“ROLE OF SONOELASTOGRAPHY IN EVALUATION OF MEDIAN NERVE CHANGES IN DIABETIC PATIENTS AND COMPARISION TO NORMAL SUBJECTS – A CASE CONTROL STUDY”

BY

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SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION & RESEARCH , TAMAKA, KOLAR, KARNATAKA

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF

DOCTOR OF MEDICINE
IN
RADIODIAGNOSIS

UNDER THE GUIDANCE OF
Dr. ANIL KUMAR SAKALECHA
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PRIOR PERMISSION TO START OF STUDY

The Institutional Ethics Committee of Sri Devaraj Urs Medical College, Tamaka, Kolar has examined and unanimously approved the synopsis entitled **"Role Of Sonoelastography In Evaluation Of Median Nerve Changes In Diabetic Patients And Comparision To Normal Subjects - A Case Control Study"** being investigated by **Dr.Soumya Z Chincholikar & Dr. Anil Kumar Sakalecha** in the Department of Radio-Diagnosis at Sri Devaraj Urs Medical College, Tamaka, Kolar. **Permission is granted by the Ethics Committee to start the study.**

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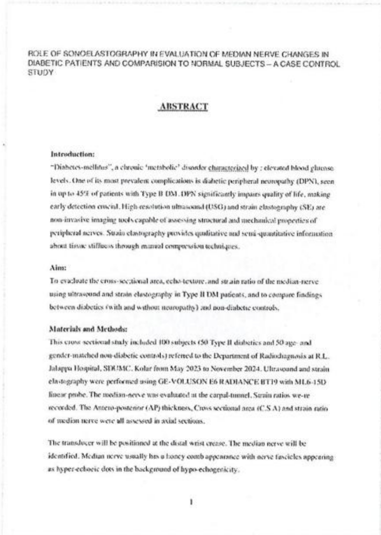

ROLE OF SONOELASTOGRAPHY IN EVALUATION OF MEDIAN NERVE CHANGES IN DIABETIC PATIENTS AND COMPARISION TO NORMAL SUBJECTS – A CASE CONTROL STUDY

ABSTRACT

Introduction:
"Diabetes-mellitus", a chronic 'metabolic' disorder characterized by : elevated blood glucose levels. One of its most prevalent complications is diabetic peripheral neuropathy (DPN), seen in up to 45% of patients with Type II DM. DPN significantly impairs quality of life, making early detection crucial. High-resolution ultrasound (USG) and strain elastography (SE) are non-invasive imaging tools capable of assessing structural and mechanical properties of peripheral nerves. Strain elastography provides qualitative and semi-quantitative information about tissue stiffness through manual compression techniques.

Aim:
To evaluate the cross-sectional area, echo-texture, and strain ratio of the median-nerve using ultrasound and strain elastography in Type II DM patients, and to compare findings between diabetics (with and without neuropathy) and non-diabetic controls.

Materials and Methods:
This cross-sectional study included 100 subjects (50 Type II diabetics and 50 age- and gender-matched non-diabetic controls) referred to the Department of Radiodiagnosis at R.L. Jalappa Hospital, SDUMC, Kolar from May 2023 to November 2024. Ultrasound and strain elastography were performed using GE-VOLUSON E6 RADIANCE BT19 with ML6-15D linear probe. The median-nerve was evaluated at the carpal-tunnel. Strain ratios we-re recorded. The Antero-posterior (AP) thickness, Cross sectional area (C.S.A) and strain ratio of median nerve were all assessed in axial sections.

The transducer will be positioned at the distal wrist crease. The median nerve will be identified. Median nerve usually has a honey comb appearance with nerve fascicles appearing as hyper-echoic dots in the background of hypo-echogenicity.

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LIST OF ABBREVIATIONS

DM	Diabetes mellitus
DPN	Diabetic polyneuropathy
NCSs	Nerve conduction tests/studies
CTS	Carpal tunnel syndrome
SE	Strain elastography
SWE	Shear wave elastography
kPa	kilopascals
m/s	meters per second
ARFI	Acoustic radiation force impulse
SWV	Shear wave velocity
CSA	Cross-sectional area
USS	Ultrasonography
PN	Peripheral neuropathy
HC	Healthy control
MNSI	Modified Michigan Neuropathy Screening Instrument
FLP	Fasting lipid profile
FBG	Fasting blood glucose
HbA1c	Glycated hemoglobin
CATL	Carpal tunnel
SD	Standard deviation
OGTT	Oral glucose tolerance test
A1C	Glycated hemoglobin
NGSP	National Glycohemoglobin Standardization Program
DCCT	Diabetes Control and Complications Trial
FPG	Fasting plasma glucose
IFG	Impaired fasting glucose
IGT	Impaired glucose tolerance
PAD	Peripheral arterial disease
NCV	Nerve conduction velocity
CIDP	Chronic inflammatory demyelinating polyneuropathy
MMN	Multifocal motor neuropathy
MADSAM	Multifocal acquired demyelinating sensory and motor neuropathy
Fig	Figure
ROI	Region of interest
APD	Anteroposterior diameter
HRU	High-resolution ultrasonography
TN	Tibial nerve
MN	Median nerve
CPN	Common peroneal nerve
wCSA	Cross-sectional area at the proximal carpal tunnel
fCSA	Cross-sectional area at the forearm
wSWE	Shear wave elastography at the wrist crease
fSWE	Shear wave elastography at the forearm
tSWE	Shear wave elastography within the carpal tunnel
AUC	Area under the curve
ROC	Receiver operating characteristic

ABSTRACT

Introduction:

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by elevated blood glucose levels. One of its most prevalent complications is diabetic peripheral neuropathy (DPN), seen in up to 45% of patients with Type II DM. DPN significantly impairs quality of life, making early detection crucial. High-resolution ultrasound (USG) and strain elastography (SE) are non-invasive imaging tools capable of assessing structural and mechanical properties of peripheral nerves. Strain elastography provides qualitative and semi-quantitative information about tissue stiffness through manual compression techniques.

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To evaluate the cross-sectional area, echotexture, and strain ratio of the median nerve using ultrasound and strain elastography in Type II DM patients, and to compare findings between diabetics (with and without neuropathy) and non-diabetic controls.

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This cross-sectional study included 100 subjects (50 Type II diabetics and 50 age- and gender-matched non-diabetic controls) referred to the Department of Radiodiagnosis at R.L. Jalappa Hospital, SDUMC, Kolar from May 2023 to November 2024. Ultrasound and strain elastography were performed using GE-VOLUSON E6 RADIANCE BT19 with ML6-15D linear probe. The median nerve was evaluated at the carpal tunnel. Strain ratios were recorded. The Antero-posterior (AP) thickness was assessed in longitudinal section, Cross sectional area (C.S.A) and strain ratio of median nerve were assessed in axial sections.

The transducer will be positioned at the distal wrist crease. The median nerve will be identified. Median nerve usually has a honey comb appearance with nerve fascicles appearing as hyper-echoeic dots in the background of hypo-echogenicity.

The AP thickness of the median nerve will be measured using the callipers.

The cross-sectional area (CSA) of the median nerve will be measured using the application of CSA under the musculoskeletal (MSK) settings. Under the MSK setting, once the median nerve is identified, AP thickness and CSA are taken. Adequate pressure is applied manually by the linear probe over the median nerve and a box with indication of green color for more than 70% is considered as adequate pressure. Now the ROI will be calculated using the formula;

ROI= AP thickness × 2/3

Once the ROI is calculated, strain elastrography application is used and ROI are placed over the median nerve and the surrounding tendon. The application gives the strain ratio. The AP thickness was assessed in longitudinal section. C.S.A and maximum strain ratio of median nerve were assessed in axial section.

In this way, ROI box and strain elastography box are kept fixed. Hence, standardising the study.

Results and Statistical Analysis:

Sample size was determined using data from Attah A.F. et al., targeting 90% power and 95% confidence interval, with 100 being total sample size. Data will be analyzed using SPSS v22. Categorical data will be expressed as frequencies and percentages, continuous data as mean ± SD. Chi-square and independent t-tests will assess significance, with $p < 0.05$ considered statistically significant.

Conclusion:

This study aims to establish the utility of ultrasound and strain elastography in early detection of diabetic neuropathy through structural and mechanical assessment of the median nerve, potentially improving disease management and patient outcomes.

Keywords: Diabetes Mellitus, Diabetic Peripheral Neuropathy, Median Nerve, Ultrasound, Strain Elastography, Cross-Sectional Area (CSA), Nerve Stiffness, High-Resolution Ultrasonography, Peripheral Nerve Imaging, Type II Diabetes.

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INTRODUCTION

INTRODUCTION

Diabetes mellitus (DM) is a persistent metabolic condition marked by hyperglycemia, arising from deficiencies in insulin production, insulin action, or both. It is a significant contributor to blindness, renal failure, myocardial infarctions, cerebrovascular accidents, and lower limb amputations. The incidence of diabetes has been increasing more swiftly in low- and middle-income nations than to high-income nations. Diabetes results in hyperglycemia, which, over time, inflicts significant damage on several bodily systems, particularly the nerves and blood vessels.

Chronic hyperglycemia is linked to prolonged damage, malfunction, and failure of several organs, including the eyes, kidneys, nerves, heart, and blood vessels. The worldwide incidence of diabetes has risen markedly in recent decades. In 2019, diabetes directly caused 1.5 million fatalities, with 48% of these deaths occurring prior to the age of 70. Furthermore, diabetes and diabetes-related kidney disease resulted in around 2 million fatalities [1,2].

Diabetic polyneuropathy (DPN) is the predominant type of neuropathy, accounting for 50–75% of non-traumatic amputations. It results in a greater number of hospitalizations than all other diabetes complications together. Painful diabetic peripheral neuropathy is frequently challenging to manage and correlates with diminished quality of life, inadequate sleep, depression, and worry. The management of neuropathy, including sufficient podiatric care for these patients, contributes to the fiscal strain on the national health system.

Consequently, the early identification of nerve dysfunction is crucial for triaging patients for referral and for initiating suitable and prompt care, thereby preventing significant consequences [3,4]. DPN is characterized by clinical symptoms such as numbness, paraesthesia, and tingling, along with indicators like loss of ankle reflex and vibration perception; diagnosis is verified with nerve conduction tests (NCSs). In recent years, emphasis has transitioned to the early identification of diabetic neuropathy during its preclinical phase. Several screening modalities employed in asymptomatic diabetics encompass the tuning fork test, monofilament testing, and nerve conduction studies (NCSs). The tuning fork and monofilament assessments exhibit high specificity and accuracy; yet, their sensitivity is limited. NCSs are laborious, uncomfortable, somewhat intrusive, and frequently poorly tolerated for successive assessments [5,6].

Peripheral neuropathy correlates with elevated intraneural pressure, making ultrasonic elastography an optimal technique for identifying early manifestations of this disorder through alterations in nerve stiffness. The predominant peripheral neuropathy in diabetes individuals is median neuropathy at the wrist. Estimates suggest that around 25% to 33% of individuals have either symptomatic or asymptomatic median mononeuropathy [7].

Diabetes affects hand function through many diseases, with the most prevalent manifestations being carpal tunnel syndrome, trigger finger, Dupuytren's contracture, and restricted joint mobility. The median nerve of the hand, essential for appropriate hand function, may be affected by metabolic abnormalities, ischemia, and/or entrapment neuropathies. Nonetheless, illness in this nerve frequently remains unacknowledged: the manifestations of carpal tunnel syndrome (CTS) are identified in 20-30% of diabetic individuals by electrophysiological assessment, whereas clinical manifestations are observed in about 5.8% of patients [8,9].

Ultrasound is an imaging modality that employs high-frequency sound waves to delineate tissue characteristics. It is readily accessible, user-friendly, and a more cost-effective imaging method. It is exceptionally secure and does not employ radiation. Ultrasound is a prevalent method for examining peripheral nerves, offering insights into nerve microstructure. Numerous peripheral nerves are situated superficially, making them readily accessible for ultrasound evaluation.

Strain elastography is a qualitative method that offers insights into the comparative stiffness of different tissues. In strain elastography, stress is delivered by repetitive manual compression of the transducer, and the degree of lesion deformation compared to adjacent normal tissue is quantified and shown in colour [10].

Elastography is an ultrasonic method utilized to assess the stiffness of biological tissue. The two predominant elastographic techniques are strain elastography (SE) and shear wave elastography (SWE). The previous, older model relies on operator-induced compression utilizing a transducer to assess tissue displacement and, consequently, its elasticity or stiffness. The outcome is displayed on a qualitative color scale, with elastic, intermediate, and stiff tissues indicated in red, green, and blue, respectively, or semi-quantitatively as a strain/elasticity ratio between structures. In the newly implemented SWE, the velocity of a transducer-induced shear wave is quantified across the tissue of interest and represented as a measurable quantity, either in kilopascals (kPa, as Young's modulus) or meters per second (m/s) [(9,10)].

Given the aforementioned pathophysiology of peripheral neuropathy characterized by elevated intraneural pressure and edema, ultrasonic elastography appears to be an optimal technique for identifying early stages of this ailment through alterations in the stiffness of the afflicted nerve.

Consequently, the present investigation was conducted to examine the relevance of sonoelastography in assessing median nerve alterations in diabetes patients and to compare these findings with those of normal participants.

AIMS & OBJECTIVES

AIMS & OBJECTIVES OF THE STUDY

1) To assess the cross-sectional area and echotexture of median nerve in diabetics and comparison with normal subjects by ultrasonography.

2) To compare the strain ratio of median nerve in diabetics and normal subjects.

3) To interpret ultrasound and strain ratio findings in diabetic patients with or without neuropathy in comparison to normal subjects.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

The term diabetes mellitus refers to disorders of improper glucose metabolism characterized by hyperglycemia. It is linked to a relative or absolute deficiency in insulin secretion, accompanied by various levels of peripheral resistance to insulin's effects. Periodically, the diabetic community reassesses the existing guidelines for the categorization, diagnosis, and screening of diabetes, incorporating new insights from research and clinical experience.

Type 2 diabetes mellitus is defined by hyperglycemia, insulin resistance, and a relative deficiency in insulin production. The pathophysiology is only poorly comprehended, characterized by heterogeneity, with both genetic variables influencing insulin secretion and sensitivity, as well as environmental factors like obesity, playing significant roles.

Type 2 diabetes

Type 2 diabetes is the predominant form of diabetes in adults, accounting for over 90 percent of cases. It is defined by hyperglycemia, often arising from a steady decline in insulin production from beta cells, compounded by underlying insulin resistance, leading to relative insulin insufficiency. The majority of individuals appear asymptomatically, with hyperglycemia identified during regular laboratory assessments, necessitating further testing. The incidence of symptomatic diabetes has diminished along with enhanced initiatives for early diagnosis via screening.

The hallmark symptoms of hyperglycemia, such as polyuria, polydipsia, nocturia, impaired vision, and weight loss, are sometimes recognized only in hindsight following the identification of increased blood glucose levels. Polyuria transpires when blood glucose levels dramatically exceed 180 mg/dL (10 mmol/L), beyond the renal threshold for glucose reabsorption, resulting in heightened urine glucose excretion. Glycosuria induces osmotic diuresis (i.e., polyuria) and hypovolemia, subsequently resulting in polydipsia. Patients who replenish their volume deficits with concentrated sugary beverages, such as regular sodas, worsen their hyperglycemia and osmotic diuresis.

Adults with type 2 diabetes may seldom exhibit a hyperosmolar hyperglycemic condition, distinguished by significant hyperglycemia, profound dehydration, and altered consciousness, however devoid of ketoacidosis. Diabetic ketoacidosis (DKA) is an unusual first manifestation of type 2 diabetes in adults, while it may arise under specific conditions, typically including severe infection or other acute illnesses.

DIAGNOSTIC CRITERIA

Fasting plasma glucose (FPG), two-hour plasma glucose from a 75-gm oral glucose tolerance test (OGTT), or glycated hemoglobin (A1C) can be utilized for diagnostic evaluation. The Oral Glucose Tolerance Test (OGTT) is hardly utilized, particularly in pregnancy, due to its impracticality.

The subsequent definitions align with the standards set out by the American Diabetes Association (ADA) and the World Health Organization (WHO). The diagnostic criteria were established based on the observed correlation between glycemic thresholds and the probability of developing retinopathy [11].

Table 1 - American Diabetes Association criteria for the diagnosis of diabetes

1. A1C ≥6.5%. The test should be performed in a laboratory using a method that is NGSP certified and standardized to the DCCT assay. *   **OR**
2. FPG ≥126 mg/dL (7 mmol/L). Fasting is defined as no caloric intake for at least 8 hours. *   **OR**
3. 2-hour plasma glucose ≥200 mg/dL (11.1 mmol/L) during an OGTT. The test should be performed as described by the World Health Organization, using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water. *   **OR**
4. In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose ≥200 mg/dL (11.1 mmol/L).

A1C: glycated haemoglobin; NGSP: National Glycohemoglobin Standardization Program; DCCT: Diabetes Control and Complications Trial; FPG: fasting plasma glucose; OGTT: oral glucose tolerance test*. In the absence of unequivocal hyperglycemia, diagnosis requires two abnormal test results from the same sample or in two separate test samples.

Table 2 - Categories of increased risk for diabetes (prediabetes)*

FPG 100 to 125 mg/dL (5.6 to 6.9 mmol/L) – IFG
2-hour post-load glucose on the 75 g OGTT 140 to 199 mg/dL (7.8 to 11.0 mmol/L) – IGT
A1C 5.7 to 6.4% (39 to 46 mmol/mol)

FPG: fasting plasma glucose; IFG: impaired fasting glucose; OGTT: oral glucose tolerance test; IGT: impaired glucose tolerance; A1C: glycated hemoglobin. For all 3 tests, risk is continuous, extending below the lower limit of the range and becoming disproportionately greater at higher ends of the range.

Pathophysiology

The pathogenesis of type 2 diabetes is multifactorial. Patients typically present with a combination of varying degrees of insulin resistance and defective insulin secretion (beta cell dysfunction). Both contribute to type 2 diabetes, with heightened demand for insulin action mediated by resistance that is not matched by insulin secretion. Its occurrence most likely represents a complex interaction among many genes and environmental factors, which are different among different populations and individuals [12].

Genetic susceptibility

More than 500 genetic variants have been robustly associated with type 2 diabetes and related to pathways of beta cell function and insulin action. There are ongoing studies of using genetics for prediction of risk of developing diabetes as well as for gaining insights into disease pathophysiology.

Role of environment

The most striking environmental risk factors in most patients who develop type 2 diabetes are increased weight gain and decreased physical activity, each of which increases the risk of diabetes.

The mechanism by which obesity induces insulin resistance is poorly understood. Inflammation may be the common mediator linking obesity to the pathogenesis of diabetes.

Drug-induced hyperglycemia

A large number of drugs can impair glucose tolerance. They act by decreasing insulin secretion, increasing hepatic glucose production, or causing resistance to the action of insulin.

Diabetes-related change in body composition

Type 2 diabetes is generally associated with overweight and obesity. These conditions can be considered not only important causes of type 2 diabetes, but also consequences of the disease itself that typically involve changes in fat distribution and muscle mass. Several studies have evaluated fat distribution in diabetic patients. A significantly higher trunk and visceral fat distribution and a reduction in total leg fat mass caused by a lower subcutaneous adipose tissue are associated with more intramuscular and intermuscular adipose tissue deposition[13].

A number of epidemiological studies conducted in different populations have investigated the distribution of muscle mass according to diabetes status, using different analytic approaches, and provide conflicting results[14]. Park et al.[15] demonstrated that in both sexes, the presence of diabetes was associated with a significantly higher appendicular (arms and leg) muscle mass. In the Invecchiare in Chianti (InCHIANTI) Study, an Italian population-based cohort study, older persons with diabetes had a larger cross-sectional calf muscle area although this difference disappeared after standardization for body mass.

Longitudinal studies have found that older adults with type 2 diabetes experience an accelerated loss of muscle mass compared to normoglycemic counterpart. In a study among 3153 older Chinese adults, participants with type 2 diabetes showed an accelerated appendicular lean mass loss over a period of 4 years, independently of the diabetes related conditions studied [16].

