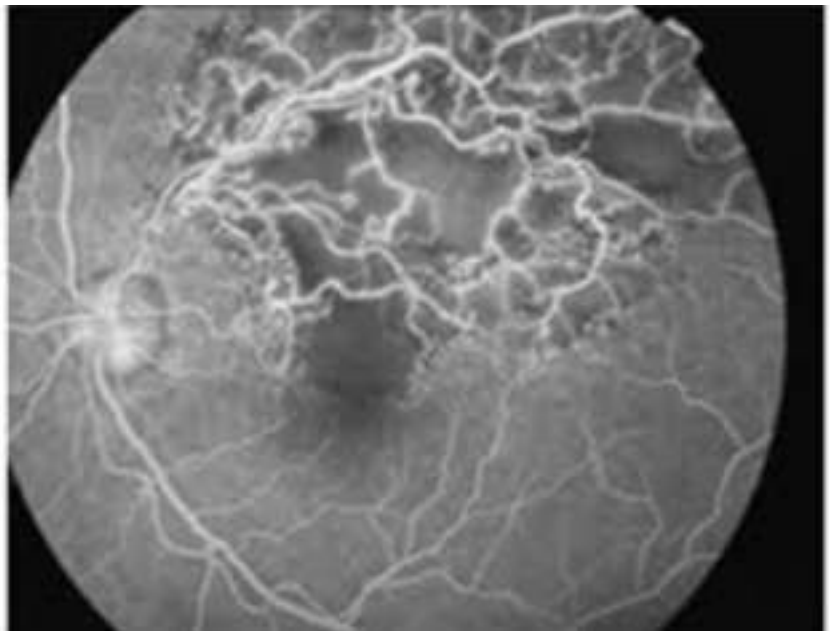


Figure 4: Filling Defect due to Capillary Non – Perfusion Areas

FFA of ST BRVO:

The absorption spectrum of the fluorescein dye is very narrow, with a peak at 490 nm (485 – 500 nm, blue visible spectrum). The fluorescence happens in the spectrum of yellow – green at 530 nm wavelength and it is emitted back as a green light. A green filter is used by the capturing device or camera which selectively saves the fluorescent image on the film digitally. In a normal eye, the molecule of sodium fluorescein crosses chorio – capillaries. Majority i.e., 80 % of the molecule circulates through plasma proteins which are blood bound but they remain within the retinal and larger choroidal vessels. This makes FA the perfect method for assessment of retinal circulation, and the inner and outer blood – retinal barrier.

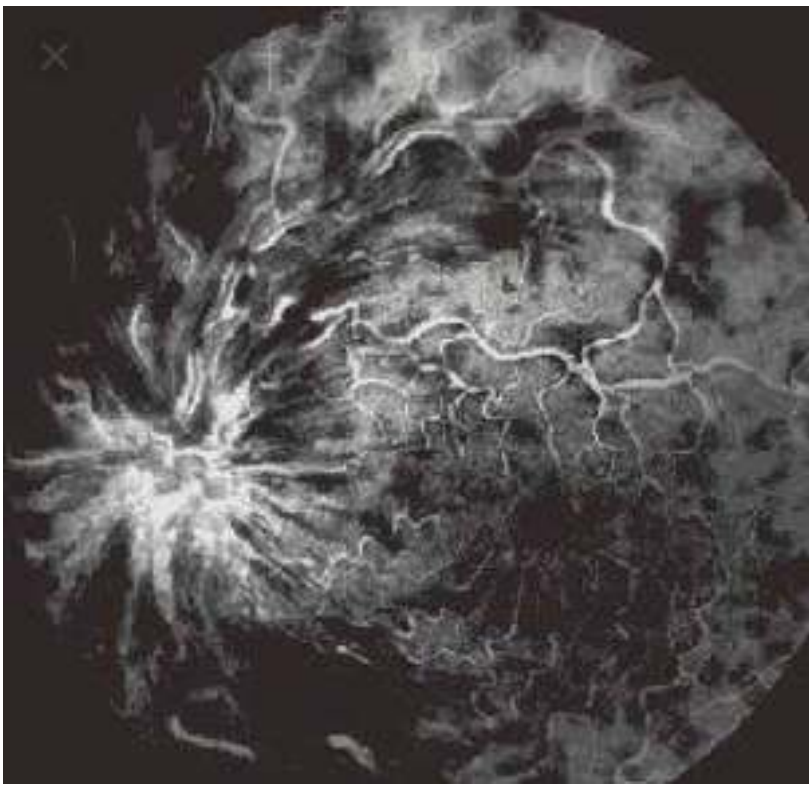


Figure 5: FFA changes in Retinal Venous Occlusions

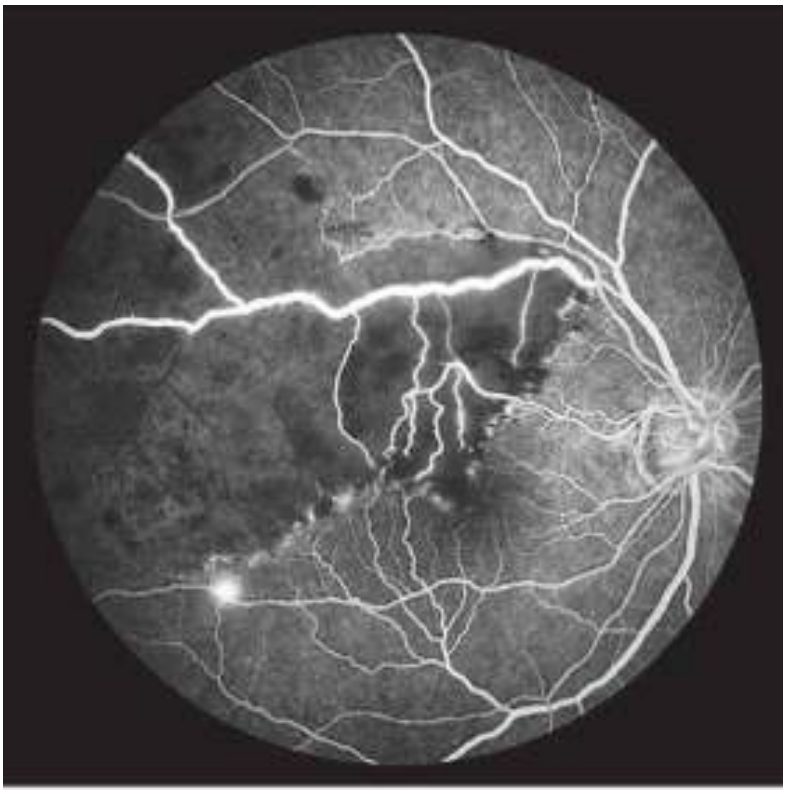


Figure 6: FFA of ST BRVO

Wedge – shaped areas of hypo – fluorescence is seen in occlusive diseases. Zones of hypo – fluorescence, secondary to focal choroidal non – perfusion are seen in Systemic diseases, such as choroidopathy of lupus, malignant HTN, giant cell arteritis[41-42].

FLUORESCEIN ANGIOGRAPHY IN CRVO:

Fluorescein angiography for CRVOs shows a delayed filling of the retinal veins. It is an important test for the assessment of non – perfusion, and neo – vascularization. In a study titled "The Central Vein Occlusion Study Group", it was found that a total of 35 % of ischemic and 10 % of non – ischemic CRVO was seen. The worst prognostic factors are for patients with poor visual acuity at presentation or more areas of non – perfusion[35].

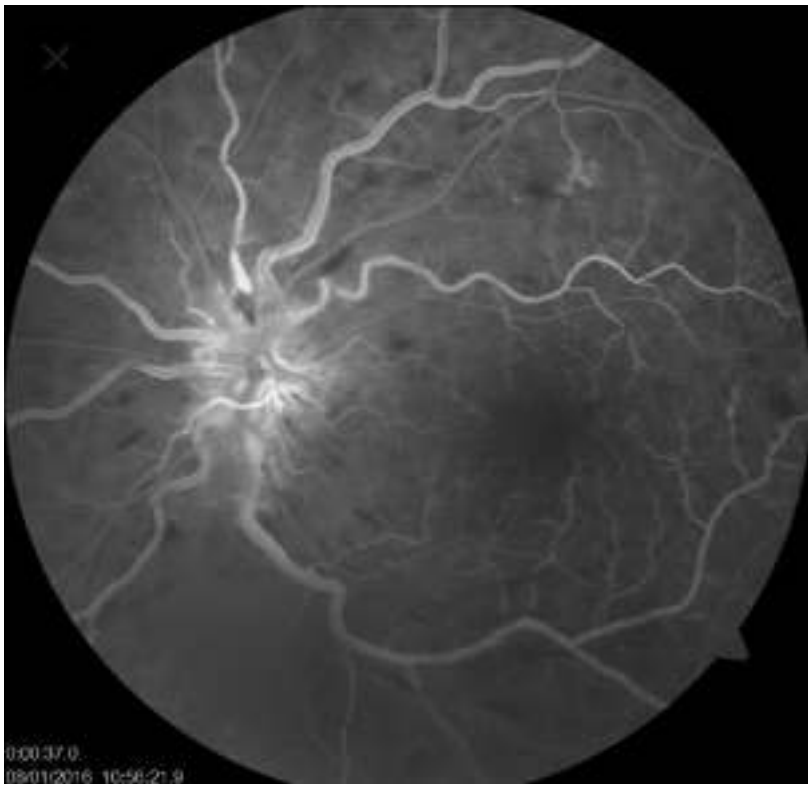


Figure 7: Fluorescein Angiography For CRVO

Hypo – fluorescence is seen on FFA in the case of ischemic CRVO, which is due to either blockage by retinal haemorrhages or due to retinal capillary non – perfusion. The macular region presents with extensive macular oedema in case of non – ischemic CRVOs, FFA may stain along capillaries. Non – perfusion capillary areas in non – ischemic CRVO is minimal or absent. Retinal changes almost return to normal state after 6 months in non – ischemic CRVO cases.

OPTICAL COHERENCE TOMOGRAPHY (OCT)[43]:

OCT is a non – invasive imaging method, based on the principle of optical reflectometry of light.

TIME DOMINANT OCT:

A scan is obtained, using a reference arm, in which various lengths are used. False colours are used to construct an image, high reflectivity is represented by bright colours. Limitations are slow accusation and limited resolution.

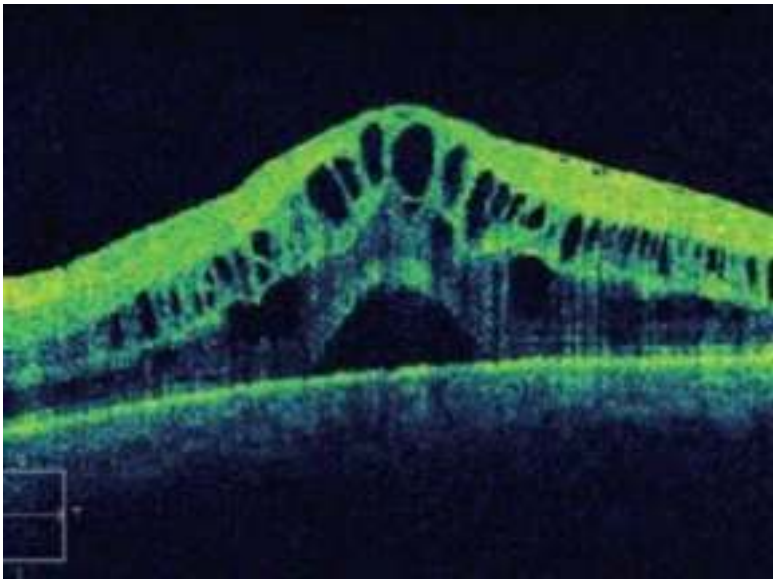


Figure 8: Macular Oedema as visualised in Oct

SPECTRAL – DOMAIN OCT:

Here echo time is measured by spectrometer and charge – coupled device. A single exposure is required and this has improved sensitivity and it is faster.

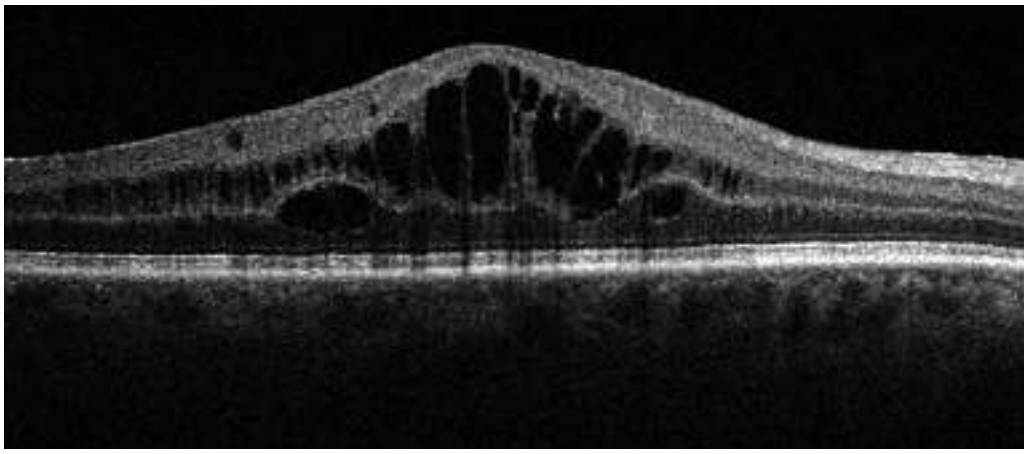


Figure 9: Macular Oedema on OCT

SWEPT – SOURCE OCT:

The advantages of this method are a high signal to noise detection technology; bandwidths range from 20 – 200 kHz. Has improved speed and scan depths, disadvantages include non – linearity and high sensitivity to movements[44].

OCT – ANGIOGRAPHY:

Optical coherence tomography – angiography (OCTA) has emerged as a non - invasive technique for imaging the micro – vasculature of the retina and the choroid. OCTA technology uses laser light reflectance of the surface of moving red blood cells to accurately depict vessels through different segmented areas of the eye, thus eliminating the need for intra – vascular dyes. The OCT scan of a patient's retina consists of multiple individual A – scans, which when compiled into a B – scan provides cross – sectional structural information. With OCTA technology, the same

tissue area is repeatedly imaged and differences are analysed between scans (over time), thus allowing one to detect zones containing high flow rates (i.e., with marked changes between scans) and zones with slower, or no flow at all, which will be similar among scans. Light is emitted through either a spectral – domain OCT (SD – OCT), with a wavelength of near 800 nm; or a swept – source OCT (SS – OCT), which utilizes a longer wavelength, close to 1050 nm. Longer wavelengths have a deeper tissue penetrance, but a slightly lower axial resolution. OCTA employs two methods for motion detection: amplitude – decorrelation or phase variance. The former detects differences in amplitude between two different OCT B – scans. Phase variance is related to the emitted light wave properties, and the variation of phase when it intercepts moving objects. To improve visualization and reduce background noise from normal small eye movements, two averaging methods – split spectrum amplitude decorrelation technique and volume averaging – were developed. These OCTA algorithms produce an image (3 mm^2 to 12 $mm^{2)}$ that is segmented, by standard, into four zones: the superficial retinal plexus, the deep retinal plexus, the outer retina and the chorio – capillaries. Applied to the optic disc, it includes its full depth.

The OCTA findings on RVO are decreased foveal and para – foveal vascular densities, areas of non – perfusion, engorged capillaries, vascular tortuosity, aneurysms, collateral vessels; there may be restricted field view and inability to show leakage[45].

DIFFERENTIAL DIAGNOSES FOR CRVO INCLUDE:

1. Ocular ischemic syndrome
2. Diabetic retinopathy
3. Radiation retinopathy
4. Hyper – viscosity retinopathy
5. HTN retinopathy

The ocular ischemic syndrome often presents with:

1. Attenuated vessels rather than dilated and tortuous veins
2. Can have choroidal non – perfusion in addition to retinal non – perfusion
3. Retinal haemorrhages are more in the mid – periphery in comparison to the posterior pole in CRVO

DM retinopathy has a positive history of DM. It is generally bilateral, characterized by micro – aneurysms, dot – blot haemorrhages, flame – shaped haemorrhages, and hard exudates.

Radiation retinopathy needs a history of radio – therapy, over the peri – orbital region.

HYPER – VISCOSITY SYNDROMES:

This bilateral disease occurs more usually in hyper – viscosity states like:

1. Waldenström's macro – globulinemia
2. Polycythaemia Vera
3. Leukaemia and
4. Multiple myeloma

If a patient presents with a CRVO in the absence of clear risk factors or presents with bilateral disease, the medical and laboratory explore the evidence of DM, hyper – viscosity syndromes, or inflammatory disease[43,44].

SYSTEMIC WORK – UP OF THE PATIENT:

- Complete history
- Physical examination
- BP
- CBC
- Coagulation factors
- Fasting blood glucose, post – prandial blood glucose
- Lipid profile
- ESR
- C – reactive protein
- Serum homo – cysteine
- Serum protein electrophoresis
- Complement factors
- Protein C, Protein S
- Cardiac evaluation

OPHTHALMOLOGICAL WORK – UP OF THE PATIENT:

- Vision is assessed using Snellen's chart and near vision charts respectively
- The anterior segment is examined using slit – lamp microscopy
- Pupils are examined using the slit – lamp as well as a handheld torchlight (an evenly bright light source) in a dimly illuminated room to avoid constriction caused by a brightly lit room; they are examined for their size, shape, position,

colour, and symmetry initially and then, the right and left eye pupils are examined individually for direct light reflex, consensual light reflex, near reflex and hence, accordingly for a relative afferent pupillary defect (RAPD)

- Funduscopic assessment is done by indirect ophthalmoscope and 20 D lens OR 90 D lens on a slit – lamp microscope

- The intra – ocular pressure is measured using non – contact tonometer

TREATMENT OF CRVO:

CRVO doesn't have any specific treatment. However, complications of CRVO like macular oedema and neo – vascularization may be treatable.

TREATMENT OF NEO – VASCULAR GLAUCOMA:

Neo – vascular glaucoma (NVG) is the most serious complication of ischemic CRVO. Abnormal neo – vascularization of the iris and angle is one of the prominent feature[46]. This is one of the important conditions leading to a painful blind eye. In a study titled "A randomized clinical trial of early PRP for ischemic central venous occlusion", it was found that in CVOs prophylactic PRP was effective in preventing the development of NVI or NVA in patients with ischemic CRVO[47]. Management of ischemic CRVO includes frequent follow – up and prompt PRP in case of development of NVI to reduce the risk of coherence topographical function [48]. It is essential to identify NVI at the pupillary border, also it's important to examine the un – dilated pupil. Gonioscopy has to be done regularly because NVA can occur without NVI. Drugs such as bevacizumab which are Intravitreal anti – vascular endothelial growth factor (VEGF) now have become a useful adjunctive therapy that helps to

quickly reduce or eliminate neo – vascularization from the anterior segment in CRVO[49]. However, VEGF use is limited by the half – life of the drug.

TREATMENT OF MACULAR OEDEMA:

In almost all patients with ischemic CRVO and some patients with non – ischemic CRVO, there is Cystoid macular oedema (CME) and resulting dysfunction of the macula[50]. Generally macular grid photo – coagulation is not employed for the treatment of CME in CRVO. Intra – vitreal triamcinolone[51], is commonly used off – label to treat CME associated with CRVO.

