

**Carotid Plaque Characterization using Doppler Ultrasound in Patients with
Diabetes, Hypertension and Hyperlipidemia**



SUPERIOR UNIVERSITY

**Thesis Submitted to
The Superior University Lahore
In Partial Fulfillment of the
Requirement for the Degree of
Master of Science in Allied Health Sciences**

**By
(HASEEB AHMAD)
SU91-MSAHW-F23-042
Session: 2023-2025**

**Faculty of Allied Health Sciences
The Superior University, Lahore**

Author's Declaration

I hereby state that my MS thesis titled “**Carotid Plaque Characterization using Doppler Ultrasound in Patients with Diabetes, Hypertension and Hyperlipidemia**” is my work and has not been submitted previously by me for taking any degree from this University,

The Superior University, Lahore,

or anywhere else in the country/world.

At any time if my statement is found to be incorrect even after my graduation, the university has the right to withdraw my MS/M.Phil. degree.

HASEEB AHMAD

Date: _____

Plagiarism Undertaking

I solemnly declare that research work presented in the thesis titled “**Carotid Plaque Characterization using Doppler Ultrasound in Patients with Diabetes, Hypertension and Hyperlipidemia**” is solely my research work with no significant contribution from any other person.

Small contribution/help wherever taken has been duly acknowledged and that complete thesis has been written by me.

I understand the zero-tolerance policy of the HEC and University,

The Superior University, Lahore,

towards plagiarism. Therefore, I as an author of the above-titled thesis declare that no portion of my thesis has been plagiarized and any material used as a reference is properly referred/cited. I undertake that if I am found guilty of any formal plagiarism in the above-titled thesis, even after awarding of MS/M.Phil. degree, the University reserves the rights to withdraw/revoke my MS/M.Phil. degree and that HEC and the University have the right to publish my name on the HEC/University website on which names of students are placed who submitted a plagiarized thesis.

Student/Author Signature: _____

Name: HASEEB AHMAD

Research Completion Certificate

This is to certify that the thesis entitled **“Carotid Plaque Characterization Using Doppler Ultrasound in Patient with Diabetes, Hypertension And Hyperlipidemia”** submitted by **“Haseeb Ahmad”** has been accepted towards the partial fulfillment of the requirement for **“MS Allied Health Sciences”**. The quality of the work contained in this thesis is adequate for the award of degree.

Signature:

Supervisor Name: Dr. Tahira Batool

Designation: Assistant Professor

Certificate of Approval

This is to certify that the research work presented in this thesis, titled “ **Carotid Plaque Characterization using Doppler Ultrasound in Patients with Diabetes, Hypertension And Hyperlipidemia**” was conducted by “**Haseeb Ahmad**” under the supervision of “**Dr. Tahira Batool**”

No part of this thesis has been submitted anywhere else for any other degree. This thesis is submitted to the Faculty of Allied Health Sciences, The Superior University, Lahore in partial fulfillment of the requirements for the degree of Master of Science in the field of “**Allied Health Sciences**” in Faculty of Allied Health Sciences at The Superior University, Lahore.

Student Name: Haseeb Ahmad

Signature: _____

Examination Committee:

Session Chair:

Signature: _____

a) External Examiner:

Signature: _____

b) Internal Examiner:

Signature: _____

c) Supervisor Name:

Signature: _____

d) PL/HOD: Dr. Hafiz Shehzad

Signature: _____

e) Dean: Prof. Dr. M. Naveed Babur

Signature: _____

f) Controller of Examination: Dr. M. Haris

Signature: _____

DEDICATION.

In the name of Allah, the Most Gracious, the Most Merciful.

I dedicate this thesis to my beloved parents, whose love, prayers, and constant support have been my greatest strength.

I am deeply grateful to my respected supervisor for their valuable guidance and encouragement throughout this work.

Above all, I thank Almighty Allah for granting me the ability, patience, and courage to complete this research

ACKNOWLEDGEMENT

.In the name of Allah, the most Gracious, the most Merciful.

I am extremely thankful to Almighty ‘Allah’ Who is the entire source of knowledge and wisdom endowed to mankind, for providing me with the acumen and vision to complete this endeavor.

I would like to express my profound gratitude to my supervisor **Dr. Tahira Batool** for her wise counsel and encouraging attitude towards this study. I am extremely grateful to him/her for immensely facilitating me during my study period by ensuring the provision of favorable circumstances and conducive environment. This project would not have been possible without his support and expert guidance.

In the end, I would like to extend my deepest gratitude to my family members. Without their encouragement, I would not have been able to complete this endeavor.

HASEEB AHMAD

TABLE OF CONTENTS

Plagiarism Undertaking.....	III
Certificate of Approval.....	V
Dedication.....	VI
TABLE OF CONTENTS.....	VIII
ABSTRACT.....	1
CHAPTER 1.....	2
INTRODUCTION.....	2
1.1. RATIONALE.....	6
CHAPTER 2.....	7
LITERATURE REVIEW.....	7
2.1. OBJECTIVE.....	10
2.2. PROBLEM STATEMENT.....	11
2.3. OPERATIONAL DEFINITIONS.....	12
CHAPTER 3.....	13
MATERIAL AND METHODS.....	13
3.1. STUDY DESIGN:.....	13
3.2. SETTINGS:.....	13
3.3. DURATION OF STUDY:.....	13
3.4. SAMPLE SIZE:.....	13
3.5. SAMPLING TECHNIQUE:.....	13
3.6. SAMPLE SELECTION.....	13
3.6.1. INCLUSION CRITERIA.....	13
3.6.2. EXCLUSION CRITERIA.....	13
3.7. EQUIPMENT:.....	14
3.8. TECHNIQUE:.....	14
3.9. DATA COLLECTION PROCEDURE.....	16
3.11. DATA ANALYSIS PROCEDURE.....	17
3.13. ETHICAL CONSIDERATIONS.....	18
CHAPTER 4.....	19
RESULTS.....	19
CHAPTER 5.....	32

DISCUSSION.....	32
5.1. CONCLUSION.....	34
5.2. RECOMMENDATION.....	35
CHAPTER 6.....	36
REFERENCES.....	36
ANNEXURE.....	41
CONSENT FORMS.....	55
CONSENT FORM (URDU).....	56
PERFORMA/QUESTIONNAIRE.....	57

LIST OF TABLES

Tables No.	Description	Page
Tables 4.1 Details	Frequency distribution of Gender	20
Tables 4.2 Details	Frequency distribution of Hypertension	21
Tables 4.3 Details	Frequency distribution of Diabetes	22
Tables 4.4 Details	Frequency distribution of Hyperlipidemia	23
Tables 4.5 Details	Frequency distribution of Plaque	24
Tables 4.6 Details	Frequency distribution of Plaque appearance	25
Tables 4.7 Details	Descriptive Statistics of variables	26
Tables 4.8 Details	Descriptive Statistics of doppler indices	26
Tables 4.9 Details	Diabetic and Plaque Crosstabulation	27
Tables 4.10 Details	Hypertension and Plaque Crosstabulation	28
Tables 4.11 Details	Hyperlipidemia and Plaque Crosstabulation	29
Tables 4.12Details	Gender and Plaque Crosstabulation	30
Tables 4.13 Details	Age and Plaque Crosstabulation	31

LIST OF FIGURES

Figure No.	Description	Page
Figure 1.1 Details	Normal Carotid Artery Anatomy	02
Figure 1.2 Details	Carotid Intima Media Thickness	03
Figure 1.3 Details	Carotid Plaque on Ultrasound	04
Figure 4.1 Details	Frequency distribution of Gender	20
Figure 4.2 Details	Frequency distribution of Hypertension	21
Figure 4.3 Details	Frequency distribution of Diabetes	22
Figure 4.4 Details	Frequency distribution of Hyperlipidemia	23
Figure 4.5 Details	Frequency distribution of Plaque	24
Figure 4.6 Details	Frequency distribution of Plaque appearance	25
Figure 4.7 Details	Diabetic and Plaque Crosstabulation	27
Figure 4.8 Details	Hypertension and Plaque Crosstabulation	28
Figure 4.9 Details	Hyperlipidemia and Plaque Crosstabulation	29
Figure 4.10 Details	Gender and Plaque Crosstabulation	30
Figure 4.11 Details	Age and Plaque Crosstabulation	31

LIST OF ABBREVIATIONS

IMT	Intima-media thickness
CCA	Common carotid artery
CVD	Cardiovascular disease
ECA	External carotid artery
CA	Common carotid artery
CVD	Cardiovascular disease
CVA	Cerebrovascular accident

ABSTRACT

Background: Carotid artery stenosis lowers the cerebral oxygen supply, impairing brain function. Due to their high oxygen requirements, short-term interruptions in the blood flow can result in irreversible cell death. Since it accounts for a large portion of cases, carotid atherosclerosis is thought to be a significant cause of ischemic stroke.

Objective: To evaluate carotid plaque using doppler ultrasound in subjects with risk factors diabetes hypertension and hyperlipidemia

Methodology: The Combined Military Hospital (CMH) served as the site of this cross-sectional descriptive study. The convenience sample method was used to collect 115 people of different ages and genders. Those with mental illness or an inability to comply with sonographic assessment were excluded; those with diabetes, hypertension, or hyperlipidemia were included. Results: 115 patients were found to meet the inclusion criteria. Frequency distributions are displayed in Tables 01–05. Male respondents made up 50.4% of the sample, while female respondents made up 49.6%. Thirty patients (26.1%) had hyperlipidemia, Hypertension was present in 99 patients (86.1%), and diabetes was present in 103 patients (89.6%). There were 100 patients (87.0%) who had plaque. Table 06 shows how the plaque looks: 12.2% hypoechoic, 10.4% heterogeneous, 7.8% with mixed features, and 67.8% calcified.

Conclusion: The analysis shows a strong correlation between carotid plaque development and severe cardiovascular risk factors—diabetes, hypertension, and hyperlipidaemia. Male patients were more commonly involved compared with females. Carotid Doppler ultrasonography was a sensitive, valid, and cost-saving non-invasive diagnostic method for the detection of carotid plaques. The prevalence of the high frequency of the plaques, especially in elderly males, emphasizes the importance of the early detection and preventive interventions to curb the impact of cerebrovascular disease among populations at risk.

Key words: Carotid intima media thickness, Peak systolic velocity, End diastolic velocity, Plaque

Chapter 1

INTRODUCTION

The common carotid artery (CCA) is one of the major elastic arteries that carry oxygenated blood to the cranial and cervical areas. Although they differ in their anatomical origins, both the left and right CCAs are symmetrical in structure and function. The left artery arises from the aortic arch, whereas the right arises from the brachiocephalic trunk at the level of the sternoclavicular joint. In the carotid sheath of deep cervical fascia, the CCA continues downward on the side of the vagus nerve and internal jugular vein, running along the trachea and oesophagus (2, 3).

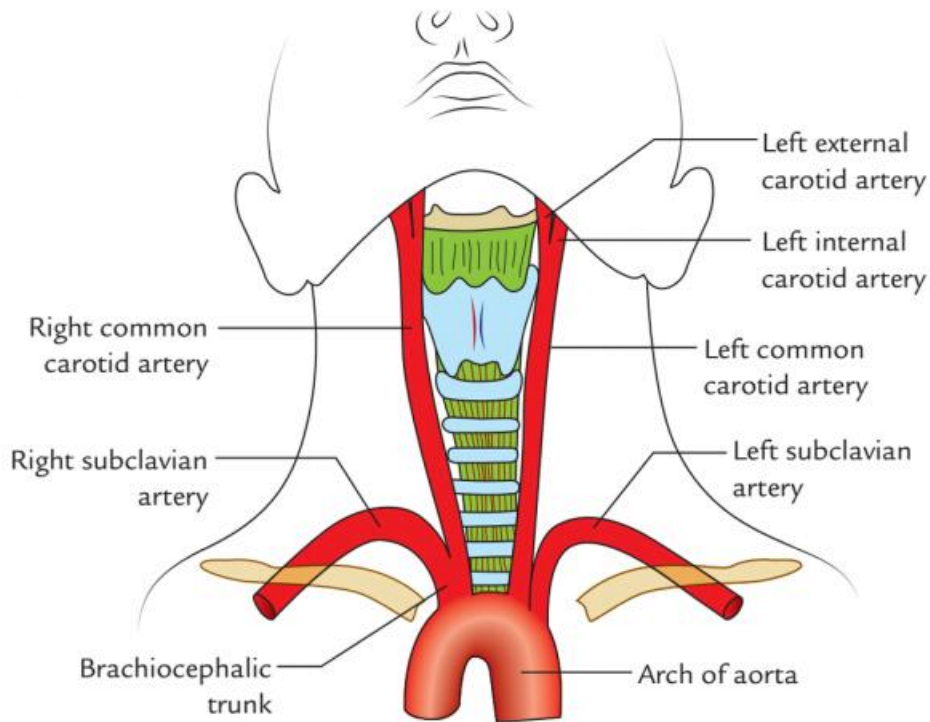


Figure 1.1: Normal anatomy of Carotid artery (4).

Carotid intima-media thickness (IMT) measurement provides a validated, noninvasive method for assessment of subclinical atherosclerosis. B-mode ultrasonography offers a simple and reproducible means of visualizing the arterial wall and measuring intimal and medial thickness in a variety of physiological and pathological states (5). Exclusion of atheromatous plaques is required during such measurements to ensure validity. Sonographically, IMT appears as two discrete echogenic lines: the interface of lumen-intima and media-adventitia

junction (6). A mean CCA-IMT of more than 1 mm is generally considered abnormal (7). Its progressive increase parallels systemic atherosclerotic burden and has been associated with subsequent cardiovascular and cerebrovascular morbidity. Raised IMT has also been observed in patients with metabolic syndrome, regardless of central obesity (8–11).



Figure 1.2: Carotid Intima Media Thickness (12).

Carotid artery intima-media thickness (CAIMT) is being used more and more as a proxy for early atherosclerosis, and a recent study has indicated that it is a powerful indicator of vascular events in the future, such as myocardial infarction and stroke. (13). Values of CAIMT, however, differ among populations according to ethnicity, habitual lifestyle, diet, and measurement methods. Age and sex also affect reference ranges and must be taken into account in clinical interpretation (14). In addition to IMT, other markers such as arterial stiffness, endothelial dysfunction, and coronary calcium scoring give evidence of early vascular changes. Evidence indicates that atherosclerotic lesions start insidiously in childhood and continue into adulthood, sometimes before the manifestation of overt cardiovascular disease signs (15).

Since atherosclerosis develops slowly over decades, early detection is important for prevention. Traditional risk factors of hypertension, obesity, hyperlipidemia, and age are still key, but they do not account for individual susceptibility (17). In fact, existing predictive systems like the Framingham Risk Score explain only some two-thirds of cardiovascular risk. Non-invasive measures like measurement of CIMT would complement risk stratification, especially in those at intermediate risk. Higher

BMI, longstanding hypertension, and older age are all regularly linked to increased IMT values, affirming its status as an early marker for disease extension (20).