Park et al.[17] using data from the Health ABC Study demonstrated that older adults with either diagnosed or undiagnosed type 2 diabetes showed excessive loss of appendicular lean mass and trunk lean fat mass compared with nondiabetic subjects. The decline in muscle mass was higher in previously undiagnosed diabetic participants suggesting that the most important loss of lean mass might happen in the early stages of the disease or when diabetes is untreated. Similarly, data from the Osteoporotic Fractures in Men (MrOS) Study showed that men with untreated diabetes, diabetes treated without insulin sensitizers, or impaired fasting glycemia had greater loss in total and appendicular lean mass even after adjustment for medical comorbidities or lifestyle factors. In contrast, the relative loss in total and appendicular lean mass in men with diabetes treated with insulin sensitizers was significantly lower than that in normoglycemic men supporting a pivotal role of insulin resistance in the pathogenesis of muscle mass loss [18].

Diabetic complications

Chronic long-term complications of diabetes have been implicated in the pathogenesis of muscle impairment in type 2 diabetic patients. Lower extremity peripheral arterial disease (PAD) may functionally impair lower limb skeletal muscles by means of decreased blood flow that could lead to muscle atrophy, fewer muscle cells, and worse oxidative metabolism. Arterial stiffening, a dysfunction in blood vessel dynamics, has been related to reduced lower extremity blood flow volume in type 2 diabetic patients as well as to reduced muscle mass decline in the general population [19].

PAD is also associated with poor nerve conduction velocity (NCV) and with impaired lower extremity functioning in persons with and without symptoms of intermittent claudication. In addition to PAD, the autonomic nervous system plays a major role in capillary recruitment, and in patients with diabetes, subclinical autonomic nervous system alterations might affect contraction by reducing blood supply to the exercising muscle [20].

Diabetic peripheral neuropathy (DPN) is another long-term detrimental complication of type 2 diabetes that directly predisposes diabetic patients to disability in daily life activities. DPN, through sensory impairment, affects position sense leading to ataxia and reduces movement perception at the ankle, which is thought to contribute impaired dynamic balance control, slow walking speed and increased risk of falling. DPN, by means of sensory and motor impairment, is involved in foot ulceration that is a common cause of lower extremity disability and amputation [21].

Ultrasound elastography

Electrodiagnostic tests are the primary diagnostic method employed in assessing peripheral neuropathy, offering insights into peripheral nerve function and the extent of myelin impairment and axonal degeneration. In recent years, diagnostic ultrasonography has become more recognized as a supplementary examination to electrodiagnostic tests. The swift progress in ultrasonic technology, especially in the creation of high-frequency transducers, has led to enhanced resolution and picture quality. Numerous clinically significant peripheral nerves are situated superficially, making them easily accessible for ultrasonography evaluation. Nerves are conventionally assessed by B mode ultrasonography [22,23].

Peripheral polyneuropathy has been thoroughly assessed utilizing conventional ultrasonography methodologies. Ultrasound examinations may aid in the diagnosis of inflammatory neuropathies, including Guillain-Barré syndrome, chronic inflammatory demyelinating polyneuropathy (CIDP), multifocal motor neuropathy (MMN), and multifocal acquired demyelinating sensory and motor neuropathy (MADSAM) [24,25]. Characteristic ultrasound features are seen in some hereditary neuropathies, including Type 1A Charcot-Marie-Tooth disease, and ultrasonography may assist in differentiating hereditary neuropathy from CIDP.9 Standard ultrasonography examinations often fail to reveal diagnostic alterations in axonal neuropathy, however several investigations have noted associations between nerve dimensions, vascularity, and functional electrophysiological evaluations [26].

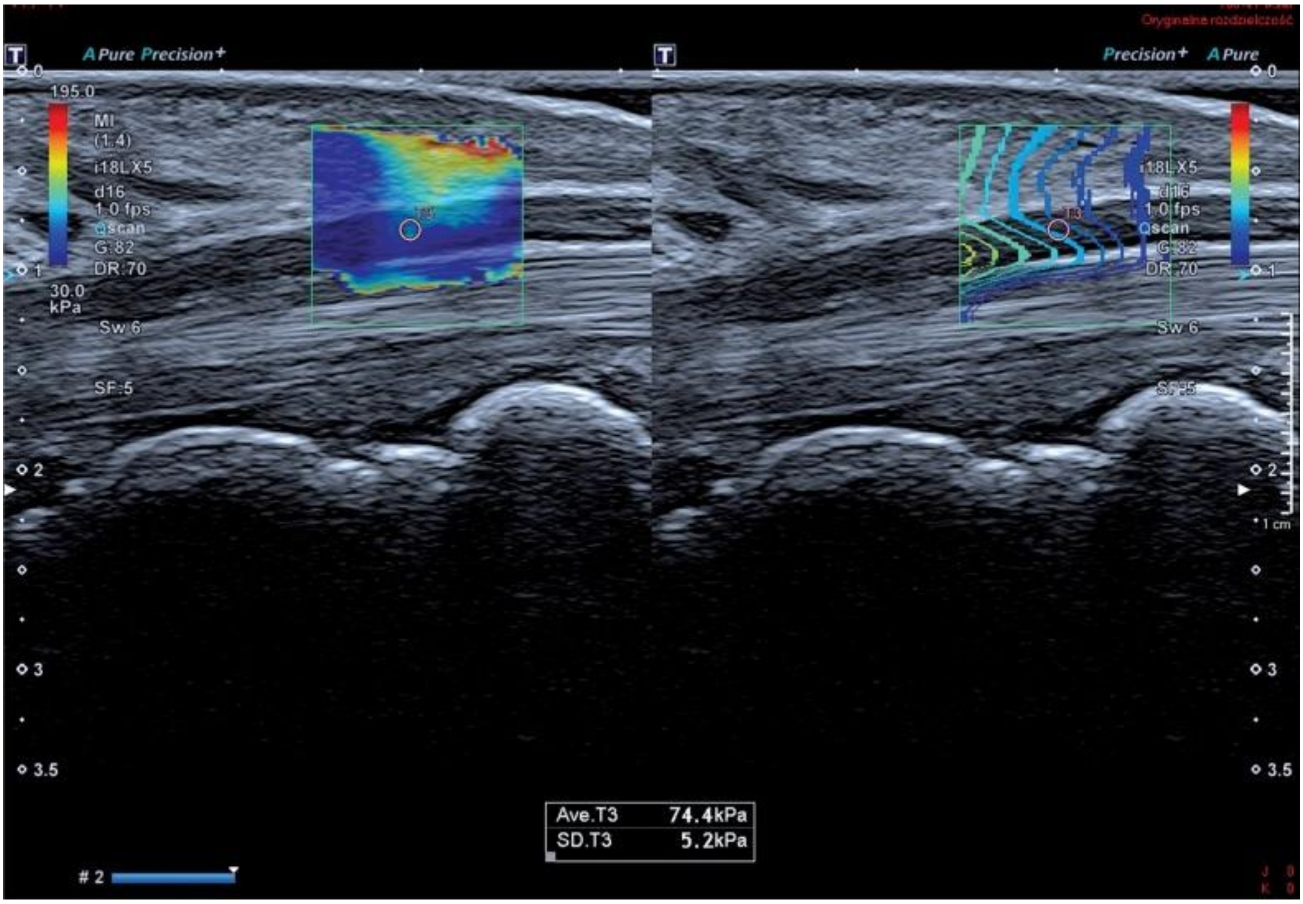

Figure 1: Shear wave elastography picture of the median nerve in the carpal tunnel of a 30-year-old male with normal nerve conduction investigations.

Ultrasound elastography is categorized into strain elastography and shear wave elastography (SWE), based on the determined physical quantity. Strain elastography can be categorized further according to the technique of tissue excitation. The initial technique for tissue excitation involves the manual compression of tissue using the transducer by the sonographer. The extent of manual compression may exhibit considerable intra- and interobserver variability. Mechanical devices have been created to enhance the standardization of force applied to tissues. Nonetheless, these gadgets are predominantly confined to research environments. The inability to exactly quantify the force applied to tissues complicates standardization, resulting in this approach offering qualitative information that is less beneficial for research requiring repeated investigations, while it may still be applicable in some therapeutic contexts [27].

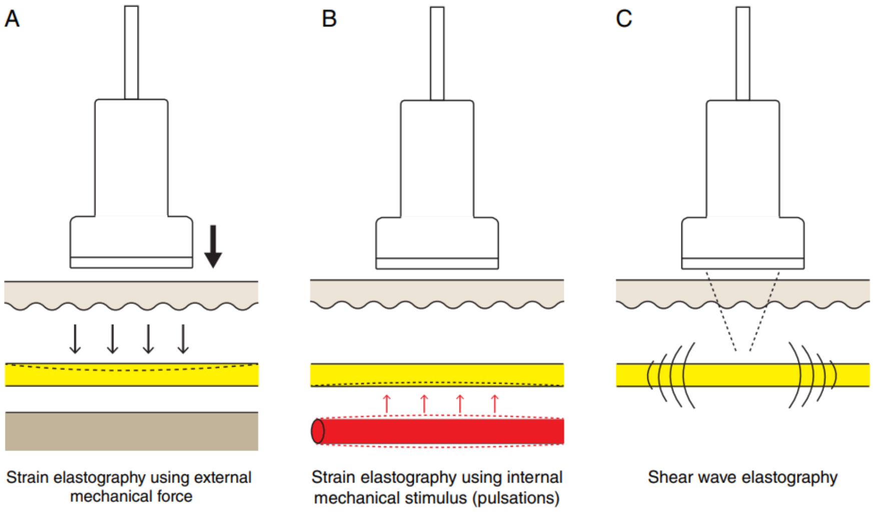

Figure 2: Depiction of ultrasonic elastography procedures employed in nerve evaluation. A, Strain ultrasound elastography employs external manual compression to generate mechanical excitation. B, Ambient strain ultrasonic elastography employs circulatory pulsations to generate mechanical stimulation. Respiration may also constitute a sort of mechanical excitation. Both modalities of strain ultrasound elastography provide a ratio indicating the distortion of the target tissue in relation to a reference tissue. C, Shear wave elastography (SWE) employs a focused acoustic radiation force produced by the ultrasonic transducer in a designated area, resulting in the creation of shear waves and subsequent tissue deformation. SWE provides a quantifiable measurement of tissue elasticity, namely shear wave velocity.

The second approach, termed ambient strain elastography, utilizes tissue oscillations generated by vascular pulsations to create tissue deformation, subsequently comparing two regions to ascertain a strain ratio.

This technology offers an advantage over traditional strain elastography that employs external compression, since it facilitates a more consistent and trustworthy assessment. Third, there exists acoustic radiation force impulse (ARFI) strain imaging. This approach generates high-intensity sonographic "push pulses," resulting in the displacement of underlying tissue. ARFI strain imaging, akin to ambient strain elastography, offers a more objective and consistent assessment of tissue compression than compression strain elastography [28].

In contrast to strain elastography, which assesses tissue displacement, shear wave elastography (SWE) generates shear waves following tissue activation using acoustic radiation force impulse (ARFI) or controlled external vibration. The shear wave velocity (SWV) is subsequently recorded, yielding a quantitative evaluation of tissue stiffness. Velocity correlates with tissue stiffness, with stiffer tissues linked to accelerated shear wave propagation. Tissue stiffness can be expressed as shear wave velocity (SWV) in meters per second (m/s) or as shear modulus in kilopascals (kPa). The correlation between shear modulus and shear wave velocity (SWV) is expressed as $G = \rho c^2$, where G denotes shear modulus, ρ represents tissue density (about 1000 kg/m^3), and c signifies SWV. The density of tissue will differ across various types of soft tissues [29,30].

Transient elastography employs controlled external vibration and serves as an alternate method to SWE, mostly utilized for evaluating liver fibrosis, with extensive validation for this application. The mechanical vibrating mechanism is incorporated into the transducer, which is also utilized to measure the SW.

Irrespective of the ultrasonic elastographic techniques employed, the outcomes can be qualitatively represented as a color elastogram that illustrates the relative variations in tissue stiffness within the designated region of interest. Conventionally, red signifies increased stiffness, whereas blue denotes less stiffness. B-mode imaging facilitates the identification of the region of interest, as the elastogram is often overlaid on the B-mode picture. Consequently, a high-quality B-mode picture serves as the fundamental prerequisite for achieving an accurate elastographic evaluation. The exception is transient elastography, which does not provide B mode pictures and so cannot assist in guiding the selection of the region of interest [31].

Median nerve

The predominant focus of current investigations on ultrasonic elastography has been the median nerve at the wrist. Numerous investigations employing both strain elastography and shear wave elastography (SWE) have been published in the past five years. In healthy control subjects, the median nerve has greater stiffness at the wrist compared to the forearm. Nonetheless, there was no notable disparity in the flexibility of the median nerve across sides, indicating that contralateral limbs may function as internal controls [32,33].

Numerous research on Carpal Tunnel Syndrome have indicated a disparity in nerve stiffness between CTS patients and control wrists, irrespective of the ultrasound elastography methods employed. Several studies have revealed a correlation between nerve stiffness and electrophysiological markers of carpal tunnel syndrome severity, indicating that greater CTS severity is related with increased nerve stiffness. Numerous cutoff values have been suggested for the diagnosis of carpal tunnel syndrome, along by corresponding sensitivity and specificity metrics [34–37].

The ratio of the stiffness of the median nerve at the carpal tunnel to that of the nerve in the forearm has been suggested as a more sensitive indicator of carpal tunnel syndrome, similar to the nerve cross-sectional area ratios frequently employed in assessing CTS and ulnar neuropathy. A ratio of 1.48 between median nerve stiffness measurements at the wrist and forearm was suggested as a diagnostic threshold for carpal tunnel syndrome, resulting in a sensitivity of 97.7% and a specificity of 100%. This figure closely aligns with the diagnostic cutoff threshold of the "swelling ratio" established when comparing the cross-sectional area at the wrist and forearm. This suggests a potential dependence between elastography and CSA. This observation necessitates targeted statistical examination in further investigations [38].

Ultrasound elastography has been investigated in particular patient demographics with carpal tunnel syndrome, including those who are pregnant and individuals undergoing hemodialysis. Generally, these investigations have revealed results analogous to idiopathic CTS. A relative increase in median nerve stiffness was seen in individuals with systemic sclerosis, leprosy, and acromegaly, despite the absence of clinically reported carpal tunnel syndrome; however, only one of the three investigations provided electrodiagnostic data. The importance of these studies is challenging to evaluate without baseline electrodiagnostic assessments. Since the conditions may be linked to peripheral nerve injury, the findings indicate that diagnostic "cutoff" values for carpal tunnel syndrome will vary among patients with other medical conditions impacting the peripheral nervous system and should not be applied indiscriminately as standalone values [39–41].

Nerve ultrasound elastography has been utilized to assess alterations in the median nerve subsequent to decompressive surgery for carpal tunnel syndrome. The rigidity of the median nerve considerably diminishes following carpal tunnel release, correlating with a reduction in symptoms. The study demonstrated that elastographic parameters exhibited more consistent improvement prior to alterations in nerve morphology, suggesting that nerve ultrasound elastography may serve as a more sensitive indicator of nerve recovery than cross-sectional area. Comparable alterations were seen following treatment with low-level laser energy and splinting. Consequently, median nerve ultrasonography elastography may serve as a viable objective metric to assess the efficacy of CTS therapies in clinical practice and trial design. An objective approach to assess treatment success is distinctly advantageous due to the significant impact of the surgical placebo effect on clinical response metrics. However, for any observations conducted before and after an intervention, it is essential to verify the repeatability of the employed techniques, especially for strain elastography methods [42,43].

It is crucial to assess if the incorporation of elastography alongside routine ultrasound imaging improves the diagnostic evaluation of individuals with CTS. Elastography enhanced the diagnostic precision of ultrasound examinations when utilized with conventional B-mode ultrasonography. Secondly, elastography may assist in the sonographic assessment of the severity of carpal tunnel syndrome, which might be pertinent to therapy selection [44,45].

In the study conducted by **Attah et al**. in the year 2019, to assess the cross-sectional area of the median nerve with B-mode ultrasonography (USS) and the occurrence of peripheral neuropathy (PN) in a group of adult diabetic Nigerians. Demographic and anthropometric data were collected from 85 adult diabetes mellitus (DM) patients and 85 age- and sex-matched apparently healthy control (HC) participants. A comprehensive physical examination was conducted on all research participants to ascertain the existence of peripheral neuropathy, and the modified Michigan Neuropathy Screening Instrument (MNSI) was employed to assess its severity. Venous blood was collected from the research participants for fasting lipid profile (FLP), fasting blood glucose (FBG), and glycated hemoglobin (HbA1c), while their median nerve cross-sectional area (MN CSA) was assessed at a location 5 cm proximal to the carpal tunnel and at the carpal tunnel using high-resolution B-mode ultrasound (USS). The data was analyzed with SPSS version 22.

The mean MN CSA was substantially greater in DM patients than in HC at 5cmCATL ($P < 0.01$) and at the CATL ($P < 0.01$) bilaterally. The existence of diabetic peripheral neuropathy (DPN) significantly elevated the MN cross-sectional area (CSA) at the CATL ($P < 0.05$), but not at 5cmCATL ($P > 0.05$). Nonetheless, the degree of diabetic peripheral neuropathy did not exert any further influence on the median nerve cross-sectional area 5 cm proximal to and at the carpal tunnel. No significant correlation was seen between MN CSA and the duration of diabetes mellitus and glycemic control. Thickening of the MN cross-sectional area at 5 cm CATL and CATL is observed in diabetes mellitus. The presence of DPN correlates with increased thickness of the MN CSA at the CATL, but not at the 5cm CATL. The severity of diabetic peripheral neuropathy, length of diabetes mellitus, and glycemic management did not exert any further influence on the cross-sectional area of the median nerve [46].

In the year 2009 **Watanabe et al**. studied the correlation between nerve conduction study (NCS) outcomes and nerve size as measured by sonography in diabetes individuals. Twenty diabetes patients (mean age ± SD, 57.1 ± 13.6 years) and twenty healthy volunteers (mean age, 61.1 ± 8.9 years) participated in this study. Patients exhibiting symptoms of carpal tunnel syndrome were excluded from the research; only those with negative Phalen test findings were included. Phalen's test is a diagnostic maneuver for carpal tunnel syndrome (CTS) that evaluates median nerve compression at the wrist. The test is performed by maintaining maximal passive wrist flexion for 30 to 60 seconds, which increases pressure within the carpal tunnel. A positive test is characterized by the reproduction of paresthesia or pain in the median nerve distribution (thumb, index, middle, and radial half of the ring finger), indicating potential median neuropathy. Subsequently, the patients were categorized into two groups: those with diabetic symmetric polyneuropathy (DPN) and those without. The cross-sectional area (CSA) was assessed in the carpal tunnel 5 cm proximal to the wrist and elbow joints of the median nerve.

A notable elevation in the CSA was seen in patients with DPN within the carpal tunnel, in comparison to control participants ($P < .01$) and patients without DPN ($P < .01$). The CSA in the carpal tunnel exhibited a notable connection with the motor nerve conduction velocity ($r = -0.473$). The cross-sectional area of the median nerve in the carpal tunnel of patients with diabetic peripheral neuropathy is larger than that of patients without diabetic peripheral neuropathy and healthy persons, and it corresponds with nerve conduction studies [5].

Bathala et al. conducted a study in year 2014 which aimed to get normative cross-sectional area (CSA) values for the median nerve using ultrasonography at specified locations and link them with electrophysiological parameters in healthy Asian individuals. Ultrasonographic examination of the median nerve was conducted on 100 healthy volunteers, with a mean age of 39 years (range, 18–75 years). The cross-sectional area of the median nerve was assessed at the wrist, mid-forearm, mid-arm, and axilla. All participants performed concurrent standardized nerve conduction tests.

The average cross-sectional areas (CSAs) of the median nerve ± standard deviation (SD) at the distal wrist crease were 7.2 ± 1 mm²; at mid-forearm 4.8 ± 0.9 mm²; at mid-arm 6.1 ± 1 mm²; and at the axilla 5.9 ± 0.9 mm². The cross-sectional area at the wrist was the most substantial compared to other levels ($P < 0.001$) and exhibited an increase with increasing age ($P < 0.002$). The normative data indicated that the cross-sectional area of the median nerve is not consistent along its length. Gender inequalities exist, and values tend to rise with age [47].