The Standard Care Versus Corticosteroid for Retinal Venous Occlusion (SCORE)[52,53]. The study concluded significant improvements in visual acuity in both treatment groups compared to the observation group.

Studies have proven the efficacy of anti – VEGF agents including bevacizumab[54,55] which is used off – label, and both ranibizumab[56] and aflibercep[57,58,59].

The GALILEO study showed 60.2 % of patients treated with aflibercept monthly for 6 months gained 15 or more letters as compared to 20.1 % in the same group[60].

Finally, the NEWTON study demonstrated that patients previously treated with bevacizumab and ranibizumab experienced longer oedema – free intervals on aflibercept (62 days on aflibercept vs 39 days on bevacizumab / ranibizumab)[61].

RANIBIZUMAB:

Ranibizumab is the most commonly used anti – VEGF agent. It is the antigen – binding Fragment (Fab) of a mono – clonal humanized anti – body. It inhibits active forms of VEGF – A[62].

BEVACIZUMAB:

It is a mono – clonal anti – body (Humanised), mechanism of action is by inhibition of biologically active forms of VEGF – A.

CLINICAL COURSE WITH OUTCOME:

Visual recovery and the prognosis are dependent on sub – type of CRVO. The extent of recovery can be assumed from the visual acuity during the initial presentation. Complete recovery may be seen in non – ischemic CRVO; however, this may occur in less than 10 % of cases[62]. In CVOS, for eyes with presenting visual acuity of 20 / 40 or more, 65 % maintained this range of visual acuity, whereas, for eyes with presenting visual acuity of less than 20 / 200, 80 % had visual acuity remaining this poor at the end of the study. With newer treatment modalities (particularly anti – VEGF agents), the natural course of CRVO is being arrested, and better visual acuity outcomes are now compared to the era of the CVOS, as evidenced by the SCORE and CRUISE studies. In the era of the CVOS study, recommended follow – up examination intervals depending on the presenting visual acuity and degree of ischemia[35,47]. With the advent of anti – VEGF therapy, most patients with CRVO are followed monthly, at least initially, while undergoing monitoring and treatment for CME[63,64].

TREATMENT OF BRVO:

The Branch Vein Occlusion Study (BVOS) analysed the effect of laser photo – coagulation on neo – vascularization, vitreous haemorrhage, and macular oedema in BRVO[65]. Peripheral sectoral scatter laser photo – coagulation reduced the rate of development of both neo – vascularization and vitreous haemorrhage[65]. It was found that no advantage in treatment before neo – vascularization occurred, even if extensive capillary non – perfusion existed. If the laser is applied to all non – perfused BRVOs, a large percentage of patients shall be treated unnecessarily. When a peripheral laser is indicated, FFA can help guide laser treatment by defining areas of peripheral capillary non – perfusion. A scattered pattern of laser is performed in the affected sector. In BVOS, patients who had 20 / 40 (6 / 12) or worse vision and macular oedema on FFA were treated with focal laser in a grid pattern over the area of leakage. Treated patients had reduced macular oedema and improved visual acuity compared to untreated controls[66-70].

The SCORE study for BRVO concluded no improvement in visual acuity outcomes with intravitreal triamcinolone compared to standard grid macular laser treatment for macular edema[71].

The (BRAVO) clinical trial found improvement of visual acuity between the 0.3 mg group and 0.5 mg group in the monthly ranibizumab groups compared with the sham group at 6 months[72]. The use of grid laser in BRVO patients has contributed to better stability by decreasing the VEGF load[73].

GUIDELINES FOR TREATMENT OF BRVO AND NEO – VASCULARIZATION:

- FFA to be done after clearing of retinal haemorrhages
- Patients to be followed up at 4 – month period if there are more than five – disc diameter of non – perfusion present
- Using Argon laser PRP, if neo – vascularization develops in areas involving the retinal sector

The VIBRANT study compared treatment with monthly aflibercept vs grid laser for BRVO – related CME. BCVA was improved by 17.0 letters in the aflibercept group vs 6.9 letters in the grid laser group[74].

The RELATE trial evaluated the benefit of grid laser[75].

COURSE AND OUTCOME:

About one – third of the patients with BRVO had a better vision 20 / 40 to 6 / 12 with no treatment. Although 2 / 3rd had macular oedema and ischemia or haemorrhage which resulted in poor visual acuity. Laser treatment improved macular oedema and vision[68,76]. With the advent of anti – VEGF therapy, treatment is often initiated earlier, and visual acuity outcomes are potentially more, compared with treatment with focal laser alone.

POOR VISUAL PROGNOSTIC FACTORS[77]:

1. Elderly patients
2. Male sex
3. Poor visual acuity
4. Increased number of pre – disposing risk factors

MATERIALS AND METHODS

SOURCE OF DATA:

Subjects attending the Vitreo – Retinal OPD of the Ophthalmology department who are newly diagnosed with BRVO or CRVO or HRVO at KLES DR. PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI.

METHOD OF COLLECTION OF DATA

STUDY DESIGN: A One Year Case – Control; prospective, comparative, non – interventional observation study.

DURATION: 1st January 2020 – 31st March 2021

TOTAL SAMPLE SIZE: 31

FORMULA USED T O CALCULATES SAMPLE SIZE:

$$n = \frac{2pq\,(z_\alpha + z_\beta)^2}{d^2}$$

where z_α is linked with the level of significance and z_β is linked with the power of the test. For 5 % level of the significance $z_\alpha = 1.96$ and $z_\beta = 0.84$ for 80 % power of the test.

% of HTN = p1 = 70.4 % (cases)

% of HTN = p2 = 35.3 % (controls)

D = p1 – p2 = 35.1 %

Using the above values, the sample size was calculated to be 31 in cases and 31 in controls. The values considered above are taken from a multi – centric study conducted at five large clinical centres[68].

SELECTION CRITERIA

INCLUSION CRITERIA:

1. Patients of either gender
2. Newly diagnosed case of either BRVO or CRVO or HRVO
3. Age between 40 to 70 years

EXCLUSION CRITERIA:

1. Previously known cases of RVO
2. Patients with pathological myopia (moderate to severe myopia accompanied by a posterior staphyloma), vaso – proliferative retinopathy, and intermediate or posterior intraocular inflammatory disease
3. Patients with coagulopathy disorders

METHODOLOGY:

In the present study a total of 62 eyes were considered, out of these 31 eyes were of cases who had CRVO, BRVO, or HRVO, patients consenting for the participation in the study and satisfying the inclusion criteria will be taken as cases. Demographic data of the cases and controls will be taken using a pre – designed Performa. The study will be done in the Ophthalmology OPD – 17, KLES DR. PRABHAKAR KORE HOSPITAL AND RESEARCH CENTRE, BELAGAVI, KARNATAKA. Assessment of patients will be done concerning medical history,

visual acuity, refractive history, and examination of anterior and posterior chambers. Patients will be asked about their previous history regarding HTN, DM, any episode of stroke, ischemic heart disease which will include a history of any previous stenting or CABG, a chronic kidney disease which includes whether the patient was on dialysis or any renal treatment, glaucoma and smoking; and investigations will be conducted accordingly; to ascertain the association of the above systemic conditions in cases.

Controls will be the patients who will be visiting the outpatient department of the hospital for ophthalmological complaints or regular or executive check – up or follow – up. Controls will be selected from those patients who do not satisfy the inclusion criteria as mentioned above, they will be selected from the same clinic and will be age and sex – matched.

They will also be asked about their previous history regarding HTN, DM, any episode of stroke, ischemic heart disease which will include a history of any previous stenting or CABG, a chronic kidney disease which will include whether the person was on dialysis or any renal treatment, glaucoma and smoking; and then the same investigations will be conducted for controls as well to ascertain the association of the systemic conditions in controls.

DATA COLLECTION:

Informed consent will be taken from both the cases and controls after explaining the procedure of the study in their language. Informed consent will be obtained from all the patients after a detailed explanation of the procedures and processes involved. If the patient is unable to read, the consent form will be explained

to the patient in his / her vernacular language. Socio – demographic information of the cases and controls will be taken. Patients will be examined in the ophthalmology OPD.

CLINICAL EXAMINATION:

A thorough clinical examination will be done and findings will be noted. Detailed history and examination of the patients will be undertaken. Other relevant past histories will be noted, history of smoking and alcohol intake, medication history will be taken.

Assessment of pre – existing co – morbidities in the patient, if any will be enquired by a history of HTN, DM, cancer, chronic kidney disease, and chronic liver disease.

OPHTHALMOLOGICAL EXAMINATION:

Vision: The near and distance vision of the cases and controls was assessed using near vision charts and Snellen's charts respectively

Anterior segment was examined using slit – lamp microscope

Pupils were examined using the slit – lamp as well as a handheld torchlight (an evenly bright light source) in a dimly illuminated room to avoid constriction caused by a brightly lit room; they were examined for their size, shape, position, colour, and symmetry initially and then, the right and left eye pupils were examined individually for direct light reflex, consensual light reflex, near reflex and hence, accordingly for a relative afferent pupillary defect (RAPD)

Funduscopy was done by indirect ophthalmoscope and 20 D lens or slit – lamp microscope and 90 D lens

Tonometry: The IOP was assessed using NCT. If the IOP was found to be high then, examination of the angle, ONH, and visual field tests was conducted in cases not known to be glaucoma patients; if it is a known case of glaucoma previous records were verified and type of glaucoma ascertained

PROCEDURE:

Blood samples were taken under strict aseptic precautions following universal safety precautions.

MEASUREMENT OF BLOOD PRESSURE:

According to Joint national committee guidelines, high BP is defined as readings of ≥ 130 mm of Hg for systolic blood pressure (SBP) measurement, or readings of ≥ 80 mm of Hg For the diastolic blood pressure (DBP) measurement; the BP of each patient was thus taken based on AHA recommended protocol. The blood pressure was taken while the patient was seated and relaxed. The history of HTN was elicited and noted[78].

BLOOD GLUCOSE MEASUREMENT:

According to ADA, the following values were used for the classification of DM[79]:

FASTING BLOOD GLUCOSE LEVELS:

Result	Fasting Plasma Glucose (FPG)
Normal	less than 100 mg / dl
Pre – Diabetes	100 mg / dl to 125 mg / dl
Diabetes	126 mg / dl or higher

FOR POST – PRANDIAL BLOOD SUGAR:

Result	Post Prandial – Blood Sugar
Normal	less than 140 mg / dl
Pre – Diabetes	140 mg / dl to 199 mg / dl
Diabetes	200 mg / dl or higher

FOR HbA1C:

Result	HbA1C
Normal	Less than 5.7 %
Pre – Diabetes	5.7 % - 6.4 %
Diabetes	6.5 % or Higher

SERUM CREATININE AND UREA[80]

CREATININE: Normal creatinine levels were considered to be 0.9 to 1.3 mg / dl in men and 0.6 to 1.1 mg / dl in women

UREA: Normal blood urea level was considered to be 6 to 24 mg / dl (2.1 to 8.5 mmol / l)

MEASUREMENTS

HEIGHT: Height was measured with tape to the nearest centimetre. Subjects were requested to stand upright without footwears with their back against the wall, heels together and eyes directed forward

WEIGHT: Weight was measured with a traditional spring balance that was kept on a firm horizontal surface. The scale was checked and calibrated with "known" weights before going for data collection. Participants were asked to wear light clothing and weight was recorded to the nearest 0.5 kg

BODY MASS INDEX (BMI): BMI was calculated using the formula:

Weight (kg) / Height (m^2) and classified as follows:

CLASSIFICATION OF BMI[81]:

Under – Weight	< 18.50
Normal	18.50 – 24.99
Over – Weight	25.00 – 29.99
Obese	≥ 30.00

HABITS

SMOKING TOBACCO: The subjects were questioned regarding their smoking habits like beedis, cigarettes. The information included frequency, duration of consumption, and also, whether they quit the habit or not. For analysis, the study subjects were grouped as follows depending upon their tobacco consumption:

CURRENT SMOKER: A person who smoked more than 100 cigarettes in his life time and is currently smoking every day or some – day at the time of study

FORMER SMOKER: A person who smoked more than 100 cigarettes in his life time and who had quit and did not smoke for the past year

NEVER SMOKER: A person who never smoked at all or smoked less than 100 cigarettes in his entire life time

SMOKE – LESS TOBACCO: The subjects were questioned regarding their smoke – less tobacco use habits like chewing tobacco, snuff inhalation, guthkha, paan with tobacco, etc. The information included frequency, duration of consumption, and also whether they quit the habit or not. For analysis, the study subjects were grouped as follows depending upon their tobacco consumption:

CURRENT USER: A person who used more than 100 smoke – less tobaccos in his life time and is currently using it every – day or some – day at the time of study

FORMER USER: A person who had used more than 100 smoke – less tobaccos in his life time and who had quit and did not use for the past year

NEVER USER: A person who never used smoke – less tobaccos at all or used less than 100 ones in his life time [82]

ALCOHOL CONSUMPTION: The subjects were questioned regarding the habit of consuming alcohol like beer, whisky, rum, etc. the information included are frequency, duration, quantity, and also if they had quit the habit or not

CURRENT USER: A person who is presently consuming alcohol

FORMER USER: A person who used to consume alcohol before one year and is abstaining from it at present

NEVER USER: A person who has never consumed alcohol in any form in his life

SERUM CHOLESTEROL[83]:

Total cholesterol levels less than 200 milligrams per decilitre (mg / dL) were considered desirable for adults. 200 – 239 mg / dL was considered borderline high and a reading of 240 mg / dL and above was considered high. LDL cholesterol levels of more than 100 mg / dL were considered high. A fasting sample was taken.

STATISTICAL ANALYSIS:

The prevalence of each risk factor in cases of RVO and among controls will be calculated as percentages and their CI will be estimated. The proportion of each risk factor among cases of RVO will be compared with its proportion among controls by Chi – square test. p – value of less than 0.05 will be considered significant, Mean and Std deviation will be calculated. Students t – test will be used to compare continuous quantitative variables. Data will be presented using suitable tables and graphs. Percentages, ratios, and rates will be calculated for categorical data.

RESULT

Table 1: Age Distribution of the Study Subjects

	Cases		Controls	
Age group	**Frequency**	**Percentage**	**Frequency**	**Percentage**
40 – 50	**3**	**9.6 %**	**3**	**9.6 %**
51 – 60	**9**	**29 %**	**9**	**29 %**
61 – 70	**19**	**61.4 %**	**19**	**61.4 %**
Total	**31**	**100 %**	**31**	**100 %**

Graph 1: Age Distribution of the Study Subjects

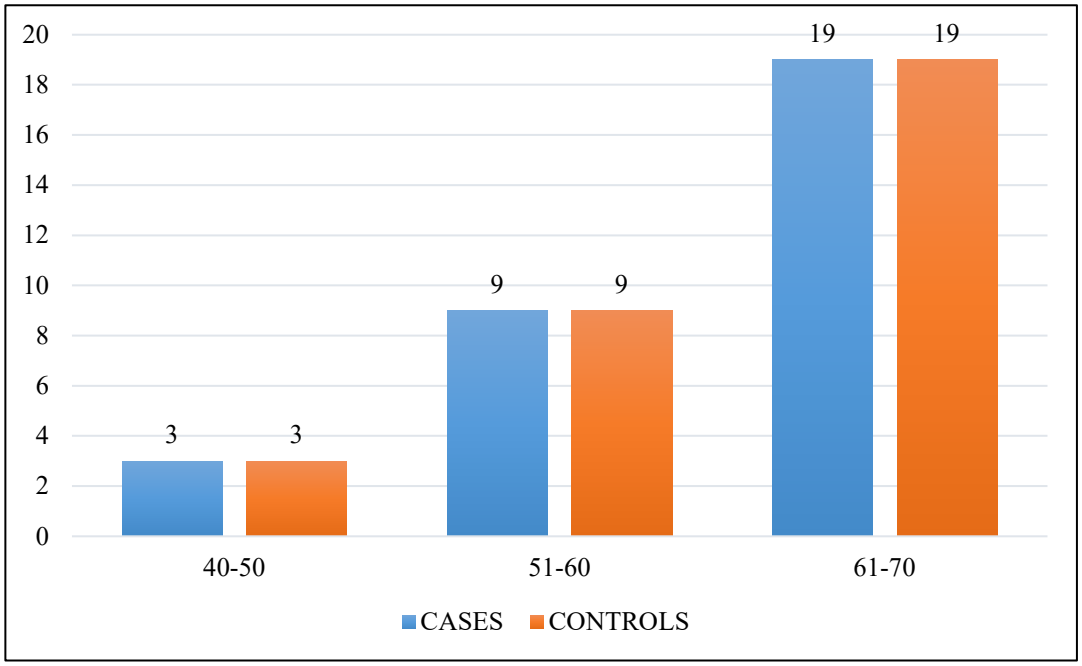


Table 2: Mean Age between the Cases and the Controls

	Cases Mean ± STD DEV	Controls Mean ± STD DEV	T value	P value
MEAN AGE	**59.93 ± 8.11**	**56.24 ± 11.62**	**1.056**	**0.295**

UNPAIRED t – TEST WAS USED TO COMPARE THE MEANS AND STANDARD DEVIATION, p – VALUE < 0.05 IS CONSIDERED AS STATISTICALLY SIGNIFICANT