In population groups, CIMT is an easily applied surrogate of atherosclerosis and has been powerfully linked with coronary artery disease and cerebral vascular events (21). Several studies have demonstrated that CIMT not only reflects established disease but also predicts cardiovascular events in the future in the general and high-risk populations, including diabetes and haemodialysis patients. Studies investigating CIMT in diabetic patients with significant renal involvement are few when compared to research in other groups. In addition, hyperlipidaemia adds significantly to wall thickening through promoting deposition of lipids in the intima, thus hastening plaque growth and atherosclerotic risk (13, 14).

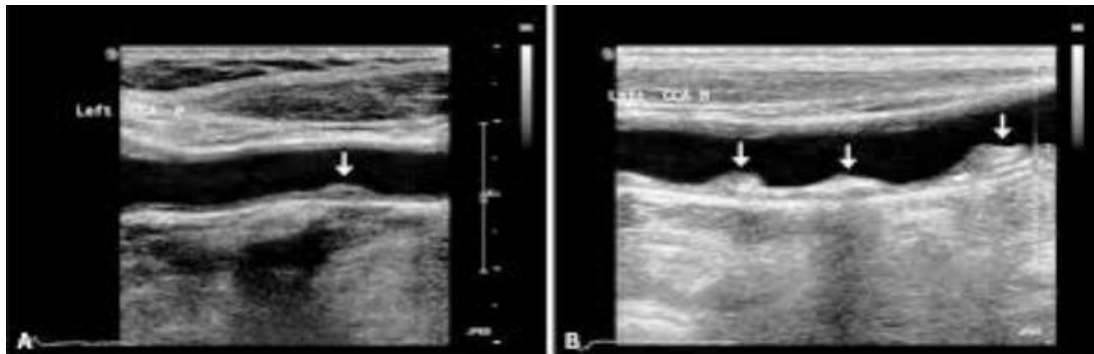


Figure 1.3: Ultrasound Imaging of Carotid Plaques (21).

Since atherosclerosis is difficult to reverse once clinical events occur, prevention of early wall thickening and plaque development has become a major clinical focus (23). The landmark study by Pignoli in 1986 was the first to demonstrate the correlation between ultrasonographical observed double-line patterns and histological confirmation of the intima-media complex. This finding introduced carotid ultrasonography as a key diagnostic modality for assessing early changes in the arterial wall (24). The method is dependable, affordable, and non-invasive. Serial CIMT provides valuable information about the course of the disease and the impact of interventions, even though elevated CIMT is a sign of systemic atherosclerotic burden and does not always portend imminent events (25).

Early longitudinal studies established the correlation between cardiovascular risk factors and CIMT (26, 27). Subsequent research and meta-analyses have further established CIMT as a powerful predictor of cardiovascular morbidity and mortality

in a wide variety of populations (28). Carotid ultrasound thus is not just a diagnostic modality, but also an epidemiological marker of vascular health (29). Since the severity of CCA-IMT mirrors systemic atherosclerosis, it has been identified as a surrogate endpoint within clinical trials that evaluate interventions with a view to lowering cardiovascular risk (30). B-mode ultrasonography provides easy and reproducible measurement of CCA-IMT in multiple carotid segments with enhanced diagnostic accuracy (31). Although distal wall measurements are generally thought to be more accurate due to improved visualization of the media–adventitia interface, IMT can be measured at both proximal and distal arterial locations (32). The mean CCA-IMT values reported in broader epidemiological surveys have shown a favorable correlation with B-mode ultrasound examinations (33). Lower values in healthy subjects and values above 1 mm are typically associated with increased cardiovascular risk (34)

Variability in CCA-IMT values between populations is believed to be due to differences in diet, lifestyle, and ethnicity. Thus, population-specific normative data should be developed, as results in one region cannot necessarily be adopted directly in another. The majority of previous reference studies were carried out in Western populations, and their use may be limited in Asian cohorts (35).

Numerous demographic groups and medical conditions have been studied in order to determine the predictive value of CCA-IMT. For instance, CCA-IMT has been linked to inflammatory processes that underpin atherogenesis in addition to being a structural marker (30, 31). Evidence that lifestyle changes such as exercise, diet control, and quitting smoking can slow the progression of CCA-IMT supports its value as a responsive biomarker in both clinical and preventive cardiology (32, 33). Despite its recognized value, CCA-IMT interpretation requires careful attention to technical variability because variations in ultrasonography equipment, measurement technique, and operator skill can impact outcomes (34). This heterogeneity has led to recommendations for standardized guidelines and population-based normative values. Standardization is especially significant in Asian populations, where diet, genetic influences, and environmental exposures vary from Western cohorts that have hitherto characterized CCA-IMT studies (35).

1.1 RATIONALE

This study aimed to increase our understanding of how risk factors appear. In addition to being a structural marker, CIMT has been linked to the inflammatory mechanisms that underpin atherogenesis. Patients who have any one of a more comprehensive assessment of CIMT reveals that it is linked to inflammatory processes that underpin atherogenesis, making it a useful diagnostic for diabetes, hypertension, and hyperlipidemia. Therapy made simpler and fatality rates would eventually be lower if carotid artery plaque is detected early.

Chapter 2

LITERATURE REVIEW

In their 2017 assessment, Colour Doppler Evaluation of Extracranial Carotid Artery in Patients with Acute Ischaemic Stroke and Correlation with Different Risk Factors, Samrin et al. highlighted Doppler parameters' diagnostic power for indicating arterial stenosis. They noted that internal-to-common carotid velocity ratio from Doppler assessment proved to be a good indicator of disease severity. Their study also found the most susceptible segment to plaque development to be the carotid bulb, with diabetes mellitus and hypertension as the major systemic causes of cerebral ischaemia (36).

Casadei et al. in 2012 examined structural plaque characteristics of carotid plaques in a cohort of outpatients to ascertain their implications for continued surveillance. They found that some plaque morphologies were more susceptible to progression and therefore deserved more intensive imaging follow-up. Their study highlighted that frequent ultrasonographic monitoring is still needed even in patients with non-critical stenosis, as plaque characteristics, as well as luminal compromise, determine future risk (37).

Sehrawat et al. in the same year assessed patients with cerebrovascular disease with Colour Doppler to relate clinical presentation to radiological signs of atherosclerosis. The findings strengthened the relationship between plaque distribution and vascular risk factors, and smoking being the strongest lifestyle determinant. Transient ischaemic attacks and hemiparesis were common in the clinical presentation, and imaging always localized the bifurcation as the site with the most prevalent plaque accumulation (38).

Ho and others in 2016 reaffirmed the worldwide burden of cardiovascular disease, fueled primarily by atherosclerosis, and highlighted the prognostic importance of plaque imaging over mere stenosis measurement. They demonstrated that ultrasonographic indicators of plaque vulnerability provided superior predictive value for stroke risk than lumen constriction alone. Advances in sonographic technology were revealed to improve plaque characterization and help clinicians identify who would benefit most from intervention (39).

In 2018, Zhang et al. looked into the prognostic value of minimal stenosis and intima-media thickness in older adults. The study demonstrated that pathological vascular alterations can independently predict adverse cardiovascular events even in the absence of obvious plaque. These findings supported the theory that subtle changes in arterial walls might serve as early indicators of systemic vascular disease (40).

In 2019, Rafaelidis et al. evaluated carotid plaques by contrasting contrast-enhanced ultrasonography with Doppler imaging. Their results demonstrated the potential for better diagnostic performance in identifying high-risk lesions through the integration of imaging modalities. The clinical benefit of multimodal imaging was demonstrated by the ability to better differentiate symptomatic from asymptomatic lesions with the addition of plaque vulnerability indices, even though stenosis remained a significant predictor (41).

Sun et al. Investigated the 2019 prevalence of plaque and vascular wall thickening in a population without any symptoms. They observed that intima-media thickness was influenced by carotid artery laterality and sex differences. Age and metabolic risk factors were closely linked to the occurrence of plaque, whereas thickened intima-media were significantly linked to the development of carotid atherosclerosis. It was determined that intima-media thickness was a trustworthy preclinical indicator (42).

In 2015, Jarauta and associates conducted a more thorough analysis of carotid intima-media thickness as a predictor of cardiovascular risk. According to their findings, arterial wall thickening is regulated by biological and metabolic factors like sex, blood pressure, age, and lipid concentrations. They also emphasized the importance of carotid IMT measurement in prevention cardiology, specifically in identifying high-risk patients prior to clinical disease (43).

In 2012, Freedman et al. investigated the long-term effects of childhood obesity on adult cardiovascular risk. They highlighted the gradual vascular burden of obesity that starts early in life by linking early-life adiposity to later increases in intima-media thickness. Given that vascular alterations signify a lifetime exposure to metabolic stressors, the study underlined the importance of addressing obesity prevention at an early stage (44).

Lastly, D'Agostino and others in 2018 assessed carotid IMT as a generalizable marker of cardiovascular wellbeing. Their cross-sectional analyses established that common and internal carotid IMT indicated systemic vascular ageing. They indicated that traditional cardiovascular risk factors accounted for only part of the variation, demonstrating that IMT captures a broad range of environmental and biological effects. This supports its application as a universal surrogate endpoint in cardiovascular research (45).

2.1. OBJECTIVE

To evaluate carotid plaque using doppler ultrasound in subjects with risk factors diabetes hypertension and hyperlipidemia

2.2. PROBLEM STATEMENT

Narrowing of the carotid artery decreases the supply of oxygen-rich blood to the brain. As the brain relies on an uninterrupted supply of oxygen in order to function effectively, even a temporary cessation of cerebral circulation can lead to drastic consequences. Neuronal injury may follow within a matter of minutes of deprivation of oxygen or blood. About 15–20% of all ischaemic strokes are due to atherosclerotic transformations in the CA (23). Furthermore, data indicate that , both the left and right CCA, defined as $\geq 50\%$ arterial stenosis, occurs in 5% to 10% of patients >65 years of age. As diagnostic imaging and therapeutic options have improved, however, the pathophysiologic mechanisms, natural history, and best methods for the prevention and management of carotid plaque remain unknown.

2.3. OPERATIONAL DEFINITION

Peak Systolic Velocity (PSV):

In spectral Doppler ultrasound, peak systolic velocity is one of the main haemodynamic indices used. It is the highest deflection that can be seen in the Doppler spectral tracing and represents the maximum speed of blood flow occurring during systole, when the cardiac cycle is at its widest. It is an important index that gives useful information regarding the severity of vascular narrowing and disturbance of flow.

Carotid Atherosclerotic Plaques:

The carotid arteries serve as the primary channels for delivering oxygenated blood from the heart to the brain. In carotid artery stenosis, the vessels are obstructed through narrowing caused by the formation of atherosclerotic plaque in the intimal layer. This narrowing, commonly referred to as carotid artery disease, leads to impaired cerebral circulation and enhances the risk for ischemic events like stroke.

Measurement of Intima–Media Thickness (IMT):

Several sonographic protocols exist to measure the intima–media thickness (IMT) of (CCA). In doing so, areas with focal atheromatous plaque need to be excluded from analysis to prevent bias. On ultrasonography, IMT is delineated by two echogenic reflections: the lumen–intima junction and the media–adventitia junction. Longitudinal images of the CA are taken, and five measurements minimum on each side are suggested, and the mean of these is used to describe the "average IMT." For best measurement, the patient lies supine with the head rotated away from the examined side. Posterior walls of both the right and left CCAs need to be evaluated, and measurements need to be made around one centimeter proximal to the carotid (22).

Chapter 3

METHODOLOGY

3.1. STUDY DESIGN:

A cross-sectional study design was used for this research, which allowed data to be gathered and examined all at once.

3.2. DATA SETTINGS:

Patients who visited the Combined Military Hospital (CMH), Kharian, within the designated study period provided all of the data.

3.3. DURATION OF STUDY:

The study's total duration, including participant selection, data collection, and analysis, was four months.

3.4. SAMPLE SIZE:

Sample size was 115

$$\text{sample size} = \frac{\text{sum of all sample size}}{\text{no of studies}} = \frac{110^{24} + 142^{25} + 93^{16}}{3} = 115$$

3.5. SAMPLING APPROACH

The research used a convenience non-probability sampling strategy, and patients who qualified for study were recruited consecutively within the data collection period.

3.6. SELECTION OF SAMPLES

3.6.1. Conditions for Eligibility

- Male and female participants were eligible.

- Patients with confirmed diagnoses of diabetes mellitus, hyperlipidaemia were included in the study sample.

3.6.2. Conditions for Non Eligibility

- Psychiatric patients or those who could not remain still during sonographic assessment were excluded.
- Patients with a previous history of carotid artery disease or who were previously subjected to endarterectomy were not recruited.
- Peak systolic velocity (PSV) values for were excluded, since no significant correlation was noted between Doppler velocity indices and carotid plaque evaluation.

3.7. EQUIPMENT

Imaging examinations were performed on Toshiba Aplio 300 and Toshiba Xario ultrasound machines that had high-frequency linear array probes between 7.5–11 MHz in power.

3.8. TECHNIQUE

Subjects were placed supine or semi-supine, with the head tilted 45° contralateral to the side being examined and slightly extended. For Doppler examination, lower-frequency linear probes (10–15 MHz) were best for optimal performance, while more high-frequency transducers had better resolution for intima–media thickness and plaque morphology. In subjects with short or muscular necks, where linear probe imaging was not possible, a curved 7 MHz transducer was used with colour Doppler to visualize the carotid bifurcation.

Grey-scale sonography was employed to evaluate for the presence, distribution, and morphologic type of plaques of the common carotid artery (CCA) and internal carotid artery (ICA). The transducer was directed caudally at the supraclavicular fossa and cranially at the mandibular level to maximize vascular visualization. Colour Doppler mapping indicated areas of abnormal flow, which were then analyzed further with pulsed-wave (PW) Doppler spectral analysis. PSV was recorded in the , proximal ICA, and at pre-, intra-, and post-stenotic locations. The

external carotid artery (ECA) was also evaluated for distinguishing it from the ICA and for allowing for potential bruits. Both vertebral arteries were Doppler interrogated to exclude subclavian steal syndrome.

3.9. DATA COLLECTION PROCEDURE

After clearance from the institutional ethics committee, participants were recruited based on the eligibility framework . Following a description of the study's objectives, anticipated advantages, and possible risks, a written informed consent form was signed. Clinical history and demographic data were recorded using a structured proforma. A Toshiba Aplio 300 machine equipped with a linear array probe (10–15 MHz) was used to perform carotid ultrasonography. Age, sex, hypertension, hyperlipidemia, IMT thickness, and sonographic abnormalities such as plaques were among the details that were noted.

3.10. DATA ANALYSIS APPROACH

Version 23 of the SPSS software was used to enter and analyze the data. All continuous variables like age, BMI, IMT, and PSV were described as mean $\pm$ standard deviation, whereas categorical data were shown as frequencies and percentages. Cross-tabulation was used to assess associations, and statistical significance was set at a p-value < 0.05 .

