Shear Wave Elastography of the Tibial Nerve in Diabetic Peripheral Neuropathy: Diagnostic Performance and Clinical Applications

Ahmed Mohamed Hasan Alsowey [1], Nesreen Mohamed Mohey Mohamed[2], Saeed Mohamed Yousef Ebrahim[3], Essam Elsayed Tharwat Mohamed [4]

1. *Professor of Radiodiagnosis, Faculty of Medicine - Zagazig University,*
2. *Professor of Radiodiagnosis, Faculty of Medicine - Zagazig University,*
3. *MBBCH, Faculty of Medicine, Zagazig University*
4. *Lecturer of Radiodiagnosis, Faculty of Medicine - Zagazig University,*

Corresponding Author: Saeed Mohamed Yousef Ebrahim

Received: 28 October 2024, **Accepted:** 17 November 2024, **Published:** 20 November 2024

Abstract

Background: Diabetic peripheral neuropathy (DPN) is one of the most frequent and disabling complications of diabetes mellitus and remains a major cause of pain, foot ulceration, and non-traumatic limb amputation. Early detection of DPN is critical, yet current reference diagnostic tools such as nerve conduction studies (NCS) are time-consuming, limited largely to large-fiber assessment, and not ideal for widespread screening. High-resolution ultrasound (HRUS) has emerged as a valuable adjunct for structural nerve assessment, but conventional B-mode and Doppler imaging provide mainly morphologic and vascular information and are relatively insensitive to early microstructural change.

Shear wave elastography (SWE) is a quantitative ultrasound technique that measures tissue stiffness by tracking the propagation speed of mechanically induced shear waves. Applied to peripheral nerves, SWE can characterize changes in intraneural stiffness related to axonal loss, endoneurial edema, and increased connective tissue. The tibial nerve, easily accessible at the ankle and frequently involved in length-dependent DPN, has become the most studied target for elastographic evaluation in diabetes. This review synthesizes the current evidence regarding tibial nerve SWE in patients with DPN. Specifically, it aims to: (1) summarize technical principles of SWE and their implications for peripheral nerve imaging; (2) describe the elastographic characteristics of the tibial nerve in healthy individuals versus diabetic patients with and without clinically or electrophysiologically confirmed DPN; (3) evaluate the diagnostic performance of tibial nerve stiffness measurements compared with clinical assessment, NCS, and conventional ultrasound; and (4) discuss the potential role of SWE as a noninvasive biomarker for early or subclinical DPN, severity grading, and longitudinal follow-up.

Published studies consistently demonstrate higher tibial nerve stiffness values in patients with established DPN than in diabetic patients without neuropathy and healthy controls, with stiffness increasing in parallel with neuropathy severity. Notably, several reports show elevated stiffness in diabetic patients without clinical or NCS evidence of neuropathy, suggesting that SWE may detect neuropathic involvement at a subclinical stage. While current data support tibial nerve SWE as a promising adjunctive tool, limitations include inter-machine variability, lack of standardized protocols, and insufficient longitudinal and outcome-based data. SWE should presently be viewed as a complementary technique integrated with clinical and neurophysiologic evaluation, with further multicenter studies needed to define robust cut-off values, normative databases, and its impact on clinical decision-making.

Keywords: *Shear Wave Elastography, Tibial Nerve, Diabetic Peripheral Neuropathy*

Introduction

Diabetic peripheral neuropathy (DPN) is one of the most common and debilitating chronic complications of diabetes mellitus, affecting up to half of the global diabetic population over their lifetime. As diabetes prevalence continues to rise worldwide, the burden of neuropathic complications is expected to escalate substantially. According to the International Diabetes Federation, 463 million individuals were living with diabetes in 2019, a figure projected to reach 700 million by 2045, making DPN an increasingly important public health and clinical concern [1]. Clinically, DPN manifests as a length-dependent, predominantly sensory neuropathy that greatly impairs mobility, sleep, psychological well-being, and increases the risk of foot ulcers and non-traumatic limb amputations [2]. These outcomes collectively underscore the necessity for early, accurate detection of peripheral nerve injury before irreversible structural damage occurs.

Despite its prevalence and impact, diagnosing DPN at an early stage remains challenging. Traditional diagnostic tools such as nerve conduction studies (NCS) are often considered the gold standard for confirming large-fiber neuropathy; however, they are time-consuming, uncomfortable, operator-dependent, and limited in their ability to detect small-fiber dysfunction, which often precedes measurable electrophysiologic abnormalities [3]. Moreover, NCS may remain normal in patients with subclinical DPN, creating a diagnostic gap during the early window when intervention could be most beneficial [4]. Clinical evaluation and bedside screening tools, although useful, lack sensitivity for subtle or early neuropathic changes, contributing to delayed diagnosis and increased risk of progression to severe neuropathic complications [5].