In the year 2014 **Kantarci et al** in his study quantified median nerve (MN) stiffness using shear wave elastography (SWE) at the carpal tunnel entrance and assessed the utility of SWE in diagnosing carpal tunnel syndrome. The study comprised 37 consecutive patients (60 wrists) with a confirmed diagnosis of carpal tunnel syndrome and 18 healthy volunteers (36 wrists). The MN cross-sectional area (CSA) was assessed using ultrasound, and stiffness was evaluated using SWE. The disparities between CTS patients and controls, as well as among subgroups based on electrodiagnostic assessments, were analyzed using the student's t-test. Interobserver variability and receiver operating characteristic analysis were conducted.

The MN stiffness was much greater in the CTS group (66.7 kPa) compared to the controls (32.0 kPa) ($P < 0.001$), and was also elevated in the severe or extreme severity group (101.4 kPa) relative to the mild or moderate severity group (55.1 kPa) ($P < 0.001$). A cut-off value of 40.4 kPa on SWE demonstrated a sensitivity of 93.3%, specificity of 88.9%, positive predictive value of 93.3%, negative predictive value of 88.9%, and an accuracy of 91.7%. The interobserver agreement for SWE measurements was exceptional. The stiffness of the median nerve at the carpal tunnel entrance was markedly elevated in individuals with carpal tunnel syndrome, for whom shear wave elastography is a notably repeatable diagnostic method [37].

Kang et al. performed the study in the study 2016 in which he examined and compared the ultrasonographic characteristics of various peripheral nerves in the upper and lower limbs of patients with diabetic peripheral neuropathy (DPN) to those of healthy controls. This research was a case-control study with 20 patients with a verified diagnosis of diabetic peripheral neuropathy and 20 healthy controls. Ultrasonography was conducted on the sural, tibial, fibular, sciatic, median, ulnar, radial, and musculocutaneous nerves. The cross-sectional area (CSA) of the nerve was assessed at several locations for each peripheral nerve. The CSAs were compared between the DPN and control groups and examined for clinical features and electrophysiological results.

The cross-sectional areas were much greater in the diabetic peripheral neuropathy group for the sural nerve, fibular nerve at the fibular head, median nerve at the carpal tunnel and mid-humerus, ulnar nerve at the cubital tunnel outlet and mid-humerus, and radial nerve at the spiral groove. The cross-sectional areas of the sural nerve, tibial nerve, and median nerve exhibited substantial correlation with electrophysiological data. The cross-sectional area of the sural nerve shown a strong connection with HbA1c levels. These results indicated ultrasonography offers valuable insights for the diagnosis and assessment of diabetic peripheral neuropathy [48].

In the systematic study done by **Zakrzewski et al.** in the year 2019, it aimed to assess the utility of strain elastography (SE) and shear wave elastography (SWE) in evaluating peripheral nerves in patients with neuropathy of diverse etiologies. Published evidence unequivocally demonstrates that ultrasound elastography can accurately diagnose various forms of peripheral neuropathies, including carpal tunnel syndrome, other entrapment neuropathies, diabetic peripheral neuropathy, and peripheral neuropathy linked to other systemic diseases, often at stages when the condition remains asymptomatic. Nonetheless, it remains uncertain if elastographic alterations in the nerves precede functional abnormalities identifiable by nerve conduction investigations.

Furthermore, less knowledge exists on the correlation between the rigidity of peripheral nerves and the severity of peripheral neuropathy along with its underlying ailment. According on the repeatability statistics, SWE appears to be superior over SE. Nonetheless, the origins of variability in peripheral nerve stiffness among healthy individuals must be elucidated, and reference value sets for specific peripheral nerves must be established. The possible confusing influence of hardening artifacts, such as bones, on the stiffness of peripheral nerves must be confirmed. Upon resolving these challenges, elastographic assessment of peripheral nerve rigidity may emerge as a dependable, readily accessible, and easy diagnostic procedure frequently conducted in patients with diverse peripheral neuropathies [49].

Aslan et al. in the year 2019 demonstrated the efficacy of shear wave elastography (SWE) in identifying morphological abnormalities of the median nerve and posterior tibial nerve in both transverse and longitudinal axes in adolescents with type 1 diabetes mellitus (DM) who do not have diabetic peripheral neuropathy (DPN). The median and posterior tibial nerves of 25 adolescents diagnosed with type 1 diabetes mellitus without diabetic peripheral neuropathy, together with 32 healthy volunteers, were assessed using shear wave elastography by two observers in both transverse and longitudinal orientations. The cross-sectional area, thickness of the nerves, and duration of the illness were recorded, and potential correlations between these parameters and SWE characteristics were examined. Interobserver and intra-observer correlations were analyzed. The threshold for statistical significance was established at P value $< .05$.

The median nerve and posterior tibial nerve exhibited reduced size, diminished thickness, and increased stiffness in the patient group, as seen by both observers across both axes. The length of the illness had a modest correlation with median nerve SWE characteristics ($r = 0.245$-0.391). The thickness and cross-sectional area exhibited no association with SWE characteristics. Adolescents with type 1 diabetes mellitus without diabetic peripheral neuropathy exhibit morphologic alterations in the median nerve and posterior tibial nerve, detectable by shear wave elastography irrespective of the imaging axis. Shear wave elastography may play a possible function in asymptomatic diabetic peripheral neuropathy; nevertheless, the reliability of the results was not as good as preferred [50].

Singh et al. in 2019 studied the efficacy of high-resolution ultrasonography (HRU) in the assessment of diabetic peripheral neuropathy (DPN). The study comprised thirty-seven adult diabetes patients with clinically confirmed diabetic peripheral neuropathy and forty-five healthy adult volunteers. High-resolution ultrasound of the right medial, ulnar, common peroneal, and posterior tibial nerves was performed. The average cross-sectional area (CSA) of the affected nerves was assessed in both groups at the same locations. The CSA was compared between the two groups, and a student's t-test was utilized to evaluate statistical significance.

The cross-sectional area of the median, ulnar, common peroneal, and posterior tibial nerves was significantly greater in individuals with diabetic peripheral neuropathy than in healthy volunteers. Sonographic data were compared with nerve conduction studies (NCS) for all examined nerves, excluding the common peroneal nerve, as NCS for CPN was not frequently performed. DPN was categorized as low or moderate to severe based on latency and velocity evaluated using NCS. The average CSA in all assessed nerves was greater in moderate to severe DPN compared to mild DPN; however, this difference was not statistically significant, except for the ulnar nerve, which exhibited a P value of < 0.0001. HRU exhibits a morphological alteration in individuals with DPN characterized by a statistically significant increase in CSAs. HRU can objectively enhance other diagnostic assessments, such as NCS. High-resolution ultrasonography of peripheral nerves may emerge as the preferred diagnostic modality for assessing DPN [51].

Wee and Simon, 2019 emphasized that Peripheral nerve diseases are often observed in clinical practice. Electrodiagnostic tests are fundamental to the assessment of nerve illnesses. Ultrasound has increasingly assumed a supplementary function in the neuromuscular clinic. Ultrasound elastography is a method that quantifies the elastic characteristics of tissues. Nerve ultrasound elastography has been investigated as a non-invasive method to assess alterations in nerve tissue composition, considering the histological abnormalities seen in sick peripheral nerves. Current studies indicate that nerve stiffness often escalates in cases of peripheral neuropathy, irrespective of the underlying cause, aligning with the reduction of pliable myelin and its substitution with connective tissue. This systematic review aims to synthesize the existing research on the application of ultrasonic elastography in assessing peripheral neuropathy. The constraints of ultrasonic elastography and deficiencies in existing literature are examined, along with potential future clinical and research uses [52].

The objective of the study done by **He et al.,** 2019 was to assess the efficacy of shear wave elastography (SWE) in identifying diabetic peripheral neuropathy affecting the median and tibial nerves. The study comprised 40 patients with diabetic peripheral neuropathy (DPN), 40 patients with diabetes mellitus (DM) without DPN, and 40 healthy individuals. High-resolution ultrasonography and shear wave elastography were conducted on the median nerve (MN) and tibial nerve (TN), measuring cross-sectional area (CSA) and nerve stiffness. ROC analysis was conducted as well.

Patients with diabetic peripheral neuropathy exhibited increased stiffness of the median and tibial nerves in comparison to healthy volunteers and diabetic mellitus patients ($P<0.001$). Bilateral examination indicated no significant difference in nerve stiffness between the left and right median nerves and tibial nerves in individuals with diabetic peripheral neuropathy ($P>0.05$). No significant difference was seen in the stiffness of the median nerve and tibial nerve on either side in individuals with diabetic peripheral neuropathy ($P>0.05$). The CSA of the tibial nerve in the DPN group was substantially greater than that in the other groups ($P<0.001$), although no significant difference in median nerve CSA was seen among the three groups ($P>0.05$). The area under the curve (AUC) of SWE (MN: 0.899, TN: 0.927) for diagnosing DPN was substantially superior than that of CSA (TN: 0.798). The ideal cut-off values in SWE for the tibial nerve and median nerve in diagnosing DPN were 4.11 m/s and 4.06 m/s, respectively, demonstrating high sensitivity and specificity. The stiffness of the median and tibial nerves was markedly elevated in patients with diabetic peripheral neuropathy (DPN). The data indicated that SWE-based stiffness assessment of the nerve is superior to CSA and can serve as an efficient supplementary approach in diagnosing DPN [53].

In year 2020, **Chen et al** investigated the use of conventional ultrasonography and real-time shear wave elastography (SWE) for the tibial nerve and the common peroneal nerve in diabetic peripheral neuropathy. Thirty-three healthy volunteers, 33 diabetic patients without diabetic peripheral neuropathy, and 30 diabetic patients with DPN participated in this study. The anteroposterior diameter (APD), cross-sectional area, and perimeter of the tibial nerve and common peroneal nerve were assessed using conventional ultrasonography, while nerve stiffness was evaluated by shear wave elastography.

The standard ultrasonography parameters and stiffness of the TN in patients with DPN were much greater than those in the other two groups ($P < 0.01$). The standard ultrasonography parameters of the CPN were markedly elevated in individuals with DPN compared to the other two groups (P <

0.01). Patients with diabetic peripheral neuropathy exhibited increased stiffness of the common peroneal nerve compared to the control group ($P < 0.05$). The comparison of all parameters for the left and right tibial nerves and common peroneal nerves across the three groups revealed no significant differences. The area under the curve for TN stiffness in diagnosing DPN was much superior to those of traditional ultrasonography parameters. The standard ultrasonography parameters and the stiffness of the TN and CPN were markedly elevated in patients with DPN. The stiffness of the tibial nerve may more effectively detect diabetic peripheral neuropathy than traditional ultrasonography measures. In summary, conventional ultrasonography and shear wave elastography of nerves provide significant diagnostic utility in the identification of diabetic peripheral neuropathy [54].

Narayan et al. in the year 2021 performed an observational study which an objective to conduct an ultrasound assessment of the cross-sectional area of peripheral nerves in individuals with suspected diabetic peripheral sensorimotor neuropathy. CSA was evaluated in relation to clinical factors and nerve conduction study metrics for early diagnosis and involvement patterns. Fifty patients with probable diabetic peripheral neuropathy resulting from Type 2 diabetes and fifty age-matched healthy controls underwent sonographic evaluations of the ulnar nerve in the lower arm, the median nerve proximal to the carpal tunnel, the common peroneal nerve proximal to the fibular head, the tibial nerve proximal to the tarsal tunnel, and the sural nerve in the lower third of the leg.

CSA was elevated in instances with DPN relative to healthy controls. The alterations in area were more pronounced with a demyelinating pattern. Probable DPN patients with normal NCS had a considerably greater number of peripheral nerves with elevated CSA compared to healthy controls. A threshold of >4 nerve thickening demonstrated a sensitivity of 86% and a specificity of 56%. The neuropathy pattern in the lower limb was axonal, whereas in the upper limb, it was demyelinating, with most cases exhibiting sonographic features indicative of concomitant compressive neuropathy. There was an elevation in the cross-sectional area of peripheral nerves in diabetes individuals. It served as a morphological marker for the classification of DPN, with alterations detected prior to NCS abnormalities. The clinical neurological manifestation in likely diabetic peripheral neuropathy may also result from compressive neuropathy in the first stages, and ultrasonography might serve as an effective diagnostic technique [55].

In 2022 study done by **Wu et al.** assessed the efficacy of conventional ultrasound and real-time shear wave elastography in evaluating median neuropathy in patients with carpal tunnel syndrome pre- and post-surgery. The Boston Carpal Tunnel Questionnaire (BCTQ) was initially administered to individuals diagnosed with carpal tunnel syndrome. All patients were assessed at three locations: the distal third of the forearm, the proximal carpal tunnel, and the distal carpal tunnel, utilizing conventional ultrasonography and shear wave elastography. Median nerve parameters were assessed in individuals with carpal tunnel syndrome one-week post-surgery.

The cross-sectional area and stiffness of the median nerve at the carpal tunnel entrance and distal carpal tunnel were markedly elevated in patients with carpal tunnel syndrome compared to healthy controls ($p < 0.001$). The cross-sectional area and stiffness of the median nerve at the carpal tunnel inlet exhibited statistically significant differences between preoperative and postoperative patients with carpal tunnel syndrome ($p < 0.001$). The cross-sectional area and stiffness of the nerve in individuals with carpal tunnel syndrome had a favourable connection with the severity of electrophysiological findings. Conventional ultrasonography and elastography are essential in diagnosing carpal tunnel syndrome and are beneficial for clinically evaluating nerve healing post-surgery [56].

Neto et al. in the year 2024, in the study emphasised, Ultrasound shear wave elastography as novel non-invasive imaging modality for the assessment of peripheral nerves. Shear wave velocity (SWV), an indirect indicator of stiffness, has potential as a biomarker for many peripheral nerve diseases. To optimize its clinical and biomechanical significance, it was essential to thoroughly comprehend the elements that affect nerve SWV readings. This systematic study sought to determine the normative range of shear wave velocity for healthy sciatic and tibial nerves and to elucidate the factors that may influence nerve SWV. An electronic search identified 17 studies suitable for inclusion, encompassing 548 healthy participants (age range, 17 to 72 years). Notwithstanding excellent reliability metrics, the documented SWV values exhibited significant variation among studies for the sciatic (1.9-9.9 m/s) and tibial (2.3-9.1 m/s) nerves. Factors including measuring closeness to joint areas, limb postures that induce axial nerve stretching, and transducer alignment with nerve fiber orientation were correlated with elevated SWV. The findings indicated region-specific neuron mechanical characteristics, non-linear elastic behaviour, and significant mechanical anisotropy. The influence of age and gender was ambiguous and requires more research. These results underscored the need of accounting for these parameters when evaluating and interpreting nerve SWE. Although elevated SWV correlates with pathological alterations in nerve tissue mechanics, the considerable diversity seen in healthy nerves underscores the necessity for standardized shear wave elastography testing techniques. Formulating recommendations for improved clinical utility and attaining a thorough comprehension of the elements that affect nerve function. SWE evaluations are essential for the progression of the discipline [57].

In the year 2024 **Martikkala et al.** investigated the correlations among median nerve shear wave elastography, MN cross-sectional area, patient complaints, and the neurophysiological severity of carpal tunnel syndrome. The best suitable venue for conducting SWE was also evaluated. This prospective research included 86 wrists from 47 consecutive patients who gave consent to undergo MN ultrasonography following an electrodiagnostic evaluation. The neurophysiological severity of carpal tunnel syndrome was evaluated based on the findings of a nerve conduction study. The MN cross-sectional area was assessed at the proximal carpal tunnel (wCSA) and the forearm (fCSA). Shear wave elastography was conducted on the median nerve (MN) in a longitudinal orientation at the wrist crease (wSWE), at the forearm (fSWE), and within the carpal tunnel (tSWE).

The wCSA and wSWE exhibited a positive correlation with the neurophysiological severity of CTS ($r = .619$, $P < .001$; $r = .582$, $P < .001$, respectively). The ideal cut-off values to differentiate between groups with normal NCS and those with CTS results were 10.5 mm^2 for the wCSA and 4.12 m/s for the wSWE. Utilizing these cut-off values, wCSA exhibited a sensitivity of 80% and a specificity of 87%, whereas wSWE demonstrated a sensitivity of 88% and a specificity of 76%. Neither tSWE nor fSWE exhibited a correlation with the neurophysiological severity of CTS, nor did they vary between NCS negative and positive groups ($P = .429$, $P = .736$, respectively). The shear wave velocity in the MN at the proximal carpal tunnel rises in carpal tunnel syndrome and corresponds with its neurophysiological severity of CT, similarly to the cross-sectional area assessed at the same location [58].

MATERIALS & METHODS

MATERIALS AND METHODS

SOURCE OF DATA

STUDY DESIGN: 100 patients, out of which 50 with Type II diabetes mellitus and 50 age and gender matched non-diabetic patients (control group) referred for ultrasonography and strain elastography to the department of Radiodiagnosis at R.L. Jalappa Hospital and Research Center attached to SDUMC, Kolar.

STUDY PERIOD: May 2023 – Nov 2024.

INCLUSION CRITERA:

1. Cases: Patients diagnosed with type II diabetes mellitus with or without neuropathy.
2. Controls: Age and gender matched subjects without diabetes mellitus and are undergoing ultrasound and strain elastography for a cause unrelated to median nerve neuropathy
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EXCLUSION CRITERIA:

3. Thyroid disorders/Rheumatoid arthritis.
4. History of surgeries for hand / wrist fractures.

METHOD OF COLLECTION OF DATA

Prior written Informed consent will be taken for their willingness to participate in the study. The patients will be included in the study if they fulfil the inclusion/exclusion criteria. Baseline data will be collected from the patients along with clinical history and relevant lab investigations. Ultrasonography and strain elastography will be performed using GE-VOLUSON E6 RADIANCE BT 19 ultrasound system equipped with strain wave point quantification, using high resolution ML6-15D linear probe.

Each participant will be seated on examination couch with a pillow on his/her lap. The forearm will be placed supine on the pillow with elbow and fingers semi-flexed during the examination of the median nerve. Following adequate positioning, gel will be applied to the anterior part of the wrist joint, over the carpal tunnel.

The volar wrist crease and pisiform bone will be used as external reference points and landmarks during scanning. The transducer will be positioned at the distal wrist crease. The median nerve will be identified. Median nerve usually has a honey comb appearance with nerve fascicles appearing as hyper-echoeic dots in the background of hypo-echogenicity.

The AP thickness of the median nerve will be measured using the callipers.

The cross-sectional area (CSA) of the median nerve will be measured using the application of CSA under the MSK settings. Under the MSK setting, once the median nerve is identified AP thickness and CSA are taken. Adequate pressure is applied manually by the linear probe over the median nerve and a box with indication of green color for more than 70% is considered as adequate pressure. Now the ROI will be calculated using the formula;

ROI= AP thickness × 2/3

Once the ROI is calculated, strain elastrography application is used and ROI are placed over the median nerve and the surrounding tendon. The application gives the strain ratio. The AP thickness, C.S.A and strain ratio of median nerve were all assessed in axial section.

In this way, ROI box and strain elastography box are kept fixed. Hence, standardising the study.

Figure 3: GE-VOLUSON E6 RADIANCE BT 19 ultrasound machine used for the study

Figure 4: ML6-15D linear probe used for the study.

SAMPLE SIZE ESTIMATION: Was estimated by using the difference in Mean median nerve CSA at carpal tunnel between DM group and Healthy group from the study Attah A F et. al.[1] as 7.9 ± 1.9 and 5.4 ± 14. Using these values at 95% Confidence limit and 90% power sample size of 32 was obtained in each group by using the below mentioned formula and Med calc sample size software. With 10% nonresponse sample size of 32 + 3.2 ≈ 35 cases will be included in each group. Rounding off to the nearest number , we will be taking sample size as 100, with 50 in normal group, 25 in diabetics without neuropathy and 25 in diabetics with neuropathy.

Sample Size Estimation Formula:

$$N = \frac{2\ SD^2 - (Z_{\alpha/2} + Z_{\beta})^2}{d^2}$$

- Where Zα/2 is the critical value of the Normal distribution at α/2 (e.g., for a confidence level of 95%, α is 0.05 and the critical value is 1.96).

- $Z\beta$ is the critical value of the Normal distribution at β (e.g., for a power of 90%, ß is 0.1 and the critical value is 1.28),
- SD is the standard deviation from previous study population variance, and
- d is the largest difference between two means

STATISTICAL METHODS

Data will be entered into Microsoft excel data sheet and will be analyzed using SPSS 22 version software. Categorical data will be represented in the form of Frequencies and proportions. Chi-square will be the test of significance. Continuous data will be represented as mean and standard deviation. Independent t test will be the test of significance to identify the mean difference between two groups. P value <0.05 was considered as statistically significant.