3.11. ETHICAL CONSIDERATIONS

The rules and regulations set by the ethical committee of the superior university lahore were followed while conducting the research and the rights of the research participants were respected.

- Written informed consent (attached) was taken from all the participants.
- All information and data collection were kept confidential during examination.
- Participants were remain anonymous throughout the study.
- The subjects were informed that there are no bio effects of Ultrasound, the procedure is non-invasive.
- Participants were also be informed that they were free to withdraw at any time during the process of the study.
- Participants were informed and instructed regarding the required aspects of the study and protocol for the proper exposure of the lower abdomen.
- Privacy of the patient were kept at top priority during the pelvic examination procedure. Data were kept under key and lock. In laptop it was kept under password

Chapter 4

RESULTS

In this research study, a total of 115 patients were selected based on inclusion criteria. The frequency distributions have been shown in Tab.01, 02, 03, 04, 05. Male ratio 50.4% was higher than females 49.6%. Out of 115 patients 99 (86.1%) patients were diagnosed with hypertension, 103 (89.6%) were with diabetes, 30 (26.1%) were with hyper-lipidemia and 100 (87.0%) patients were diagnosed with plaque. Table 06 Shows 12.2% had hypoechoic plaques, 10.4% had heterogenous, 68.7% had calcified, and 8.7% had mixed plaque appearance.

The Descriptive statistics have been shown in (Tab.07). The mean age of patients was 57.0783 with, the standard deviation was calculated at 15.48012. Of note outliers within the age distribution of patients were 25 years for the youngest patient while the oldest patient was 90 years old. The mean BMI of patients was 27.9130 with, the standard deviation was calculated at 2.85794. The mean IMT of patients was 1.1991 with, the standard deviation was calculated at 0.07314. Table 08 shown that The mean PSV of RCC was 70.1478 with, the standard deviation 12.35104. The mean PSV of RIC was 64.9478 with, the standard deviation 17.70709. The mean PSV of LCC was 73.2087 with, the standard deviation 13.79709. The mean PSV of LIC was 70.6087 with, the standard deviation 30.23002.

Table 09, 10, 11, 12 and 13 shows crosstabulation between risk factors and plaque formation, which shows out of 115 patients who developed carotid plaques, 99(86.1%) had hypertension, 103(89.6%) had diabetes, and 30(26.1%) had hyperlipidemia. shows out of 115 patients with carotid plaque 58(50.4%) were male and 57(49.6%) were females. Table 13 shows that out of 115 patients with carotid plaque, 18 were in age group 1, 37 were in age group 2 and 60 were in age group 3. The results of all the cross tabulation indicates a strong association between all the risk factors and plaque formation.

Table 4.1: Frequency distribution of Gender

	Frequency	Percent
Male	58	50.4
Female	57	49.6
Total	115	100.0

Table 4.1: Shows male ratio 50.4% was higher than females 49.6%

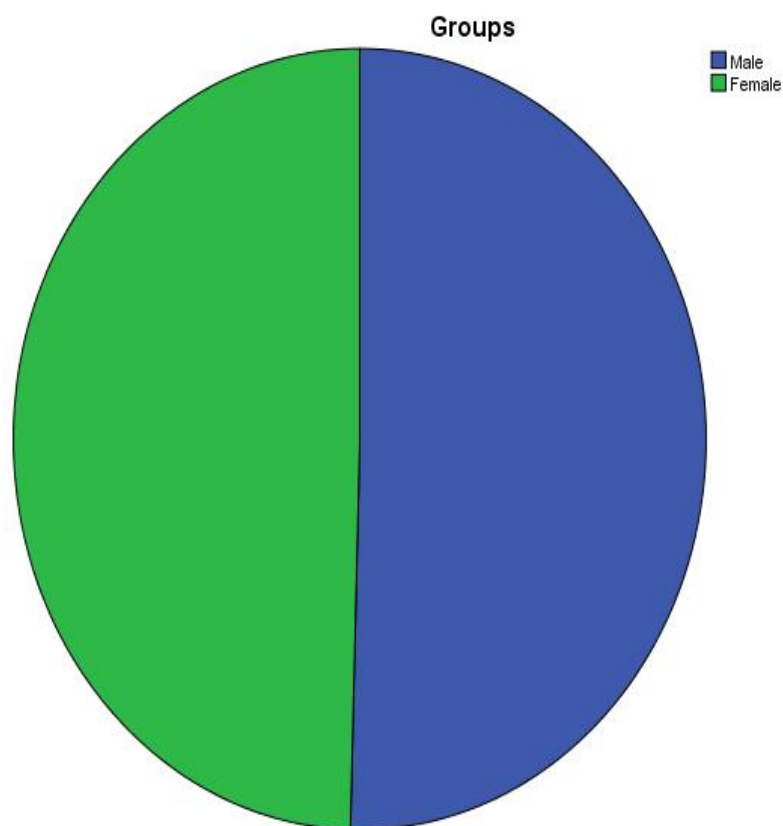


Figure 4.1: Frequency distribution of gender in patients.

Table 4.2: Frequency distribution of Hypertension

	Frequency	Percent
Yes	99	86.1
No	16	13.9
Total	115	100.0

Table 4.2: Shows 99 (86.1%) patients were diagnosed with hypertension.

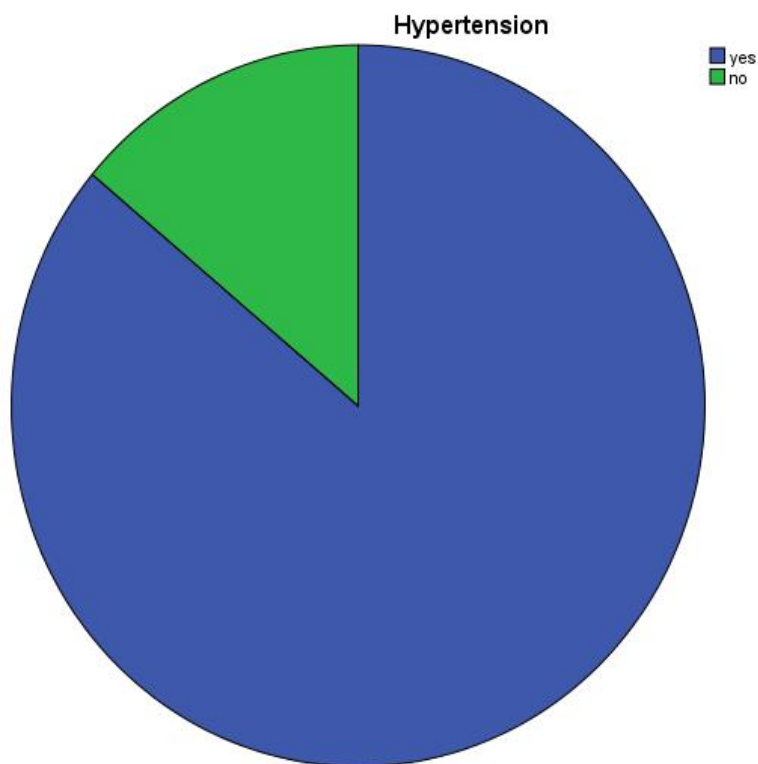


Figure 4.2: Frequency distribution of hypertension in patients.

Table 4. 3: Frequency distribution of Diabetes

	Frequency	Percent
Yes	103	89.6
No	12	10.4
Total	115	100.0

Table 4.3: Shows 103 (89.6%) patients were diagnosed with diabetes.

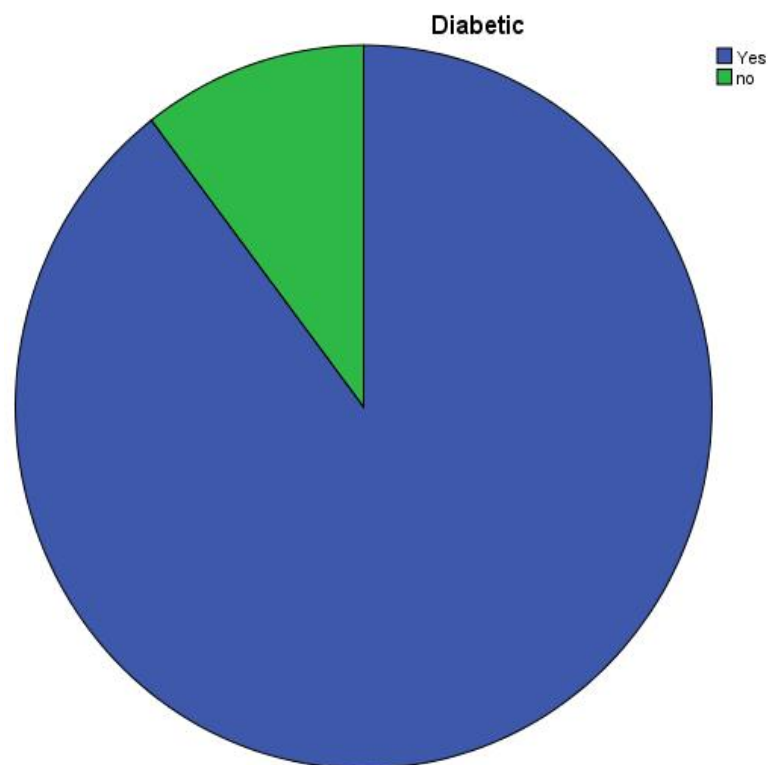


Figure 4.3: Frequency distribution of diabetes in patients.

Table 4.4: Frequency distribution of Hyper-lipidemia

	Frequency	Percent
Yes	30	26.1
No	85	73.9
Total	115	100.0

Table 4.4: Shows 30 (26.1%) patients were diagnosed with hyper-lipidemia.

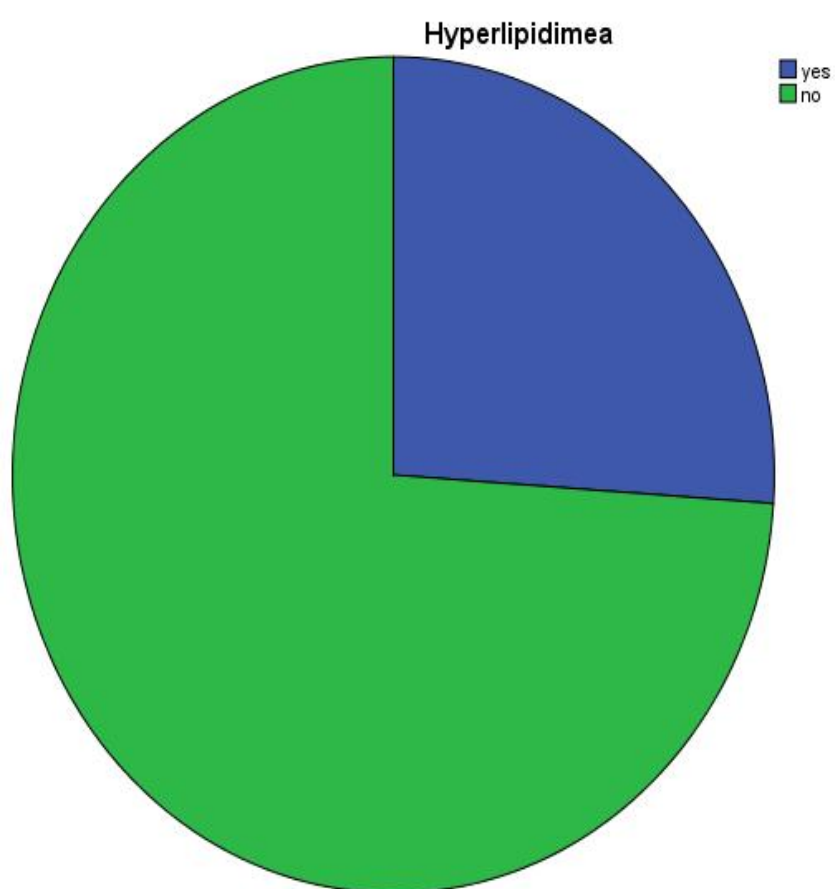


Figure 4.4: Frequency distribution of hyper-lipidemia. in patients.

Table 4.5: Frequency distribution of Plaque

	Frequency	Percent
Yes	100	87.0
No	15	13.0
Total	115	100.0

Table 4.5: Shows 100 (87.0%) patients were diagnosed with plaque.

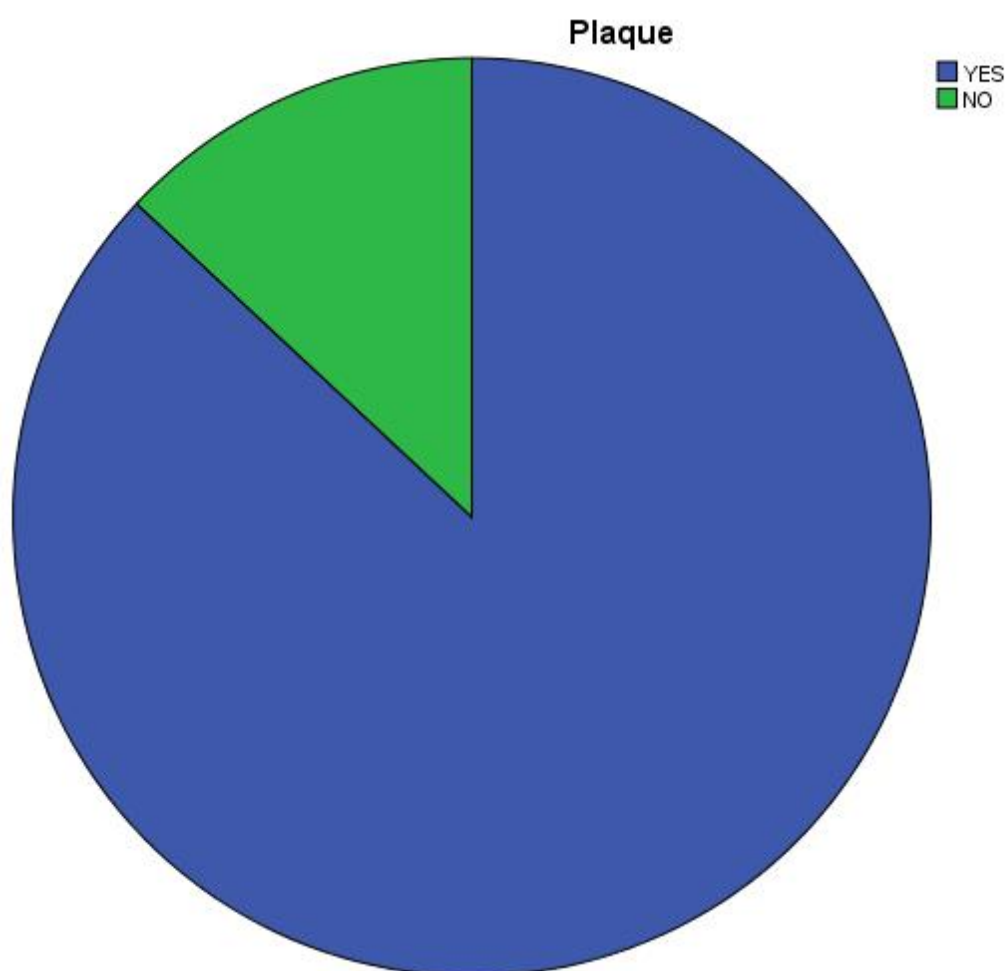


Figure 4.5: Frequency distribution of plaque in patients.