Graph 2: Mean Age between the Cases and the Controls

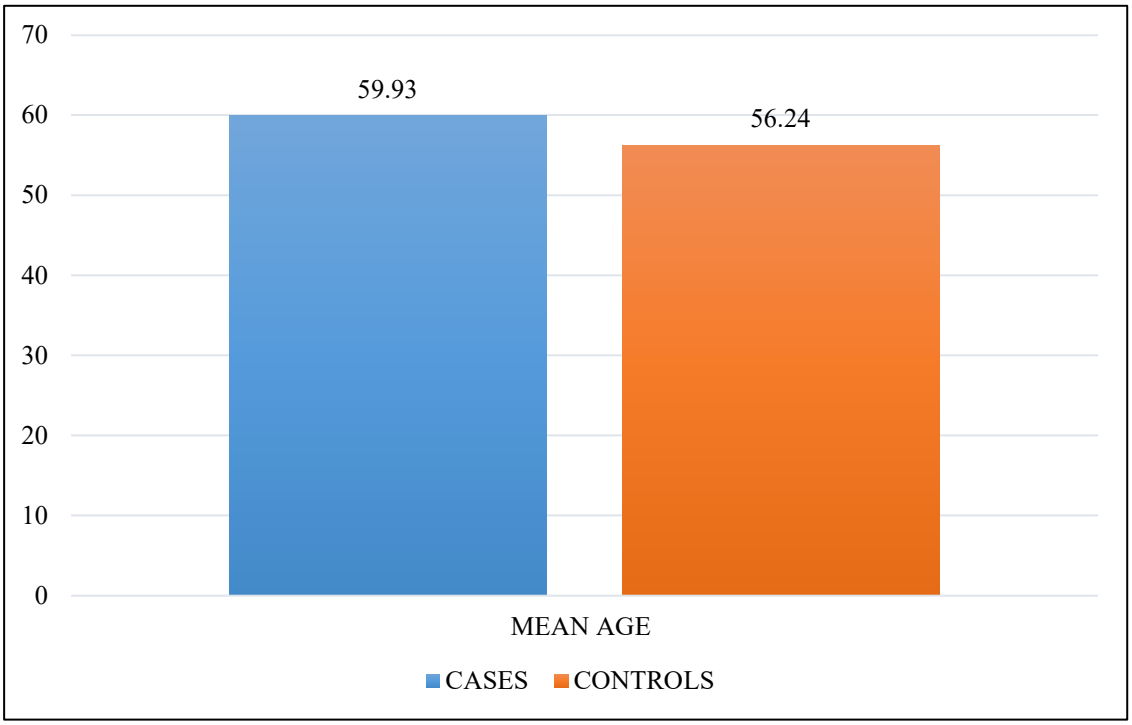


Table 3: Gender – Wise Distribution of The Study Subjects

Gender	Frequency	Percentage
Male	36	58 %
Female	26	42 %
Total	62	100 %

Graph 3: Gender – Wise Distribution of The Study Subjects

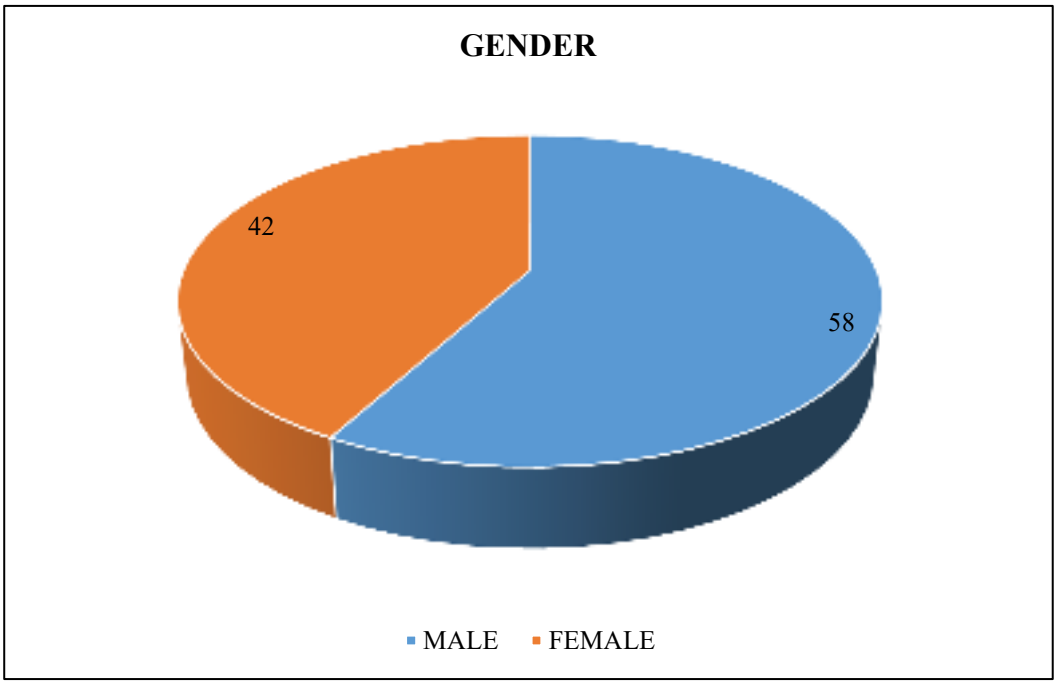


Table 4: Gender – Wise Distribution of The Study Subjects

	Cases		Controls	
Gender	**Frequency**	**Percentage**	**Frequency**	**Percentage**
Male	**18**	**58 %**	**18**	**58 %**
Female	**13**	**42 %**	**13**	**42 %**
Total	**31**	**100 %**	**31**	**100 %**

Graph 4: Gender – Wise Distribution of The Study Subjects

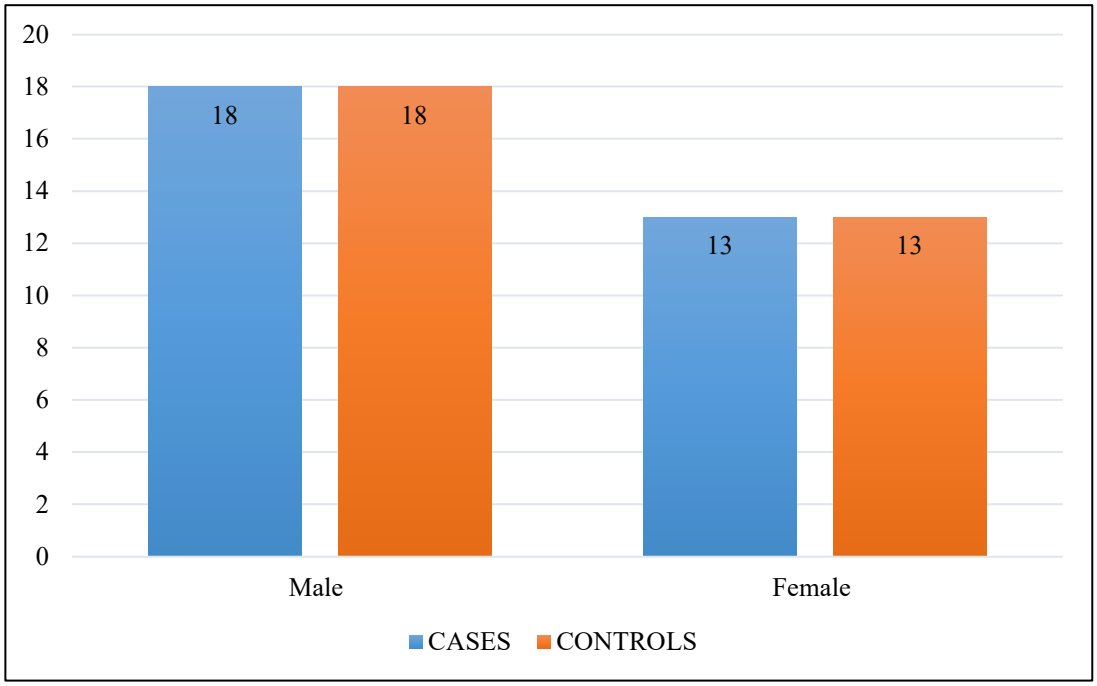


Table 5: Laterality of the Eye Affected in The Study Subjects

	LEFT		RIGHT		P VALUE
	N	%	N	%	
Cases	13	42 %	18	58 %	0.304
Controls	15	48 %	16	52 %	
Total	28		34		

CHI – SQUARE TEST WAS USED AND p – VALUE < 0.05 WAS CONSIDERED STATISTICALLY SIGNIFICANT

Graph 5: Laterality of the Eye Affected in the Study Subjects

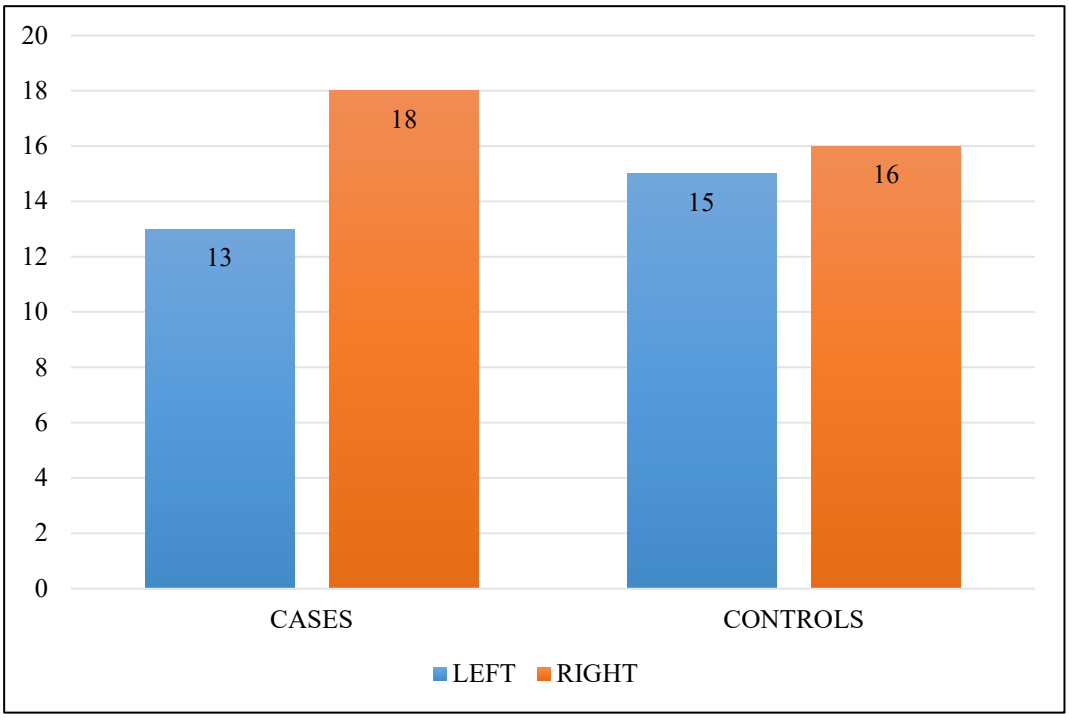


Table 6: Presence of Co – Morbidities in Cases VS Controls

	Cases		Controls	
Co - Morbidities	**Frequency**	**Percentage**	**Frequency**	**Percentage**
HTN	**23**	**74 %**	**11**	**35.4 %**
DM	**21**	**61.2 %**	**12**	**38.7 %**
History Of Heart Disease	**4**	**13 %**	**2**	**7 %**
Obesity	**22**	**71 %**	**10**	**32.2 %**
Glaucoma	**6**	**19.3 %**	**0**	**0**
Hyper – Lipidaemia	**16**	**51.6 %**	**6**	**19.3 %**

Graph 6: Presence of Co – Morbidities in Cases VS Controls

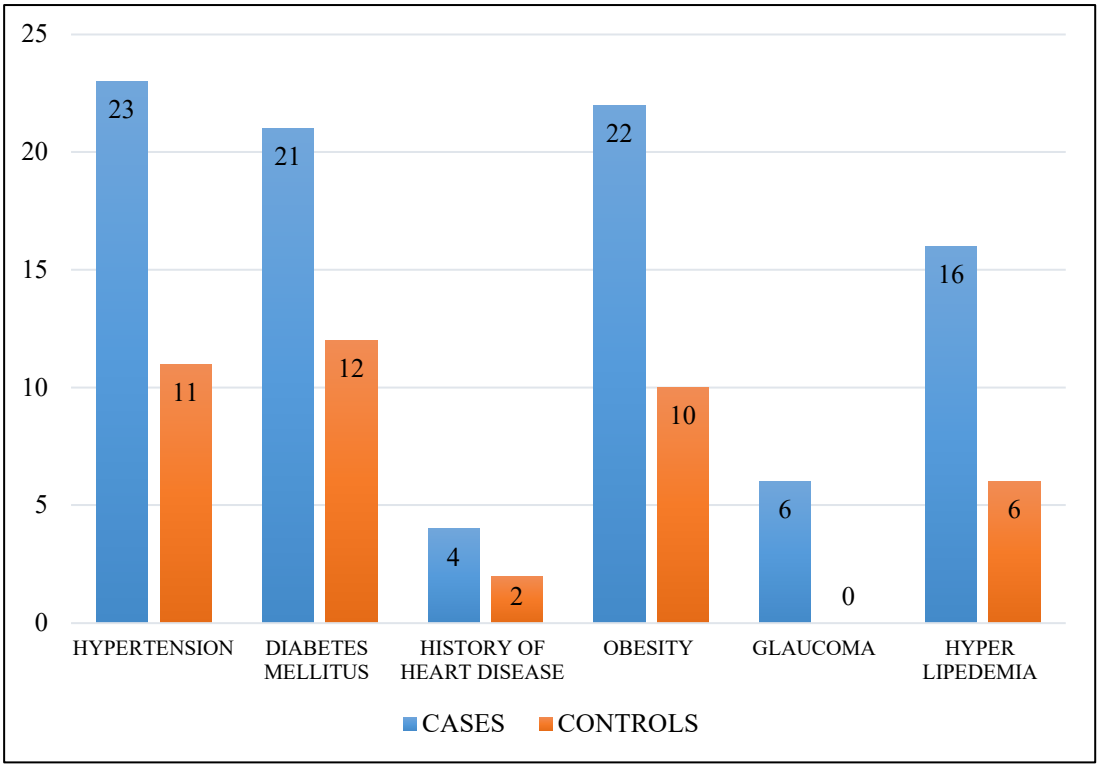


Table 7: Type of Glaucoma in the Study Subjects

	Cases		Controls	
	Frequency	**Percentage**	**Frequency**	**Percentage**
Primary open angle glaucoma	**6**	**100 %**	**0**	**0**
Primary angle closure glaucoma	**0**	**0**	**0**	**0**
Total	**6**	**100 %**	**0**	**0**

Graph 7: Type of Glaucoma in the Study Subjects

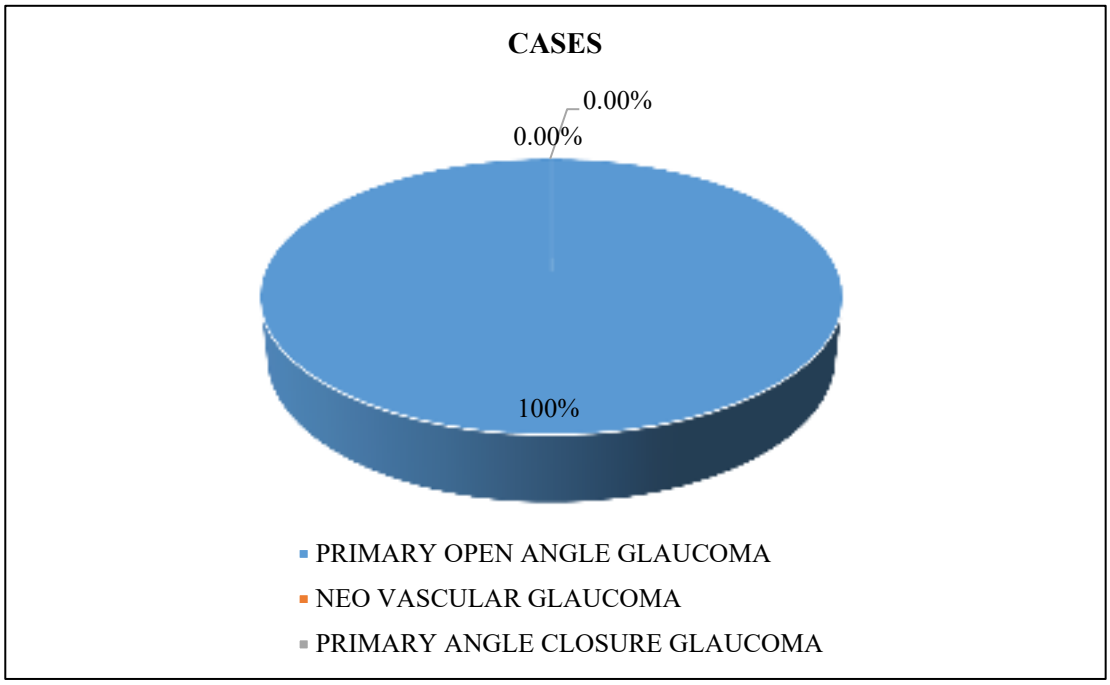


Table 8: Habits of Cases VS Control in The Study Subjects

	Cases		Controls		Chi Square	P Value
Habits	**Frequency**	**Percentage**	**Frequency**	**Percentage**		
Smoking	**19**	**61.3 %**	**8**	**25.8 %**	**7.939**	**0.002****
Non-smoking	**12**	**38.7 %**	**23**	**74.2 %**		
Total	**31**	**100 %**	**31**	**100 %**		

CHI – SQUARE TEST WAS USED, p – VALUE < 0.05 WAS CONSIDERED STATISTICALLY SIGNIFICANT

Graph 8: Showing Habits in Cases VS Control in The Study Subjects

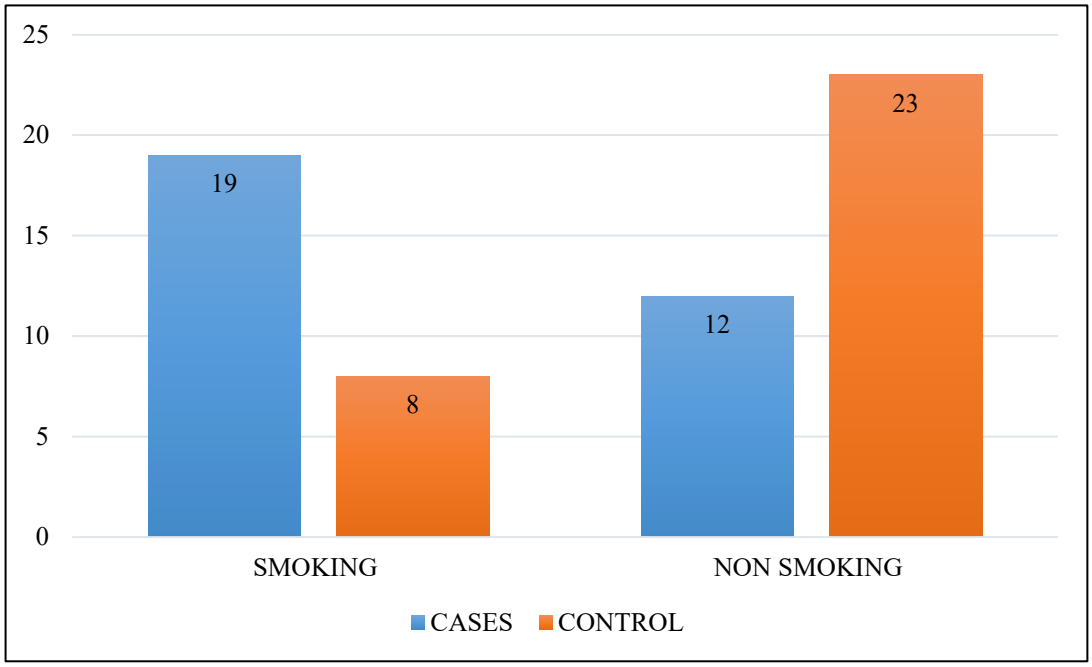


Table 9: Types of CVO In the Study Population in Cases and Controls

	Cases	
	Frequency	**Percentage**
ISCHEMIC CRVO	**5**	**16.5 %**
NON – ISCHEMIC CRVO	**4**	**13.5 %**
SUPERIOR HRVO	**1**	**3 %**
INFERIOR HRVO	**4**	**13.5 %**
ST BRVO	**8**	**26.2 %**
IT BRVO	**5**	**16.5 %**
MT BRVO	**3**	**10.8 %**
	31	**100 %**