RESULTS

RESULTS

The study comprised a total of 100 participants, categorized into three age groups. The highest proportion of participants (68 individuals) were aged between 46-55 years, with a mean age of **49.71 years**. The younger group (>45 years) had a mean age of **44.07 years**, while the oldest group (56-65 years) had a mean age of **58.47 years**.

1) Age Distribution

Table 3:

Age	Frequency
>45	15
46-55	68
56-65	17

Table 3 shows age group and number of participants belonging to those age groups.

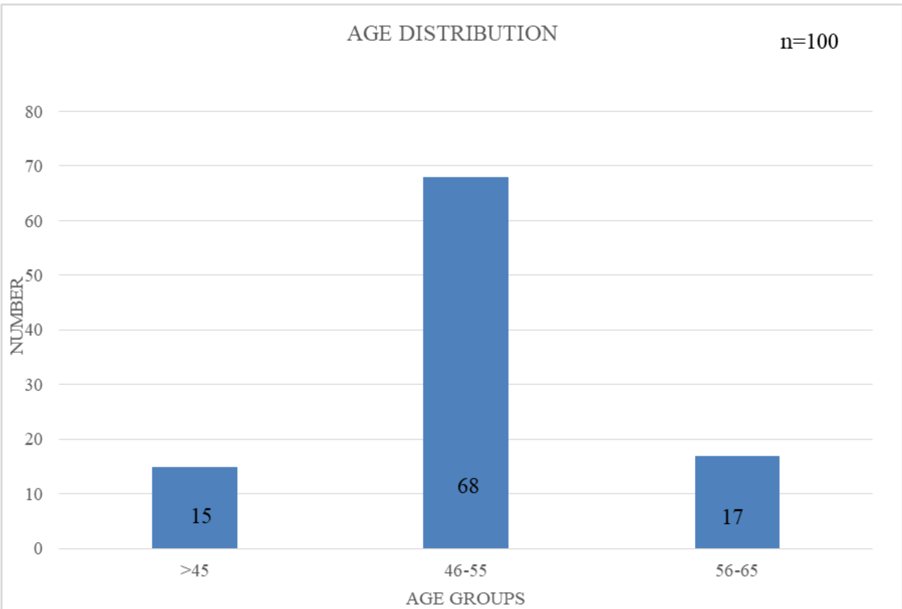

Figure 5: Graphical representation showing age distribution and number among different age groups.

2) Gender

Table 4:

Gender	Frequency
Female	49
Male	51

Table 4 shows number of participants belonging to male and female genders.

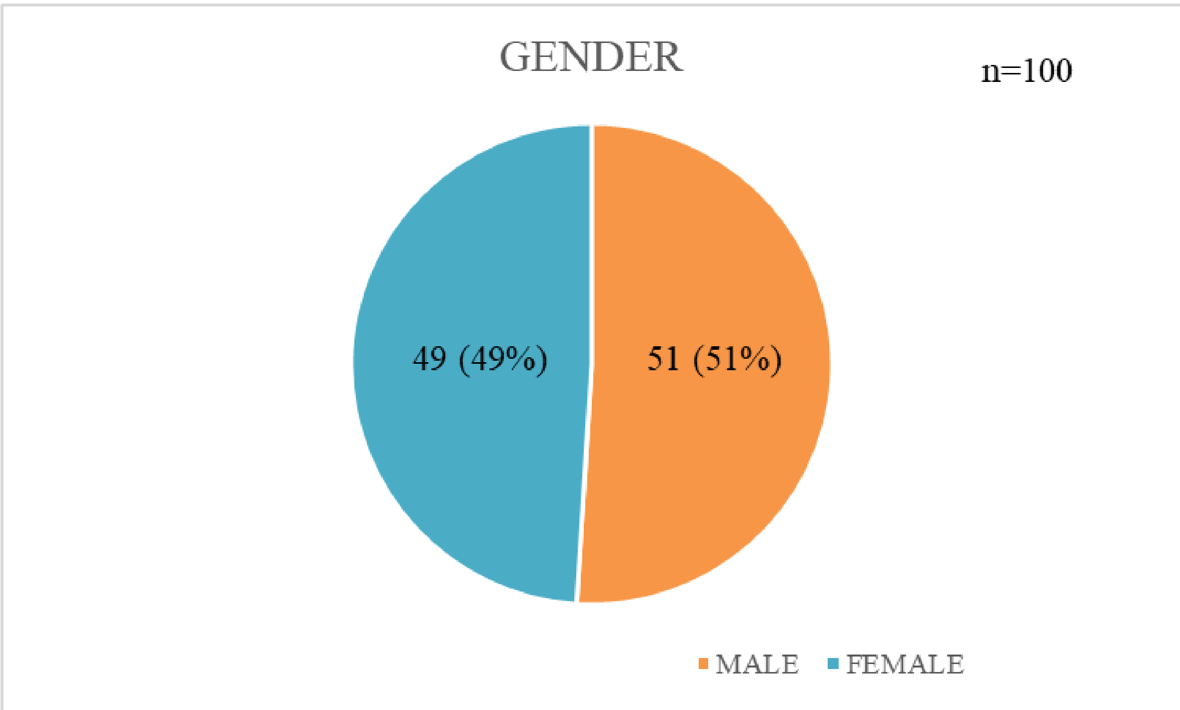

Figure 6: Among the participants, there were **51 males and 49 females**, ensuring a near-equal gender distribution across the study population.

3) Groups between normal subjects, diabetics with neuropathy and diabetics without neuropathy.

Table 5:

Group	Total
Normal	50
Diabetic Neuropathy	25
Diabetic without neuropathy	25

Table 5 shows number of participants belonging to different groups: normal, diabetics with neuropathy and diabetics without neuropathy.

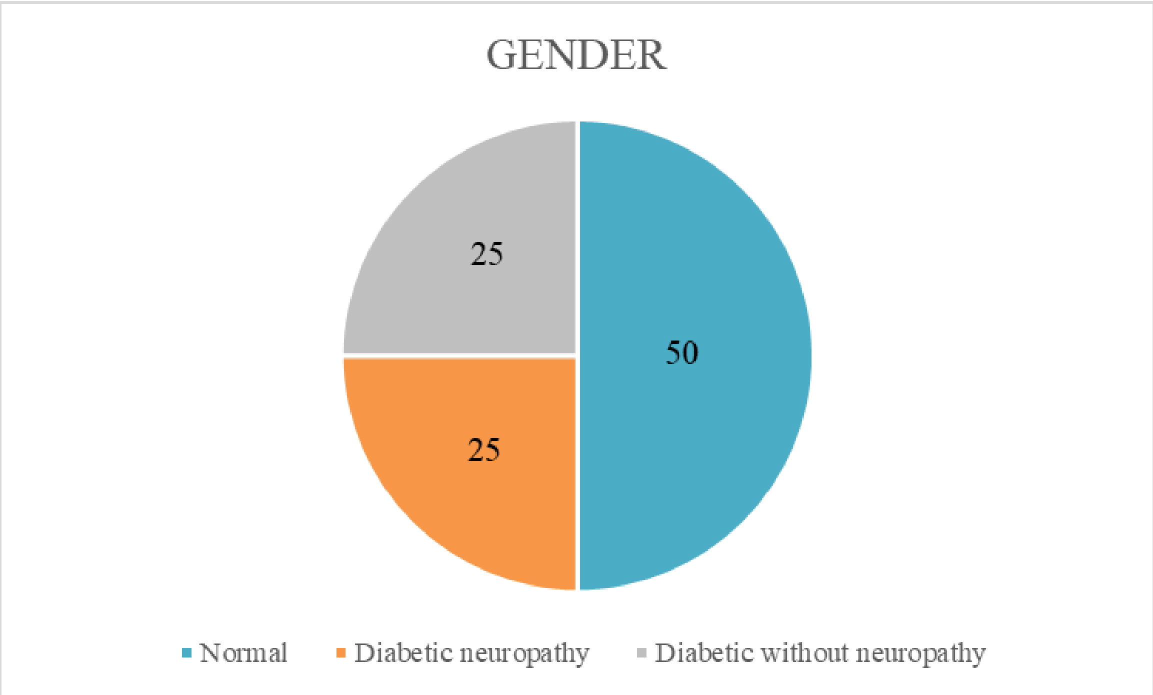

Figure 7: Pie chart showing distribution of participants among different groups.

4) Median Nerve

The sonoelastographic parameters of the median nerve revealed the following mean values:

- **Anteroposterior (AP) Thickness: 1.547 mm** (Standard Deviation: 0.3289)
- **Cross-Sectional Diameter: 0.067 cm²** (Standard Deviation: 0.0241)
- **Strain Ratio: 1.0425** (Standard Deviation: 0.4523)

These values indicate variations in nerve structure between diabetic and non-diabetic individuals. Further subgroup analysis is needed to determine the significance of these findings.

Table 6:

Nerve parameters	Mean ± St deviation
AP Thickness (mm)	1.547 ± 0.328896
Cross Section Area (cm^2)	0.067 ± 0.024142
Strain Ratio	1.0425 ± 0.452344

Table 6 shows mean values of different parameters of median nerve: AP thickness, C.S.A and strain ratio of median nerve.

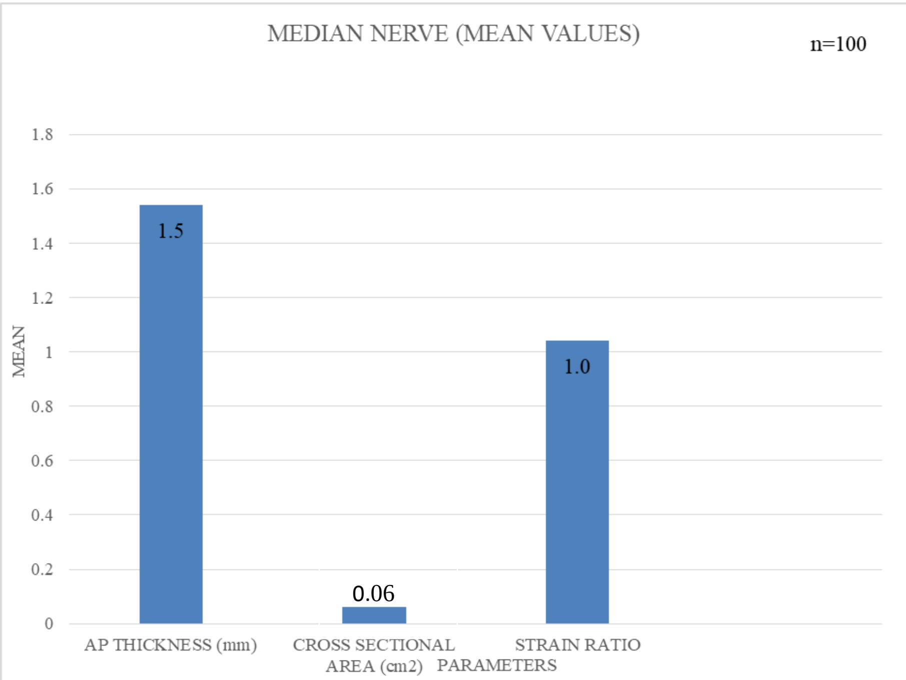

Figure 8: Graphical representation showing mean value of AP thickness, CSA and strain ratio of median nerve.

5) Echogenicity

Table 7:

Groups	**Number**	**Echogenicity**
Normal subjects	**50**	**Normal**
Diabetics without neuropathy	**25**	**Maintained**
Diabetics with neuropathy	**20**	**Altered**
Diabetics with neuropathy	**5**	**Maintained**

Table 7 shows distribution of type of echogenicity of median nerve in different groups: normal, diabetics without neuropathy and diabetics with neuropathy.

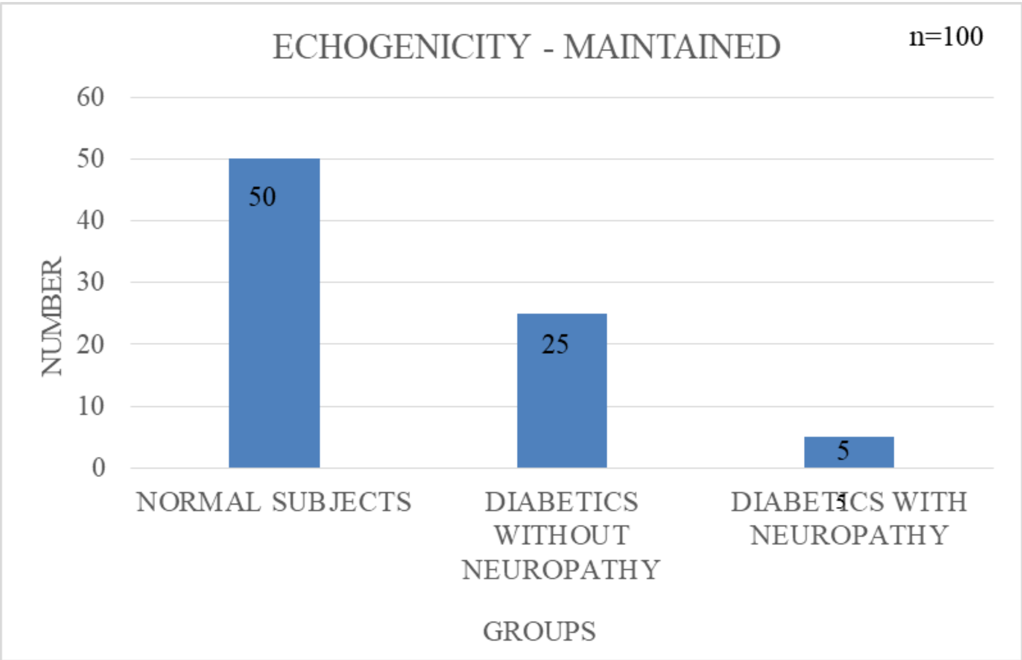

Figure 9: Graphical representation showing maintained echogenicity of median nerve among different groups: normal, diabetics without neuropathy and diabetics with neuropathy.

6) HbA1c

Table 8:

HbA1c	Mean ± St deviation
Total	5.804 ± 1.128
Female	5.859 ± 1.101
Male	5.751 ± 1.1609

Table 8 shows distribution of mean value of HbA1c in different groups: total, female and male.

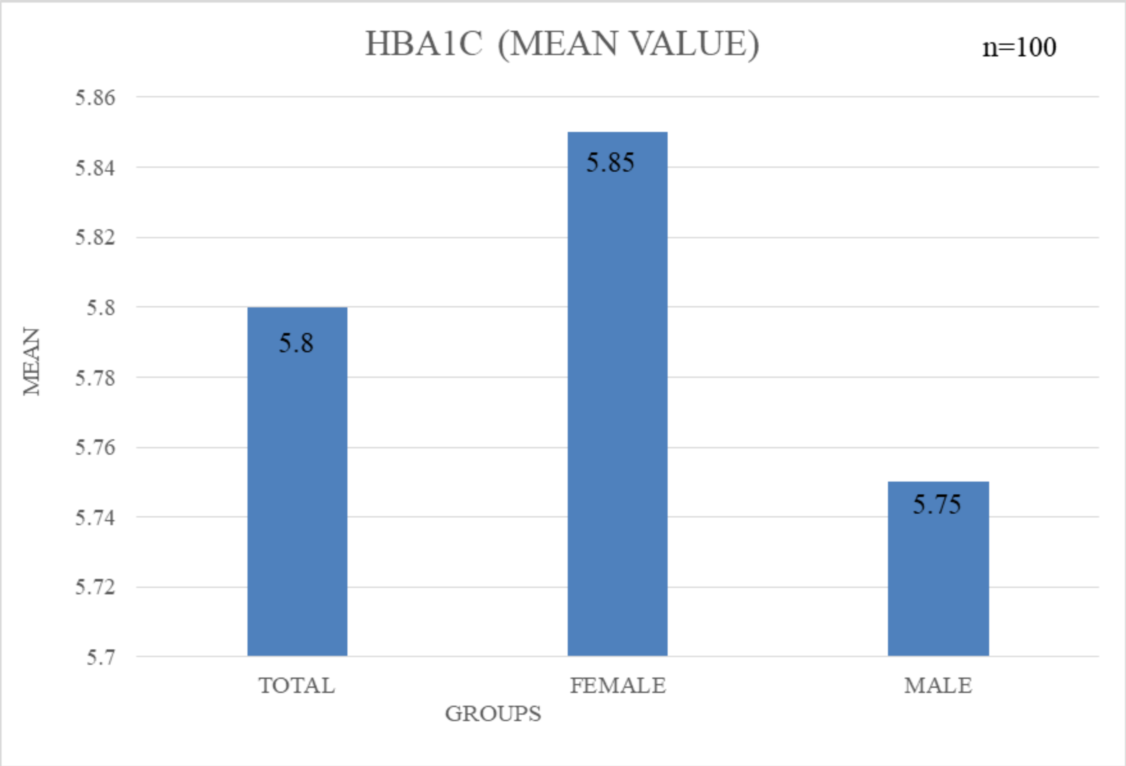

Figure 10: Graphical representation showing mean value of HBA1c in total, female and male groups. The overall mean HbA1c was **5.804** (Standard Deviation: 1.128). Gender-based analysis showed: **Females:** 5.859 ± 1.101, **Males:** 5.751 ± 1.1609

7) FBS

Table 9:

FBS (mg/dL)	Mean ± St deviation
Total	117.58 ± 33.806
Female	118.388 ± 35.591
Male	116.804 ± 32.335

Table 9 shows distribution of mean value of FBS in different groups: total, female and male.

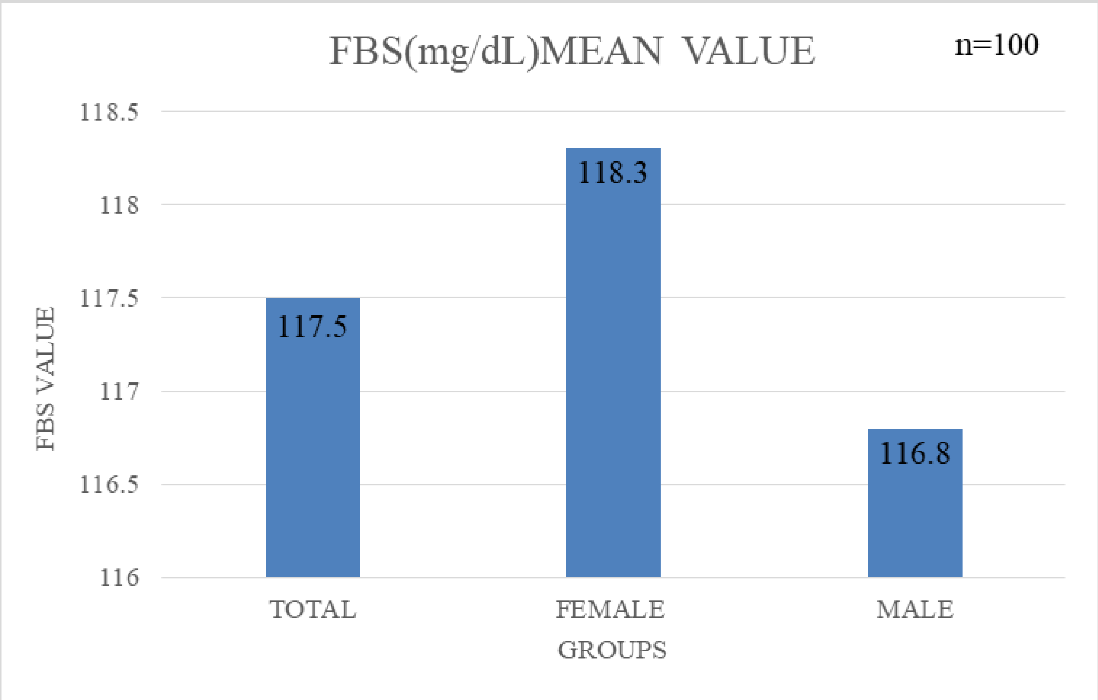

Figure 11: Graphical representation showing mean values of FBS in total, female and male groups. The mean FBS level across all subjects was **117.58 mg/dL** with: **Females:** 118.388 ± 35.591 mg/dL ; **Males:** 116.804 ± 32.335 mg/ dL.