Table 4.6: Frequency distribution of Plaque Appearance

		Frequency	Percent
	Hyperechoic	14	12.2
	Heterogeneous	12	10.4
	Calcified	79	68.7
	Mixed	10	8.7
	Total	115	100.0

Table 4.6: Shows 12.2% had hyperechoic plaques, 10.4% had heterogenous, 68.7% had calcified, and 8.7% had mixed plaque appearance.

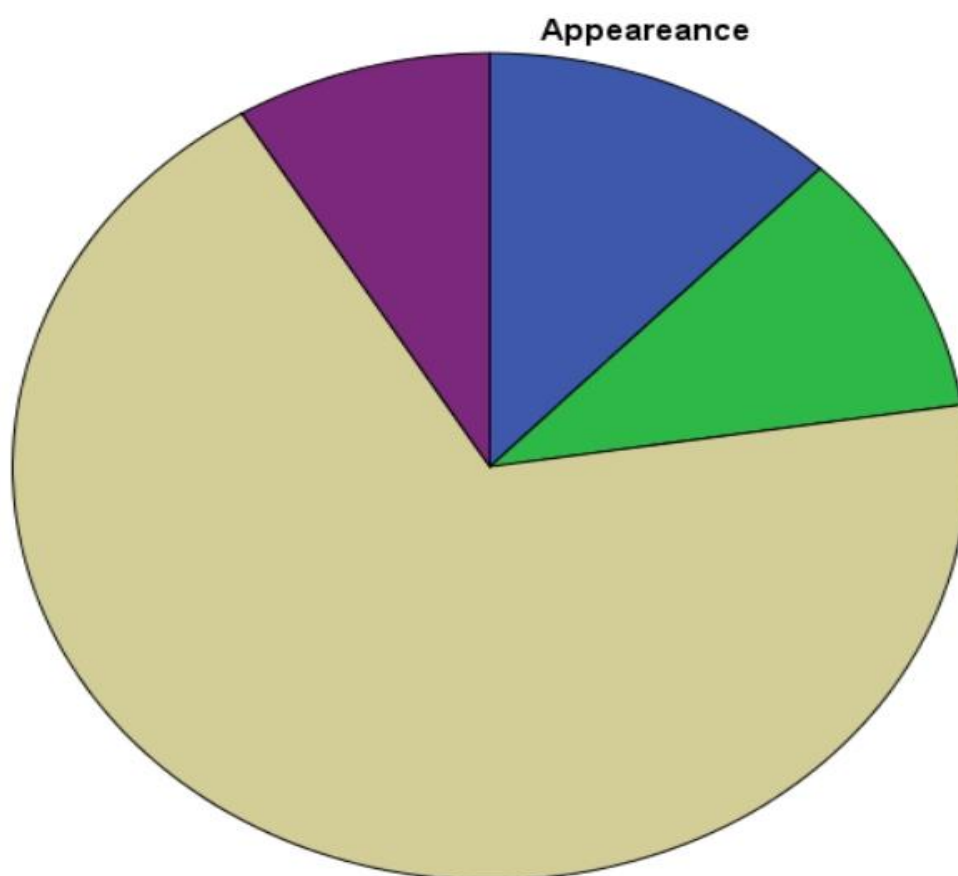


Figure 4.6: Frequency distribution of plaque appearance.

Table 4.7: Descriptive Statistics of variables

	N	Minimum	Maximum	Mean	Std. Deviation
BMI	115	16.00	38.00	27.9130	2.85794
IMT	115	1.10	1.30	1.1991	.07314
Age	115	25.00	90.00	57.0783	15.48012

Table 4.7: Demonstrated the mean, maximum, minimum and Std. Deviation of variables(Age, IMT and BMI)

Table 4. 8: Descriptive Statistics of doppler indices

	N	Minimum	Maximum	Mean	Std. Deviation
Right common Carotid Artery PSV	115	36.00	97.00	70.1478	12.35104
Right Internal Carotid Artery PSV	115	29.00	112.00	64.9478	17.70709
Left common carotid Artery PSV	115	39.00	108.00	73.2087	13.79709
Left Internal Carotid Artery PSV	115	42.00	260.00	70.6087	30.23002

Table 4.8: Demonstrated the mean, maximum, minimum and Std. Deviation of doppler indices

Table 4.9: Plaque and Diabetic Crosstabulation

			Diabetic		Total
			Yes	No	
Plaque	YES	Count	103	12	115
		% within Plaque	89.6%	10.4%	100.0 %
Total		Count	103	12	115
		% within Plaque	89.6%	10.4%	100.0 %

Table 4.9: shows out of 115 patients with carotid plaque 103(89.6%) had diabetes

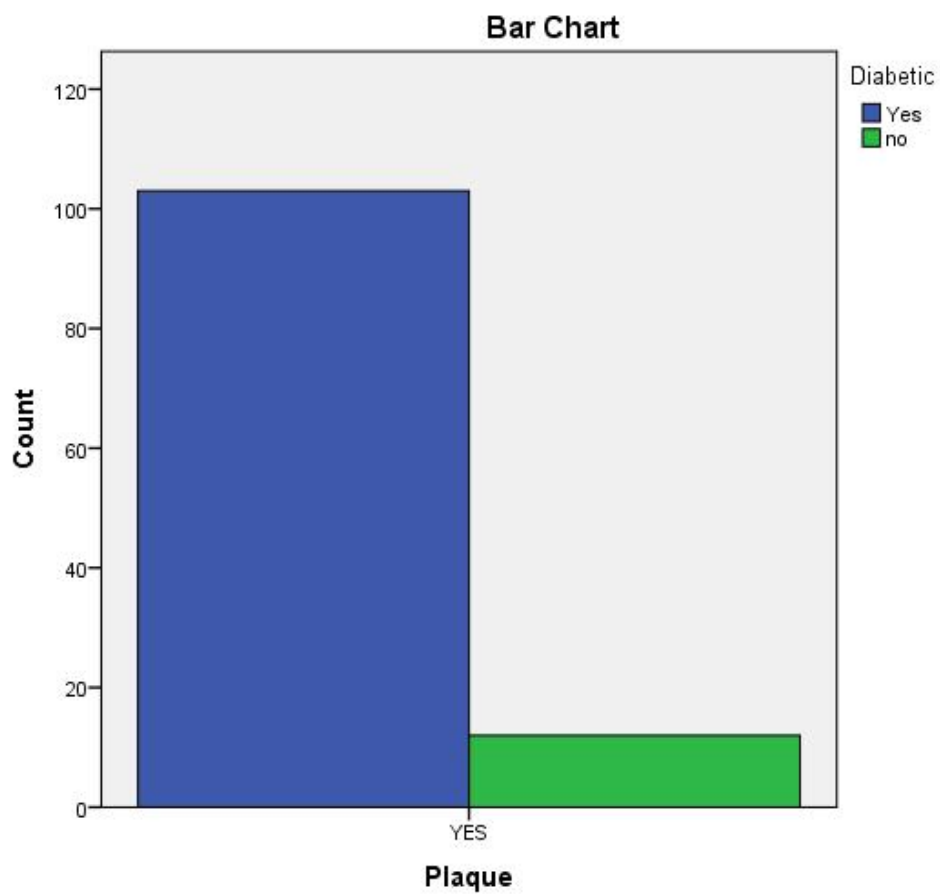


Figure 4.7: Diabetic and Plaque Crosstabulation

Table 4.10: Plaque and Hypertension Cross-tabulation

			Hypertension		Total
			Yes	No	
Plaque	YES	Count	99	16	115
		% within Plaque	86.1%	13.9%	100.0%
Total		Count	99	16	115
		% within Plaque	86.1%	13.9%	100.0%

Table 4.10 : shows out of 115 patients with carotid plaque 99(86.1%) had hypertension

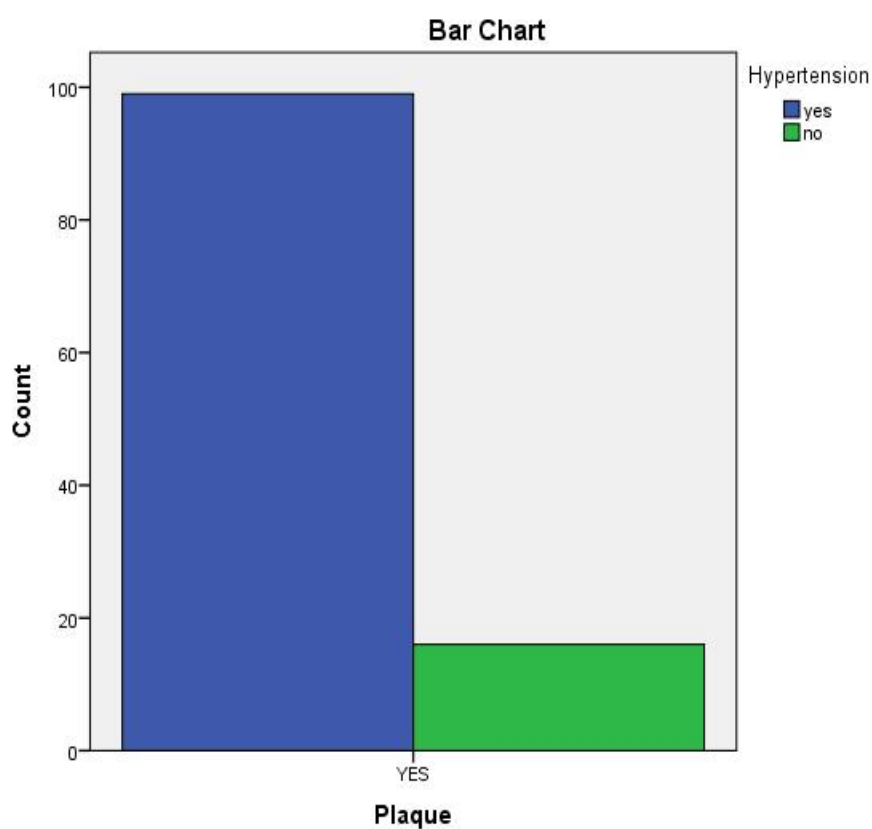


Figure 4.8: Hypertension and Plaque Cross-tabulation

Table 4.11: Plaque and Hyperlipidemia Cross-tabulation

			Hypertension		Total
			Yes	No	
Plaque	YES	Count	30	85	115
		% within Plaque	26.1%	73.9%	100.0%
Total		Count	30	85	115
		% within Plaque	26.1%	73.9%	100.0%

Table 4.11: shows out of 115 patients with carotid plaque 30(26.1%) had hyperlipidemia

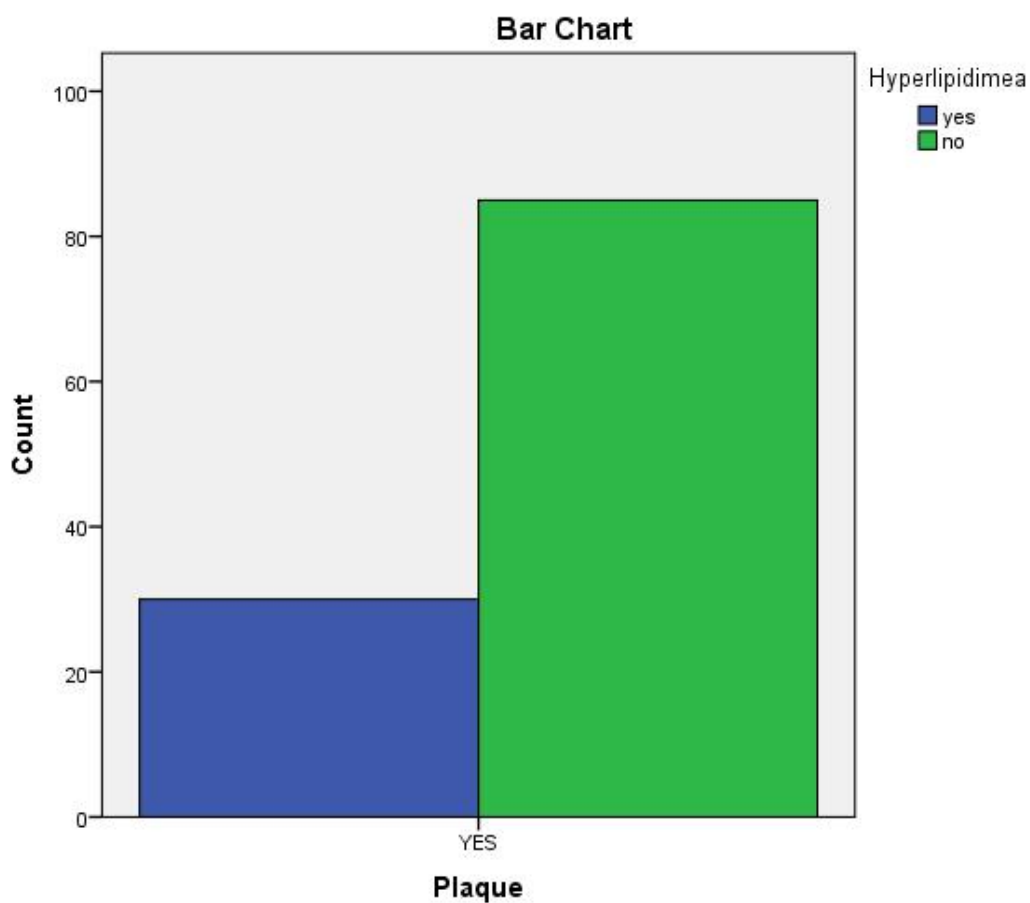


Figure 4.9: Hyper-lipidemia and Plaque Cross-tabulation

Table 4.12: Plaque and Gender Cross-tabulation

			Gender		Total
			Male	Female	
Plaque	YES	Count	58	57	115
		% within Plaque	50.4%	49.6%	100.0%
Total		Count	58	57	115
		% within Plaque	50.4%	49.6%	100.0%

Table 4.12: shows out of 115 patients with carotid plaque 58(50.4%) were male and 57(49.6%) were females.