Graph 9: Types of CVO In Cases VS Control in The Study Subjects

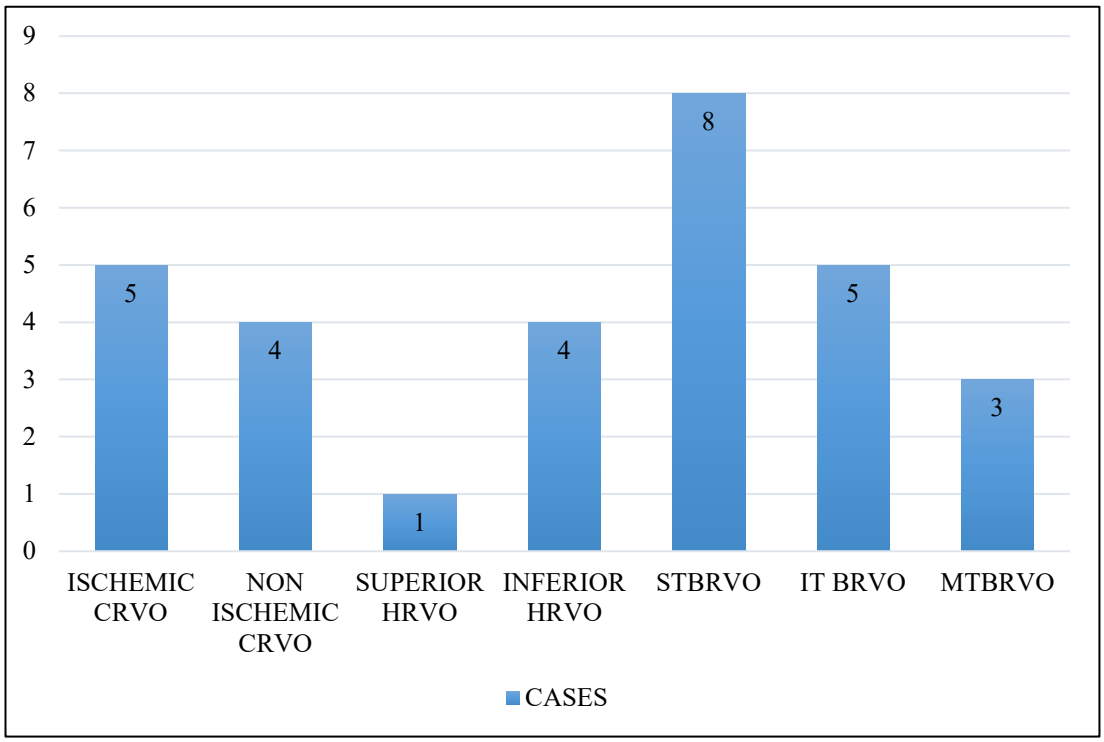
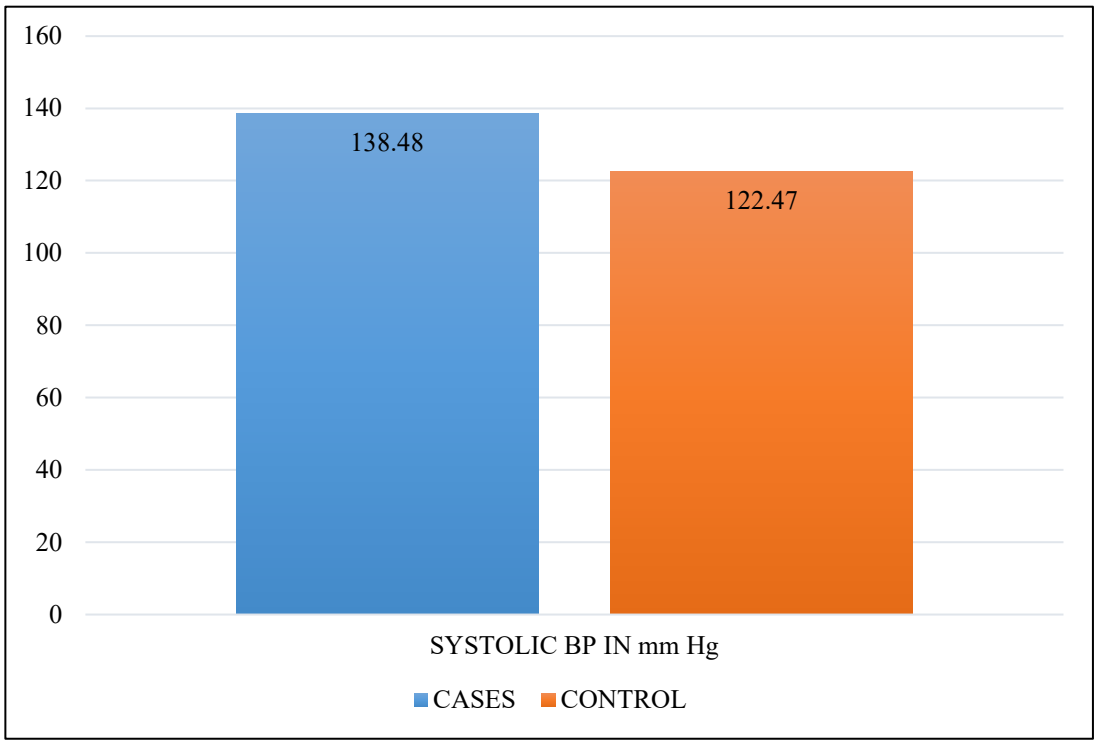


Table 10: Comparison of Mean Values of Investigations in Cases and Controls

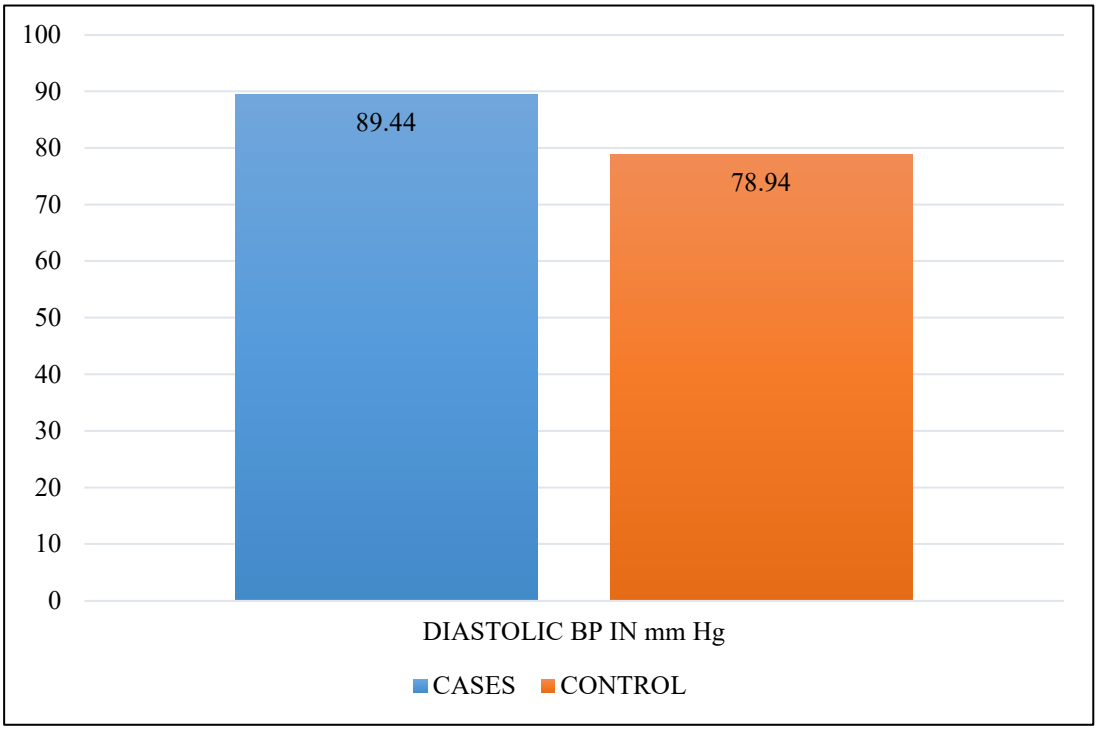
	Mean ± Std Dev	**Mean ± Std Dev**	**t – value**	**p – value**
	Cases	**Controls**		
SYSTOLIC BP IN mm Hg	138.48 ± 14.51	122.47 ± 10.66	4.95	**<0.001****
DIASTOLIC BP IN mm Hg	89.44 ± 10.54	78.94 ± 11.45	3.77	**0.003***
HbA1c	7.95 ± 1.22	6.14 ± 0.69	7.74	**<0.001***
SERUM CREATININE	1.02 ± 0.24	0.96 ± 0.02	1.38	0.170
BLOOD UREA	18.45 ± 3.45	16.74 ± 4.58	1.66	0.102
BODY MASS INDEX	28.45 ± 4.66	22.48 ± 3.14	5.91	**0.0001***
INTRA – OCULAR PRESSURE	22.5 ± 4.6	17.55 ± 3.58	4.77	**0.001***
CHOLESTEROL	243.41 ± 35.11	210.14 ± 30.44	3.98	**0.001***

UNPAIRED t – TEST WAS USED TO COMPARE THE MEAN AND STD DEVIATION, p – VALUE <0.05 IS CONSIDERED AS STATISTICALLY SIGNIFICANT

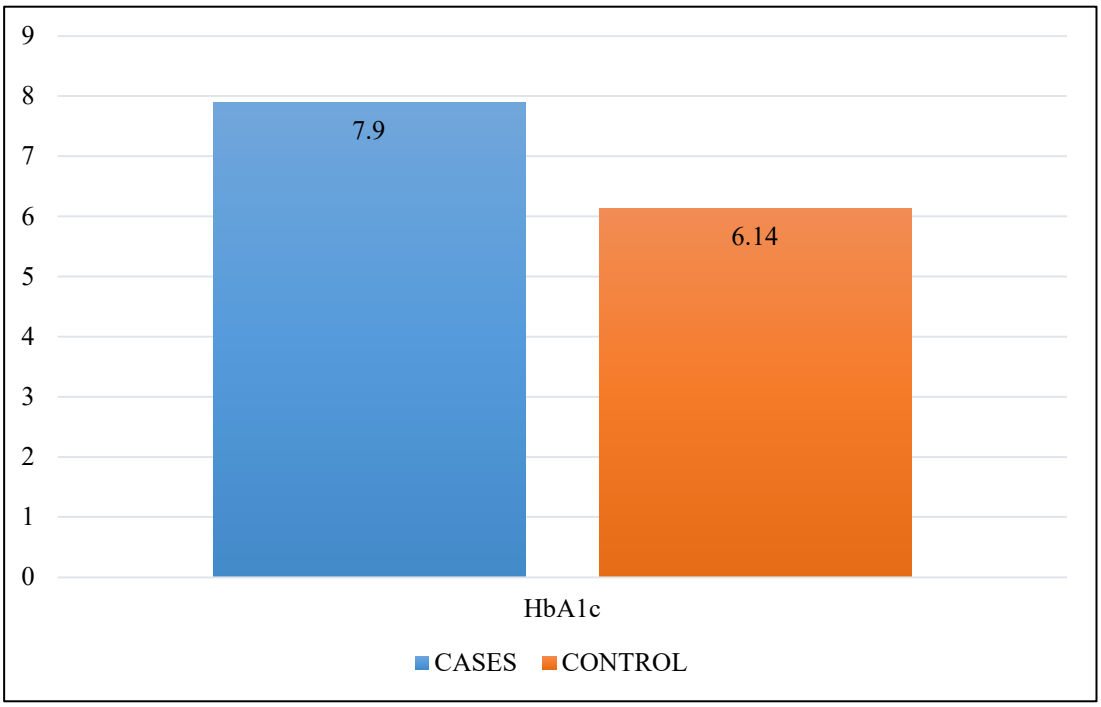
Graph 10: Comparison of Mean Systolic Bp among Cases and Controls



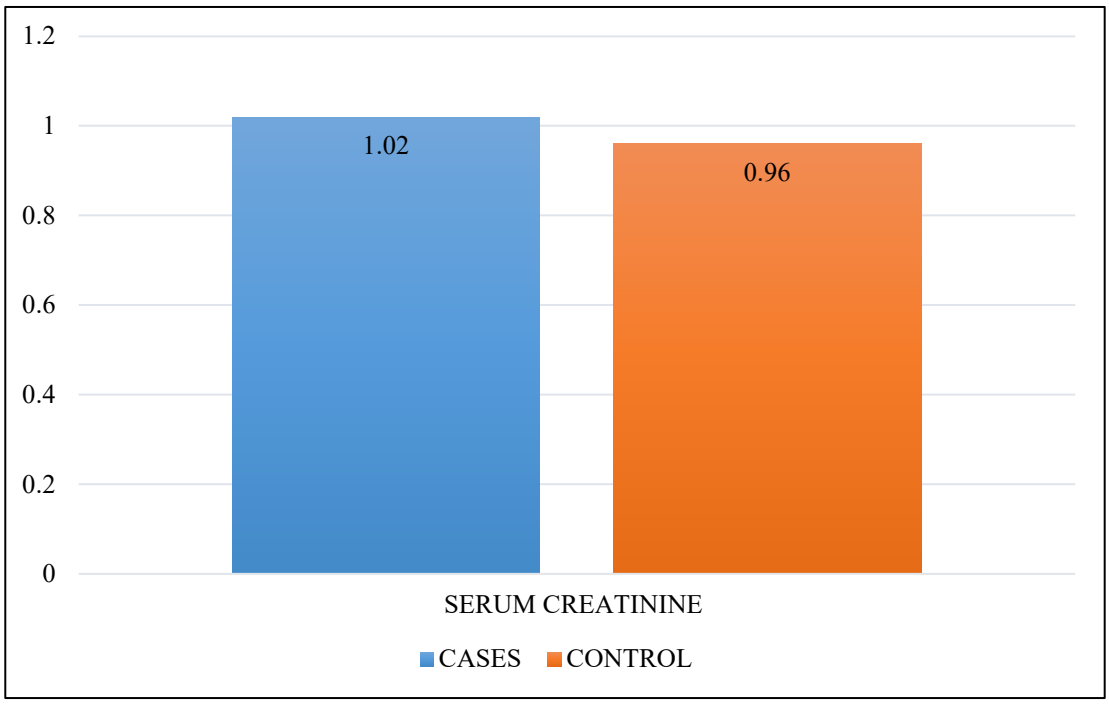
Graph 11: Comparison of Mean Diastolic BP Among Cases and Controls



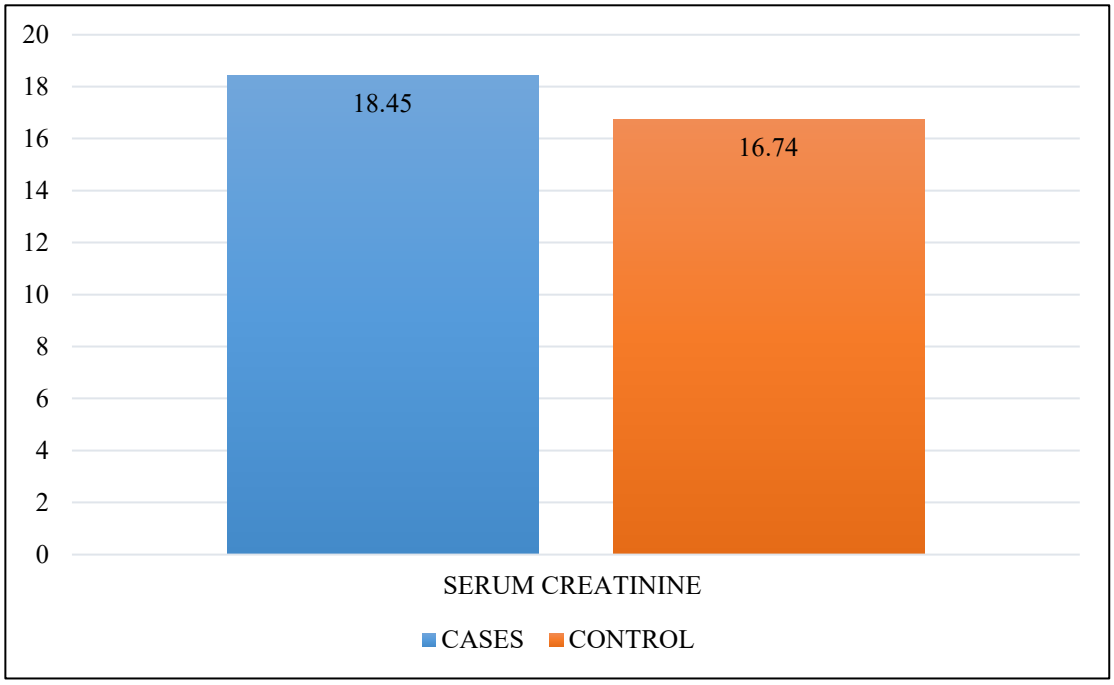
Graph 12: Comparison of Mean Hba1c amongst the Cases and Controls



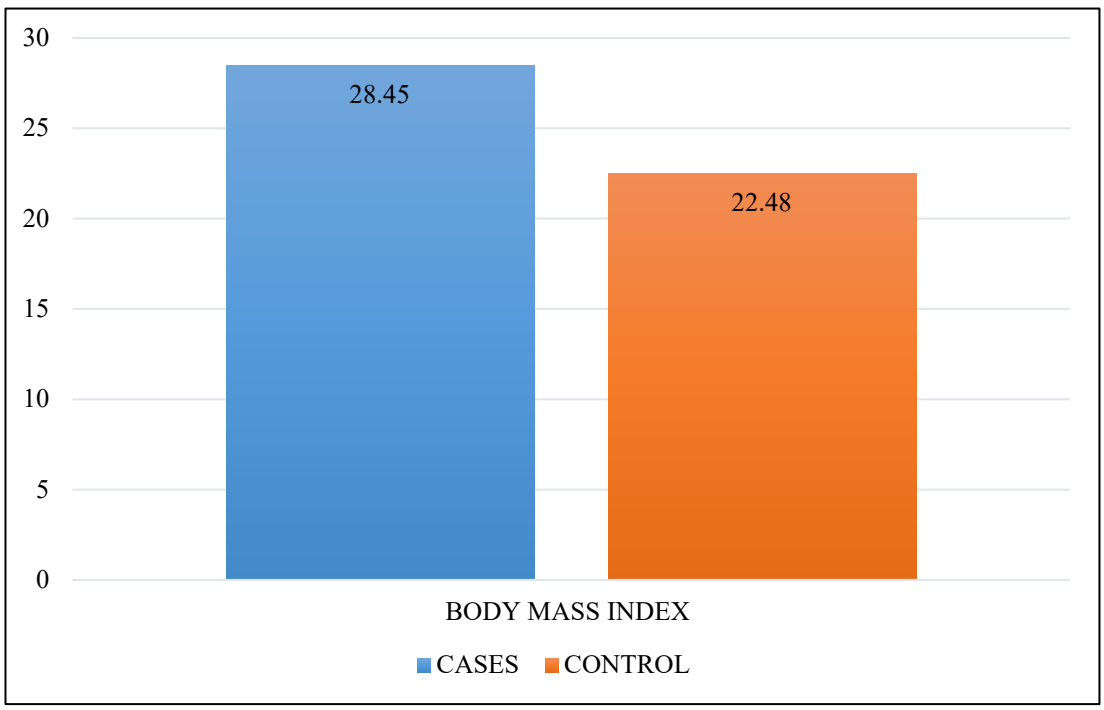
Graph 13: Comparison of Mean Serum Creatinine Levels amongst the Cases and Controls