8) Duration of Diabetes Mellitus (years)

- The average duration of diabetes was **4.25 years** (Standard Deviation: 4.635), with males (4.333 years) showing slightly longer disease duration than females (4.163 years).
- The duration of diabetes increased with age, with the longest mean duration (**10.118 years**) observed in the **56-65 years** group.

Table 10:

Duration of DM (years)	Mean ± St deviation
Total	4.25 ± 4.635
Female	4.163 ± 4.351
Male	4.333 ± 4.934

Table 10: Shows mean value of duration of Diabetes mellitus in different groups: total, female and male.

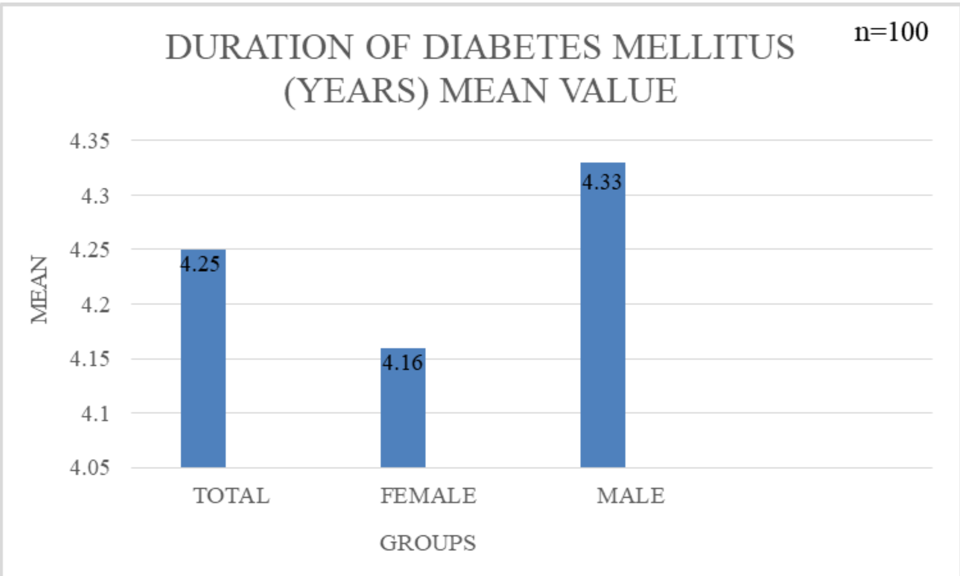

Figure 12: Graphical representation showing mean values of duration of Diabetes mellitus in total, female and male groups.

9) Diabetes Mellitus status

DM	Female	Male	Total
NA	24 (24%)	26 (26%)	50 (50%)
Controlled	13 (13%)	14 (14%)	27 (27%)
Uncontrolled	12 (12%)	11 (11%)	23 (23%)
Total	25	25	50

Table 11 - Controlled diabetes: 27 individuals (13% females, 14% males)

- Uncontrolled diabetes: 23 individuals (12% females, 11% males)

- Non-diabetic subjects: 50 individuals

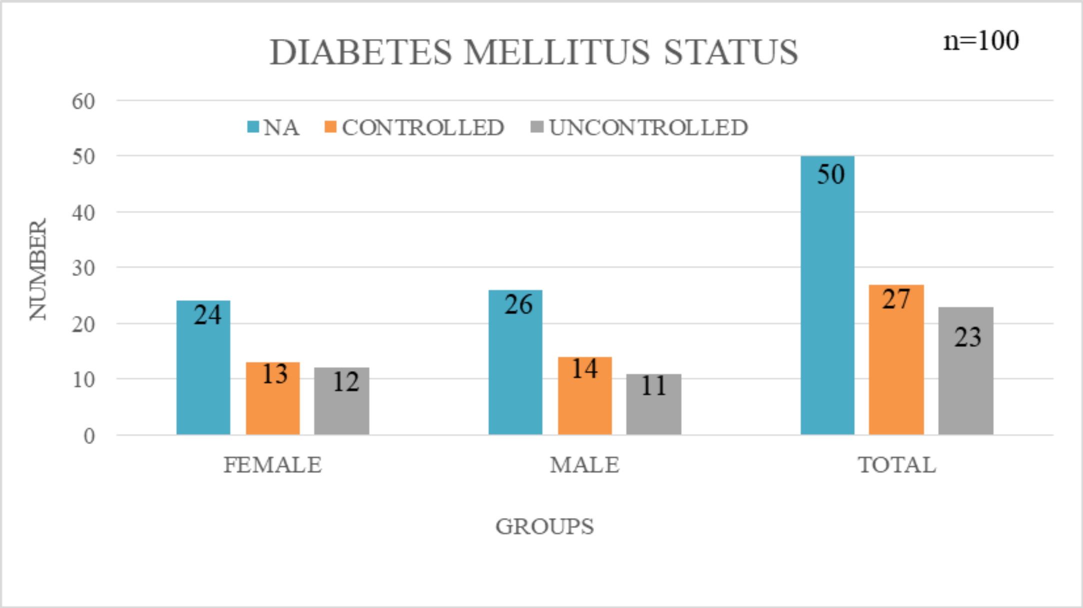

Figure 13: Graphical representation showing status of Diabetes mellitus: controlled, uncontrolled in female, male groups and in general.

10) Correlations

Table 12.1:

Characteristics	No. of subjects	Mean ± SD	Range
Age (years)	100	12.03 ± 3.67	42-62
FBS (mg/dL)	100	117.58 ± 33.86	88-220
HbA1c	100	5.804 ± 1.128	4.5- 8.5
Duration of diabetes (years)	100	4.25 ± 4.63	0-13

Table 12.2:

Age	FBS (mean)	HbA1c (mean)	Duration of DM (mean)
>45	103.2	5.13	8 months
46-55	115.832	5.735	3.5 years
56-65	137.176	6.676	10.1 years

Table 12.3:

Variables	N	Pearson's correlation	p-value
Age and FBS	100	0.399	<0.0001***
Age and HbA1c	100	0.547	<0.0001***
Age and Duration of DM	100	0.765	<0.0001***
HbA1c and Duration of DM	100	0.889	<0.0001***
FBs and HbA1c	100	0.907	<0.0001***

A Pearson's correlation analysis was performed to assess the relationships among various parameters:

- **Age vs. FBS:** r = 0.399, **p < 0.0001** (significant positive correlation)
- **Age vs. HbA1c:** r = 0.547, **p < 0.0001** (strong positive correlation)
- **Age vs. Duration of Diabetes:** r = 0.765, **p < 0.0001** (very strong correlation)
- **HbA1c vs. Duration of Diabetes:** r = 0.889, **p < 0.0001** (very strong correlation)
- **FBS vs. HbA1c:** r = 0.907, **p < 0.0001** (highly significant correlation)

The study highlights significant structural changes in the median nerve among diabetic patients, as reflected in sonoelastographic parameters such as AP diameter, cross-sectional area, and strain ratio. Additionally, the strong correlations between age, HbA1c, FBS, and diabetes duration suggest a progressive impact of diabetes on nerve function. The findings underscore the potential of sonoelastography as a non-invasive diagnostic tool for early detection of diabetic neuropathy.

CASE 1

Figure 14.1: Ultrasound grey scale image, longitudinal section showing AP thickness of median nerve - 1.3 mm in a normal subject

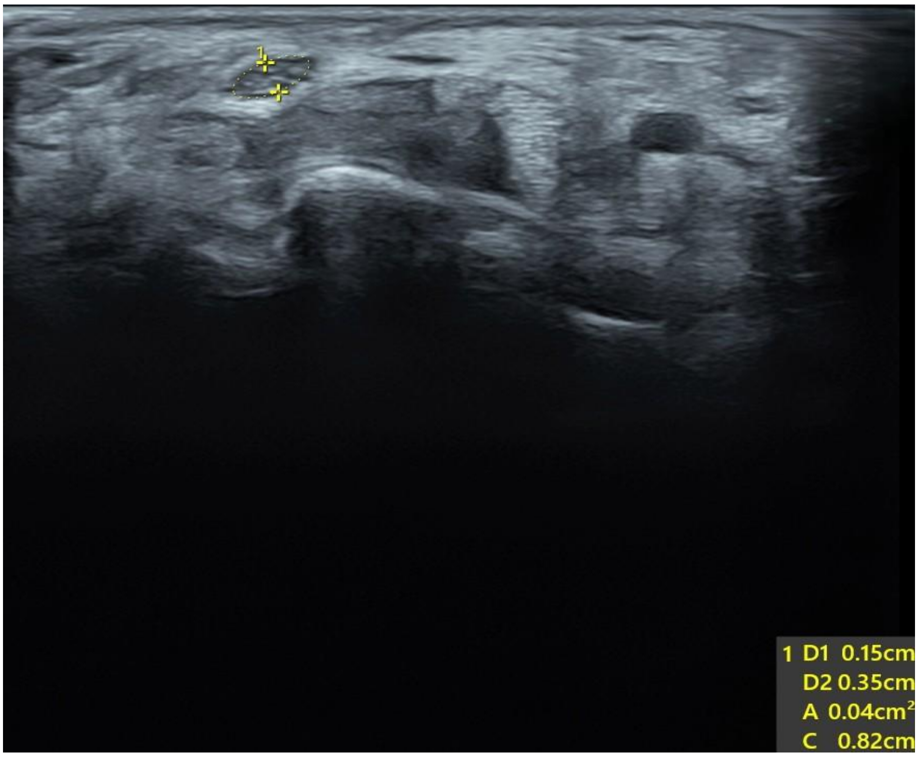

Figure 14.2 : Ultrasound grey scale image , axial section showing C.S.A of median nerve - 0.04 cm^2 in a normal subject

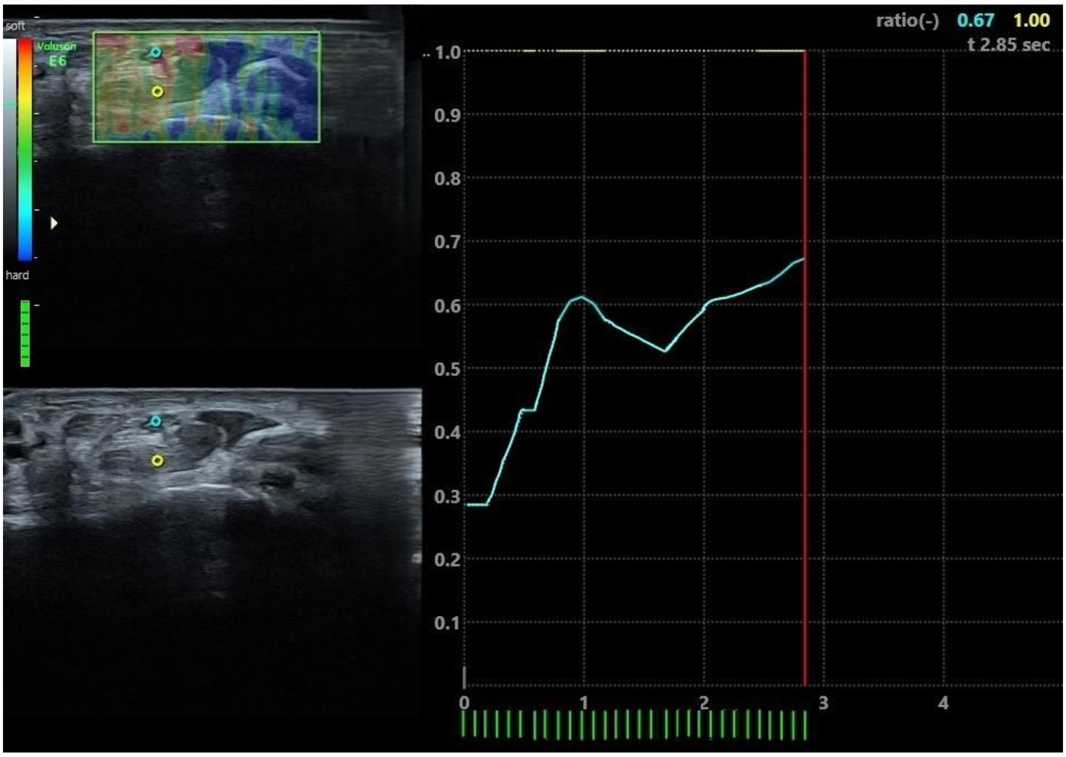

Figure 14.3 : Strain elastography image, axial section showing strain ratio of median nerve - 0.6 in a normal subject.

CASE-2

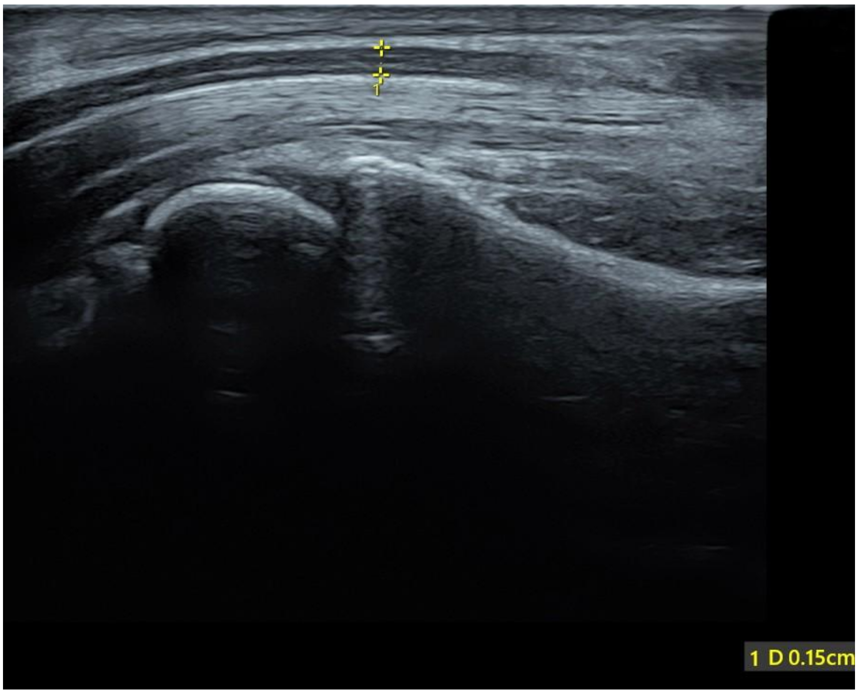

Figure 15.1 : Ultrasound grey scale image, longitudinal section showing AP thickness of median nerve - 1.5 mm in a diabetic patient without neuropathy

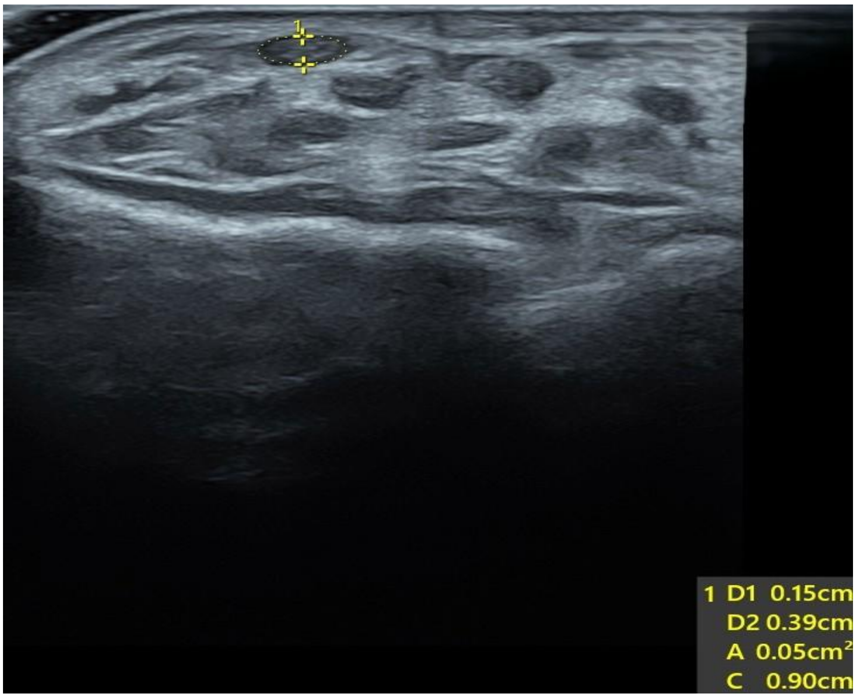

Figure 15.2 : Ultrasound grey scale image, axial section showing C.S.A of median nerve - 0.05 cm^2 in a diabetic patient without neuropathy.

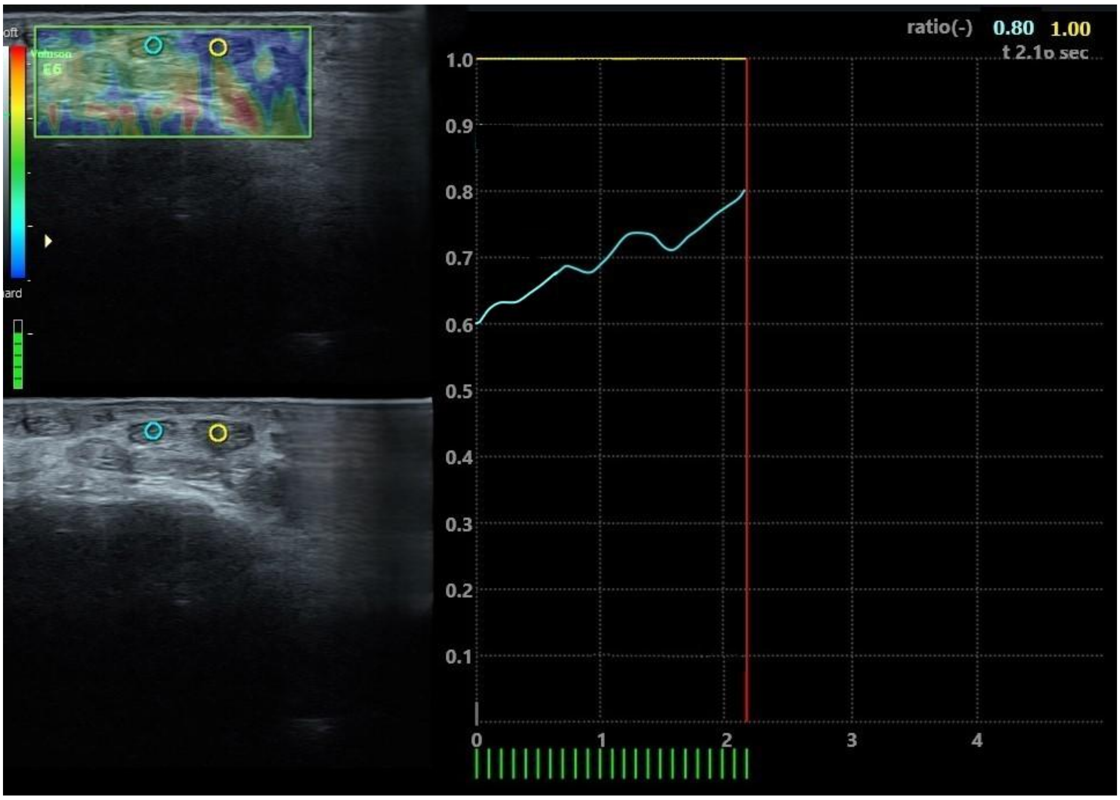

Figure 15.3 : Strain elastography image, axial section showing strain ratio of median nerve - 0.8 in a diabetic patient without neuropathy.