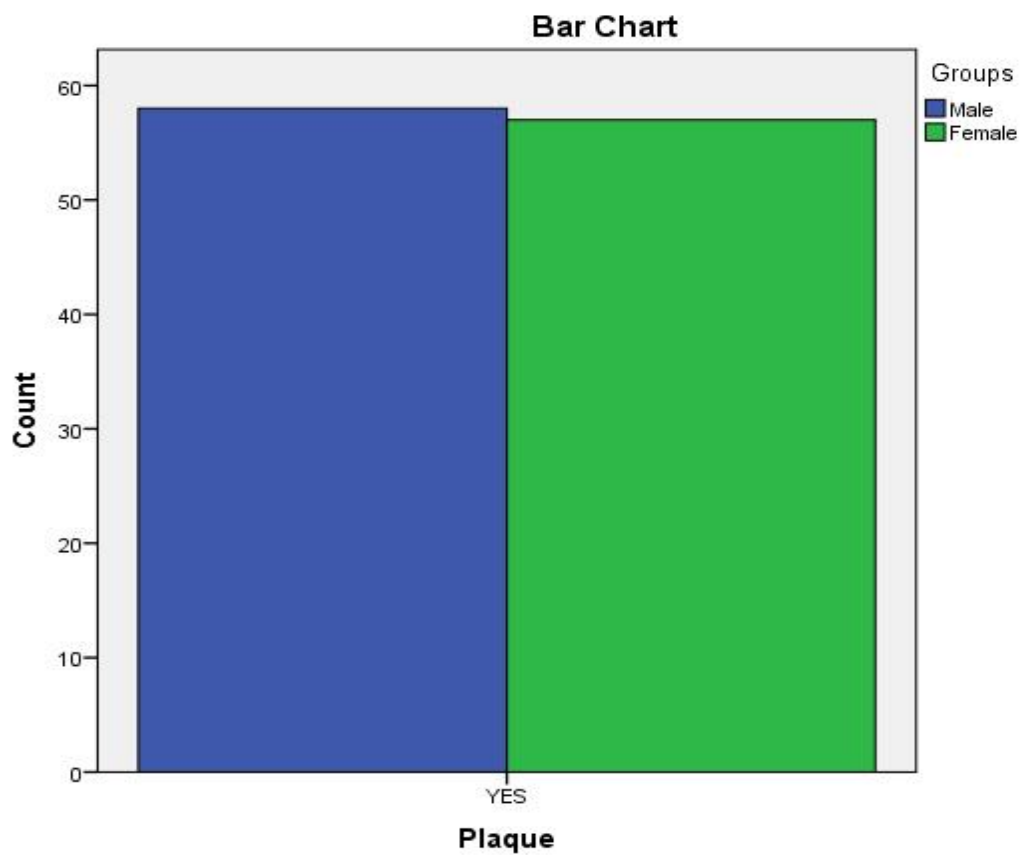


Figure 4.10 : Gender and Plaque Cross-tabulation

Table 4.13: Plaque and Age groups Cross-tabulation

			Age Groups			Total
			1	2	3	
Plaque	YES	Count	18	37	60	115
		% within Plaque	15.7%	32.2%	52.2%	100.0%
Total		Count	18	37	60	115
		% within Plaque	15.7%	32.2%	52.2%	100.0%

Table 4.13: shows out of 115 patients with carotid plaque, 18 were in age group 1, 37 were in age group 2 and 60 were in age group 3.

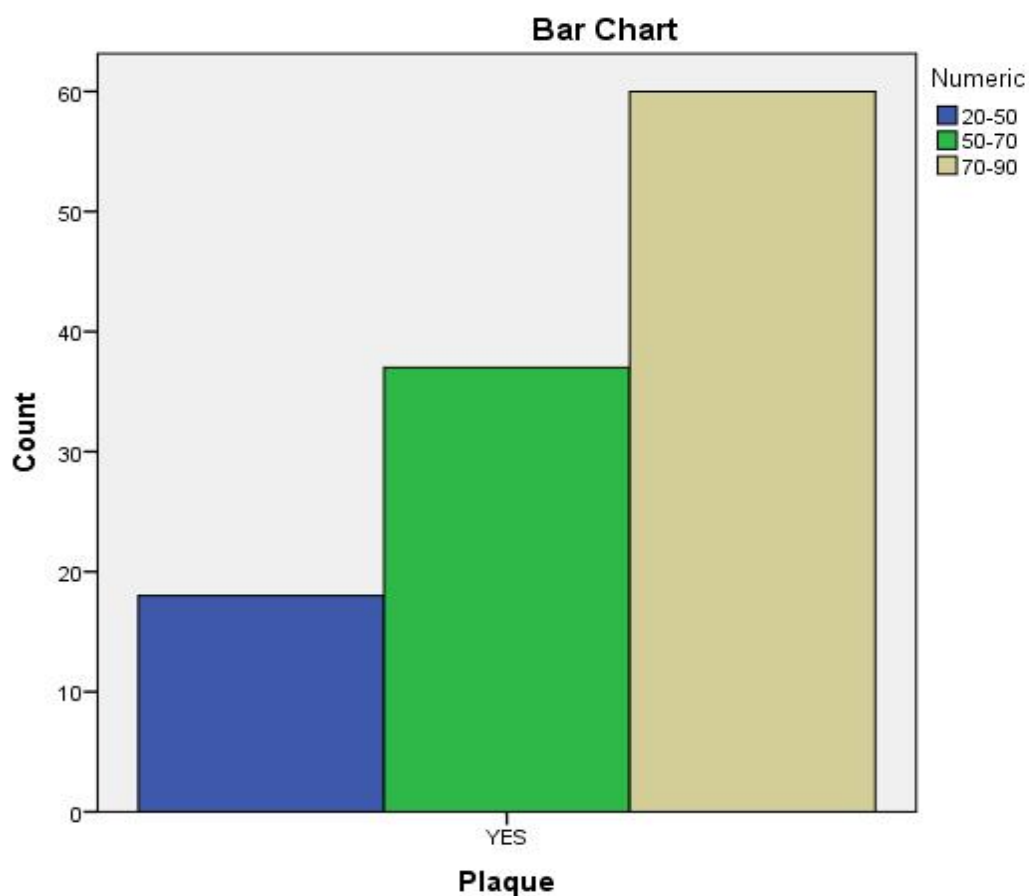


Figure 4.11: Age groups and Plaque Cross-tabulation

CHAPTER 5

DISCUSSION

Atherosclerotic plaque is a chronic, progressive systemic disease closely associated with advanced age, diabetes, hyperlipidemia, hypertension and gender history. Our study demonstrated that age was an independent risk factor for carotid plaque formation in elderly patients. Advanced age has been shown to cause disorders in cerebrovascular function, leading to the development of diseases such as hypertension and cerebral amyloidosis. Hypertension can in turn damage vascular endothelial cells and induce coagulation of platelets and mononuclear macrophages, leading to atherosclerotic plaque formation. Diabetes may cause insulin resistance and hyperinsulinemia; thus, diabetes is also an important risk factor for atherosclerosis formation (23).

Carotid artery intima media thickness is a surrogate marker of atherosclerosis and can diagnose early atherosclerosis. We should know normal value of CAIMT to take a decision whether the person is having an abnormal thickening of the carotid artery or not. The CIMT in this population is strongly dependent on age, sex, and other risk factors so we present the values by sex and risk factors. Our study is helpful for taking this decision. In this study, we found that the CAIMT of normal healthy persons (including all group) was 1.1991 ± 0.073 , which is nearly similar to a study by Wong M in which was 2.1791 ± 0.199 they conducted study on 110 patients with different age groups (8).

Temelkova-Kurktschiev et al conducted a study in the population of 112 individuals, 50 males and 62 females. They concluded that there is a strong correlation between plaque and gender (23). Our work shows that plaque is positively correlated to sex, with invariably higher in men than in women, and this was found at all ages. Interestingly, this finding is not borne out in the international literature. The mean total body mass index (BMI) was 27.91 ± 2.85 kg/m² (Table 7) which is comparable to other authors (24). Young Finns et.al conducted a study in the population studied 150 individuals, 94 males and 56 females. which a meaningful statistical difference ($P < 0.001$) in mean IMT (m-IMT) and maximum IMT (M-IMT) emerged between males and females (mean age: 31.7 ± 5.0 years) was noted (21). Our study also showed that CAIMT was positively correlated with age and was higher in the age

group of 71-90 years, than lower age groups and sex and also higher CAIMT was seen in male sex than age matched female sex in this study. Engelen L, et.al have demonstrated a steeper increase in risk of CV events later in life (>50 years). In particular, it grows higher with increasing age in studies which was conducted by Touboul PJ, in which they resulted that increase risk of higher flow in CMIT were always higher in men than women (20).

The majority of our patients had all the risk factors for developing plaque i.e older age, male gender, familial hypercholesterolemia, hypertension, and diabetes. The hemodynamic disturbances and dysfunctional endothelial cells strengthen the interaction between leukocytes and endothelial cells. The oxidation reaction and glycosylation of low-density lipoprotein and a series of chain reactions caused by the deposition of advanced glycosylation end products result in CP formation. This finding is comparable to all other studies done on Asian and American reports (28).

5.1. CONCLUSION

This study concludes that there was significant association between carotid plaques and diabetes, hypertension and hyper-lipidemia. By using the sample size, we found evidence of carotid plaque in the majority of subjects with possible risk factors. Men were more likely to develop carotid plaques than females. Carotid Doppler ultrasound is a cheap noninvasive modality to detect carotid artery plaques due to its sensitivity, precision, and reliability. Since there is a high frequency of carotid plaques noted in older male adults, appropriate screening with prophylactic management can save many diseases from a lower middle-income class.

5.2. RECOMMENDATION And LIMITATION

To properly evaluate the relationship between carotid plaques and the risk factors associated with them, more research with a bigger sample size is advised, utilizing laboratory indicators and ultrasonography as variables. It is critical to acknowledge this study's limitations. As a cross-sectional descriptive study, it could only demonstrate correlations rather than a causal relationship between risk factors and carotid plaque development. Due to the small sample size and the fact that it was only collected from one health care facility, the results might not be as widely applicable as they could be. Selection bias might have been introduced through the use of a convenience sampling technique. Furthermore, although they may have an impact on carotid intima-media thickness (CIMT) and plaque formation, confounding variables like medication use, dietary intake, physical activity, and smoking habits were not assessed. A more thorough hemodynamic evaluation was also hampered by the lack of PSV data for certain carotid artery segments.

CHAPTER 6

REFERENCES

1. Wikstrand J. Methodological considerations of ultrasound measurement of carotid artery intima-media thickness and lumen diameter. *Clinical physiology and functional imaging*. 2007 Nov;27(6):341-5.
2. Touboul PJ, Grobbee DE, Ruijter HD. Assessment of subclinical atherosclerosis by carotid intima media thickness: technical issues. *European journal of preventive cardiology*. 2012 Aug 1;19(2_suppl):18-24.
3. Lian S, Luo Z, Feng C, Li S, Li S. APRIL: Anatomical prior-guided reinforcement learning for accurate carotid lumen diameter and intima-media thickness measurement. *Medical Image Analysis*. 2021 Jul 1;71:102040.
4. Centuri3n OA. Carotid intima-media thickness as a cardiovascular risk factor and imaging pathway of atherosclerosis. *Critical pathways in cardiology*. 2016 Dec 1;15(4):152-60.
5. Henareh L, Jogestrand T, Agewall S. Glucose intolerance is associated with C-reactive protein and intima-media anatomy of the common carotid artery in patients with coronary heart disease. *Diabetic Medicine*. 2005 Sep;22(9):1212-7.
6. Touboul PJ, Hennerici MG, Meairs S, Adams H, Amarenco P, Bornstein N, Csiba L, Desvarieux M, Ebrahim S, Fatar M, Hernandez RH. Mannheim carotid intima-media thickness consensus (2004–2006). *Cerebrovascular diseases*. 2007;23(1):75-80.
7. Touboul PJ, Hennerici MG, Meairs S, Adams H, Amarenco P, Bornstein N, Csiba L, Desvarieux M, Ebrahim S, Hernandez RH, Jaff M. Mannheim carotid intima-media thickness and plaque consensus (2004–2006–2011). *Cerebrovascular diseases*. 2012;34(4):290-6.
8. Wong M, Edelstein J, Wollman J, Bond MG. Ultrasonic-pathological comparison of the human arterial wall. Verification of intima-media thickness. *Arteriosclerosis and thrombosis: a journal of vascular biology*. 1993 Apr;13(4):482-6.
9. Piconi S, Trabattoni D, Luraghi C, Perilli E, Borelli M, Pacei M, Rizzardini G, Lattuada A, Bray DH, Catalano M, Sparaco A. Treatment of periodontal disease results in improvements in endothelial dysfunction and reduction of the carotid intima-media thickness. *The FASEB Journal*. 2009 Apr;23(4):1196-204.

10. Jegelevičius D, Lukoševičius A. Ultrasonic measurements of human carotid artery wall intima-media thickness. *Ultragarsas/Ultrasound*. 2002 Aug 28;43(2):43-7.
11. Riley WA. Carotid intima-media thickness: risk assessment and scanning protocol. *European heart journal*. 2002 Jun 1;23(12):916-8.
12. Jarauta E, Mateo-Gallego R, Bea A, Burillo E, Calmarza P, Civeira F. Carotid intima-media thickness in subjects with no cardiovascular risk factors. *Revista Española de Cardiología (English Edition)*. 2010 Jan 1;63(1):97-102.
13. Rundek T, Blanton SH, Bartels S, Dong C, Raval A, Demmer RT, Cabral D, Elkind MS, Sacco RL, Desvarieux M. Traditional risk factors are not major contributors to the variance in carotid intima-media thickness. *Stroke*. 2013 Aug;44(8):2101-8.
14. Engelen L, Ferreira I, Stehouwer CD, Boutouyrie P, Laurent S, Reference Values for Arterial Measurements Collaboration. Reference intervals for common carotid intima-media thickness measured with echotracking: relation with risk factors. *European heart journal*. 2013 Aug 7;34(30):2368-80.
15. Tonstad S, Joakimsen O, Stensland-Bugge E, Leren TP, Ose L, Russell D, Bønaa KH. Risk factors related to carotid intima-media thickness and plaque in children with familial hypercholesterolemia and control subjects. *Arteriosclerosis, thrombosis, and vascular biology*. 1996 Aug;16(8):984-91.
16. Gnasso A, Irace C, Mattioli PL, Pujia A. Carotid intima-media thickness and coronary heart disease risk factors. *Atherosclerosis*. 1996 Jan 5;119(1):7-15.
17. Touboul PJ, Hennerici MG, Meairs S, Adams H, Amarenco P, Bornstein N, Csiba L, Desvarieux M, Ebrahim S, Fatar M, Hernandez RH. Mannheim carotid intima-media thickness consensus (2004–2006). *Cerebrovascular diseases*. 2007;23(1):75-80.
18. Touboul PJ, Hennerici MG, Meairs S, Adams H, Amarenco P, Bornstein N, Csiba L, Desvarieux M, Ebrahim S, Hernandez RH, Jaff M. Mannheim carotid intima-media thickness and plaque consensus (2004–2006–2011). *Cerebrovascular diseases*. 2012;34(4):290-6.
19. Wong M, Edelstein J, Wollman J, Bond MG. Ultrasonic-pathological comparison of the human arterial wall. Verification of intima-media thickness. *Arteriosclerosis and thrombosis: a journal of vascular biology*. 1993 Apr;13(4):482-6.