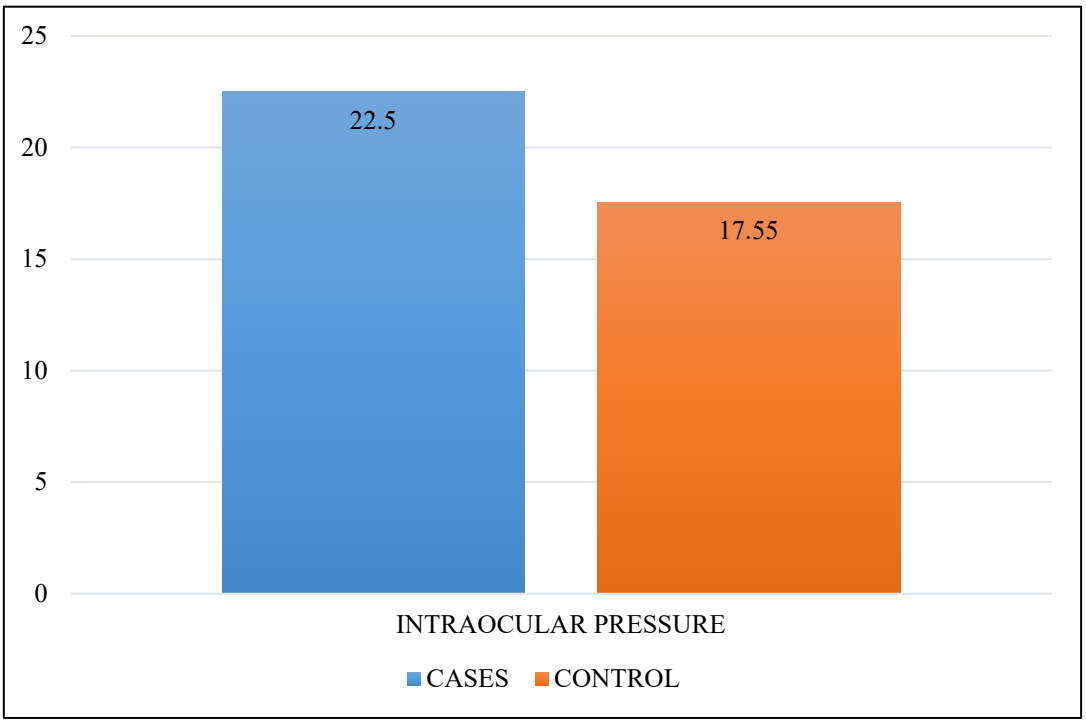
Graph 14: Comparison of Mean Blood Urea Levels amongst the Cases and Controls



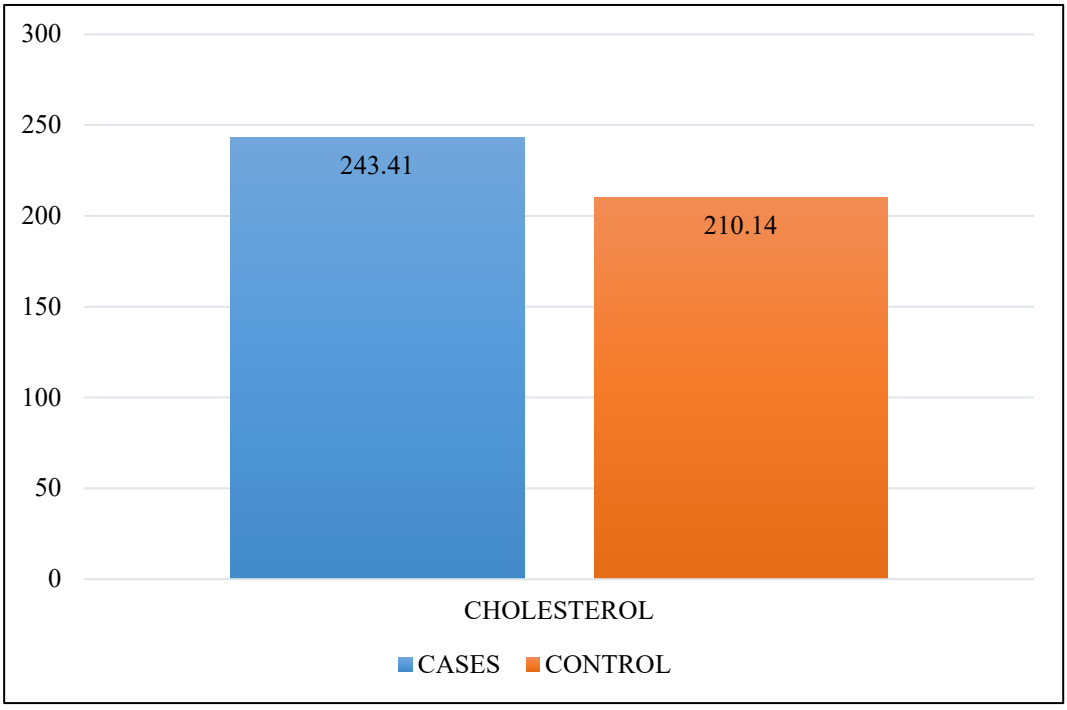
Graph 15: Comparison of Mean Body Mass Index Amongst the Cases and Controls



Graph 16: Comparison of Mean IOP amongst the Cases and Controls



Graph 17: Comparison of Mean Cholesterol Levels amongst the Cases and Controls



DISCUSSION

In the present study majority of the patients belonged to the age group of 61 – 70, i.e., 19, 61.4 %, further 9 patients i.e., 29 % of the study subjects belonged to the age group of 51 – 60 years and 9.6 % of patients belonged to the age group of 40 – 50 years, which is similar to the study by Roger at al[4] which stated that RVO is more prevalent (58%) among above 65 – year – old patients. In the present study, 58 % i.e., 18 patients in each group were males and 42 %, i.e., 13 patients in each group were females, which opposes the study by Roger et al[4] which states that females were 25 % less likely to have RVO.

In the present study 42 % i.e., 13 patients' left eyes were affected in cases and 58 %, i.e., 18 patients' right eyes were affected in cases. In controls, 48 % i.e., 15 patients' left eyes were affected, right eyes were affected in 16 patients i.e., 52 % of the patients in the control group. In our study, no significance was noted in the laterality of the affected eye. There was no significant age and sex – wise distribution noted.

In the present study, overall, the cases had more co – morbidities, 23 (74 %) of the study participants in cases were HTN, followed by 22 (71 %) patients who were obese, followed by 21 (61.2 %) of patients who were DM, 6 (19.3 %) of the study subjects of cases were having glaucoma, 16 (51.6 %) of study subjects in cases were having hyper – lipidaemia, 4 (13 %) of study subjects were having a history of heart disease. The controls had a lower number of co – morbidities, where 11 (35.4 %) of the participants were HTN, 12 (38.7 %), had DM, 10 (32.2 %) had obesity, and only 2 (7 %) of the patients in the control group had a history of heart disease. No patient in

the control group had glaucoma, 6 (19.3 %) of the patients had hyper – lipidaemia. A similar result was found by a study by Mohamed et al[73] which stated that 48 % of RVO is connected to HTN.

HYPERTENSION

In the present study the mean systolic BP was 138.48 ± 14.51 in cases and 122.47 ± 10.66 in controls, this was statistically significant with a t – test value of 4.95 and p – value of 0.001.

The mean diastolic BP in the cases was 89.44 ± 10.54, whereas the mean systolic BP in the controls was 78.94 ± 11.45, this mean difference was statistically significant with a t – value of 3.77 and a p – value of 0.003.

In the study by Shrestha R K, 54 % of the patients had HTN followed by DM which was 8 %, BRAVO'S prevalence was twice that of CRVO[12].

In the study by Bum Joo Choo et al, HTN was seen in 17.3 % in CRVO and BRVO it was 15.2 %, which was followed by BRVO[13].

In another study by Suthasinee Sinawat et al, who studied systemic ab – normalities association with RVO, the researchers found that HTN was seen in 27.55 % of patients[14].

A Korean study by Jin Y B et al found that heart diseases and HTN were significantly associated with BRVO and DM was more common in CRVO[15].

In another study by Robert D Sperduto et al, the results showed that systemic HTN and history of DM were associated with an increased risk of HRVO[16].

According to a study done in National Eye Institute, Bethesda, an increased risk of CRVO was found in people with systemic HTN[17].

In a meta – analysis by Paul R A et al who considered 2,916 cases and controls of about 28,646; a total of 21 studies were included, which found that HTN (OR 3.5) and hyper – lipidaemia (OR 2.5) had a significant association with RVO[21].

In another study by F Martinez et al, the general population and those patients with RVO were compared, the researchers found that the prevalence of HTN was significantly higher in the RVO population, also the proportion of un – diagnosed HTN was more in RVO group[28].

A study by Călugăru D concluded that risk factors for the occurrence of CRVO are to a certain extent similar to those of cardio – vascular diseases (e.g., arterio – sclerosis, arterial systemic HTN, DM, dys – lipidaemia). Hyper – homo – cysteinemia is an essential risk factor for arterio – sclerosis intervening also directly in the local mechanism of causing venous and arterial occlusions. Ocular HTN and glaucoma are risk factors significantly associated with the pathogenesis of CRVO[84].

In another study which was done in the general Japanese population was titled "The Hisayama study[8]", which studied the prevalence and systemic risk factors for RVO after age and sex adjusting. the authors found that RVO had a significant association with HTN and haematocrit values. HTN and high / normal BP were associated with RVO. Other important factors which were associated with RVO were higher diastolic BP, hyper – lipidaemia, higher BMI; higher diastolic BP was an independent significant risk factor[8].

DIABETES MELLITUS

In our study, 61.2 % of the patients were DM, which was almost twice the number noted in another study[86] where 38 % had type 2 DM, in contrast to this; another study by Mohamed et al[87] concluded that only 5 % of RVO cases had been associated with DM.

In the present study, the mean HbA1c in the cases was 7.95 ± 1.22, whereas the mean HbA1c in the controls was 6.14 ± 0.69, this mean difference was statistically significant with the t – value of 7.74 and p – value of 0.001.

According to a study by J G Santigo et al, a total of 19,648 patients (13,571 DM; 6,077 non – DM) were reviewed. The authors found that CRVO prevalence in DM and non – DM were 0.5 % and 0.4 % respectively. PRP and disc neo – vascularisation was commonly seen in these patients[18].

Yun Wang et al conducted a meta – analysis with 1,48,654 cases and 23,768,820 controls, 37 publications data were pooled and analysed. It was concluded that there is a significant association between risk of RVO and DM and HTN with an odds ratio of 1.68, sub – group analysis showed that DM association with CRVO was statistically significant[19]. In a study by Mirko Di Capua et al, DM was significantly more frequent in patients than in controls[8].

In a study by M L Shahsuvaryan et al, DM was associated with an increased risk for CRVO[10]. Shrestha R K et al studied the association of systemic diseases with RVO. The study was done in 100 patients, in these patients with CRVO and BRVO, DM was seen in 84 % of the patients, and DM was significantly associated with CVO[12]. In the study by Bum Joo Choo et al, 1,511 BRVO patients and 417 CRVO

patients were studied, DM was the most commonly associated disease. CRVO group's prevalence of DM and CKD was higher than the BRVO group[13].

Suthasinee Sinawat et al studied systemic ab – normalities associated with RVO and they found that DM was seen in 16.33 % of patients[14.] In another study by Robert D Sperduto et al, the results showed that systemic HTN and history of DM were associated with increased risk of HRVO. The risk of CRVO increased with a history of DM[16].

In a study by R L Funderburk et al, the authors evaluated a series of patients with RVO to determine whether DM patients with RVO developed retinal neo – vascularization more frequently than non – DM. Retinal neo – vascularization occurred in 68.8 % of DM after CRVO compared with 27.8 % of non – DM[88].

GLAUCOMA

In the present study, a total of 6 patients were suffering from glaucoma, of which 6 (100 %) were suffering from POAG. In another similar study[74], 6 patients (12 %) out of 50 had open angle glaucoma, in contrast, another study[33] concluded that glaucoma is the most common ocular risk factor associated with RVO. Among the patients with glaucoma, 16 % had good visual outcomes and 84 % had a poor visual outcome. Among the patients without glaucoma, 52 % had vision improvement and 48 % had worsening of vision.

In a study by Davis R et al, it was found that among glaucoma patients who were separated from the remainder of the population, there was a marked difference in the incidence rate of RVO in the same period (1.85 and 17.3 per 1000 respectively)[89.]

In another meta – analytical study by Xue Yin et al, it was found that glaucoma is a significant risk factor for RVO. It was indicated that OAG (POAG / COAG) could increase the incidence of RVO, particularly for CRVO. Meanwhile, there was less association between PACG and RVO, especially for BRVO[90].

In a study by S S Hayreh et al, it was found that the prevalence of glaucoma / OHT was found to be significantly ($P < 0.0001$) higher in patients with CRVO and HRVO than in the general population. There was no significant difference in the proportion of patients with glaucoma / OHT among the various types of CRVO / HRVO ($P = 0.156$). 48 % of all patients had lower IOP (≥ 2 mm Hg) in the CRVO / HRVO eye than in the fellow (un – involved) eye at their initial evaluation[9].

In another prospective study by Hirota A et al, done in 433 glaucoma patients, 18 (4.2 %) subsequently presented with RVO, 9 had CRVO and 9 exhibited BRVO. 7 of 87 (8.1 %) patients with PACG exhibited RVO, showing the highest incidence among glaucoma types. The incidence of RVO detected by the general OPD clinic was 0.59 % during the same period[91].

In the present study, 19 (61.3 %) of the study subjects in cases were smokers and 12 (38.7 %) were non – smokers, whereas in the control group only 8 (25.8 %) study subjects were smokers and 23 (74.2 %) were non – smokers. The association was statistically significant with chi – square value of 7.939 and p – value of 0.002. In another study[74], 12 patients (50 %) out of 24 males were smokers, which is similar to the study conducted by Klein et al[6] which concluded that smoking as a risk factor, increases the development of RVO by 3 times. Among the smokers, 42 % had good visual outcomes and 58 % had poor visual outcome[6].

In a study by Sherpa D done in 50 patients, 19 (38 %) were CRVO and 31 (62 %) were BRVO. Out of 50 patients of RVO, 12 patients had glaucoma. 1 of the patients had POAG and 1 had PACG. The authors concluded that association of glaucoma as a risk factor for RVO, also the authors opined that evaluation of RVO in glaucoma patients would play an important role in prevention complications[92].

In the present study, ischemic CRVO was seen in 5 (16.5 %) of the cases, non – ischemic CRVO was seen in 4 (13.5 %) of the cases, superior HRVO was seen in only 1 (3 %) of the cases, inferior HRVO was seen in 4 (13.5 %) of the patients. ST BRVO was seen in maximum number with 8 (26.2 %) patients, IT BRVO was seen in 5 (16.5 %) patients and MT BRVO was seen in 3 (10.8 %) of the cases.

In a study by Ponto et al, it was found that out of 92 patients with RVO, 46 (50 %) had CRVO, 31 (33.7 %) had BRVO, and 15 (16.3 %) had HRVO[93].

The mean IOP in the cases was 22.5 ± 4.6, whereas the mean IOP in the controls was 17.55 ± 3.58, this mean difference was statistically significant with a t – value of 4.77 and p – value of 0.001.

In a study by J Frucht et al, where the IOP of 59 patients with RVO was studied. CRVO was diagnosed in 24 patients and BRVO was diagnosed in the rest. The IOP of sex and age – matched group of controls was also studied for comparison. The IOPs in the CRVO group were significantly different from those of the matched controls ($p < 0.001$), this was in agreement with our study. Further in the same study, a statistically significant difference of a lesser degree ($p < 0.05$) was also found in a comparison of the IOP of the BRVO group with those of their sex and age – matched group of controls[94].

S S Hayreh et al found that the prevalence of higher IOP was found to be significantly ($P < 0.0001$) higher in patients with CRVO and HRVO[9].

According to the study conducted by The Eye Disease Case – Control Study Group at The University of Illinois, Chicago[61]. Higher levels of IOP were significantly associated with BRVO.

SERUM CREATININE

The mean serum creatinine in the cases was 1.02 ± 0.24, whereas the mean creatinine in the controls was 6.14 ± 0.69, this mean difference was statistically not significant with a t – value of 1.38 and p – value of 0.170. The mean blood urea in the cases was 18.45 ± 3.45, whereas the mean blood urea in the controls was 16.74 ± 4.58, this mean difference was statistically not significant with a t – value of 1.66 and p – value of 0.102. The normal serum creatinine levels for adult men are 0.74 to 1.35 mg / dL (65.4 to 119.3 micromoles / L) and for adult women it ranged from 0.59 to 1.04 mg / dL.

In one of the studies by R Klien et al, it was found that the 15 – year cumulative incidence of BRVOs was higher in persons who had higher serum creatinine, this was unlike our study results[95].

In another study by Bum Joo Cho et al, in patients of CRVO, blood urea and serum creatinine were significantly elevated ($P = 0.002$). In the Blue Mountains Eye Study, the serum creatinine level was not associated with the development of RVO in a 10 – year follow – up period, which was in agreement with our study[1].

In another study by Steven Martin et al, a thorough multiple linear regression analysis revealed significant relationships between homo – cysteine levels and the presence of RVO ($p = 0.0002$) and serum creatinine ($p = 0.001$)[96].

BODY MASS INDEX

In the present study, the mean BMI in the cases was 28.45 ± 4.66, whereas the mean BMI in the controls was 22.48 ± 3.14, this mean difference was statistically significant with a t – value of 5.91 and p – value of 0.0001.

In a study by Lam H D, it was found that an increased risk of BRVO was found in patients with a history of systemic HTN, hyper – lipidaemia, and increased BMI[97].

In another study by Suguda C et al, the authors concluded that factors predicting the incidence of RVO included obesity with an odds ratio of 2.16. In another study by Yu Yen Chen et al, concluded that RVO patients were observed to have obesity[98].

Yu Yen et al demonstrated that obesity is one of the major risk factors of RVO[99].

CHOLESTEROL / HYPER – LIPIDEMIA

The mean cholesterol levels in the cases were 243.41 ± 35.11, whereas the mean cholesterol levels in the controls were 210.14 ± 30.44, this mean difference was statistically significant with a t – value of 3.98 and p – value of 0.001.

Multiple studies have studied the risk factors evaluation HTN[38] (76 %) was found to be the most common risk factor associated with RVO, these findings correlated with the Beaver Dam eye study[6] which suggest that after controlling for age, the incidence of BRVO was associated with HTN, serum lipids, BMI, WBC count, alcohol consumption, aspirin use, glaucoma, IOP, ocular HTN.

The mean values of various investigations of the patients in cases and control groups were compared using unpaired student t – test, invariably the mean values were higher in cases, out of 8 variables, 6 were also statistically significant. The following table compares different risk factors:

Table 11: Comparison of Various Studies

Study	No of pts	Mean age (Yrs)	CRVO / BRVO ratio	HT present	DM Present	Hyper – lipidaemia	Glaucoma	Smoking	Obesity
Present Study	31	59.93	9 / 22	74 %	61.2 %	15.6 %	19.3 %	61.3 %	71 %
Tony Yuan – Ting Chen et al	805	-	-	83.6 %	19.8 %	29.8 %	12.4 %	19.2 %	11.1 %
Mirko Di Capua et al	117	-	62 / 48	79.2 %	29.2 %	-	-	26 %	83.3 %
Shrestha RK et al	100	-	34 / 66	54 %	8 %	-	-	-	-
Sinawat S et al	100	36.5 ± 8.7	70 / 30	55.1 %	22 %	5 %	-	-	-
Karolina Kaźmierc Zak et al	23	-	-	81.8 %	6.1 %	21.7 %	-	(4.3 %)	-
Suthasine e Sinawat et al	98		68 / 30	27.55 %	16.33 %	7 %	-	15	-

CONCLUSION

The elderly age group, age greater than 60 years, are more prone to retinal venous occlusion diseases. Males were more affected by RVOs in the present study.