CASE 3

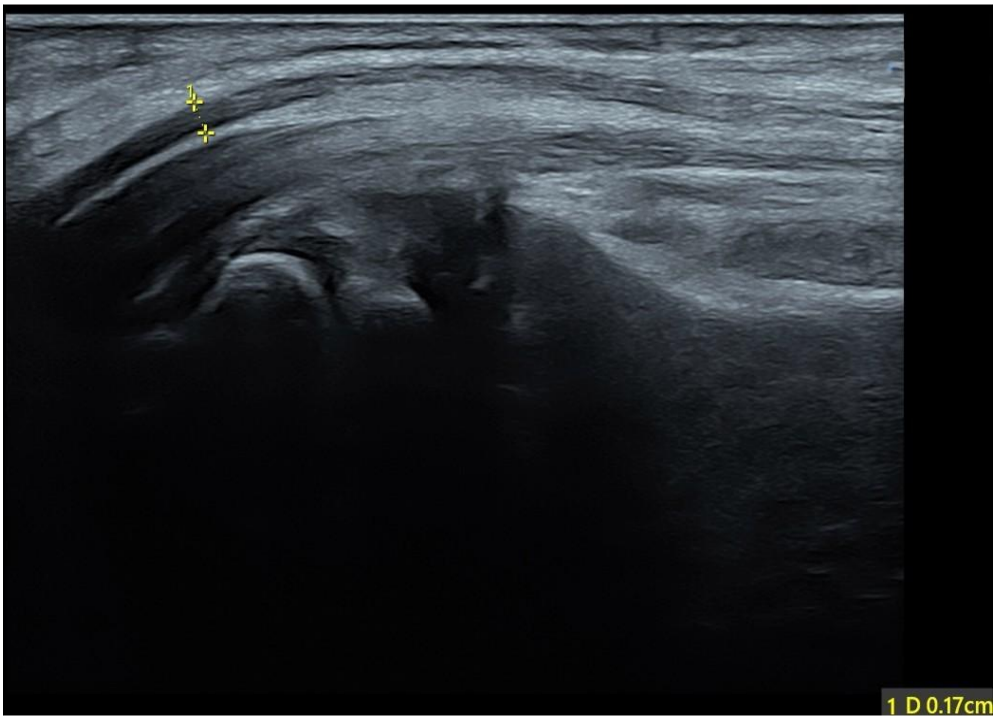

Figure 16.1 : Ultrasound grey scale image, longitudinal section showing AP thickness of median nerve - 1.7 mm in a diabetic patient without neuropathy

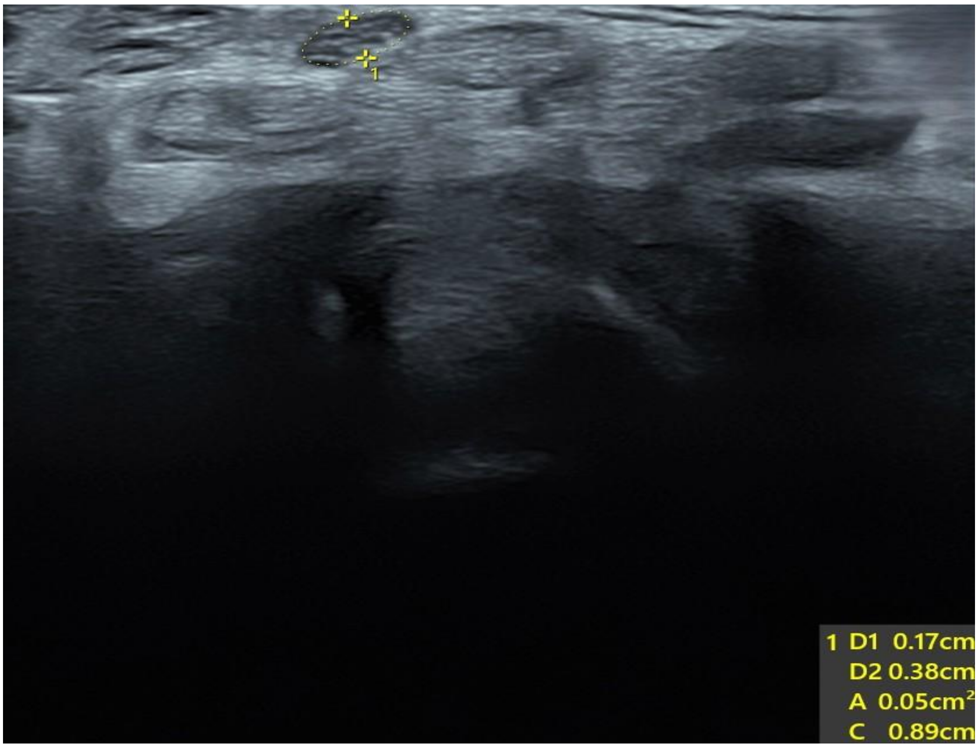

Figure 16.2 : Ultrasound grey scale image, axial section showing C.S.A of median nerve - 0.05 cm^2 in a diabetic patient without neuropathy.

Figure 16.3 : Strain elastography image, axial section showing strain ratio of median nerve - 0.79 in a diabetic patient without neuropathy.

CASE 4

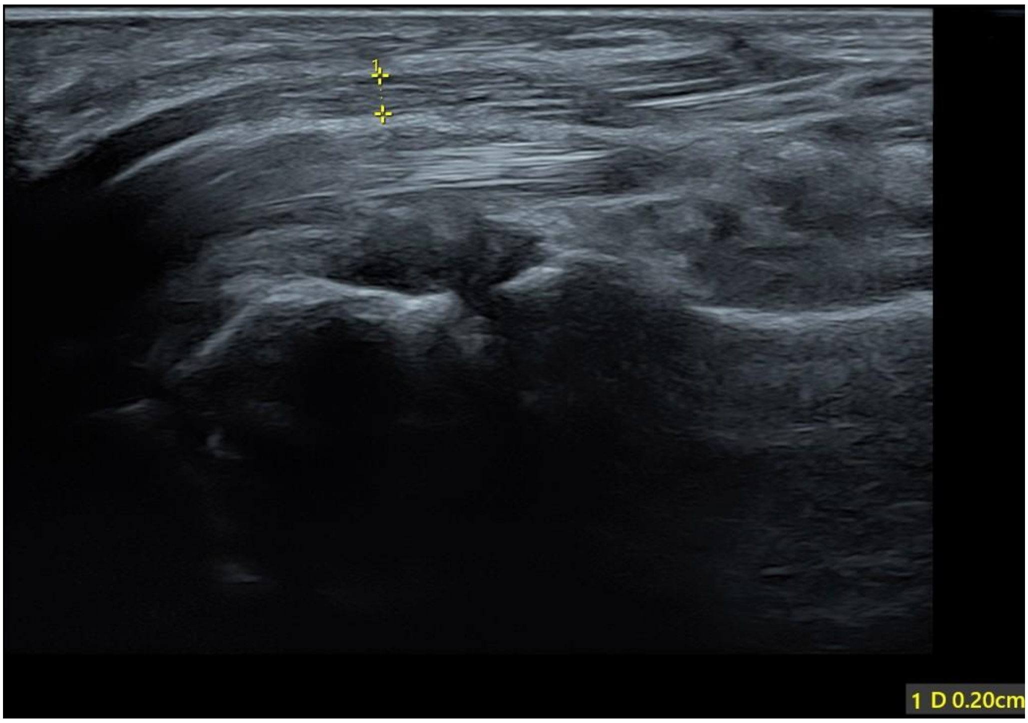

Figure 17.1 : Ultrasound grey scale image, longitudinal section showing AP thickness of median nerve - 2.0 mm in a diabetic patient with neuropathy

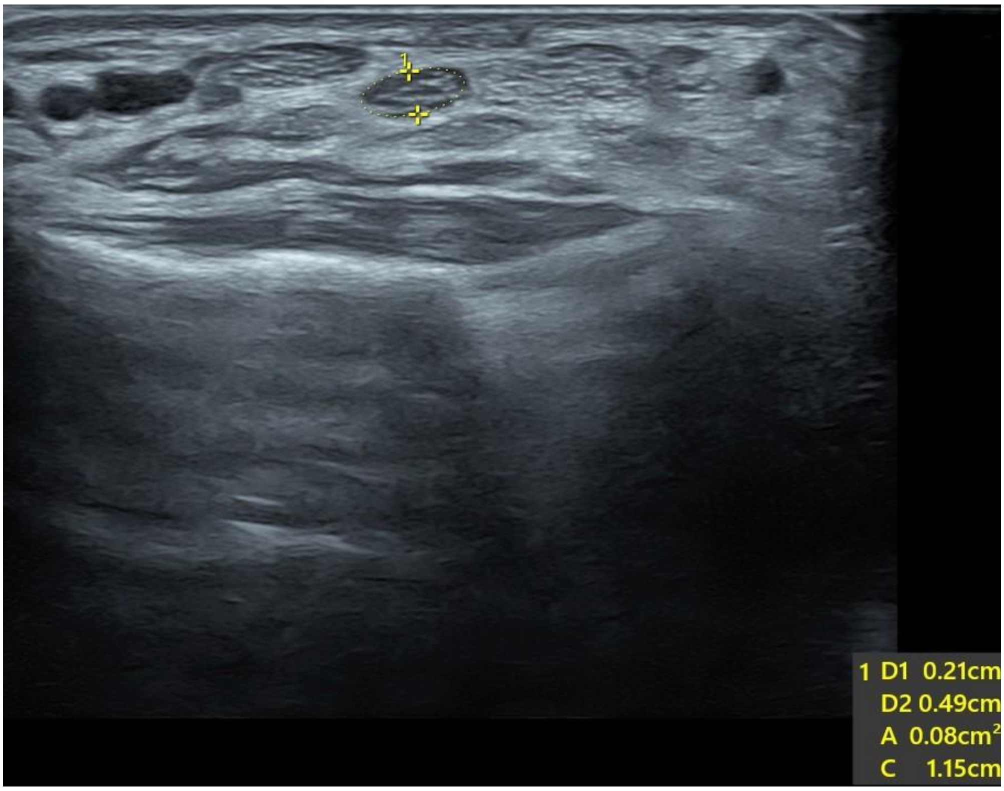

Figure 17.2 : Ultrasound grey scale image, axial section showing C.S.A of median nerve - 0.08 cm^2 in a diabetic patient with neuropathy.

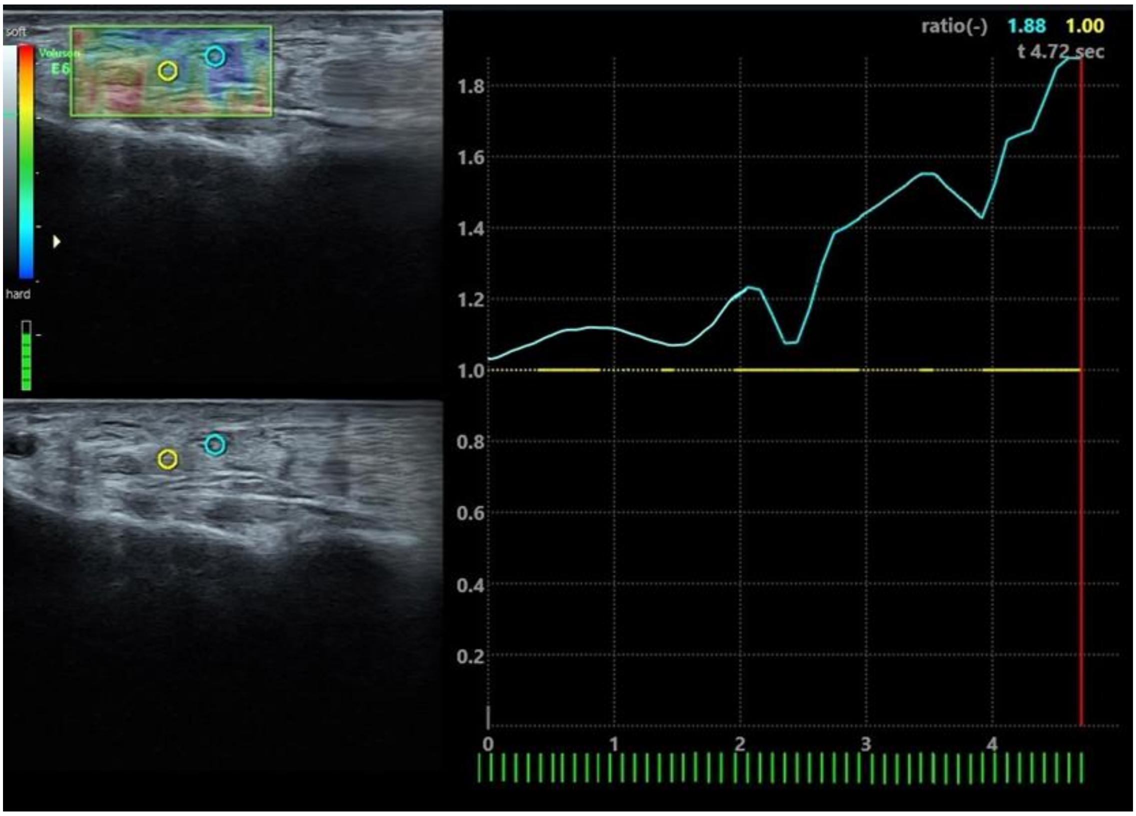

Figure 17.3 : Strain elastography image, axial section showing strain ratio of median nerve - 1.8 in a diabetic patient with neuropathy**.**

CASE 5

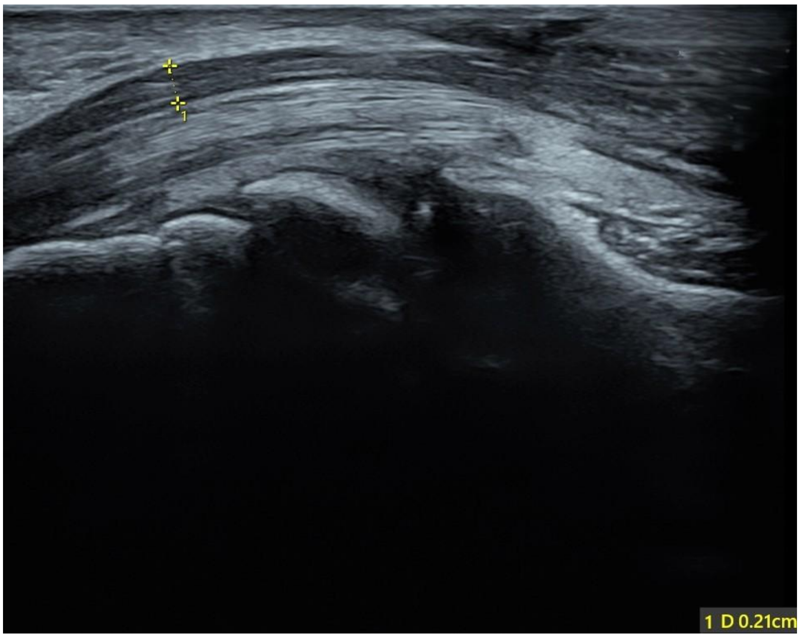

Figure 18.1 : Ultrasound grey scale image, longitudinal section showing AP thickness of median nerve - 2.1 mm in a diabetic patient with neuropathy

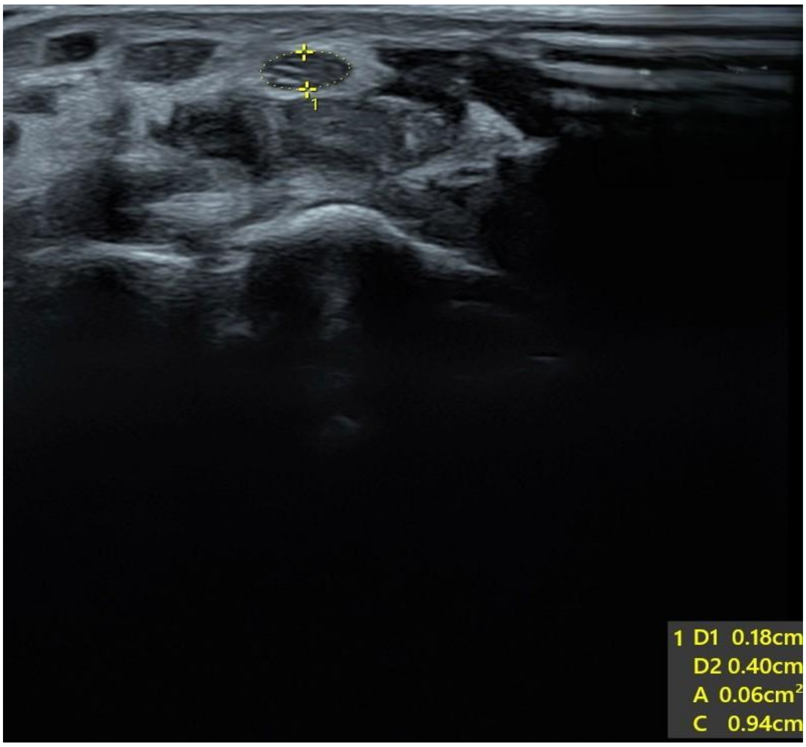

Figure 18.2 : Ultrasound grey scale image, axial section showing C.S.A of median nerve - 0.06 cm^2 in a diabetic patient with neuropathy

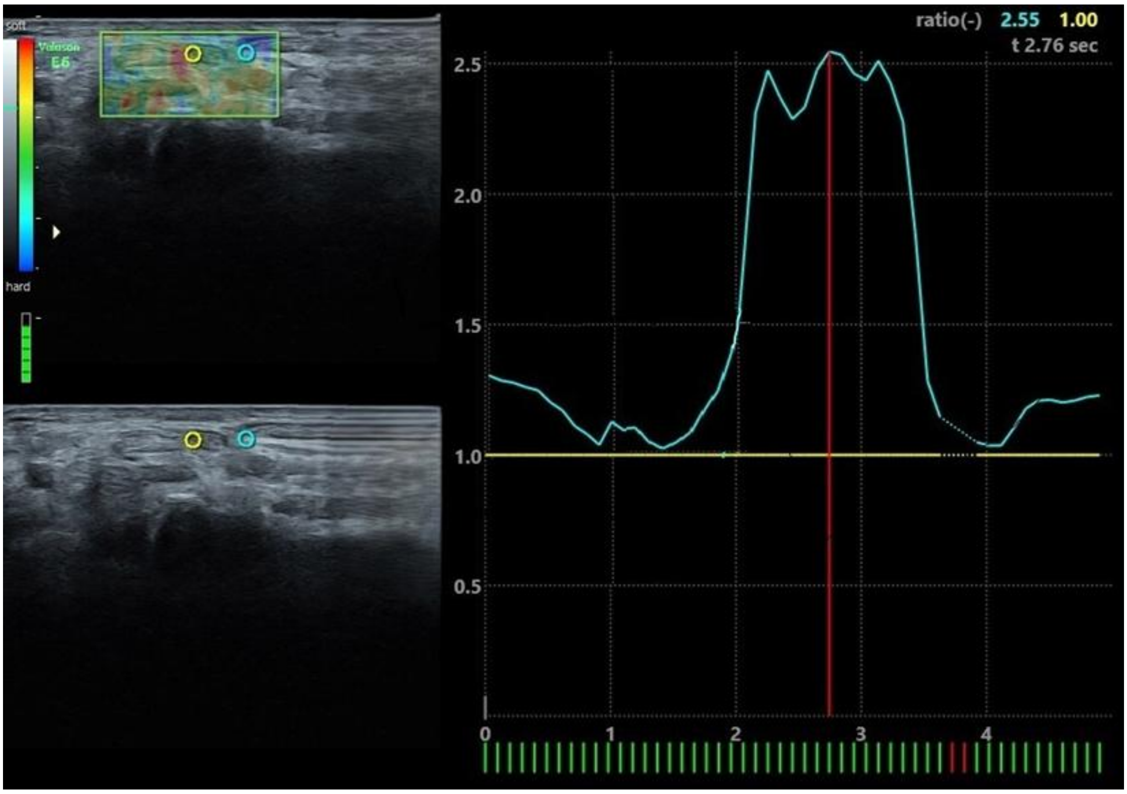

Figure 18.3 : Strain elastography image, axial section showing strain ratio of median nerve - 2.5 in a diabetic patient with neuropathy.

DISCUSSION

DISCUSSION

Our study examined 100 volunteers categorized into three unique age groups, with the majority (68 individuals) aged between 46 and 55 years, yielding a mean age of 49.71 years. The youngest cohort (>45 years) exhibited a mean age of 44.07 years, whilst the oldest cohort (56–65 years) demonstrated a mean age of 58.47 years. The gender distribution was almost equal, consisting of 51 males and 49 females, so assuring balanced representation for analysis. Participants were classified into three groups: 50 healthy individuals, 25 diabetic patients without neuropathy, and 25 diabetic patients with neuropathy, with both diabetic cohorts exhibiting almost equal male and female representation.

The sonoelastographic evaluation of the median nerve exhibited notable structural features. The average anteroposterior (AP) thickness of the median nerve was 1.547 mm (SD = 0.3289), the mean cross-sectional area was 0.067 cm^2 (SD = 0.0241), and the strain ratio averaged 1.0425 (SD = 0.4523).

The echogenicity of median nerve was maintained in normal subjects and diabetics without neuropathy, whereas it was altered in most cases of diabetics with neuropathy. The findings indicate small but quantifiable variations in nerve structure that may be associated with diabetes state, suggesting that sonoelastography might serve as a non-invasive diagnostic tool for the early identification of diabetic neuropathy.