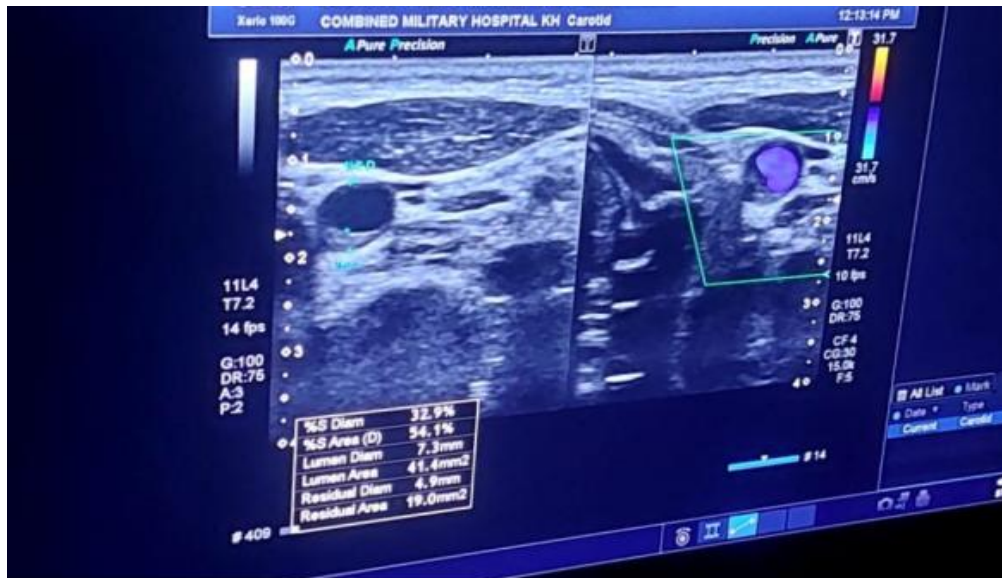
20. Piconi S, Trabattoni D, Luraghi C, Perilli E, Borelli M, Pacei M, Rizzardini G, Lattuada A, Bray DH, Catalano M, Sparaco A. Treatment of periodontal disease results in improvements in endothelial dysfunction and reduction of the carotid intima-media thickness. *The FASEB Journal*. 2009 Apr;23(4):1196-204.
21. Jegelevičius D, Lukoševičius A. Ultrasonic measurements of human carotid artery wall intima-media thickness. *Ultragarsas/Ultrasound*. 2002 Aug 28;43(2):43-7.
22. Riley WA. Carotid intima-media thickness: risk assessment and scanning protocol. *European heart journal*. 2002 Jun 1;23(12):916-8.
23. Jarauta E, Mateo-Gallego R, Bea A, Burillo E, Calmarza P, Civeira F. Carotid intima-media thickness in subjects with no cardiovascular risk factors. *Revista Española de Cardiología (English Edition)*. 2010 Jan 1;63(1):97-102.
24. Rundek T, Blanton SH, Bartels S, Dong C, Raval A, Demmer RT, Cabral D, Elkind MS, Sacco RL, Desvarieux M. Traditional risk factors are not major contributors to the variance in carotid intima-media thickness. *Stroke*. 2013 Aug;44(8):2101-8.
25. Engelen L, Ferreira I, Stehouwer CD, Boutouyrie P, Laurent S, Reference Values for Arterial Measurements Collaboration. Reference intervals for common carotid intima-media thickness measured with echotracking: relation with risk factors. *European heart journal*. 2013 Aug 7;34(30):2368-80.
26. Tonstad S, Joakimsen O, Stensland-Bugge E, Leren TP, Ose L, Russell D, Bønaa KH. Risk factors related to carotid intima-media thickness and plaque in children with familial hypercholesterolemia and control subjects. *Arteriosclerosis, thrombosis, and vascular biology*. 1996 Aug;16(8):984-91.
27. Gnasso A, Irace C, Mattioli PL, Pujia A. Carotid intima-media thickness and coronary heart disease risk factors. *Atherosclerosis*. 1996 Jan 5;119(1):7-15.
28. Lorenz MW, von Kegler S, Steinmetz H, Markus HS, Sitzer M. Carotid intima-media thickening indicates a higher vascular risk across a wide age range: prospective data from the Carotid Atherosclerosis Progression Study (CAPS). *Stroke*. 2006 Jan 1;37(1):87-92.
29. Luo X, Yang Y, Cao T, Li Z. Differences in left and right carotid intima-media thickness and the associated risk factors. *Clinical radiology*. 2011 May 1;66(5):393-8.

30. Aminbakhsh A, Mancini GJ. Carotid intima-media thickness measurements: what defines an abnormality? A systematic review. *Clinical and Investigative Medicine*. 1999 Aug 1;22(4):149.
31. Jarauta E, Mateo-Gallego R, Bea A, Burillo E, Calmarza P, Civeira F. Carotid intima-media thickness in subjects with no cardiovascular risk factors. *Revista Española de Cardiología (English Edition)*. 2010 Jan 1;63(1):97-102.
32. Su TC, Chien KL, Jeng JS, Chen MF, Hsu HC, Torng PL, Sung FC, Lee YT. Age-and gender-associated determinants of carotid intima-media thickness: a community-based study. *Journal of atherosclerosis and thrombosis*. 2012;19(9):872-80.
33. Pauciullo P, Iannuzzi A, Sartorio R, Irace C, Covetti G, Di Costanzo A, Rubba P. Increased intima-media thickness of the common carotid artery in hypercholesterolemic children. *Arteriosclerosis and thrombosis: a journal of vascular biology*. 1994 Jul;14(7):1075-9.
34. Freedman DS, Dietz WH, Tang R, Mensah GA, Bond MG, Urbina EM, Srinivasan S, Berenson GS. The relation of obesity throughout life to carotid intima-media thickness in adulthood: the Bogalusa Heart Study. *International journal of obesity*. 2004 Jan;28(1):159-66.
35. Allan PL, Mowbray PI, Lee AJ, Fowkes FG. Relationship between carotid intima-media thickness and symptomatic and asymptomatic peripheral arterial disease: the Edinburgh Artery Study. *Stroke*. 1997 Feb;28(2):348-53.
36. Lemne C, Jogestrand T, de Faire U. Carotid intima-media thickness and plaque in borderline hypertension. *Stroke*. 1995 Jan;26(1):34-9.
37. Jегelevičius D, Lukoševičius A. Ultrasonic measurements of human carotid artery wall intima-media thickness. *Ultragarsas/Ultrasound*. 2002 Aug 28;43(2):43-7.
38. Riley WA. Carotid intima-media thickness: risk assessment and scanning protocol. *European heart journal*. 2002 Jun 1;23(12):916-8.
39. Jarauta E, Mateo-Gallego R, Bea A, Burillo E, Calmarza P, Civeira F. Carotid intima-media thickness in subjects with no cardiovascular risk factors. *Revista Española de Cardiología (English Edition)*. 2010 Jan 1;63(1):97-102.
40. Rundek T, Blanton SH, Bartels S, Dong C, Raval A, Demmer RT, Cabral D, Elkind MS, Sacco RL, Desvarieux M. Traditional risk factors are not major

- contributors to the variance in carotid intima-media thickness. *Stroke*. 2013 Aug;44(8):2101-8.
41. Engelen L, Ferreira I, Stehouwer CD, Boutouyrie P, Laurent S, Reference Values for Arterial Measurements Collaboration. Reference intervals for common carotid intima-media thickness measured with echotracking: relation with risk factors. *European heart journal*. 2013 Aug 7;34(30):2368-80.
 42. Tonstad S, Joakimsen O, Stensland-Bugge E, Leren TP, Ose L, Russell D, Bønaa KH. Risk factors related to carotid intima-media thickness and plaque in children with familial hypercholesterolemia and control subjects. *Arteriosclerosis, thrombosis, and vascular biology*. 1996 Aug;16(8):984-91.
 43. Gnasso A, Irace C, Mattioli PL, Pujia A. Carotid intima-media thickness and coronary heart disease risk factors. *Atherosclerosis*. 1996 Jan 5;119(1):7-15.
 44. Lorenz MW, von Kegler S, Steinmetz H, Markus HS, Sitzer M. Carotid intima-media thickening indicates a higher vascular risk across a wide age range: prospective data from the Carotid Atherosclerosis Progression Study (CAPS). *Stroke*. 2006 Jan 1;37(1):87-92.
 45. Centurión OA. Carotid intima-media thickness as a cardiovascular risk factor and imaging pathway of atherosclerosis. *Critical pathways in cardiology*. 2016 Dec 1;15(4):152-60.

ANNEXURE

Image 1:



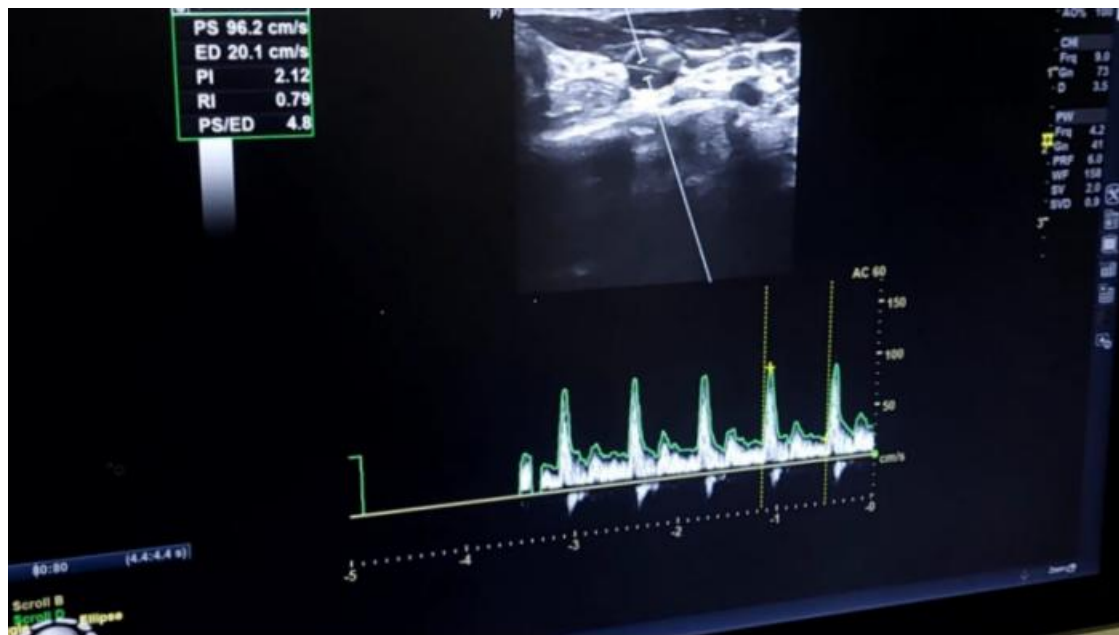
Ultrasound image shows hyperechoic plaque in common carotid artery

Image 2:



Ultrasound image shows hyperechoic plaque at carotid bifurcation

Image 3:

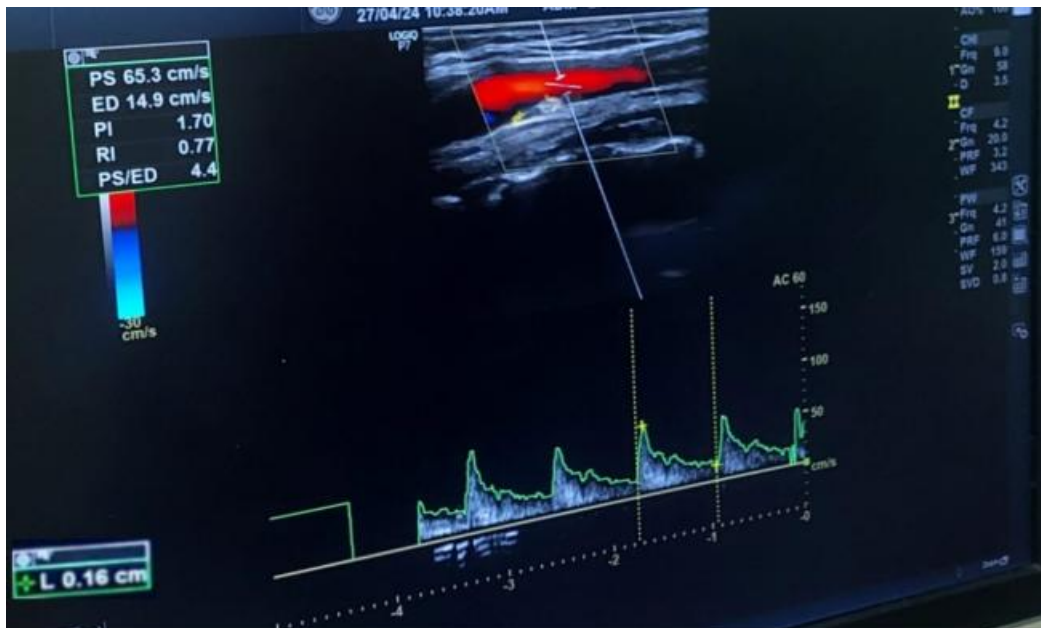


Ultrasound image shows eccentric hyperechoic plaque in common carotid artery

Image 4:



Ultrasound image shows hyperechoic plaque in common carotid artery



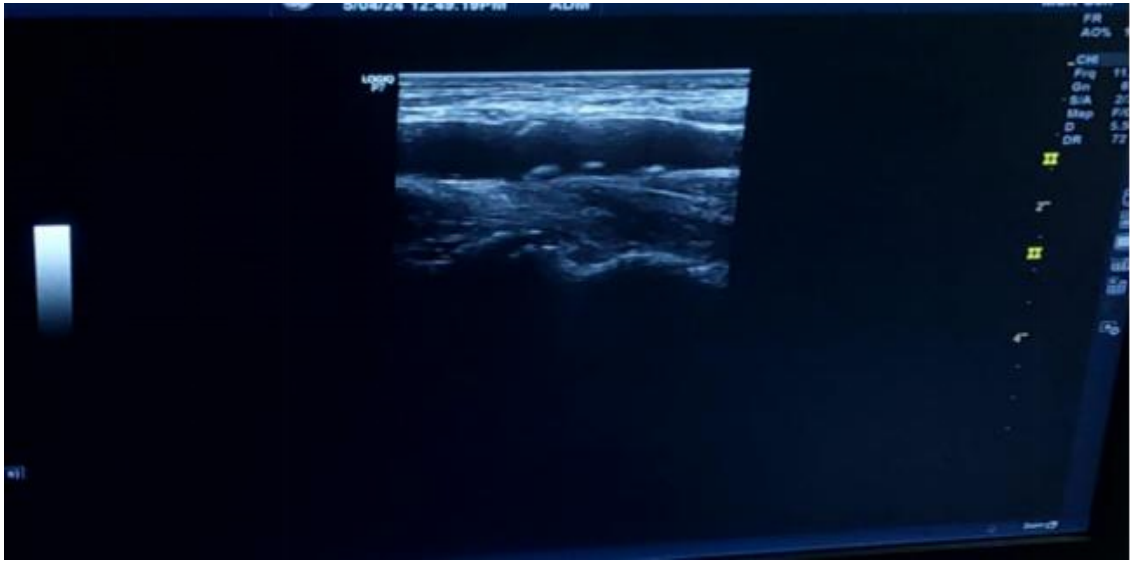
Ultrasound image shows heterogenous plaque at carotid bifurcation