Cases had a greater number of co – morbidities as compared to controls. Primary open angle glaucoma was the most common type of glaucoma associated with RVOs and there was a significant correlation between smoking and RVOs.

BRVOs are more common than CRVOs. ST BRVO was the most common type of RVO. Ischemic CRVO was more common than non – ischemic CRVO.

In these cases, the systolic Blood Pressure and diastolic Blood Pressure were higher, which was significant statistically. In cases, the HbA1c levels were higher, which was significant statistically. Serum creatinine and blood urea levels were slightly higher in cases but this was not significant statistically. BMI, IOP, and cholesterol levels were higher in cases, and this was statistically significant.

SUMMARY

- In the present study majority of the patients belonged to the age group of 61 – 70, i.e., 19, 61.4 %, further 9 patients i.e., 29 % of the study subjects belonged to the age group of 51 – 60 years and 9.6 % of the patients belonged to the age group of 40 – 50 years
- In the present study, 58 % i.e., 18 patients in each group were males and 42 %, i.e., 13 patients in each group were females
- In the present study 42 % i.e., 13 patients' left eyes were affected and 58 %, i.e., 18 patients' right eyes were affected in cases
- In controls, 48 % i.e., 15 patients' left eyes were affected, right eyes were affected in 16 patients i.e., 52 % of the patients. In our study, no significance was noted in the laterality of the affected eye. There was no significant age and sex – wise distribution noted
- In the present study, overall, the cases had more co – morbidities, 23 (74 %) of the study participants in cases had HTN, followed by 22 (71 %) patients who were obese, followed by 21 (61.2 %) of patients who had DM
- 6 (19.3 %) of the study subjects of cases were having glaucoma, 16 (51.6 %) of the study subjects in cases were having hyper – lipidaemia, 4 (13 %) of study subjects were having a history of heart disease
- The controls had a lower number of co – morbidities where 11 (35.4 %) of the participants had HTN, 12 (38.7 %) had DM, 10 (32.2 %) had obesity, only 2 (7%) of the patients in the control group had a history of heart disease. No patient in the control group had glaucoma, 6 (19.3 %) of the patients had hyper – lipidaemia

- In the present study, 19 (61.3 %) of the study subjects in cases were smokers and 12 (38.7 %) were non – smokers, whereas in the control group only 8 (25.8 %) study subjects were smokers and 23 (74.2 %) were non – smokers. The association was statistically significant with chi – square value of 7.939 and p – value of 0.002
- In the present study, ischemic CRVO was seen in 5 (16.5 %) of the cases, non – ischemic CRVO was seen in 4 (13.5 %) of the cases, superior HRVO was seen in only 1 (3 %) of the cases, inferior HRVO was seen in 4 (13.5 %) of the patients. ST BRVO was seen in maximum number with 8 (26.2 %) patients, IT BRVO was seen in 5 (16.5 %) patients and MT BRVO was seen in 4 (10.8 %) of the cases
- The mean systolic BP in the cases was 138.48 ± 14.51, whereas the mean systolic BP in the controls was 122.47 ± 10.66, this difference in mean was statistically significant with a t – value of 4.95 and p – value of < 0.001
- The mean diastolic BP in the cases was 89.44 ± 10.54, whereas the mean diastolic BP in the controls was 78.94 ± 11.45, this difference in mean was statistically significant with a t – value of 3.77 and p – value of 0.003
- The mean HbA1c in the cases was 7.95 ± 1.22, whereas the mean HbA1c value in the controls was 6.14 ± 0.69, this difference in mean was statistically significant with a t – value of 7.74 and p – value of 0.001
- The mean serum creatinine levels in the cases were 1.02 ± 0.24, whereas the mean creatinine levels in the controls were 6.14 ± 0.69, this difference in the mean was statistically not significant with a t – value of 1.38 and p – value of 0.170

- The mean blood urea in the cases was 18.45 ± 3.45, whereas the mean blood urea in the controls was 16.74 ± 4.58, this difference in mean was statistically not significant with a t – value of 1.66 and p – value of 0.102
- The mean BMI in the cases was 28.45 ± 4.66, whereas the mean BMI in the controls was 22.48 ± 3.14, this difference in mean was statistically significant with a t – value of 5.91 and p – value of 0.0001

BIBLIOGRAPHY

1. Cugati S, Wang JJ, Rochtchina E, et al. Ten-year incidence of retinal vein occlusion in an older population: the Blue Mountains Eye Study. Arch Ophthalmol 2006;124(5):726–32.

2. Green WR, Chan CC, Hutchins GM, et al. Central retinal vein occlusion: a prospective histopathologic study of 29 eyes in 28 cases. Retina 1981;1(1):27–55.

3. Frangieh GT, Green WR, Barraquer-Somers E, et al. Histopathologic study of nine branch retinal vein occlusions. Arch Ophthalmol 1982;100(7):1132–40.

4. Rogers S, McIntosh RL, Cheung N, et al. The prevalence of retinal vein occlusion: pooled data from population studies from the United States, Europe Asia, and Australia. Ophthalmology 2010;117(2):313–19.

5. Ho M, Liu DT, Lam DS, et al. Retinal vein occlusions, from basics to the latest treatment. Retina 2015;36(3):432–48.

6. Klein R, Moss SE, Meuer SM, et al. The 15-year cumulative incidence of retinal vein occlusion: the Beaver Dam Eye Study. Arch Ophthalmol 2008;126(4):513–18.

7. Marcucci R, Bertini L, Giusti B, Brunelli T, Fedi S, Cellai AP, Poli D, Pepe G, Abbate R, Prisco D. Thrombophilic risk factors in patients with central retinal vein occlusion. Thrombosis and haemostasis. 2001; 86(09): 722-6.

8. Prisco D, Marcucci R. Retinal vein thrombosis: risk factors, pathogenesis, and therapeutic approach. Pathophysiology of haemostasis and thrombosis. 2002 Sep 1;32(5-6):308-11.

9. Hayreh SS, Zimmerman B, McCarthy MJ, Podhajsky P. Systemic diseases associated with various types of retinal vein occlusion. American journal of ophthalmology. 2001 Jan 1;131(1):61-77.

10. Shahsuvaryan ML, Melkonyan AK. Central retinal vein occlusion risk profile: a case – control study. European journal of ophthalmology. 2003 Jun;13(5):445-52.

11. Chen TY, Uppuluri S, Zarbin MA, Bhagat N. Systemic and Ocular comorbidities in Central Retinal Vein Occlusion Patients Older and Younger than 40 Years Old. Investigative Ophthalmology & Visual Science. 2020 Jun 10;61(7):1329-31.

12. Shrestha RK, Shrestha JK, Koirala S, Shah DN. Association of systemic diseases with retinal vein occlusive disease. JNMA; the journal of the Nepal Medical Association. 2006 Apr 1;45(162):244-8.

13. Cho BJ, Bae SH, Park SM, Shin MC, Park IW, Kim HK, Kwon S. Comparison of systemic conditions at diagnosis between central retinal vein occlusion and branch retinal vein occlusion. PloS one. 2019 Aug 8;14(8): e0220880.

14. Sinawat S, Bunyavee C, Ratanapakorn T, Sinawat S, Laovirojjanakul W, Yospaiboon Y. Systemic abnormalities associated with retinal vein occlusion in young patients. Clinical Ophthalmology (Auckland, NZ). 2017; 11:441.

15. Jin YB, Park HS, Toon IH. Comparison of Risk Factors Associated with Central and Branch Retinal Vein Obstruction. Journal of the Korean Ophthalmological Society. 1998 Oct 1;39(10):2343-52.

16. Sperduto RD, Hiller R, Chew E, Seigel D, Blair N, Burton TC, Farber MD, Gragoudas ES, Haller J, Seddon JM, Yannuzzi LA. Risk factors for Hemi retinal vein occlusion: comparison with risk factors for central and branch retinal vein occlusion: the eye disease case-control study. Ophthalmology. 1998 May 1;105(5):765-71.

17. Risk Factors for Central Retinal Vein Occlusion. Arch Ophthalmol. 1996;114(5):545–554.

18. Santiago JG, Walia S, Sun JK, Cavallerano JD, Haddad ZA, Aiello LP, Silva PS. Influence of diabetes and diabetes type on anatomic and visual outcomes following central rein vein occlusion. Eye. 2014 Mar;28(3):259-68.

19. Wang Y, Wu S, Wen F, Cao Q. Diabetes mellitus as a risk factor for retinal vein occlusion: A meta-analysis. Medicine. 2020 Feb;99(9).

20. Kaźmierczak K, Żuchowski P, Łapińska-Duczmal K, Zabel K, Sikorska Z, Pol M, Seredyka-Burduk M, Sikorski BL, Malukiewicz G. Role of Advanced Glycation End Products in Assessment of Diabetes Mellitus as a Risk Factor for Retinal Vein Occlusion. Applied Sciences. 2021 Jan;11(17):7934.

21. O'Mahoney PR, Wong DT, Ray JG. Retinal vein occlusion and traditional risk factors for atherosclerosis. Archives of Ophthalmology. 2008 May 1;126(5):692-9.

22. Salaun N, Delyfer MN, Rougier MB, Korobelnik JF. Assessment of risk factors for retinal vein occlusions in patients under 60 years of age. Journal francais d'ophtalmologie. 2007 Nov 1;30(9):918-23.

23. Kuo JZ, Lai CC, Ong FS, Shih CP, Yeung L, Chen TL, Chen KJ, Wu WC. Central retinal vein occlusion in a young Chinese population: risk factors and associated morbidity and mortality. Retina (Philadelphia, Pa.). 2010 Mar;30(3):479.

24. Paik DW, Han K, Kang SW, Ham DI, Kim SJ, Chung TY, Lim DH. Differential effect of obesity on the incidence of retinal vein occlusion with and without diabetes: a Korean nationwide cohort study. Scientific reports. 2020 Jun 29;10(1):1-9.

25. Risk Factors for Branch Retinal Vein Occlusion. American Journal of Ophthalmology September, 1993;116: 286-296.

26. Yong Un Shin, Heeyon Cho, Jong Min Kim, Kunho Bae, Min Ho Kang, Jae Pil Shin, Eunwoo Nam, Se Woong Kang. Prevalence and associated factors of retinal vein occlusion in the Korean National Health and Nutritional Examination Survey, 2008-2012. A cross-sectional observational study. Medicine 2016; 95:44 (e5185).

27. Kassiani Giannaki, Marianna Politou, Alexandros Rouvas, EfrossyniMerkouri, AnthiTravlou, Panayiotis Theodosiadis, and ArgyriGialeraki. Retinal vein occlusion: genetic predisposition and systemic risk factors. Blood Coagulation and Fibrinolysis 2013; 24:279-83, DOI: 10.1097/MBC.0b013e32835bfda1.

28. F Martinez, E Furio, MJ Fabia, AV Perez, V Gonzalez-Albert, G Rojo-Martinez, MT Martinez-Larrad, FJ Soriguer, M Serrano-Rios, FJ Chaves, JC Martin-Escudero, MJ Garcia-Fuster. Risk Factors associated with retinal vein occlusion. The International Journal of Clinical Practice, 2014; 68; 7:871-81, doi: 10.1111/ijcp.12390

29. Sohan Singh Hayreh, Bridget Zimmerman, Mark J. McCarthy, and Patricia Podhajsky. Systemic Diseases Associated with Various Types of Retinal Vein Occlusion. American Journal of Ophthalmology, 2001; 131; 1: 61-77.

30. Yun-Shin Chang, Shih-Feng Weng, Chun Chang, Jhi-Joung Wang, Sung-Huei Tseng, Jiu-Yao Wang, and Ren-Long Jan. Risk of Retinal Vein Occlusion Following End-Stage Renal Disease. Medicine Journal,2016; 95; 16; e3474.

31. K. A. Ponto, H. Elbaz, T. Peto, D. Laubert-Reh, H. Binder, P. S. Wild, K. Lackner, N. Pfeiffer and A. Mirshahi. Prevalence and risk factors of retinal vein occlusion: the Gutenberg Health Study. Journal of Thrombosis and Haemostasis 2015; 13: 1254-63.

32. B Fiebai, CS Ejimadu, RD Komolafe; Department of Ophthalmology, University of Port Harcourt Teaching Hospital, Nigeria. Incidence and risk factors for retinal vein occlusion at the University of Port Harcourt Teaching Hospital, Port Harcourt, Nigeria. Nigerian Journal of Clinical Practice, 2014; 17:4.

33. Katarzyna Michalska-Malecka, Dorota Spiewak, LudwilaSlowinska-Lozynska and Justyna Sierocka-Stepien. Influence of hemorheological factors on the

development of retinal vein occlusion. Clinical Hemorheology and Microcirculation,2016; 63: 69-76, doi: 10.3233/CH-162056

34. Bertelsen M, Linneberg A, Christoffersen N, et al. Mortality in patients with central retinal vein occlusion. Ophthalmology 2014;121(3):637–42.

35. Natural history and clinical management of central retinal vein occlusion. The Central Vein Occlusion Study Group. Arch Ophthalmol 1997;115(4):486.

36. Hayreh SS, Klugman MR, Beri M, Kimura AE, Podhajsky P. Differentiation of ischemic from non-ischemic central retinal vein occlusion during the early acute phase. Graefe's archive for clinical and experimental ophthalmology. 1990 May 1;228(3):201-17.

37. May DR, Klein ML, Peyman GA, et al. Xenon arc pan-retinal photocoagulation for central retinal vein occlusion: a randomized prospective study. Br J Ophthalmol 1979;63(11): 725–34.

38. Magargal LE, Brown GC, Augsburger JJ, et al. Neovascular glaucoma following central retinal vein occlusion. Ophthalmology 1981;88(11):1095–101.

39. Fong AC, Schatz H, McDonald HR, et al. Central retinal vein occlusion in young adults (papillophlebitis). Retina 1992;12(1):3–11. 20. Chan CK, Ip MS, Vanveldhuisen PC, et al. SCORE Study report #11: incidences of neovascular events in eyes with retinal vein occlusion. Ophthalmology 2011;118(7):1364–72.

40. Werner JU, Böhm F, Lang GE, Dreyhaupt J, Lang GK, Enders C. Comparison of foveal avascular zone between optical coherence tomography angiography

and fluorescein angiography in patients with retinal vein occlusion. PloS one. 2019 Jun 4;14(6): e0217849.

41. Tsui I, Kaines A, Havunjian MA, et al. Ischemic index and neovascularization in central retinal vein occlusion. Retina 2011;31(1):105–10.

42. Nicheolson L, Vazquez-alfageme C, Ramu J, et al. Validation of concentric rings method as a topographic measure of retinal nonperfusion in ultra-widefield fluorescein angiography. Am J Ophthalmol 2015;160(6):1217–25. e2.

43. Catier A, Tadayoni R, Paques M, et al. Characterization of macular edema from various etiologies by optical coherence tomography. Am J Ophthalmol 2005;140(2):200–6.

44. Lerche RC, Schaudig U, Scholz F, et al. Structural changes of the retina in retinal vein occlusion – imaging and quantification with optical coherence tomography. OphthalmicSurg Lasers 2001;32(4):272–80.

45. Tsai G, Banaee T, Conti FF, Singh RP. Optical coherence tomography angiography in eyes with retinal vein occlusion. Journal of ophthalmic & vision research. 2018 Jul;13(3):315.

46. Guven D, Sayinalp N, Kalayci D, et al. Risk factors in central retinal vein occlusion and activated protein C resistance. Eur J Ophthalmol 1999;9(1):43–8.

47. A randomized clinical trial of early panretinal photocoagulation for ischemic central vein occlusion. The Central Vein Occlusion Study Group N report. Ophthalmology 1995;102(10):1434–44.

48. Tsui I, Kaines A, Havunjian MA, et al. Ischemic index and neovascularization in central retinal vein occlusion. Retina 2011;31(1):105–10.

49. Kahook MY, Schuman JS, Noecker RJ. Intravitreal bevacizumab in a patient with neovascular glaucoma. Ophthalmic Surg Lasers Imaging 2006;37 (2):144–6.

50. Evaluation of grid pattern photocoagulation for macular edema in central vein occlusion. The Central Vein Occlusion Study Group M report. Ophthalmology 1995;102(10):1425–33.

51. Cekic O, Chang S, Tseng JJ, et al. Intravitreal triamcinolone treatment for macular edema associated with central retinal vein occlusion and hemiretinal vein occlusion. Retina 2005;25(7):846–50.

52. Ip MS, Scott IU, VanVeldhuisen PC, et al. A randomized trial comparing the efficacy and safety of intravitreal triamcinolone with observation to treat vision loss associated with macular edema secondary to central retinal vein occlusion: the Standard Care vs corticosteroid for Retinal Vein Occlusion (SCORE) study report 5. Arch Ophthalmol 2009;127(9):1101–14.

53. Haller JA, Bandello F, Belfort R Jr, et al. Dexamethasone intravitreal implant in patients with macular edema related to branch or central retinal vein occlusion twelve-month study results. Ophthalmology 2011;118(12):2453–60.

54. Priglinger SG, Wolf AH, Kreutzer TC, et al. Intravitreal bevacizumab injections for treatment of central retinal vein occlusion: six-month results of a prospective trial. Retina 2007;27(8):1004–12.

55. Epstein DL, Algvere PV, von Wendt G, et al. Bevacizumab for macular edema in central retinal vein occlusion: a prospective, randomized, double masked clinical study. Ophthalmology 2012;119(6):1184–9.

56. Campochiaro PA, Brown DM, Awh CC, et al. Sustained benefits from ranibizumab for macular edema following central retinal vein occlusion: twelve-month outcomes of a phase III study. Ophthalmology 2011;118(10):2041–9.

57. Brown DM, Heier JS, Clark WL, et al. Intravitreal aflibercept injection for macular edema secondary to central retinal vein occlusion: 1-year results from the phase 3 Copernicus Study. Am J Ophthalmol 2013;155(3):429–37. e7.

58. Heier JS, Campochiaro PA, Yau L, et al. Ranibizumab for macular edema due to retinal vein occlusions: long-term follow-up in the HORIZON Trial. Ophthalmology 2012;119(4):802–9.

59. Larsen M, Waldstein SM, Boscia F, et al; CRYSTAL Study Group. Individualized ranibizumab regimen driven by stabilization criteria for central retinal vein occlusion: twelve-month results of the CRYSTAL study. Ophthalmology 2016; 123:1101–11.

60. Holz FG, Roider J, Ogura Y, et al. VEGF Trap-Eye for macular oedema secondary to central retinal vein occlusion: 6-month results of the phase III GALILEO study. Br J Ophthalmol 2013; 97:278–84.

61. Khurana R. NEWTON study: intravitreal aflibercept injection for previously treated macular edema associated with central retinal vein occlusions. Paper

presented at American Academy of Ophthalmology 2014 Annual Meeting; October 17–21, 2014; Chicago.