Biochemical parameters were evaluated, revealing an overall mean HbA1c level of 5.804 (SD = 1.128), with females exhibiting a slightly elevated level of 5.859 compared to males at 5.751. The average fasting blood sugar (FBS) levels in the study cohort were 117.58 mg/dL (SD = 33.806), with females exhibiting a slightly higher mean than men. The mean duration of diabetes was 4.25 years (SD = 4.635), with a significant trend of prolonged duration in older individuals; the 56–65 years age group had a mean length of 10.118 years, and men displayed a somewhat longer disease duration than females.

A Pearson correlation study revealed substantial positive correlations across many parameters. Age exhibited a modest positive link with FBS ($r = 0.399$) and a greater positive correlation with HbA1c ($r = 0.547$), with a very significant correlation with the duration of diabetes ($r = 0.765$). The link between HbA1c and the duration of diabetes was robust ($r = 0.889$), and there was a highly significant association between FBS and HbA1c ($r = 0.907$), with all p-values below 0.0001. These strong connections underscore the gradual effect of diabetes on metabolic regulation and neuronal function.

Results of our study enhance the existing data that sonoelastography can identify minor structural alterations in the median nerve linked to diabetes, particularly with diabetic neuropathy. It also revealed that diabetic individuals, especially those with neuropathy, had modifications in nerve characteristics, including a mean AP thickness of 1.547 mm, a cross-sectional area of 0.067 cm^2, and a strain ratio of 1.0425, indicating heightened nerve stiffness. These findings align with other studies, like the work by Zheng et al. published in the British Journal of Radiology in 2025, which also revealed that sonoelastography is a sensitive instrument for assessing median nerve alterations in diabetic patients. [59]

Our study elucidates notable changes in the median nerve of individuals with diabetes when contrasted with healthy subjects, as evaluated through sonoelastographic metrics. It revealed that diabetic patients, regardless of the presence of neuropathy, exhibited notable variations in median nerve parameters, including the anteroposterior diameter, cross-sectional area, and strain ratio. Furthermore, significant correlations were identified among age, fasting blood sugar levels, HbA1c, and the duration of diabetes.

Previous research has similarly indicated that there is a correlation between diabetic peripheral neuropathy (DPN) and heightened nerve stiffness. Ying He and colleagues in the year 2019 conducted an investigation on median and tibial nerves in patients suffering from diabetic peripheral neuropathy (DPN). Their findings revealed that nerve stiffness, as assessed by shear wave elastography, was markedly elevated in diabetic patients when compared to healthy controls. Furthermore, the results of receiver operating characteristic (ROC) analyses indicated a strong diagnostic performance for the identification of DPN. In contrast to previous studies where the region of interest (ROI) and elastography box placement were variable, our study employed a fixed ROI and elastography box for all measurements. This methodological standardization minimizes user bias and enhances the reproducibility and reliability of our results, thereby representing a methodological improvement and making our study more robust and superior in design. [53]

Mine Aslan and colleagues studied peripheral nerves in adolescents with type 1 diabetes who did not exhibit overt signs of neuropathy. It is noteworthy that, even in cases where diabetic peripheral neuropathy (DPN) is not clinically evident, the research revealed that both the median and posterior tibial nerves demonstrated a reduction in cross-sectional area, accompanied by an increase in stiffness when compared to their counterparts in healthy individuals. While the relationship with disease duration was found to be only modest, these subclinical alterations further highlight the significant potential of elastographic measurements in identifying early nerve involvement prior to the manifestation of clinical symptoms. [50]

Jiang et al. in the year 2017 has demonstrated that two-dimensional shear wave elastography serves a dual purpose: it effectively distinguishes diabetic patients from control subjects while also establishing a correlation with the severity of neuropathy. Similarly, **Chen et al.** documented that the integration of conventional ultrasound with elastography enhanced the diagnostic precision for diabetic polyneuropathy. This finding implies that heightened nerve stiffness could serve as a dependable surrogate marker for nerve dysfunction associated with diabetes. [54] Our study findings align with their findings. Numerous studies consistently indicate that individuals with diabetes exhibit heightened nerve stiffness. This phenomenon is likely attributable to the metabolic and microvascular alterations that occur as a consequence of prolonged hyperglycemia. The structural alterations identified through sonoelastography exhibit a significant correlation with glycemic indices and the duration of the disease. Furthermore, these findings suggest the potential of sonoelastography as a noninvasive instrument for the early detection and ongoing monitoring of diabetic neuropathy. Subsequent investigations involving more extensive cohorts and longitudinal methodologies will be essential to substantiate these findings further. Additionally, such research will facilitate the incorporation of sonoelastography into standard clinical practice for the effective management of patients with diabetes.

Findings of our research was the establishment of significant correlations between clinical parameters and the morphology of nerves. Notable positive correlations were identified among age, HbA1c, fasting blood sugar, and the duration of diabetes. This suggests that extended exposure to hyperglycemia, along with its associated metabolic ramifications, may result in gradual structural alterations in nerve tissue. A similar study conducted by **Zheng et al**. in 2025 elucidated that the stiffness of the median nerve, as evidenced by elevated strain ratio, correlated with inadequate glycemic control and an extended duration of the disease. In a comparative analysis, both our study and the BJR study highlight the significant utility of sonoelastography as a non-invasive imaging technique. This modality not only quantifies the physical properties of the nerve but also establishes correlations between these properties and clinically relevant biochemical markers, including HbA1c and FBS. It also shows compelling evidence suggesting that increased strain ratio may act as an early marker for neuropathic alterations. Our findings further elaborate on this premise by demonstrating that, even within a rigorously defined cohort matched for age and gender, there exists a distinct gradation of nerve stiffness that correlates with both metabolic regulation and the duration of the disease. Both studies underscore the potential of sonoelastography as a supplementary diagnostic instrument in the assessment of diabetic neuropathy, supporting the notion of its capability to identify early alterations in nerve function linked to diabetes. This approach offers an objective and reproducible metric that demonstrates a strong correlation with metabolic regulation and the duration of the disease.

In our study, we observed that healthy individuals and most diabetic patients without neuropathy had a strain ratio below 1.0. In contrast, diabetic patients with clinically evident neuropathy typically showed a strain ratio above 1.0. This indicates that a strain ratio exceeding 1.0 may be indicative of early neuropathic alterations. Among diabetic patients without neuropathy, an increasing trend in anteroposterior thickness and cross-sectional area of the median nerve was noted, along with a borderline strain ratio. Therefore, routine monitoring in such patients could aid in the early detection and prevention of neuropathy before clinical signs manifest, allowing timely intervention. However, current literature on the use of strain elastography for assessing median nerve changes remains limited.

In conclusion, the research highlights that diabetes, especially in its uncontrolled state, is linked to considerable changes in the morphology of the median nerve, as evaluated through sonoelastography. The relationships that have been observed among age, glycemic control markers such as HbA1c and fasting blood sugar (FBS), and the duration of diabetes indicate that these variables collectively play a significant role in the structural alterations occurring within the nerve. Comparable stratifications have been documented in various studies, which have indicated that modifications in nerve stiffness and cross-sectional areas may occur prior to the manifestation of clinical symptoms, thus providing an opportunity for timely therapeutic intervention.

CONCLUSION

CONCLUSION

Our study highlights the utility of ultrasound-based sonoelastography in detecting early structural changes in the median nerve of diabetic patients. Significant alterations—including increased anteroposterior (AP) thickness, enlarged cross-sectional area, and modified strain ratio—were particularly evident in diabetics with neuropathy, confirming the detrimental impact of diabetes on peripheral nerve structure.

A strong positive correlation was observed between disease duration and nerve changes, with the maximum mean disease duration (10.1 years) seen in older adults (56–65 years). This underlines the progressive nature of diabetic neuropathy and the cumulative effect of chronic hyperglycemia over time. Furthermore, biochemical parameters such as HbA1c and fasting blood sugar (FBS) showed strong correlations with age and duration of diabetes, notably with HbA1c demonstrating an exceptionally high correlation with disease duration, emphasizing the central role of glycemic control of nerves.

Advancing age was consistently associated with worsening glycemic control and longer diabetes duration, indicating that older diabetic adults are at significantly higher risk for neuropathic complications. This further supports the need for proactive screening in aging diabetic populations.

In our study, based on the findings, we have observed that, in normal subjects and most of diabetic patients without neuropathy, strain ratio less than 1.0 was obtained. In majority of diabetic patients with neuropathy, strain ratio more than 1.0 was obtained. So based on this, we can consider, strain ratio more than 1.0 as abnormal.

Our research provides compelling evidence that sonoelastography can serve as an adjunct to traditional assessments, facilitating early identification and management of diabetic neuropathy. The strong associations among age, glycemic indices, disease duration, and nerve morphology underscore the need for multidisciplinary strategies in diabetic care.

SUMMARY

SUMMARY

Our study evaluated median nerve with ultrasound & sonoelastography of 100 participants who were categorized into three groups: healthy individuals, diabetics without neuropathy, and diabetics with neuropathy. The predominant grouping comprised individuals aged 46–55, with an equitable gender distribution. The duration of diabetes was much longer in older subjects, highlighting the progressive nature of diabetic nerve injury in combination with the observed relationships. These findings underscore the promise of sonoelastography as both a diagnostic instrument for early identification and a method to assess the effects of glycemic management on nerve function in diabetic patients.

This cross-sectional study investigated the relationship between diabetic status, demographic variables, and sonoelastographic parameters of the median nerve in a cohort of 100 participants aged between 42 and 65 years. Our study aimed to evaluate how diabetes, particularly diabetic neuropathy, affects nerve structure and function and correlates with glycemic parameters, age, and duration of disease.

The age distribution showed that the majority of participants (68%) were in the 46–55 years group, with a mean age of 49.71 years. Gender distribution was nearly equal, with 51 males and 49 females. The participants were stratified into three clinical categories: 50% were non-diabetics (normal), 25% were diabetics with neuropathy, and 25% were diabetics without neuropathy.

Sonoelastographic evaluation of the median nerve provided key structural metrics. The average anteroposterior (AP) thickness was 1.54 mm, the cross-sectional area was 0.067 cm^2, and the mean strain ratio was 1.04. These values indicated measurable differences in nerve architecture, particularly in patients with diabetic neuropathy. The subgroup with neuropathy demonstrated altered echogenicity, reflecting the impact of chronic hyperglycemia on nerve integrity.

Biochemical parameters provided insight into the participants' metabolic status. The mean HbA1c was 5.804, with slightly higher values in females than males. Similarly, fasting blood sugar (FBS) values were comparable between genders, averaging around 117.5 mg/dL. These values, while modestly elevated, demonstrated good control in most diabetic subjects, but a significant subset (23%) had uncontrolled diabetes.

The duration of diabetes showed wide variability, with an overall mean of 4.25 years. The longest duration (10.1 years) was observed in the 56–65 age group, supporting the hypothesis of progressive nerve degeneration with advancing age and prolonged diabetes. This is reinforced by echogenicity findings, which showed that most diabetic neuropathy cases had altered nerve signals.

Following analysis demonstrated strong correlations:

- Age was significantly and positively correlated with FBS, HbA1c and duration of diabetes, indicating age-related worsening of diabetic control and disease progression.
- HbA1c had a very strong correlation with duration of diabetes, affirming that chronic hyperglycemia worsens with time.
- The strongest relationship was between FBS and HbA1c, underscoring the mutual reinforcement of these two glycemic markers.

Above analysis demonstrates a consistent pattern: increased age and longer diabetes duration are associated with worsening glycemic control and measurable structural changes in the median nerve. The observed echogenicity differences and sonoelastographic values support the use of imaging as a supplementary diagnostic tool in early detection of neuropathy. This is particularly relevant in borderline or asymptomatic diabetic patients, who might not yet show clinical signs but already exhibit subtle nerve changes on imaging.

In our study, it was observed that the strain ratio was less than 1.0 in normal subjects as well as in the majority of diabetic patients without neuropathy. In contrast, diabetic patients with neuropathy predominantly demonstrated a strain ratio greater than 1.0. Based on these findings, a strain ratio exceeding 1.0 may be considered abnormal and indicative of possible neuropathic changes. This could aid in the early, subclinical detection of neuropathy in diabetic patients who have not yet developed clinical symptoms, thereby facilitating timely intervention and improved management before the onset of overt neuropathy. Incorporating sonoelastography into routine evaluation may be essential for the timely detection and potential prevention of neuropathic progression in this population.

LIMITATIONS & RECOMMENDATIONS

LIMITATIONS AND RECOMMENDATIONS

The study has many limitations that must be acknowledged while analyzing its results.

- There is a need to develop more standardized sonoelastographic criteria that can facilitate the early diagnosis and ongoing monitoring of diabetic neuropathy.
- Incorporating nerve conduction studies alongside median nerve sonoelastography may improve diagnostic accuracy and overall reliability of assessments. If strain elastography yields results that would have similar accuracy as nerve conduction studies, nerve conduction study may be later on replaced by sonoelatography.
- The restricted demographic diversity indicates that the results may not be widely applicable to more heterogeneous groups.
- In this study, the strain ratio was calculated using the median nerve and the adjacent tendon. Future research could explore the use of alternative reference tissues, such as perineural fat or a combination of fat and tendon, to determine the most suitable reference for strain ratio measurements. This may help establish whether tendon or fat provides a more reliable comparison for evaluating median nerve stiffness.
- Given the high prevalence of diabetes mellitus in India, there is a pressing need for further region-specific studies to better understand the utility of sonoelastography in this context.
- Ultrasound and strain elastography are operator dependent.
- The strain ratio may vary depending on the amount of manual pressure applied by the ultrasound probe during the examination.
- Further studies involving larger cohorts, particularly within the Indian population, are necessary to validate and strengthen the findings of our research.

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ANNEXURE

PROFORMA

"ROLE OF SONOELASTOGRAPHY IN EVALUATION OF MEDIAN NERVE CHANGES IN DIABETIC PATIENTS AND COMPARISON TO NORMAL SUBJECTS - A CASE CONTROL STUDY."

DEMOGRAPHIC DETAILS:

Name:

Hospital no:

USG no:

Gender:

Age:

Address:

CLINICAL HISTORY:

DIABETIC: YES/NO

IF YES, DURATION OF DIABETES:

TREATMENT HISTORY: Insulin/Oral hypoglycemic agents

PERSONAL HISTORY:

FAMILY HISTORY:

SYSTEMIC EXAMINATION:

LOCAL EXAMINATION:

LAB INVESTIGATION:

1. FBS:

2. HbA1c:

CLINICAL DIAGNOSIS:

ULTRASOUND FINDINGS:

AP thickness of median nerve

Cross sectional area of median nerve:

Echotexture of median nerve:

STRAIN ELASTOGRAPHY FINDINGS:

MEDIAN NERVE

PARAMETER	**CASES/CONTROLS DOMINANT HAND**
STRAIN RATIO	

INFORMED CONSENT

Study title: "ROLE OF SONOELASTOGRAPHY IN EVALUATION OF MEDIAN NERVE CHANGES IN DIABETIC PATIENTS AND COMPARISION TO NORMAL SUBJECTS – A CASE CONTROL STUDY."

Chief researcher/ PG guide's name: Dr. ANIL KUMAR SAKALECHA

Principal investigator: Dr. SOUMYA CHINCHOLIKAR

Name of the subject:

Age :

Gender :

a. I have been informed in my own language that this study involves ultrasound and strain elastography as a part of the procedure. I have been explained thoroughly and understand the procedure.
b. I understand that the medical information produced by this study will become part of institutional record and will be kept confidential by the said institute.
c. I understand that my participation is voluntary and may refuse to participate or may withdraw my consent and discontinue participation at any time without prejudice to my present or future care at this institution.
d. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s).
e. I confirm that Dr. ANIL KUMAR SAKALECHA / Dr. SOUMYA CHINCHOLIKAR has explained to me the purpose of research and the study procedure that I will undergo and the possible risks and discomforts that I may experience, in my own language. I hereby agree to give valid consent to participate as a subject in this research project.

Participant's signature/thumb impression

Signature of the witness: Date:

1)

2)

I have explained to ___________________________ (subject) the purpose of the research, the possible risk and benefits to the best of my ability.

Chief Researcher/ Guide signature Date:

ಸಮ್ಮತಿ ಪತ್ರ:

ಈ ಕೆಳಗೆ ಸಹಿ ಮಾಡಿರುವ ------------------- ಆದ ನಾನು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವ ಸಲುವಾಗಿ ವೈದ್ಯಕೀಯ ಪ್ರಯೋಗ ಪರೀಕ್ಷೆಗೆ ಒಳಪಡಲು ನನ್ನ ವೈಯ್ಯಕ್ತಿಕ ವಿವರಗಳನ್ನು ನೀಡಲು ಸಮ್ಮತಿಸಿರುತ್ತೇನೆ.

ಈ ಅಧ್ಯಯನದ ಉದ್ದೇಶ, ಅಧ್ಯಯನದ ಸಂದರ್ಭದಲ್ಲಿನೀಡುವ ಮತ್ತು ಸಂಗ್ರಹಿಸುವ ಮಾಹಿತಿಯಗೋಪ್ಯತೆಯ ಬಗ್ಗೆ ನನಗೆ ನನ್ನ ಸ್ಥಳೀಯ ಭಾಷೆಯಲ್ಲಿ ಓದಿ ಹೇಳಲಾಗಿದೆ/ವಿವರಿಸಲಾಗಿದೆ ಮತ್ತು ನಾನು ಇದನ್ನು ಅರ್ಥ ಮಾಡಿಕೊಂಡಿರುತೇನೆ. ಈ ಅಧ್ಯಯನದ ವಿವಿಧ ಅಂಶಗಳ ಬಗ್ಗೆ ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳುವ ಅವಕಾಶವನ್ನು ನನಗೆ ನೀಡಲಾಗಿದೆ ಮತ್ತು ನನ್ನ ಪ್ರಶ್ನೆಗಳಿಗೆ ತೃಪ್ತಿಕರವಾದ ಉತ್ತರಗಳು ದೊರೆತಿರುತವೆ. ಈ ಅಧ್ಯಯನದ ಮೂಲಕ ಸಂಗ್ರಹಿಸಿರುವ ಮಾಹಿತಿಯನ್ನು ಸಂಶೋಧನೆಯ ಉದ್ದೇಶಕ್ಕೆ ಮಾತ್ರ ಬಳಸತಕ್ಕದ್ದು.

ಈ ಅಧ್ಯಯನದಿಂದ ಯಾವುದೇ ಸಂದರ್ಭದಲ್ಲಿಹಿಂದೆ ಸರಿಯುವ ಸ್ವಾತಂತ್ರ್ಯ ನನಗಿದೆ ಎಂಬುದನ್ನೂ, ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವುದರಿಂದ ನನಗೆ ಯಾವುದೇ ಹೆಚ್ಚುವರಿ ವೆಚ್ಚ ತಗಲುವುದಿಲ್ಲವೆಂಬುದನ್ನು ತಿಳಿದಿರುತ್ತೇನೆ.

ಪರೀಕ್ಷಾರ್ಥಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ/ಹೆಬ್ಬೆಟ್ಟಿನ ಗುರುತು

ಸಾಕ್ಷಿಗಳಹೆಸರುಮತ್ತು ಸಹಿ

1. ದಿನಾಂಕ:

2. ದಿನಾಂಕ:

ಸಂದರ್ಶಕರಹೆಸರುಮತ್ತು ಸಹಿ

ದಿನಾಂಕ:

ಪ್ರಧಾನಪರೀಕ್ಷಕರಹೆಸರುಮತ್ತು ಸಹಿ

ದಿನಾಂಕ:

PATIENT INFORMATION SHEET

ROLE OF SONOELASTOGRAPHY IN EVALUATION OF MEDIAN NERVE CHANGES IN DIABETIC PATIENTS AND COMPARISION TO NORMAL SUBJECTS – A CASE CONTROL STUDY."