Image 6:



Ultrasound image shows heterogenous plaque in common carotid artery

Image 7:



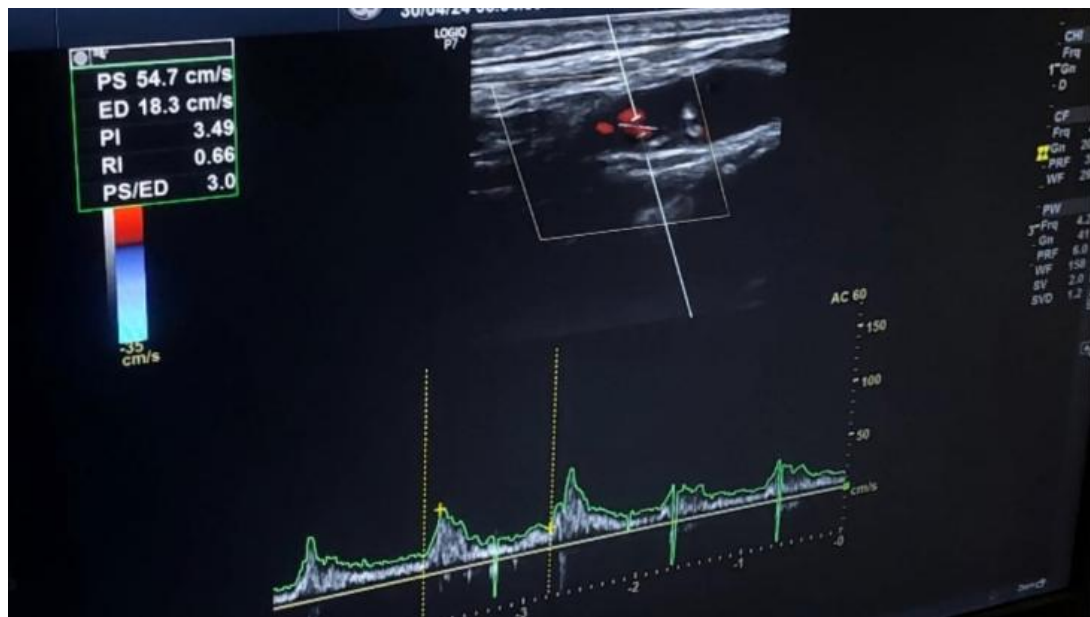
Ultrasound image shows mixed plaque at ICA

Image 8:



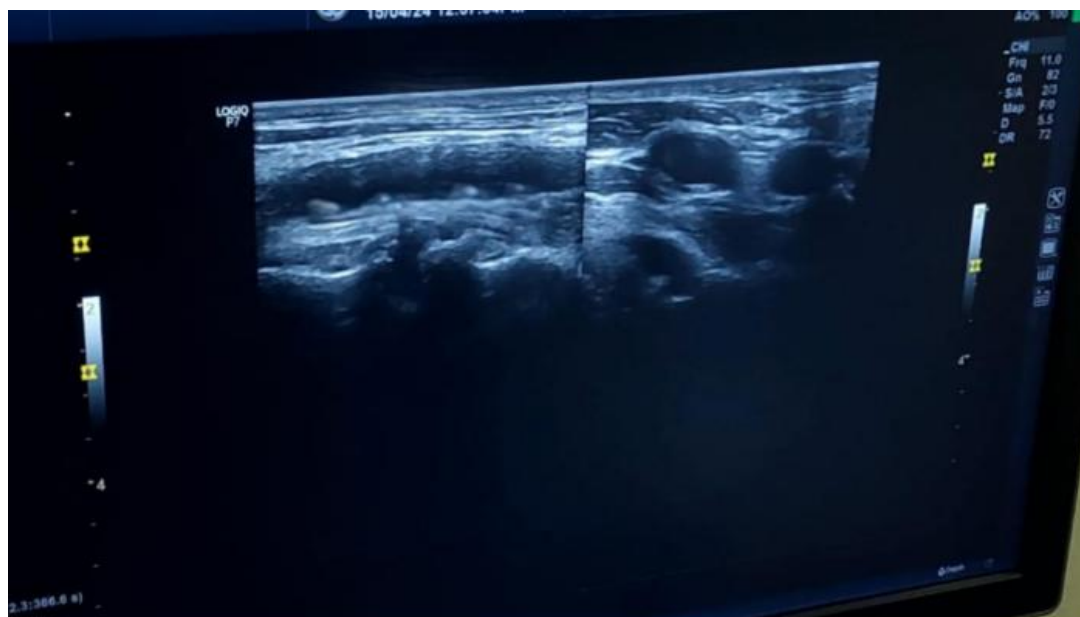
Ultrasound image shows heterogenous plaque at carotid bulb

Image 09:



Ultrasound image shows calcified plaque adherent to carotid wall

Image 10:



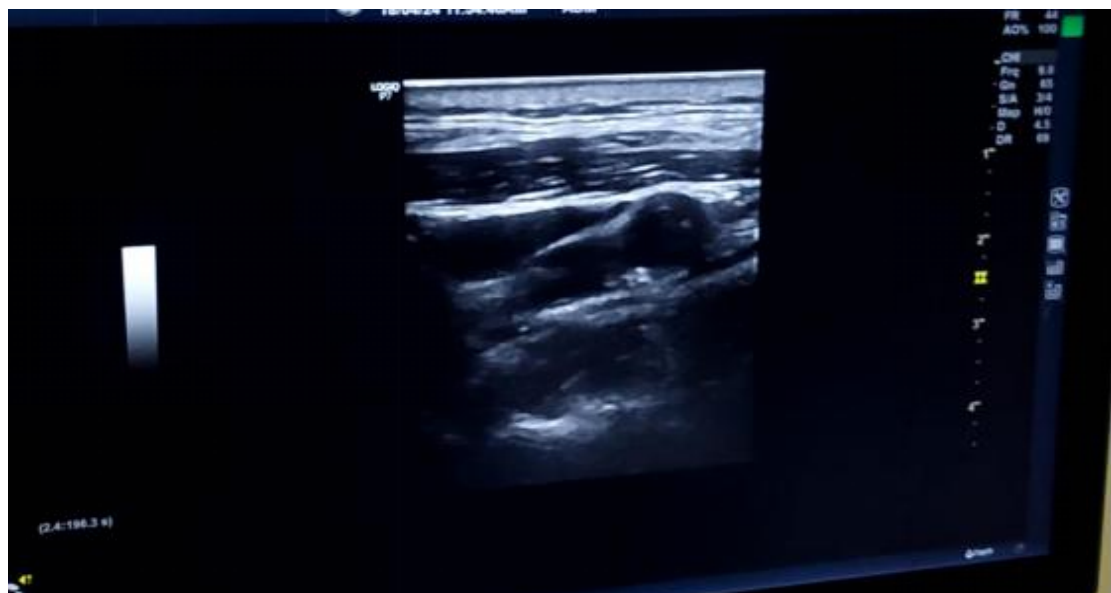
Ultrasound image shows multiple hyperechoic plaques in common carotid artery

Image 11:



Ultrasound image shows hyperechoic plaque at carotid bifurcation

Image 12:



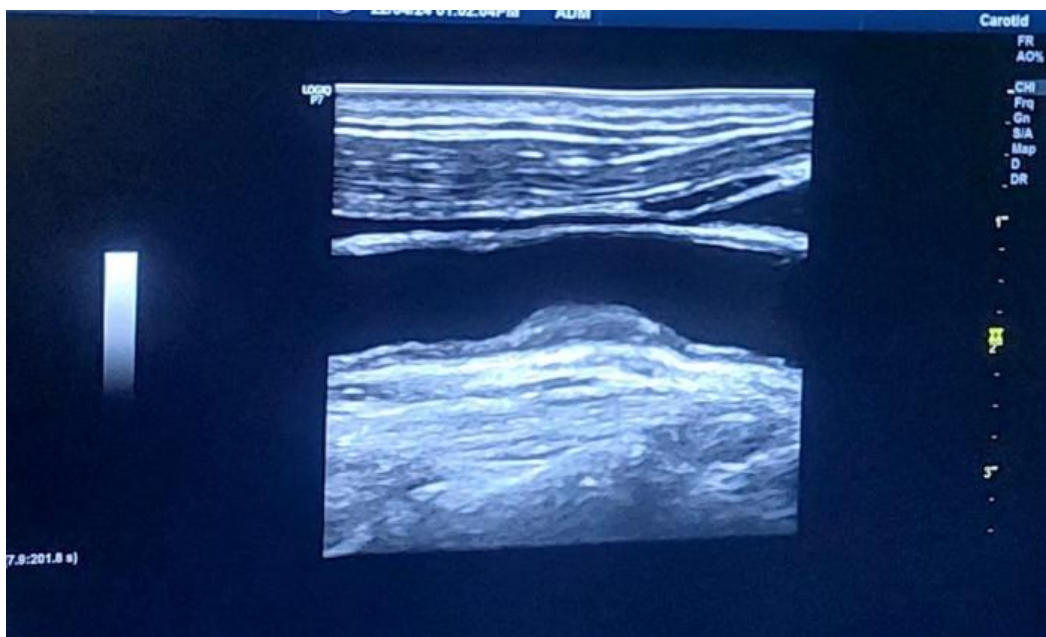
Ultrasound image shows hyperechoic plaques at carotid bulb

Image 13:



Ultrasound image shows heterogenous plaques in common carotid artery

Image 14:



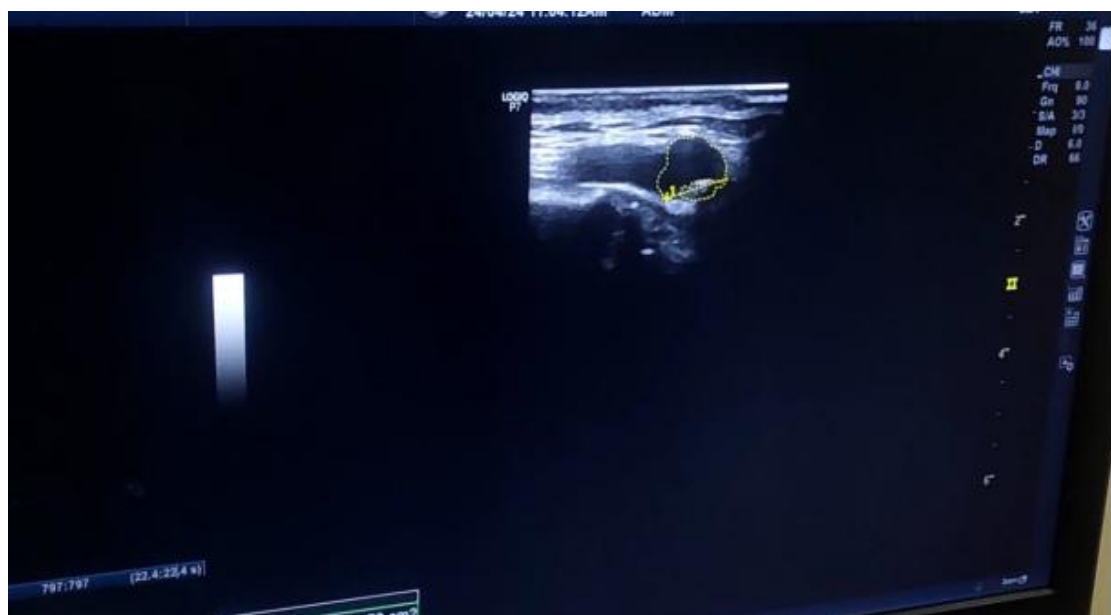
Ultrasound image shows heterogenous plaque in common carotid artery

Image 15:



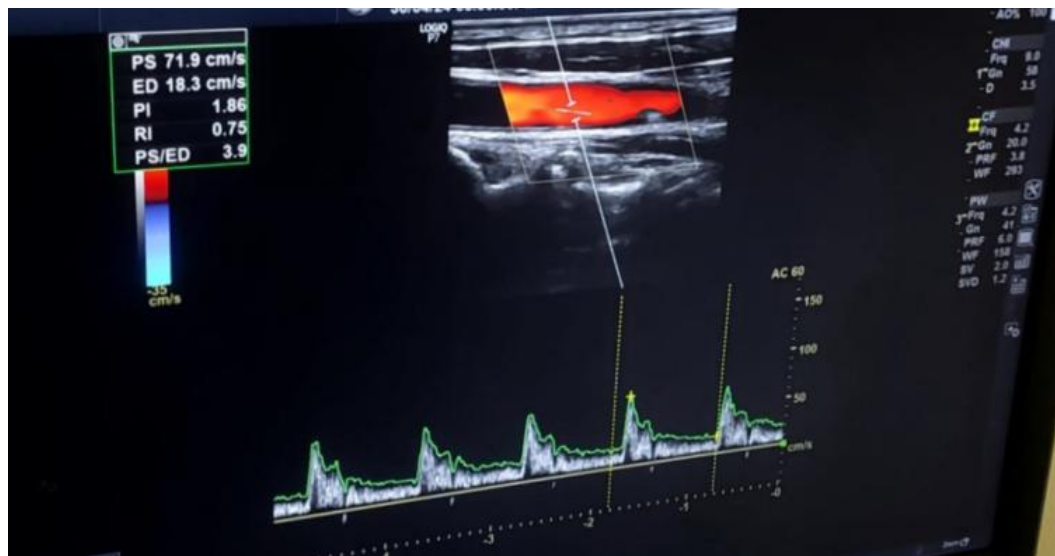
Ultrasound image shows hyperechoic irregular plaque in common carotid artery

Image 16:



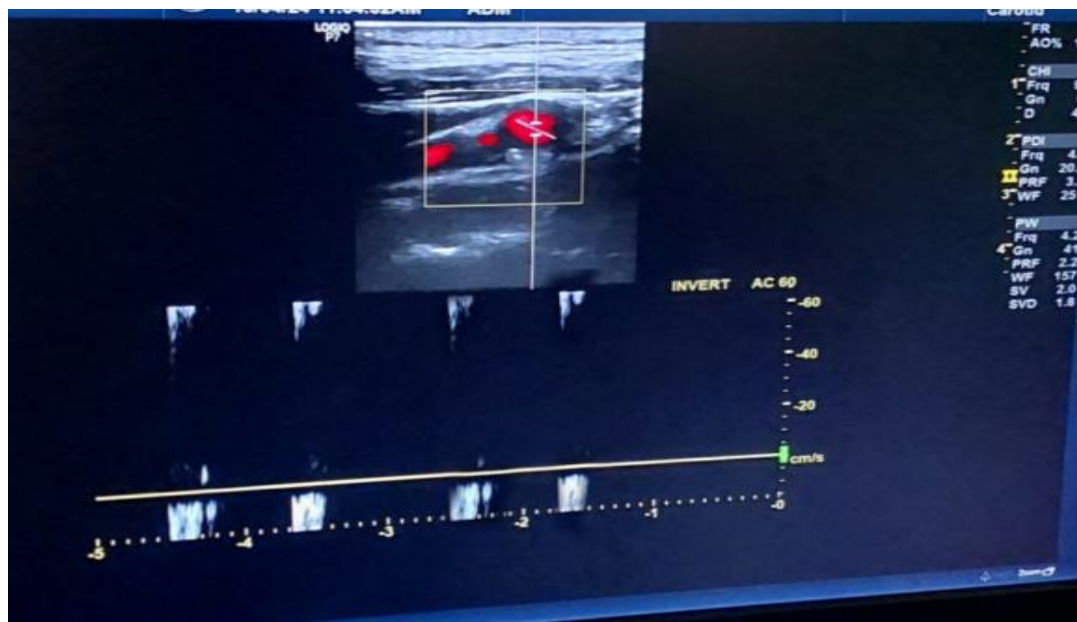
Ultrasound image shows hyperechoic plaque in common carotid artery

Image 17:



Ultrasound image shows hyperechoic plaque in common carotid artery

Image 18:



Ultrasound image shows hyperechoic plaque at carotid bifurcation

Image 19:



Ultrasound image shows hypoechoic plaque at carotid bulb

Image 20:



Ultrasound image shows heterogeneous plaque at carotid bifurcation

Image 21:



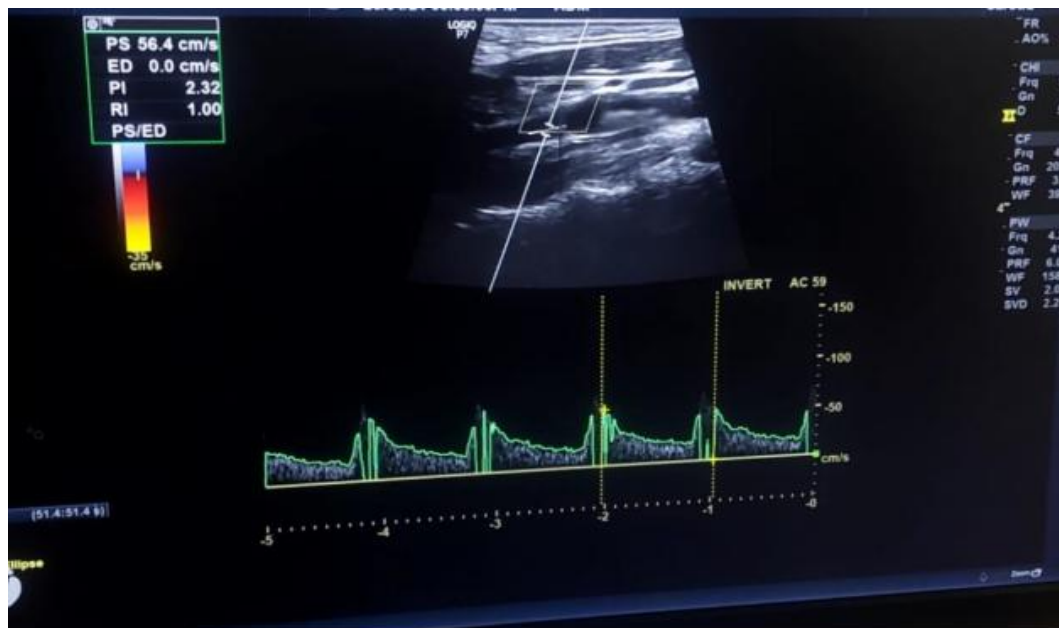
Ultrasound image shows heterogenous plaque at carotid bifurcation

Image 22:



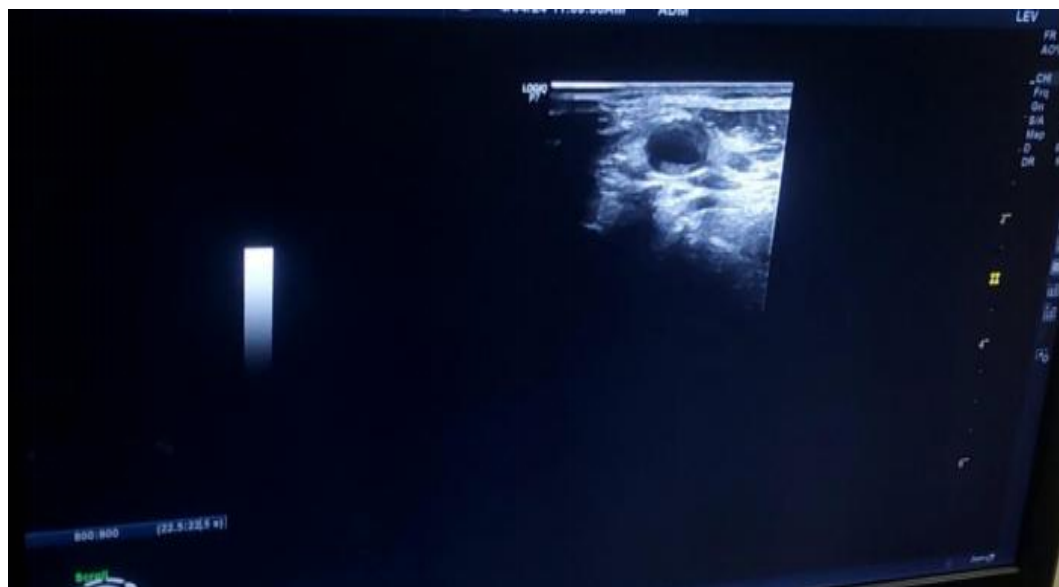
Ultrasound image shows heterogeneous plaque in common carotid artery

Image23:



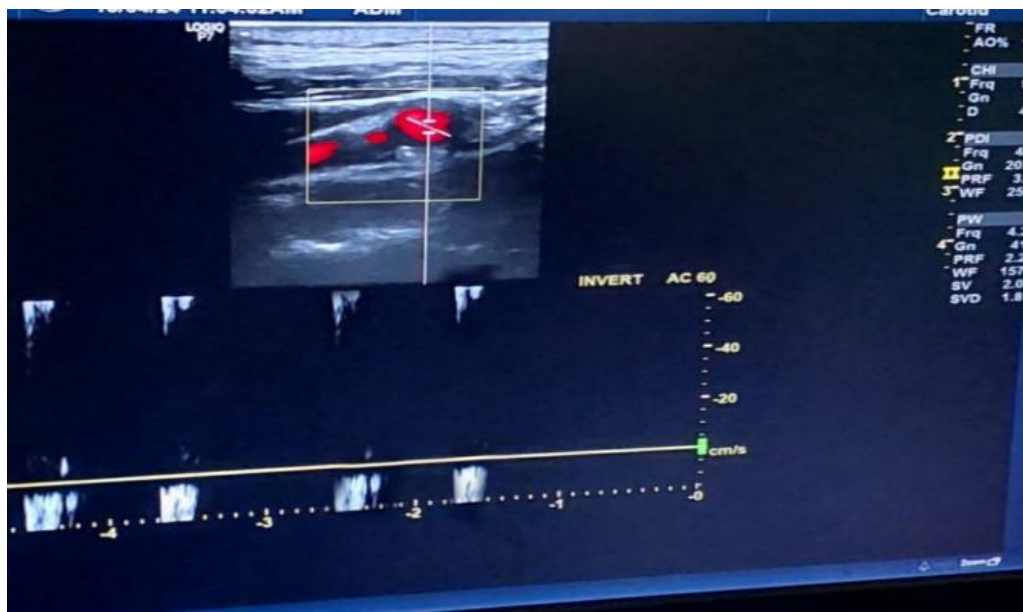
Ultrasound image shows heterogenous plaque in common carotid artery

Image 24:



Ultrasound image shows hyper-echoic plaque in at common carotid artery

Image 25:



Ultrasound image shows hyper-echoic plaque at carotid bifurcation

CONSENT FORMS

Description of the Research and Your Participation

You are invited to participate in a research study conducted by Haseeb Ahmad . The purpose of this research is to evaluate the “Carotid Plaque Characterization using Doppler Ultrasound in Patients with Diabetes, Hypertension and Hyperlipidemia”

Risks and Discomforts

There are no known risks associated with these scans. There is no invasive procedure. your data in this survey will be kept private and confidential and only be used for research purpose.

Potential Benefits

Your health status would be recognized among higher officials and health policies and programs may initiate for awareness, screening and treatment.

Protection of Confidentiality

The identity of patients will be kept private and confidential. Your all information will be kept private. Your personal identity such as name and location will not be revealed in any publication resulting from this study.

Voluntary Participation

Your participation in this research study is voluntary. You may choose not to participate and you may withdraw your consent to participate any time. You will not be penalized in any way should you decide not you participate or to withdraw from this study.

CONSENT

I have read this consent form and have been given the opportunity to ask questions.

I give my consent to participate in this study.

Participant’s Signature _ Date: A copy of this consent form should be given to the participant.

CONSENT FORM (URDU)

تحقیق میں شرکت کا دعوت نامہ

عنوان: ہائی بلڈ پریشر، ذیابیطس، اور ہائپر لیپیڈیمیا کے خطرے والے عوامل کے ساتھ ڈوپلر الٹراساؤنڈ کی تشخیص

نقصانات اور تکلیف: اس تحقیق سے کسی قسم کے نقصان یا تکلیف کا اندیشہ نہیں ہے۔

ممکنہ فوائد: آپ کو ایک اہم تحقیق میں حصہ لینے کا موقع دیا جائے گا۔

رازداری کا تحفظ: ہم آپ کی معلومات کے تحفظ کے لیے وہ سب کچھ کریں گے جو ہم کر سکتے ہیں۔ تحقیق کے متعلق اکٹھی کی گئی تمام معلومات کو انتہائی خفیہ رکھا جائے گا۔ ڈیٹا انٹری اور تجزیے کے دوران آپ کے متعلق وہ تمام معلومات جن سے آپ کی شناخت ہو سکتی ہو کو ختم کر دیا جائے گا۔ اس تحقیق کے نتیجے میں شائع ہونے والی کسی بھی اشاعت میں آپ کی شناخت کو ظاہر نہیں کیا جائے گا۔

رضاکارانہ شمولیت: اس تحقیقی مطالعہ میں آپ کی شرکت رضاکارانہ ہے۔ آپ کو شرکت نہ کرنے اور کسی بھی وقت بغیر وجہ بتانے اس تحقیق میں شمولیت کو چھوڑنے کا اختیار ہے۔ شرکت نہ کرنے یا اس میں شمولیت کو چھوڑنے کی صورت میں آپ کے خلاف کوئی کارروائی نہیں کی جائے گی

درج ذیل معلومات تحقیق میں شامل ہونے والوں کے لیے پڑھیں اور ان کا جواب دیے گئے خانوں میں درج کریں
میں نے معلوماتی شیٹ جو کہ تحقیق کی وضاحت کر رہی ہے کو سمجھ لیا ہے اور مجھے تحقیق کے سوالات کرنے کا موقع دیا گیا تھا۔

میں سمجھ گئی ہوں کہ میری شرکت رضاکارانہ ہے اور یہ کہ میں کسی بھی وقت اپنا ارادہ بدل سکتی ہوں اور تحقیق سے دستبردار سکتی

میں سمجھ گئی ہوں کہ میرے جوابات خفیہ رکھے جائیں گے۔ میں محققین کو اس بات کی اجازت دیتی ہوں کہ وہ جوابات کو جانچ سکیں۔

میں اوپر بتائی گئی تحقیق میں شامل ہونے کے لیے رضامند ہوں اور محققین کو اپنا پتہ تبدیل ہونے کی صورت میں مطلع کروں گی

رضا مندی: میں نے یہ اجازت نامہ پڑھا ہے اور مجھے سوال پوچھنے کا موقع دیا گیا ہے۔ میں اس سٹڈی میں شرکت کے راضی ہوں۔

شرکت کنندہ کا نام _____ دستخط _____ تاریخ _____

PERFORMA/QUESTIONNAIRE

**“Carotid Plaque Characterization using Doppler Ultrasound in Patients with Diabetes,
Hypertension and Hyperlipidemia”.**

Date: ____/____/____

Name: _____

Age: _____ years

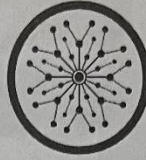
Gender: _____

BMI: _____

Diabetic	Yes	No
Hypertensive Patients	Yes	No
Hyperlipidemia	Yes	No
Ultrasound Findings		

Right CCA. PSV cm/sec	Right ICA PSV cm/sec	Left CCA PSV cm/sec	Left ICA PSV cm/sec	Appearance of plaque	IMT

October 29, 2025



PLAGIARISM CERTIFICATE

For MPhil and PhD

This is certified that the thesis entitled: **Carotid Plaque Characterization Using Doppler Ultrasound With Diabetes, Hypertension And Hyperlipidemia.**

Student Name: Haseeb Ahmad

Reg /Roll No: SU91-MSAHW-F23-042

Submitted on date: 27.10.2025

We have checked this document and found 10% plagiarism/similarity, 3% single source, and 0% AI in the document. It is certified that this document meets the HEC criteria.

Supervisor Signature

Research Project / Thesis



Librarian

Hassan Khalil
29-10-2025
Superior Information Resource Center

Report: CAROTID PLAQUE CHARACTERIZATION USING
DOPPLER ULTRASOUND WITH DIABETES, HYPERTENSION AND
HYPERLIPIDEMIA (HASEEB AHMAD)

ORIGINALITY REPORT

10 %	6 %	4 %	7 %
SIMILARITY INDEX	INTERNET SOURCES	PUBLICATIONS	STUDENT PAPERS

PRIMARY SOURCES

1	Submitted to Higher Education Commission Pakistan Student Paper	3%
2	Submitted to University of Okara Student Paper	3%
3	Submitted to Health and Care Professions Council Student Paper	<1%
4	academic.oup.com Internet Source	<1%
5	www.pertanika.upm.edu.my Internet Source	<1%
6	Campbell, I. C., D. Weiss, J. D. Suever, R. Virmani, A. Veneziani, R. P. Vito, J. N. Oshinski, and W. R. Taylor. "Biomechanical Modeling and Morphology Analysis Indicates Plaque Rupture Due to Mechanical Failure Unlikely in Atherosclerosis-Prone Mice", AJP Heart and Circulatory Physiology, 2012. Publication	<1%
7	www.panafrican-med-journal.com Internet Source	<1%
8	Adeyinka Tella. "Predictors of Library and Information Science Undergraduates"	<1%