In recent years, high-resolution ultrasound (HRUS) has gained prominence as a complementary imaging modality capable of evaluating peripheral nerves in vivo. HRUS is non-invasive, accessible, and provides morphological information including nerve cross-sectional area, echogenicity, fascicular pattern, and vascularity [6]. However, conventional B-mode ultrasound primarily depicts structural alterations and may not reliably detect microstructural or biomechanical changes occurring early in DPN. This limitation has stimulated interest in more advanced sonographic techniques capable of quantifying tissue mechanical properties in a reproducible manner.

Shear wave elastography (SWE) has emerged as a clinically relevant quantitative imaging tool for characterizing tissue stiffness by measuring the velocity of propagating shear waves. SWE has already matured in fields such as liver fibrosis assessment and musculoskeletal imaging, offering reproducible and objective measures of tissue elasticity [7]. Applied to peripheral nerves, SWE provides insight into biomechanical alterations related to edema, fibrosis, and axonal degeneration—changes that may precede overt morphologic abnormalities visible on standard ultrasound. Early studies have demonstrated increased stiffness of the tibial nerve in diabetic patients with and without clinically established neuropathy, suggesting that SWE may detect subclinical neuropathic involvement earlier than NCS or conventional ultrasound [8].

Aim

The aim of this review is to evaluate the diagnostic performance and clinical applications of tibial nerve shear wave elastography in diabetic peripheral neuropathy. This includes analysis of its quantitative capabilities, correlation with clinical and electrophysiologic findings, and potential utility in early detection, severity stratification, and follow-up monitoring.

Research Gap

Although promising, SWE remains underutilized in routine DPN assessment due to limited standardization of acquisition protocols, variability in cutoff values, and lack of large multicenter prospective studies. Additionally, the role of tibial nerve SWE in identifying early or subclinical neuropathy requires further evidence before integration into screening pathways. This review addresses

these gaps by synthesizing current validated findings and defining the clinical landscape in which tibial nerve SWE may contribute most effectively to DPN evaluation [8,9].

Diabetic Peripheral Neuropathy: Epidemiology and Clinical Significance

Diabetic peripheral neuropathy (DPN) is one of the most common and disabling complications of diabetes, affecting nearly 50%–66% of individuals with long-standing disease, based on estimates from large population studies and international diabetes surveillance reports. Global projections indicate that the increasing prevalence of diabetes will be accompanied by a proportional increase in neuropathic morbidity, including ulceration and amputations. The slow and often silent progression of nerve injury means many patients remain undiagnosed until late stages, when irreversible nerve damage has occurred. These epidemiologic patterns highlight the need for diagnostic tools capable of detecting neuropathy early. [10–13]

The pathophysiology of DPN is multifactorial, driven by chronic hyperglycemia that induces oxidative stress, mitochondrial dysfunction, microvascular ischemia, and pro-inflammatory pathway activation. These mechanisms impair axonal transport, damage Schwann cells, and disrupt endoneurial microcirculation, initiating a cascade that culminates in axonal degeneration and demyelination. Distal nerve segments are particularly vulnerable due to their high metabolic demands and length, explaining the classical length-dependent pattern of sensory loss in DPN. Biochemical and microstructural changes frequently precede clinical symptoms, underscoring the need for methods capable of detecting these early abnormalities. [14–16]

Clinically, DPN encompasses a broad spectrum ranging from asymptomatic subclinical dysfunction to severe neuropathic pain, sensory deficits, gait imbalance, and autonomic disturbance. Early disease often involves small-fiber pathology, manifesting as subtle changes in pain and temperature sensitivity that may escape routine bedside testing. With progression, large-fiber dysfunction produces diminished vibration sensation, proprioceptive loss, and muscle weakness, contributing to impaired mobility and quality of life. The variability and overlap in clinical presentations necessitate objective diagnostic tools capable of detecting early changes across multiple nerve fiber types. [17–18]

Routine screening tools such as monofilament testing, vibration perception assessment, and symptom questionnaires lack sensitivity for early neuropathy and frequently miss subclinical disease. Although nerve conduction studies remain the gold standard for evaluating large-fiber involvement, they do not reliably detect small-fiber dysfunction and may remain normal in early DPN. In addition, they are time-consuming, uncomfortable, and less practical for large-scale screening. These limitations create a diagnostic gap and justify the need for more sensitive, noninvasive imaging approaches for early detection. [19–21]

The tibial nerve is a valuable target for neuropathy assessment due to its distal location, functional relevance, and excellent accessibility to ultrasound. High-resolution ultrasonography has shown that tibial nerve enlargement, fascicular distortion, and altered echogenicity occur in DPN and correlate with disease severity. However, these morphological changes generally emerge only after substantial nerve injury, limiting the utility of B-mode ultrasound for early diagnosis. Consequently, there is a growing need for quantitative imaging techniques that detect microstructural and biomechanical changes at earlier stages. [22–23]

Shear wave elastography (SWE) has shown promise in this regard, as multiple studies have demonstrated increased tibial nerve stiffness in patients with DPN. Importantly, stiffness elevation has also been reported in diabetic patients without clinical symptoms or electrophysiologic abnormalities, suggesting that SWE may detect subclinical neuropathy. Such findings indicate that SWE may capture early changes related to fibrosis, endoneurial edema, and loss of nerve elasticity—alterations that precede visible structural abnormalities on conventional ultrasound. This highlights the potential of tibial nerve SWE as an early diagnostic biomarker for DPN. [8,24]