Principal Investigator: DR. SOUMYA CHINCHOLIKAR/ Dr. ANIL KUMAR SAKALECHA

I, **DR. SOUMYA CHINCHOLIKAR,** post-graduate student in Department of Radio-Diagnosis at Sri Devaraj Urs Medical College. I will be conducting a study titled "ROLE OF SONOELASTOGRAPHY IN EVALUATION OF MEDIAN NERVE CHANGES IN DIABETIC PATIENTS AND COMPARISION TO NORMAL SUBJECTS – A CASE CONTROL STUDY." for my dissertation under the guidance of Dr. Anil Kumar Sakalecha, Professor, Department of Radiodiagnosis. In this study, we will assess the role of ultrasound and strain elastography in evaluating the cross-sectional area of median nerve in diabetic and non-diabetic subjects. You will not be paid any financial compensation for participating in this research project.

All of your personal data will be kept confidential and will be used only for research purpose by this institution. You are free to participate in the study. You can also withdraw from the study at any point of time without giving any reasons whatsoever. Your refusal to participate will not prejudice you to any present or future care at this institution.

Name and Signature of the Principal Investigator

Date

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಮಧುಮೇಹ ರೋಗಿಗಳಲ್ಲಿ ಮಧ್ಯಮ ನರ ಬದಲಾವಣೆಗಳ ಮೌಲ್ಯಮಾಪನ ಮತ್ತು ಸಾಮಾನ್ಯ ವಿಷಯಗಳಿಗೆ ಹೋಲಿಕೆಯಲ್ಲಿ ಸೋನೋಎಲಾಸ್ಟೋಗ್ರಫಿಯ ಪಾತ್ರ - ಒಂದು ಪ್ರಕರಣ ನಿಯಂತ್ರಣ ಅಧ್ಯಯನ."

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ: ಡಾ. ಸೌಮ್ಯ ಚಿಂಚೋಲಿಕರ್ / ಡಾ. ಅನಿಲ್ ಕುಮಾರ್ ಸಕಲೇಚಾ

ನಾನು, ಡಾ. ಸೌಮ್ಯ ಚಿಂಚೋಲಿಕರ್, ಶ್ರೀ ದೇವರಾಜ್ ಉರ್ಸ್ ವೈದ್ಯಕೀಯ ಕಾಲೇಜಿನಲ್ಲಿ ರೇಡಿಯೋ ರೋಗನಿರ್ಣಯ ವಿಭಾಗದಲ್ಲಿ ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿನಿ. ನಾನು "ಮಧುಮೇಹ ರೋಗಿಗಳಲ್ಲಿ ಮಧ್ಯಮ ನರ ಬದಲಾವಣೆಗಳ ಮೌಲ್ಯಮಾಪನ ಮತ್ತು ಸಾಮಾನ್ಯ ವಿಷಯಗಳಿಗೆ ಹೋಲಿಕೆಯಲ್ಲಿ ಸೋನೋಎಲಾಸ್ಟೋಗ್ರಫಿಯ ಪಾತ್ರ - ಒಂದು ಪ್ರಕರಣ ನಿಯಂತ್ರಣ ಅಧ್ಯಯನ" ಎಂಬ ಶೀರ್ಷಿಕೆಯ ಅಧ್ಯಯನವನ್ನು ನಡೆಸಲಿದ್ದೇನೆ. ರೇಡಿಯೋ ಡಯಾಗ್ನೋಸಿಸ್ ವಿಭಾಗದ ಪ್ರಾಧ್ಯಾಪಕರಾದ ಡಾ. ಅನಿಲ್ ಕುಮಾರ್ ಸಕಲೇಚಾ ಅವರ ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ನನ್ನ ಪ್ರಬಂಧಕ್ಕಾಗಿ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ, ಮಧುಮೇಹ ಮತ್ತು ಮಧುಮೇಹವಲ್ಲದ ವಿಷಯಗಳಲ್ಲಿ ಮಧ್ಯದ ನರಗಳ ಅಡ್ಡ-ವಿಭಾಗದ ಪ್ರದೇಶವನ್ನು ಮೌಲ್ಯಮಾಪನ ಮಾಡುವಲ್ಲಿ ಅಲ್ಟ್ರಾಸೌಂಡ್ ಮತ್ತು ಸ್ಟ್ರೈನ್ ಎಲಾಸ್ಟೋಗ್ರಫಿಯ ಪಾತ್ರವನ್ನು ನಾವು ನಿರ್ಣಯಿಸುತ್ತೇವೆ. ಈ ಸಂಶೋಧನಾ ಯೋಜನೆಯಲ್ಲಿ ಭಾಗವಹಿಸಿದ್ದಕ್ಕಾಗಿ ನಿಮಗೆ ಯಾವುದೇ ಹಣಕಾಸಿನ ಪರಿಹಾರವನ್ನು ನೀಡಲಾಗುವುದಿಲ್ಲ.

ನಿಮ್ಮ ಎಲ್ಲಾ ವೈಯಕ್ತಿಕ ಡೇಟಾವನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ ಮತ್ತು ಈ ಸಂಸ್ಥೆಯು ಸಂಶೋಧನಾ ಉದ್ದೇಶಕ್ಕಾಗಿ ಮಾತ್ರ ಬಳಸುತ್ತದೆ. ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಮುಕ್ತರಾಗಿದ್ದೀರಿ. ಯಾವುದೇ ಕಾರಣಗಳನ್ನು ನೀಡದೆ ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು. ಭಾಗವಹಿಸಲು ನಿಮ್ಮ ನಿರಾಕರಣೆಯು ಈ ಸಂಸ್ಥೆಯಲ್ಲಿನ ಯಾವುದೇ ಪ್ರಸ್ತುತ ಅಥವಾ ಭವಿಷ್ಯದ ಆರೈಕೆಗೆ ನಿಮ್ಮನ್ನು ಹಾನಿಗೊಳಿಸುವುದಿಲ್ಲ.

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ

MASTER CHART

KEY TO MASTER CHART

- **AP Thickness – Anteroposterior thickness**
- **C.S.A - Cross-sectional Area**
- **Echogenicity**
- **Strain Ratio**
- **DM - Diabetes Mellitus**
- **HbA1c – Glycated haemoglobin**
- **FBS - Fasting Blood Sugar**
- **Glycemic Control**

SNO	UHID	AGE	GENDER	GROUP	AP THICKNESS (mm)	CROSS SECTION AREA (cm^2)	ECHOGENICITY	STRAIN RATIO	Hba1c	FBS (mg/dL)	DURATION OF DM (Years)	CONTROLLED/ UNCONTROLLED DM	CORRELATIONS
1	616211	45	FEMALE	NORMAL	1.3	0.04	MAINTAINED	0.6	5	95	0	NA	NA
2	599799	55	MALE	NORMAL	1.6	0.05	MAINTAINED	0.8	5	90	0	NA	NA
3	612546	50	MALE	NORMAL	1.5	0.05	MAINTAINED	0.5	4.5	97	0	NA	NA
4	497507	45	MALE	NORMAL	1.8	0.05	MAINTAINED	0.5	5	99	0	NA	NA
5	613577	45	FEMALE	NORMAL	1.1	0.05	MAINTAINED	0.5	5	98	0	NA	NA
6	614233	44	MALE	NORMAL	1.3	0.05	MAINTAINED	0.7	4.5	88	0	NA	NA
7	615216	44	FEMALE	NORMAL	1.4	0.05	MAINTAINED	0.9	4.8	92	0	NA	NA
8	612040	49	MALE	NORMAL	1.4	0.06	MAINTAINED	0.8	4.5	90	0	NA	NA
9	613045	49	FEMALE	NORMAL	1.1	0.04	MAINTAINED	0.6	5	98	0	NA	NA
10	614263	50	MALE	NORMAL	1.1	0.04	MAINTAINED	0.8	4.8	95	0	NA	NA
11	614192	48	FEMALE	DIABETIC NEUROPATHY	1.8	0.06	ALTERED	1.3	7.5	160	8	UNCONTROLLED	PRESENT
12	612061	50	MALE	DIABETIC NEUROPATHY	1.4	0.05	ALTERED	2.2	8	180	15	UNCONTROLLED	PRESENT
13	602728	60	MALE	DIABETIC NEUROPATHY	1.7	0.07	ALTERED	2.2	8.5	185	18	UNCONTROLLED	PRESENT
14	613723	49	MALE	DIABETIC NEUROPATHY	2.3	0.12	ALTERED	1.25	8	200	8	UNCONTROLLED	PRESENT
15	614117	49	FEMALE	DIABETIC NEUROPATHY	2.1	0.08	ALTERED	1.9	8.5	220	7	UNCONTROLLED	PRESENT
16	614516	50	MALE	DIABETIC NEUROPATHY	1.4	0.05	MAINTAINED	1.4	7	150	6	CONTROLLED	PRESENT
17	611105	50	FEMALE	DIABETIC NEUROPATHY	1.7	0.07	MAINTAINED	2.2	6	110	8	CONTROLLED	PRESENT
18	424516	52	MALE	DIABETIC NEUROPATHY	2.3	0.12	ALTERED	1.25	7	150	9	UNCONTROLLED	PRESENT
19	609278	52	FEMALE	DIABETIC NEUROPATHY	1.8	0.08	ALTERED	1.3	7	140	8	UNCONTROLLED	PRESENT
20	612212	53	MALE	DIABETIC NEUROPATHY	2	0.09	ALTERED	1.5	7.5	160	10	UNCONTROLLED	PRESENT
21	454514	53	FEMALE	DIABETIC NEUROPATHY	2	0.08	ALTERED	1.88	8	200	10	UNCONTROLLED	PRESENT
22	564516	55	MALE	DIABETIC NEUROPATHY	2.1	0.12	ALTERED	1.3	7	170	11	UNCONTROLLED	PRESENT
23	654516	55	FEMALE	DIABETIC NEUROPATHY	1.9	0.13	ALTERED	1.7	7.5	160	11	UNCONTROLLED	PRESENT
24	614513	54	FEMALE	DIABETIC NEUROPATHY	1.7	0.12	ALTERED	2	7	180	12	UNCONTROLLED	PRESENT
25	714514	54	MALE	DIABETIC NEUROPATHY	1.5	0.08	MAINTAINED	1.4	6	112	10	CONTROLLED	PRESENT
26	514528	56	FEMALE	DIABETIC NEUROPATHY	1.4	0.05	ALTERED	1.3	8	220	11	UNCONTROLLED	PRESENT
27	614516	56	MALE	DIABETIC NEUROPATHY	1.2	0.04	ALTERED	1.5	7.5	200	11	UNCONTROLLED	PRESENT
28	514529	57	FEMALE	DIABETIC NEUROPATHY	1.4	0.07	ALTERED	1.2	6	110	10	CONTROLLED	PRESENT
29	814532	57	MALE	DIABETIC NEUROPATHY	1.5	0.08	ALTERED	1.4	6.1	112	10	CONTROLLED	PRESENT
30	614528	59	FEMALE	DIABETIC NEUROPATHY	1.6	0.13	MAINTAINED	1.7	6.2	114	9	CONTROLLED	PRESENT
31	614530	59	MALE	DIABETIC NEUROPATHY	1.4	0.06	MAINTAINED	1.4	6.4	117	9	CONTROLLED	PRESENT
32	714534	60	MALE	DIABETIC NEUROPATHY	2.1	0.06	ALTERED	2.55	6	110	10	CONTROLLED	PRESENT
33	614548	60	FEMALE	DIABETIC NEUROPATHY	2.3	0.12	ALTERED	1.25	6.4	114	10	CONTROLLED	PRESENT
34	714535	62	MALE	DIABETIC NEUROPATHY	1.9	0.12	ALTERED	2	6.2	116	10	CONTROLLED	PRESENT
35	554516	62	FEMALE	DIABETIC NEUROPATHY	1.8	0.08	ALTERED	1.9	6.4	110	10	CONTROLLED	PRESENT
36	609882	45	FEMALE	DIABETIC	1.6	0.08	MAINTAINED	0.99	7	165	5	UNCONTROLLED	PRESENT
37	610247	45	MALE	DIABETIC	2	0.11	MAINTAINED	0.92	7	160	6	UNCONTROLLED	PRESENT
38	614552	60	MALE	DIABETIC	1.4	0.07	MAINTAINED	1	6.5	125	10	CONTROLLED	PRESENT
39	554564	60	FEMALE	DIABETIC	1.4	0.07	MAINTAINED	0.98	7.5	150	10	UNCONTROLLED	PRESENT
40	582348	47	MALE	DIABETIC	1.5	0.05	MAINTAINED	0.89	6.8	125	5	CONTROLLED	PRESENT

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41	614516	47	FEMALE	DIABETIC	1.4	0.07	MAINTAINED	0.95	6.5	112	5	CONTROLLED	PRESENT
42	524526	48	FEMALE	DIABETIC	1.7	0.05	MAINTAINED	0.79	6.2	110	5	CONTROLLED	PRESENT
43	424532	48	MALE	DIABETIC	1.6	0.1	MAINTAINED	0.91	6.4	115	6	CONTROLLED	PRESENT
44	724545	50	FEMALE	DIABETIC	1.8	0.1	MAINTAINED	0.97	7	164	7	UNCONTROLLED	PRESENT
45	554554	50	MALE	DIABETIC	1.4	0.07	MAINTAINED	0.88	6.5	110	5	CONTROLLED	PRESENT
46	454562	51	FEMALE	DIABETIC	1.4	0.08	MAINTAINED	0.82	6.4	110	6	CONTROLLED	PRESENT
47	564574	51	MALE	DIABETIC	1.6	0.07	MAINTAINED	0.95	6.5	112	5	CONTROLLED	PRESENT
48	654555	52	FEMALE	DIABETIC	2.3	0.1	MAINTAINED	1	7.5	180	8	UNCONTROLLED	PRESENT
49	614515	52	MALE	DIABETIC	1.6	0.07	MAINTAINED	0.97	6.2	100	7	CONTROLLED	PRESENT
50	714526	54	MALE	DIABETIC	2.3	0.1	MAINTAINED	1.1	7	170	8	UNCONTROLLED	PRESENT
51	514535	54	FEMALE	DIABETIC	1.8	0.1	MAINTAINED	1.2	6.8	155	6	UNCONTROLLED	PRESENT
52	614542	55	FEMALE	DIABETIC	2.3	0.1	MAINTAINED	0.99	6.2	110	7	CONTROLLED	PRESENT
53	514555	55	MALE	DIABETIC	1.6	0.07	MAINTAINED	1	6	100	6	CONTROLLED	PRESENT
54	814562	53	FEMALE	DIABETIC	1.4	0.08	MAINTAINED	0.94	6.2	110	7	CONTROLLED	PRESENT
55	614568	53	MALE	DIABETIC	2.3	0.1	MAINTAINED	1	6.4	114	8	CONTROLLED	PRESENT
56	614572	56	MALE	DIABETIC	1.4	0.07	MAINTAINED	0.89	6.8	152	9	UNCONTROLLED	PRESENT
57	714568	56	FEMALE	DIABETIC	1.6	0.08	MAINTAINED	1	6	112	8	CONTROLLED	PRESENT
58	614566	57	MALE	DIABETIC	1.8	0.1	MAINTAINED	1.2	7	165	9	UNCONTROLLED	PRESENT
59	714582	57	FEMALE	DIABETIC	2.3	0.1	MAINTAINED	0.96	6	120	8	CONTROLLED	PRESENT
60	554591	52	FEMALE	DIABETIC	1.6	0.07	MAINTAINED	1	6	110	8	CONTROLLED	PRESENT
61	654516	44	FEMALE	NORMAL	1.6	0.05	MAINTAINED	0.8	5	90	0	NA	NA
62	729882	44	MALE	NORMAL	1.5	0.05	MAINTAINED	0.3	5	97	0	NA	NA
63	750247	47	FEMALE	NORMAL	1.8	0.05	MAINTAINED	0.5	4.5	99	0	NA	NA
64	704552	47	MALE	NORMAL	1.1	0.05	MAINTAINED	0.5	5	98	0	NA	NA
65	774564	42	FEMALE	NORMAL	1.3	0.05	MAINTAINED	0.5	5	88	0	NA	NA
66	682348	42	MALE	NORMAL	1.4	0.05	MAINTAINED	0.8	4.5	92	0	NA	NA
67	654516	45	FEMALE	NORMAL	1.1	0.04	MAINTAINED	0.6	4.8	90	0	NA	NA
68	714526	45	MALE	NORMAL	1.4	0.05	MAINTAINED	0.8	4.5	97	0	NA	NA
69	764532	43	FEMALE	NORMAL	1.3	0.05	MAINTAINED	0.9	5	99	0	NA	NA
70	724548	43	MALE	NORMAL	1.4	0.06	MAINTAINED	0.8	4.8	98	0	NA	NA
71	554558	46	MALE	NORMAL	1.3	0.05	MAINTAINED	0.5	5	88	0	NA	NA
72	764562	46	FEMALE	NORMAL	1.4	0.05	MAINTAINED	0.8	4.8	92	0	NA	NA
73	784574	49	FEMALE	NORMAL	1.1	0.04	MAINTAINED	0.6	5	90	0	NA	NA
74	794555	49	MALE	NORMAL	1.3	0.05	MAINTAINED	0.9	4.5	98	0	NA	NA
75	744515	46	FEMALE	NORMAL	1.4	0.06	MAINTAINED	0.8	5	95	0	NA	NA
76	714556	46	MALE	NORMAL	1.6	0.05	MAINTAINED	0.9	5	98	0	NA	NA
77	784535	47	FEMALE	NORMAL	1.3	0.05	MAINTAINED	0.9	5	88	0	NA	NA
78	794542	47	MALE	NORMAL	1.3	0.05	MAINTAINED	0.5	4.5	92	0	NA	NA
79	754555	48	FEMALE	NORMAL	1.4	0.05	MAINTAINED	0.8	5	90	0	NA	NA
80	814572	48	FEMALE	NORMAL	1.1	0.04	MAINTAINED	0.6	5	98	0	NA	NA

SNO	UHID	AGE	GENDER	GROUP	AP THICKNESS (mm)	CROSS SECTION AREA (cm^2)	ECHOGENICITY	STRAIN RATIO	Hba1c	FBS (mg/dL)	DURATION OF DM (Years)	CONTROLLED/ UNCONTROLLED DM	CORRELATIONS
81	744568	48	MALE	NORMAL	1.3	0.05	MAINTAINED	0.9	4.5	95	0	NA	NA
82	764572	50	FEMALE	NORMAL	1.4	0.06	MAINTAINED	0.8	4.8	98	0	NA	NA
83	714558	50	MALE	NORMAL	1.4	0.05	MAINTAINED	0.8	4.5	88	0	NA	NA
84	744566	49	MALE	NORMAL	1.3	0.05	MAINTAINED	0.5	5	92	0	NA	NA
85	714576	50	FEMALE	NORMAL	1.4	0.05	MAINTAINED	0.8	4.8	90	0	NA	NA
86	784591	47	FEMALE	NORMAL	1.4	0.05	MAINTAINED	0.8	5	98	0	NA	NA
87	891242	47	MALE	NORMAL	1.4	0.05	MAINTAINED	0.8	4.5	95	0	NA	NA
88	881248	46	MALE	NORMAL	1.3	0.05	MAINTAINED	0.5	4.8	98	0	NA	NA
89	701155	47	FEMALE	NORMAL	1.3	0.05	MAINTAINED	0.9	4.5	95	0	NA	NA
90	863142	52	MALE	NORMAL	1.4	0.06	MAINTAINED	0.8	5	90	0	NA	NA
91	855138	50	FEMALE	NORMAL	1.1	0.04	MAINTAINED	0.6	5	97	0	NA	NA
92	823185	50	MALE	NORMAL	1.1	0.04	MAINTAINED	0.6	4.5	95	0	NA	NA
93	862325	46	MALE	NORMAL	1.3	0.05	MAINTAINED	0.5	5	90	0	NA	NA
94	841427	46	FEMALE	NORMAL	1.4	0.05	MAINTAINED	0.8	5	97	0	NA	NA
95	784523	48	MALE	NORMAL	1.1	0.04	MAINTAINED	0.6	4.5	99	0	NA	NA
96	794987	48	FEMALE	NORMAL	1.4	0.05	MAINTAINED	0.8	4.8	98	0	NA	NA
97	800124	47	FEMALE	NORMAL	1.3	0.05	MAINTAINED	0.5	4.5	88	0	NA	NA
98	820245	49	FEMALE	NORMAL	1.4	0.05	MAINTAINED	0.8	5	92	0	NA	NA
99	854516	47	MALE	NORMAL	1.4	0.05	MAINTAINED	0.8	4.8	90	0	NA	NA
100	844516	48	MALE	NORMAL	1.4	0.05	MAINTAINED	0.8	4.8	98	0	NA	NA