62. Quinlan PM, Elman MJ, Bhatt AK, et al. The natural course of central retinal vein occlusion. Am J Ophthalmol 1990;110(2):118–23.

63. Baseline and early natural history report. The Central Vein Occlusion Study. Arch Ophthalmol 1993;111(8):1087–95.

64. Duker JS, Brown GC. Anterior location of the crossing artery in branch retinal vein occlusion. Arch Ophthalmol 1989;107(7):998–1000.

65. Argon laser scatter photocoagulation for prevention of neovascularization and vitreous hemorrhage in branch vein occlusion. A randomized clinical trial. Branch Vein Occlusion Study Group. Arch Ophthalmol 1986;104(1):34–41.

66. Spaide RF, Lee JK, Klancnik JK Jr, et al. Optical coherence tomography of branch retinal vein occlusion. Retina 2003;23(3):34353. Ehlers JP, Fekrat S. Retinal vein occlusion: beyond the acute event. SurvOphthalmol 2011;56(4):281–99.

67. Argon laser photocoagulation for macular edema in branch vein occlusion. The Branch Vein Occlusion Study Group. Am J Ophthalmol 1984; 98:271–82.

68. Risk Factors for Branch Retinal Vein Occlusion. American Journal of Ophthalmology September, 1993;116: 286-296.

69. Esrick E, Subramanian ML, Heier JS, et al. Multiple laser treatments for macular edema attributable to branch retinal vein occlusion. Am J Ophthalmol 2005;139(4):653–7.

70. Prasad PS, Oliver SC, Coffee RE, et al. Ultra-wide-field angiographic characteristics of branch retinal and hemicentral retinal vein occlusion. Ophthalmology 2010;117(4): 780–4.

71. Scott IU, Ip MS, VanVeldhuisen PC, et al. A randomized trial comparing the efficacy and safety of intravitreal triamcinolone with standard care to treat vision loss associated with macular edema secondary to branch retinal vein occlusion: the Standard Care vs Corticosteroid for Retinal Vein Occlusion (SCORE) study report 6. ArchOphthalmol 2009;127(9):1115–28.

72. Brown DM, Campochiaro PA, Bhisitkul RB, et al. Sustained benefits from ranibizumab for macular edema following branch retinal vein occlusion: 12-month outcomes of a phase III study. Ophthalmology 2011;118(8):1594–602.

73. Ehlers JP, Decroos FC, Fekrat S. Intravitreal bevacizumab for macular edema secondary to branch retinal vein occlusion. Retina 2011;31(9):1856–62.

74. Campochiaro PA, et al. Intravitreal aflibercept for macular edema following branch retinal vein occlusion: the 24-week results of the VIBRANT study. Ophthalmology 2015; 122:538–44.

75. Campochiaro PA, et al. Scatter photocoagulation does not reduce macular edema or treatment burden in patients with retinal vein occlusion: the RELATE Trial. Ophthalmology 2015;122(7):1426–37.

76. Tadayoni R, Waldstein SM, Boscia F, et al. BRIGHTER study group. Individualized stabilization criteria-driven ranibizumab versus laser in branch retinal vein occlusion: six-month results of BRIGHTER. Ophthalmology 2016; 123:1332–44.

77. Glacet-Bernard A, Coscas G, Chabanel A, et al. Prognostic factors for retinal vein occlusion: prospective study of 175 cases. Ophthalmology1996; 103(4):551–60.

78. Chobanion AV, Bakris GL, Black HR. The seventh report of the joint national committee on prevention, detection, evaluation and treatment of high blood pressure, The JNC 8 report, JAMA 2003;289:2560-72.

79. American Diabetes Association. 2. Classification and diagnosis of diabetes: standards of medical care in diabetes—2020. Diabetes Care. 2020 Jan 1;43(Supplement 1): S14-31.

80. Salazar JH. Overview of urea and creatinine. Laboratory medicine. 2014 Feb 1;45(1): e19-20.

81. Centre for Disease Control (CDC). Body Mass Index: Consideration for Practitioners. www.cdc.gov/obesity/downloads/BMIforPactitioners.pdf

82. Centre for disease control (CDC). Definition of smoking. http://dhds.cdc.gov/guides/healthtopics/indicator?I=smokingstatus

83. Stone NJ, Robinson JG, Lichtenstein AH, Bairey Merz CN, Blum CB, Eckel RH, Goldberg AC, Gordon D, Levy D, Lloyd-Jones DM, McBride P. 2013 ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Journal of the American College of Cardiology. 2014 Jul 1;63(25 Part B):2889-934.

84. Călugăru D. Risk factors in central retinal vein occlusion. Oftalmologia (Bucharest, Romania: 1990). 2011 Jan 1;55(2):27-37.

85. Arakawa S, Yasuda M, Nagata M, et al. Nine-year incidence and risk factors for retinal vein occlusion in a general Japanese population: the Hisayama Study. Invest Ophthalmol Vis Sci 2011;52(8):5905–9.

86. Battaglia Parodi M, Saviano S, Bergamini L, Ravalico G: Grid laser treatment of macular edema in macular branch retinal vein occlusion. Doc Ophthalmol 1999, 97(3–4):427–431.

87. Faghihi H, Piri N, Esfahani M, Aalami Z, Lashay A, Piri N, Faghihi S: Intravitreal bevacizumab vs. a combination of bevacizumab and triamcinolone vs. sham treatment in Central Retinal Vein Occlusion. American Academy of Ophthalmology Meeting Archives; 2008:271. http://aao.scientificposters.com/epsView.cfm?8o86zyfE5SMw0%2FURIHEaEPee1tSQzRJzYw6PAGSTN9mNNuonO4x%2FYg%3D%3D

88. Funderburk RL, Feinberg EB. Diabetes as a risk factor for retinal neovascularization in retinal vein occlusion. Annals of ophthalmology. 1989 Feb 1;21(2):65-6.

89. David R, Zangwill L, Badarna M, Yassur Y. Epidemiology of retinal vein occlusion and its association with glaucoma and increased intraocular pressure. Ophthalmologica. 1988;197(2):69-74.

90. Yin X, Li J, Zhang B, Lu P. Association of glaucoma with risk of retinal vein occlusion: A meta-analysis. Acta ophthalmologica. 2019 Nov;97(7):652-9.

91. Hirota A, Mishima HK, Kiuchi Y. Incidence of retinal vein occlusion at the Glaucoma Clinic of Hiroshima University. Ophthalmologica. 1997;211(5):288-91.

92. Sherpa D, Shakya S, Shrestha JK. Association of primary glaucomas with retinal vein occlusion. Kathmandu University medical journal (KUMJ). 2008 Jan 1;6(1):49-54.

93. Ponto KA, Scharrer I, Binder H, Korb C, Rosner AK, Ehlers TO, Rieser N, Grübel NC, Rossmann H, Wild PS, Feltgen N. Hypertension and multiple cardiovascular risk factors increase the risk for retinal vein occlusions: results from the Gutenberg Retinal Vein Occlusion Study. Journal of hypertension. 2019 Jul 1;37(7):1372-83.

94. Frucht JO, Shapiro AM, Merin SA. Intraocular pressure in retinal vein occlusion. British journal of ophthalmology. 1984 Jan 1;68(1):26-8.

95. Klein R, Moss SE, Meuer SM, Klein BE. The 15-year cumulative incidence of retinal vein occlusion: the Beaver Dam Eye Study. Archives of ophthalmology. 2008 Apr 1;126(4):513-8.

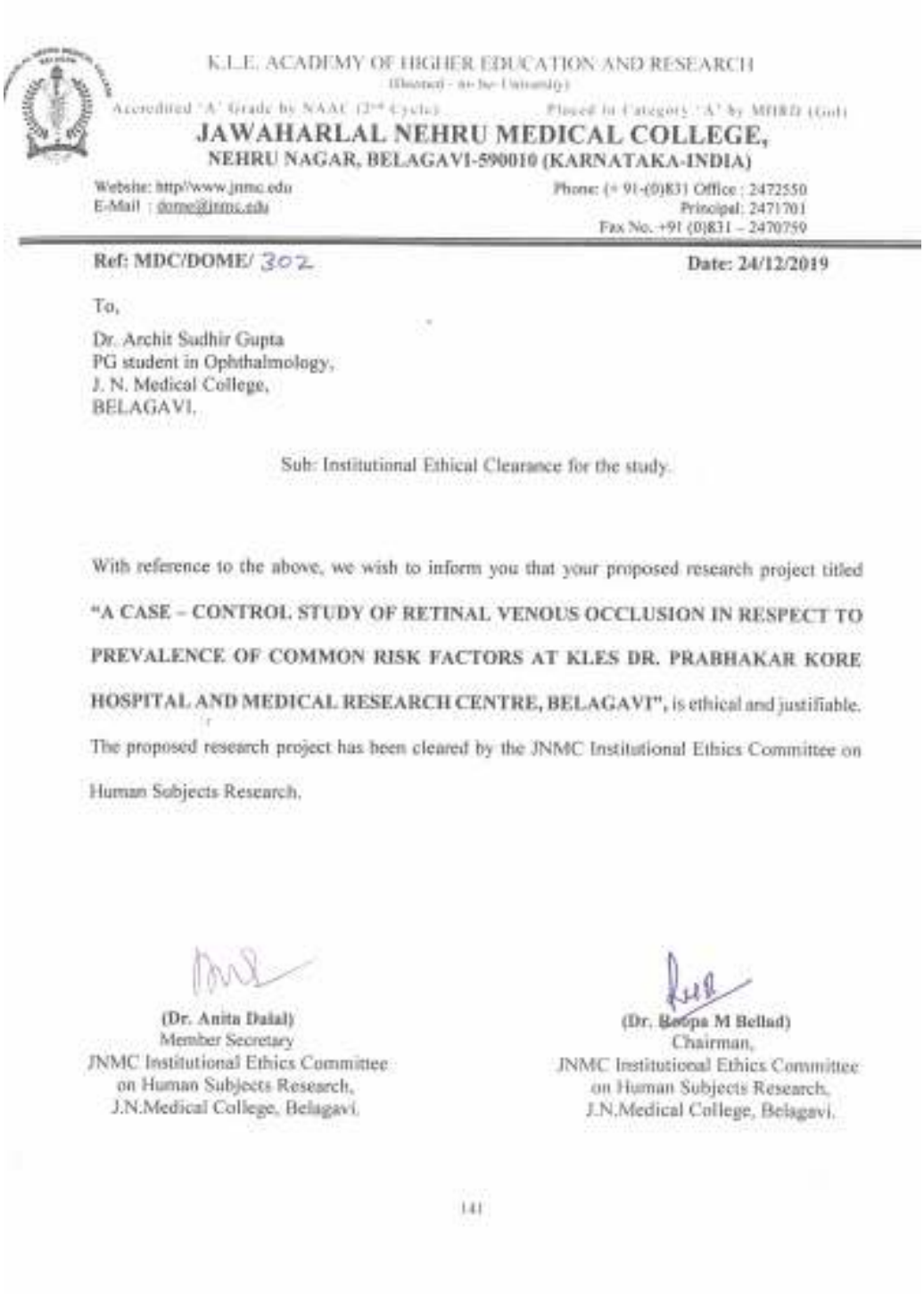
96. Martin SC, Rauz S, Marr JE, Martin N, Jones AF, Dodson PM. Plasma total homocysteine and retinal vascular disease. Eye. 2000 Jul;14(4):590-3.

97. Lam HD, Lahey JM, Kearney JJ, Ng RR, Lehmer JM, Tanaka SC. Young patients with branch retinal vein occlusion: a review of 60 cases. Retina. 2010 Oct 1;30(9):1520-3.

98. Chen YY, Yen YF, Lin JX, Feng SC, Wei LC, Lai YJ, Shen YC. Risk of ischemic stroke, hemorrhagic stroke, and all-cause mortality in retinal vein occlusion: a nationwide population-based cohort study. Journal of ophthalmology. 2018 Sep 9;2018.

99. Chen YY, Sheu SJ, Hu HY, Chu D, Chou P. Association between retinal vein occlusion and an increased risk of acute myocardial infarction: a nationwide population-based follow-up study. PLoS One. 2017 Sep 12;12(9): e0184016.

ANNEXURES I - ETHICAL CLEARANCE



K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed - to-be University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)

JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)

Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu

Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 302 **Date: 24/12/2019**

To,

Dr. Archit Sudhir Gupta
PG student in Ophthalmology,
J. N. Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"A CASE – CONTROL STUDY OF RETINAL VENOUS OCCLUSION IN RESPECT TO PREVALENCE OF COMMON RISK FACTORS AT KLES DR. PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI"**, is ethical and justifiable.

The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Anita Dalal)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

141

ANNEXURE II – INFORMED CONSENT

STUDY ID NO:

TITLE OF THE STUDY: *"A CASE - CONTROL STUDY OF RETINAL VENOUS OCCLUSION IN RESPECT TO PREVALENCE OF COMMON RISK FACTORS AT KLES DR PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI"*

PRINCIPLE INVESTIGATOR: ***Dr Archit Sudhir Gupta***

GUIDE: ***Dr Arvind Laxman Tenagi***

INTRODUCTION AND PURPOSE: This study is designed to determine the age and sex – specific risk factors for all phenotypes of RVOs as well as their determinants in a study in KLES Dr Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

PROCEDURE: I request you to kindly participate in the study titled *"A CASE - CONTROL STUDY OF RETINAL VENOUS OCCLUSION IN RESPECT TO PREVALENCE OF COMMON RISK FACTORS AT KLES DR PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI".* If you agree to participate in the study, please provide the details about the study. We will do a detailed history and examination and investigations to find out the association of common risk factors to RVOs.

BENEFITS: The knowledge about the association of conditions such as hypertension, diabetes mellitus, stroke, ischemic heart disease, chronic kidney disease, glaucoma, body mass index, lipid profile and smoking with RVOs can be very helpful in making the people at risk and the general population aware about the occurrences and

complications of RVOs beforehand to seek appropriate medical attention at the earliest and prevent any untoward incident.

RISKS: No proven side effects.

ALTERNATIVES: If the patient is not willing to take part in the study, his / her treatment or any other further investigations the patient wants to undergo, in future, in KLE will not be affected by his / her decision.

VOLUNTARY PARTICIPATION / WITHDRAWAL: Taking part in this study is voluntary. I may choose not to take part in this study, or if I decide to take part, I can later change my mind and withdraw from the study. My decision will not change the present or future health care or other services that I receive. The study doctor or the sponsor may stop my participation in this study. I will tell of any important new findings that may change my willingness to continue to take part. If I choose not to take part in the study. I will receive the standard treatment for patients with my condition.

COMPENSATION: If I become injured as a result of taking part in this study, treatment will be offered to me. No reimbursement, compensation or free medical care is given.

CONFIDENTIALITY: All information collected about me during the study will be kept confidential to the extent permitted by the law. The code numbers will identify me in this research record. Information from this study may be published but my identity will be confidential in any publication.

CONSENT TO PARTICIPATE IN RESEARCH STUDY: I voluntarily agree to take part in this study by signing on the line below. I may withdraw at any time. I am

not giving up any of my legal rights by signing this form. My signature below indicated that I have read this entire consent form or it has been read to me, and has been explained to me in my vernacular language and had all my questions answered. I will be given a copy of this consent form. Signature / Left Thumbprint of the Participant or legally authorized representative.

Participant's Name:

Signature / Left Thumb impression:

Name of the legally authorized representative:

Signature / Left Thumb impression:

Witness's Name:

Signature / Left Thumb impression:

Investigators Name and Signature:

Date and Place:

In this research record, information from this study may be published but my identity will be confidential in any publication.

QUESTIONS:

If any enquiries in the future or case of research – related injury illness, you may contact the following people:

1. PRINCIPLE INVESTIGATOR: DR ARCHIT SUDHIR GUPTA, Post – Graduate student, Department of Ophthalmology, Jawaharlal Nehru Medical College, Belagavi. Ph: 9881977551.
2. GUIDE: DR ARVIND LAXMAN TENAGI **MBBS, MS, FGO** Professor and Head, Department of Ophthalmology, Jawaharlal Nehru Medical College, KAHER, Belagavi. Ph: 9844009667.
3. CO – GUIDE: DR VIVEK B WANI **MBBS, MS, FRCSEd** Consultant, Vitreo – Retinal Department, KLES Dr PKH and MRC, Belagavi. Ph: 9449563506.

For any further queries, you may contact:

4. DR ROOPA BELLAD **MBBS, DCH, MD** Professor, Department of Paediatrics, Chairman of JNMC Institutional Ethics Committee on Human Subjects Research, Jawaharlal Nehru Medical College, Belagavi.

ANNEXURE III – PROFORMA

DATE: ☐☐ / ☐☐ / ☐☐☐☐

PATIENT'S / CONTROL'S NAME:

☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

AGE: Years

SEX: ☐ (1-Male; 2-Female)

SL. No: ☐

ADDRESS: ☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

CONTACT NUMBER: ☐☐☐☐☐☐☐☐☐☐

OP NUMBER: ☐☐☐☐☐☐☐☐

IP NUMBER: ☐☐☐☐☐☐☐☐

DATE OF EXAMINATION: ☐☐ / ☐☐ / ☐☐☐☐

IS THE PATIENT / CONTROL ELIGIBLE FOR THE STUDY? (1-YES; 2-NO) ☐

HAS INFORMED CONSENT BEEN GIVEN? (1-YES; 2-NO) ☐

GUIDE'S NAME: DR ARVIND LAXMAN TENAGI

GUIDE'S SIGNATURE:

CO – GUIDE'S NAME: DR VIVEK B WANI

CO-GUIDE'S SIGNATURE:

CHIEF COMPLAINTS OF THE CASE:

DIMINUTION OF VISION: RE ☐

Duration: _____ Days / Months / Years

LE ☐

Duration: _____ Days / Months / Years

HISTORY OF PRESENT ILLNESS:

1. DIMINUTION OF VISION 1 – Gradual; 2 – Sudden ☐

1 – Progressive; 2 – Static ☐

PAST HISTORY:

TRAUMA TO THE EYE: 1 – Present; 2 – Absent ☐

OCULAR SURGERY: 1 – Present; 2 – Absent ☐

Type of Surgery:

Duration: ☐☐ Months / Years

HYPERTENSION: 1 – Present; 2 – Absent ☐

Duration: ☐☐ Months / Years

Medications:

DIABETES: 1 – Type 1; 2 – Type 2; 3 – None ☐

Duration: ☐☐ Months / Years

Medications:

History of Stroke: 1 – Yes; 2 – No

History of Ischemic Heart Disease: 1 – Yes; 2 – No ☐

History of Chronic Kidney Disease: 1- Yes; 2- No ☐

Glaucoma Treatment: RE LE ☐

Smoking / Tobacco Addiction: 1 – Present; 2 – Absent ☐

If Yes, No. of Years:

OCULAR EXAMINATION:

1) Visual Acuity:

	RIGHT EYE	LEFT EYE
DISTANT		
PINHOLE		
NEAR		
AIDED	|	|

2. Adnexa (1 – Normal; 2 – Abnormal)	☐	☐
3. Sclera (1 – Normal; 2 – Congested)	☐	☐
4. Conjunctiva (1 – Normal; 2 – Conjunctival Congestion; 3 – Ciliary Congestion; 4 – Chemosis)	☐	☐
5. Cornea (1 – Normal; 2 – Opacity; 3 – Vascularization)	☐	☐
6. Anterior chamber (1 - Normal Depth; 2 – Shallow; 3 – Deep)	☐	☐
7. Iris (1 – Normal, Color & Pattern; 2 – Abnormal)	☐	☐
8. Pupil Size: in mm Shape: 1 – Round & Regular; 2 – Abnormal Reaction: Direct (1 – Present, 2 – Absent) Indirect (1 – Present, 2 – Absent) Near Reflex (1 – Present, 2 – Absent)	☐ ☐ ☐ ☐	☐ ☐ ☐ ☐
9. Lens Clarity: 1 – Clear ,2 – Opaque Cataract - (1), PCIOL - (2) IOP:	☐ ☐	☐ ☐

FUNDUS	RIGHT EYE	LEFT EYE
GLOW		
MEDIA		
DISC		
CUP: DISC RATIO		
BLOOD VESSELS		
BACKGROUND		
MACULA		

Body Weight: Kgs

Height: m

Body Mass Index:

Blood pressure: mm of Hg

Random blood sugar: mg/dl

HbA1c levels:

Hyper – lipidaemia / Hyper – cholesterolaemia:

ECG: Done / Not Done

Lipid Profile: Done / Not Done

Renal Profile: Done / Not Done

ANNEXURE IV – MASTER CHART (CASES)

SERIAL NUMBER	IP / OP NUMBER	AGE in YEARS	GENDER	LATERALITY OF EYE AFFECTED; R = RIGHT, L = LEFT	HYPERTENSION; 0 = NO, 1 = YES	DIABETES MELLITUS; 0= NO, 1= YES	HEART DISEASE; 0 = NO, 1 = YES	OBESITY; 0 = NO, 1 = YES	GLAUCOMA; 0 = NO, 1 = YES	HYPERLIPAEDEMIA; 0 = NO, 1 = YES	TYPE OF GLAUCOMA; 1 = PRIMARY OPEN ANGLE, 2 = PRIMARY ANGLE CLOSURE	HABITS; 0 = NON SMOKING, 1 = SMOKING	ISCHEMIC CRVO = 1, NON ISCHEMIC CRVO = 2, SUPERIOR HRVO = 3, INFERIOR HRVO = 4, ST BRVO = 5, IT BRVO = 6, MT BRVO = 7	SYSTOLIC BP mm Hg	DIASTOLIC BP mm Hg	HbA1C	SERUM CREATININE	BLOOD UREA	BODY MASS INDEX	INTRA OCULAR PRESSURE mm Hg	SERUM CHOLESTEROL
1	5912383	41	M	R	1	1	0	1	1	1	1	1	1	138	88	6.6	1.2	20	24.5	26.8	245
2	5944737	55	M	L	1	1	0	1	0	1	0	1	4	124	74	8.4	1.1	18	26.4	22.5	237
3	5956218	61	M	L	0	0	0	0	0	0	0	1	4	126	76	7.3	1	20	30	24	244
4	5954105	64	F	R	1	1	0	1	1	1	1	1	6	148	96	10.4	1.2	22	33.5	27.4	258
5	5977379	67	M	R	1	1	0	1	0	1	0	1	1	152	84	7.8	0.9	24	31.5	22	250
6	1032494	42	M	R	0	0	0	0	0	0	0	1	6	154	86	7.4	1.4	21	33	21.9	260
7	1309056	65	M	L	1	1	1	1	0	1	0	1	3	130	78	7.6	0.8	20	27.6	23.4	266
8	6123257	68	F	L	1	1	0	1	1	1	1	1	4	132	90	9.2	0.8	19	29.4	28.6	270
9	1063565	46	F	R	0	0	0	0	0	0	0	1	1	144	88	7.5	0.6	15	24.1	18.6	230
10	6152702	69	F	R	1	1	1	1	0	1	0	0	6	145	84	8.4	0.8	16	22.3	19	205
11	5982346	54	M	R	1	1	0	1	1	0	1	1	4	128	76	8.6	0.8	18	26.6	28.1	206
12	1057780	66	M	L	1	1	0	1	0	0	0	1	1	130	96	9.1	1.2	18	28.5	29.4	244
13	6106233	58	M	L	1	1	1	1	0	1	0	1	2	128	88	5.1	1.5	19	27.4	27.4	258
14	6027680	68	M	L	1	1	0	1	1	1	1	1	6	136	86	8.4	1.2	18	28.6	30.2	205
15	5986120	69	M	R	1	0	0	0	0	0	0	0	1	128	84	9.4	1.1	18	23.5	21.5	224

16	1060387	54	M	R	1	0	0	0	0	0	0	0	5	134	88	8.1	1.6	20	29.4	22.4	265
17	6115846	54	M	R	1	1	0	1	0	1	0	0	6	136	84	8	1	21	26.4	20	245
18	5935914	70	F	L	0	0	0	0	1	0	1	1	7	124	86	7	0.9	21	22.5	30	237
19	6115846	69	F	L	1	1	0	1	0	1	0	0	5	126	84	8.5	1.4	18	21.2	26	244
20	6116177	52	F	R	0	0	0	0	0	1	0	0	6	158	94	8.4	0.6	17	20.4	19	258
21	6033776	66	F	R	0	0	0	0	0	0	0	1	2	148	96	7.5	1.2	16	31.5	19	205
22	6035289	64	M	R	1	1	0	1	0	1	0	0	5	146	96	8	0.8	18	33.6	16.4	209
23	1035607	51	M	L	0	0	0	1	0	1	0	0	7	128	84	7.2	0.7	17	30.4	19	220
24	1056516	63	F	L	1	1	1	1	0	1	0	1	5	134	88	7.6	1.4	16	30.6	18.6	226
25	1039283	67	F	L	1	1	0	1	0	1	0	0	2	144	86	6.5	0.8	18	30.4	17.5	230
26	1045112	53	F	R	0	0	0	0	0	0	0	1	5	142	100	7.4	0.7	17	32.1	27	300
27	1046013	64	M	R	1	1	0	1	0	1	0	0	5	146	84	7.9	0.9	20	30.4	31.6	250
28	5899998	61	M	L	1	1	0	1	0	0	0	1	2	144	110	6.5	1.2	21	31.7	27.4	290
29	1051327	54	F	R	1	1	0	1	0	1	0	1	5	146	106	7.9	0.9	22	30.6	19	305
30	1045109	62	M	R	1	1	0	1	0	0	0	1	7	136	104	7.4	0.9	25	31.4	26	280
31	1047315	61	F	R	1	1	0	1	0	1	0	0	5	144	108	7.4	0.8	20	30.4	19.4	294

CONTROLS

SERIAL NUMBER	IP / OP NUMBER	AGE in YEARS	GENDER	LATERALITY OF EYE AFFECTED; R = RIGHT, L = LEFT	HYPERTENSION; 0 = NO, 1 = YES	DIABETES MELLITUS; 0= NO, 1= YES	HEART DISEASE; 0 = NO, 1 = YES	OBESITY; 0 = NO, 1 = YES	GLAUCOMA; 0 = NO, 1 = YES	HYPERLIPAEDEMIA; 0 = NO, 1 = YES	TYPE OF GLAUCOMA; 1 = PRIMARY OPEN ANGLE, 2 = PRIMARY ANGLE CLOSURE	HABITS; 0 = NON SMOKING, 1 = SMOKING	ISCHEMIC CRVO = 1, NON ISCHEMIC CRVO = 2, SUPERIOR HRVO = 3, INFERIOR HRVO = 4, ST BRVO = 5, IT BRVO = 6, MT BRVO = 7	SYSTOLIC BP mm Hg	DIASTOLIC BP mm Hg	HbA1C	SERUM CREATININE	BLOOD UREA	BODY MASS INDEX	INTRA OCULAR PRESSURE mm Hg	SERUM CHOLESTEROL
1	1017162	41	M	R	0	0	0	1	0	0	1	0	0	124	72	6.6	0.8	17	25	16.8	245
2	1017458	51	M	L	0	1	0	1	0	0	0	0	0	124	74	8.4	0.9	18	26	11.5	220
3	1024725	61	M	L	0	0	0	0	0	0	0	0	0	110	76	6.3	1	20	20	14	200
4	1017585	60	F	R	0	0	0	0	0	0	1	0	0	122	70	8	0.9	18	23	17.4	221
5	1017121	62	M	L	1	1	0	0	0	0	0	0	0	126	74	6.8	0.9	16	22	11	198
6	1027327	42	M	R	0	0	0	0	0	0	0	0	0	124	72	6.4	1.4	17	22	11.9	196
7	1028638	61	M	L	1	0	1	1	0	0	0	0	0	116	70	6.6	0.8	16	28	13.4	180
8	1028278	63	F	L	1	0	0	1	0	0	1	0	0	160	110	7.4	0.8	19	29	18.6	166
9	1029196	40	F	R	0	0	0	0	0	0	0	0	0	124	74	6.5	0.6	15	24	18.6	174
10	952960	60	F	L	0	0	1	0	0	0	0	0	0	120	74	8.4	0.8	16	22	19	200
11	981244	44	M	R	0	1	0	0	0	0	1	1	0	128	76	8.6	1.3	18	27	18.1	198
12	1029193	61	M	L	0	0	0	0	0	0	0	1	0	120	70	5.1	1	18	29	19.4	175
13	1003629	58	M	L	0	1	0	1	0	0	0	0	0	128	72	5.1	1.1	14	27	17.4	188
14	979276	68	M	L	0	0	0	1	0	1	1	0	0	120	68	8.4	1.2	18	29	30.1	205
15	951648	60	M	R	0	0	0	0	0	0	0	0	0	126	72	5.9	0.9	18	23	18	224

16	974820	54	M	R	0	0	0	0	0	0	0	1	0	124	76	6	0.7	16	21	16	265
17	952964	54	M	R	1	1	0	1	0	0	0	0	0	124	74	8	0.9	18	26	10	245
18	977793	61	F	L	0	0	0	0	0	0	1	1	0	124	78	6	0.9	18	23	21	237
19	1029198	61	F	L	1	1	0	1	0	1	0	0	0	126	84	6.4	0.7	18	21	16	274
20	1008233	52	F	R	0	0	0	0	0	1	0	0	0	126	64	8.4	0.6	17	20	19	258
21	1003711	70	F	R	0	0	0	0	0	0	0	1	0	144	98	6.5	0.9	16	22	19	205
22	982972	64	M	R	0	0	0	0	0	0	0	0	0	130	96	8	0.8	16	23	16.4	209
23	951626	51	M	L	0	0	0	0	0	0	0	0	0	128	84	6.2	0.7	17	20	19	220
24	1029208	63	F	L	1	1	0	0	0	0	0	1	0	118	88	6.6	1.2	22	21	18.6	226
25	1028924	62	F	L	0	0	0	1	0	0	0	0	0	120	86	6.5	0.8	18	20	17.5	200
26	1028923	57	F	R	0	0	0	0	0	0	0	1	0	110	68	6.4	0.7	15	22	17	240
27	1027121	60	M	R	1	1	0	0	0	1	0	0	0	120	84	6.9	0.9	19	20	20.5	165
28	1027328	61	M	L	1	1	0	0	0	0	0	1	0	126	76	6.5	1.2	16	22	17.4	150
29	1017481	51	F	R	1	1	0	0	0	1	0	0	0	124	72	6.9	0.9	16	21	19	204
30	1017222	62	M	R	1	1	0	0	0	0	0	0	0	124	72	6.4	0.9	10	21	16	280
31	1017171	61	F	R	1	1	0	1	0	1	0	0	0	112	70	6.4	0.8	15	29	19.4	200

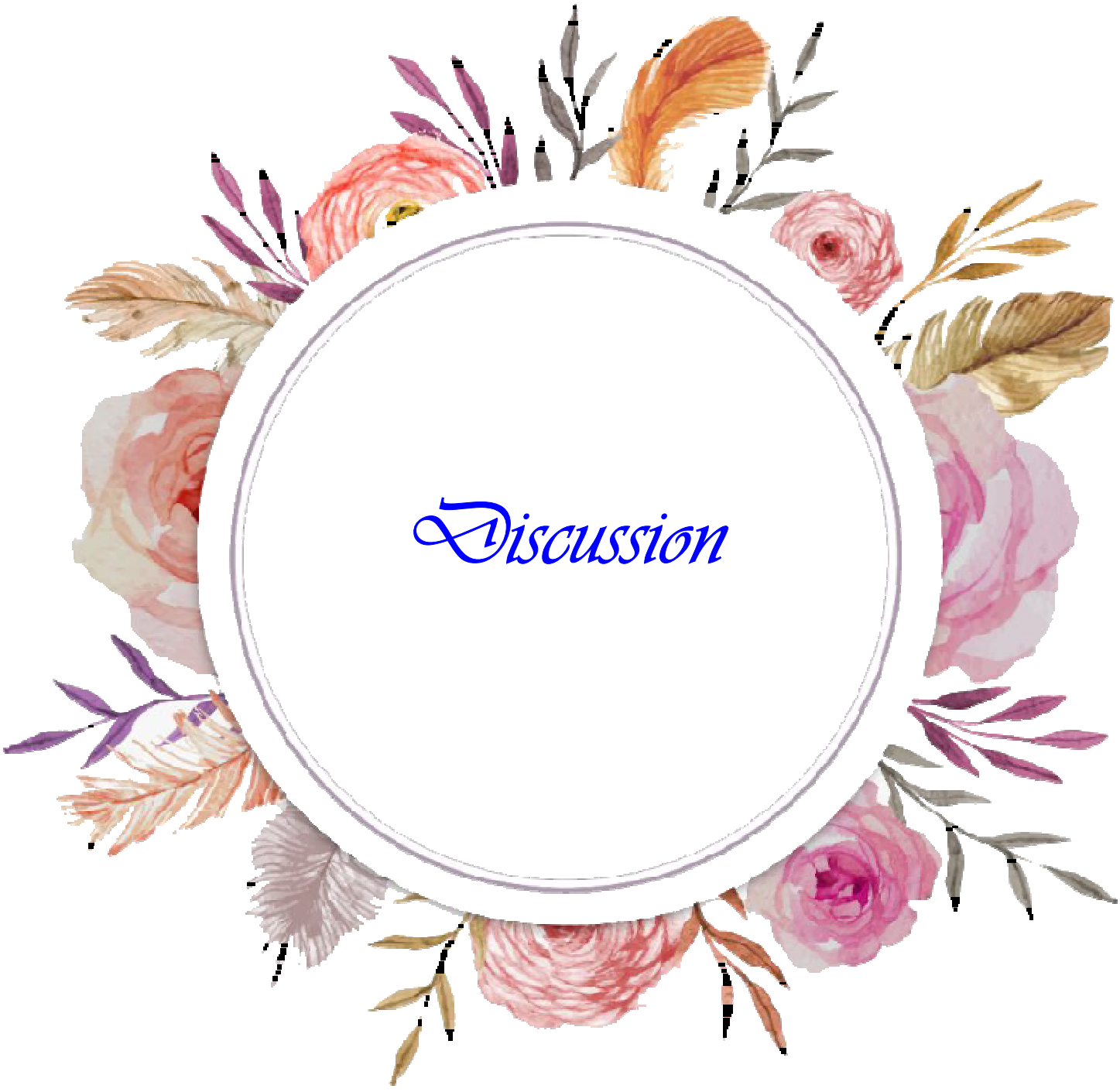

Introduction

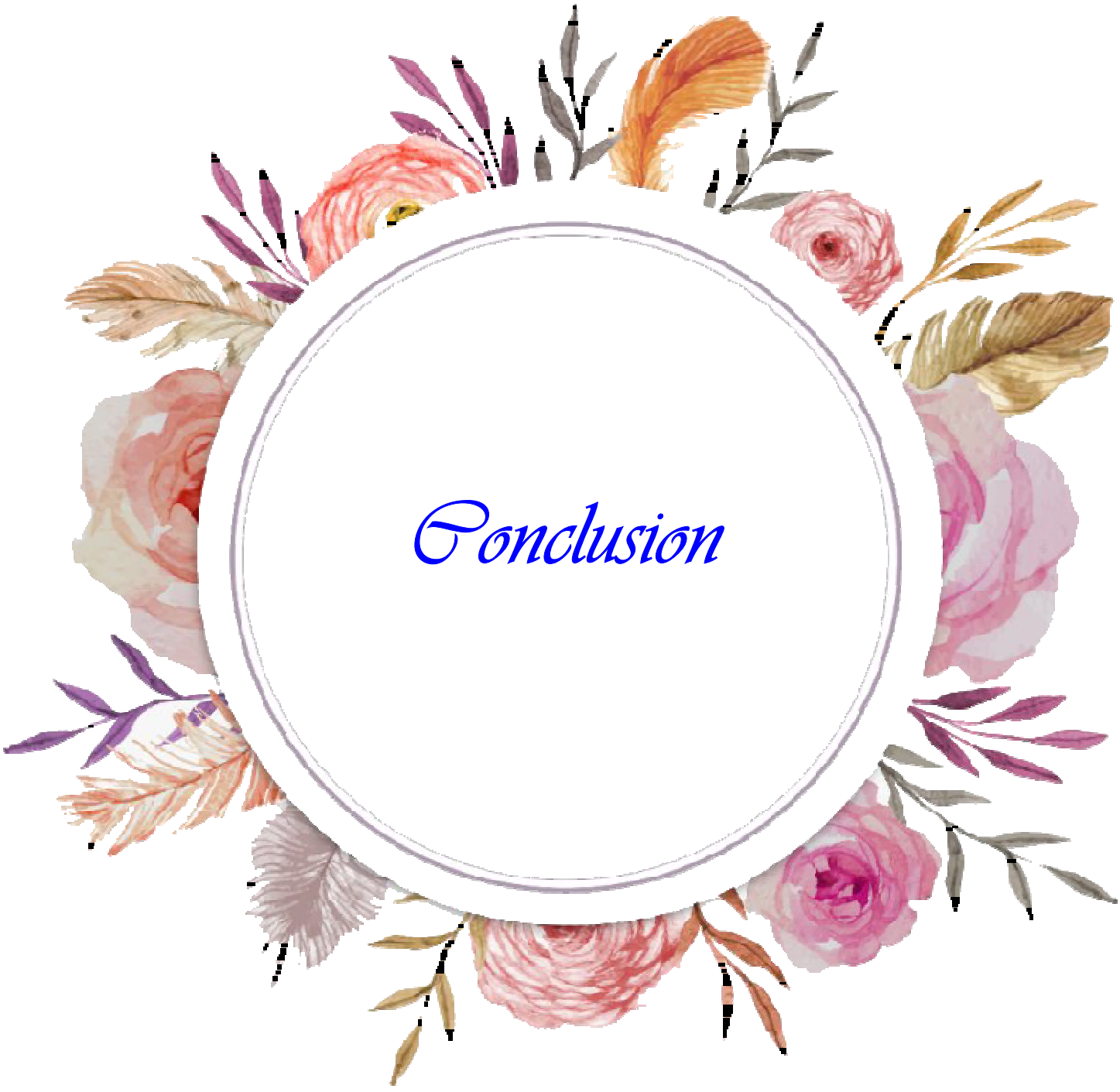

Aims and objectives


Review of literature


Materials &
Methods


Results


Discussion


Conclusion


Summary


Bibliography

Annexures