From a radiologic perspective, SWE provides quantitative, reproducible, and operator-independent assessment of tissue stiffness, offering advantages over standard sonography that relies heavily on visual

interpretation. By quantifying biomechanical alterations in peripheral nerves, SWE may overcome the diagnostic delays inherent in current techniques. As evidence grows and standardized protocols are developed, tibial nerve SWE may become an integral component of multimodal neuropathy evaluation, supporting earlier diagnosis, improved monitoring, and more effective clinical decision-making. [25]

High-Resolution Ultrasound in Peripheral Nerve Evaluation

High-resolution ultrasonography (HRUS) has become an important adjunct to clinical and electrophysiologic assessment of peripheral neuropathies due to its ability to visualize nerves in real time with excellent spatial resolution. Modern high-frequency linear probes allow detailed visualization of peripheral nerves, revealing their fascicular structure, echogenicity, and cross-sectional area in ways that were not possible with earlier ultrasound systems. HRUS is noninvasive, widely available, inexpensive, and capable of scanning the entire course of a nerve within minutes, which makes it particularly well-suited for evaluating neuropathies in large diabetic populations. Furthermore, the modality provides dynamic imaging, allowing assessment of nerve mobility and surrounding tissue relationships, features that can aid identification of entrapment and other mechanical contributors to neuropathy. [26–27]

Structurally, normal peripheral nerves appear on short-axis imaging as honeycomb-like structures composed of hypoechoic fascicles surrounded by hyperechoic perineurial tissue, while long-axis views reveal a characteristic bundle-of-straws appearance. Deviations from these patterns provide valuable clues regarding neuropathic involvement. In diabetic patients, HRUS studies have demonstrated nerve enlargement, loss of fascicular definition, increased intraneural hypoechogenicity, and changes in vascularity, reflecting chronic metabolic and ischemic injury. These features correlate with clinical severity and electrophysiological abnormalities, making HRUS a useful tool for both diagnosis and monitoring of peripheral nerve pathology. However, morphological changes typically appear only after substantial structural injury, limiting HRUS utility in early or subclinical disease detection. [28–29]

In addition to structural assessment, HRUS allows measurement of nerve cross-sectional area (CSA), which is frequently increased in patients with diabetic peripheral neuropathy. CSA enlargement likely reflects endoneurial edema, inflammatory infiltration, microangiopathic damage, and intraneural fibrosis. Studies have shown that tibial nerve CSA measured at the ankle correlates with DPN severity, although reported cutoff values vary widely due to differences in technique, probe frequency, patient positioning, and anatomic reference points. This variability reduces the standardization necessary for clinical screening and underscores the need for complementary quantitative methods capable of assessing nerve biomechanical properties rather than morphology alone. [30–31]

Color and power Doppler imaging can further enhance HRUS evaluation by measuring intraneural vascularity. Increased vascularity may be seen in inflammatory neuropathies, while reduced flow may accompany chronic ischemic damage as seen in longstanding diabetic neuropathy. Despite these capabilities, Doppler findings often lack sensitivity and specificity for DPN because vascular changes may be subtle and operator-dependent. Moreover, Doppler does not provide quantitative information regarding nerve stiffness or elasticity, limiting its usefulness for detecting early biomechanical alterations that precede morphologic abnormalities. [32]

One of the major limitations of conventional HRUS is that it primarily characterizes structural changes and cannot reliably evaluate the tissue mechanical properties affected early in DPN progression. Nerve stiffness increases due to fibrosis, axonal loss, and altered endoneurial pressure—changes not readily depicted by B-mode or Doppler ultrasound. Without the ability to quantify these mechanical alterations, conventional HRUS may miss the earliest phases of neuropathic injury. This limitation has driven the evolution of advanced sonographic techniques—particularly shear wave elastography—that offer quantitative insight into nerve stiffness and biomechanical integrity. Such adjunctive tools are crucial for bridging the diagnostic gap between clinical manifestations and early tissue changes in diabetic neuropathy. [33]

Because of its accessibility, lack of ionizing radiation, and compatibility with bedside or outpatient evaluation, HRUS continues to play an increasingly significant role in neuromuscular imaging. When

integrated with shear wave elastography, HRUS transforms from a purely morphological tool into a multimodal assessment technique capable of evaluating both structure and biomechanical function. This combination provides a more comprehensive evaluation of peripheral nerves and enhances diagnostic confidence in settings where clinical and electrophysiological findings may be inconclusive. As research advances, HRUS—particularly when augmented by elastography—may become a central pillar in the noninvasive assessment of diabetic neuropathy. [34]

Principles of Ultrasound Elastography and Its Application to Peripheral Nerves

Ultrasound elastography is a noninvasive imaging technique designed to measure tissue stiffness by evaluating the mechanical response of tissues to an applied force. First introduced in the 1990s, elastography has evolved significantly, becoming an important diagnostic tool in evaluating conditions such as liver fibrosis, breast lesions, thyroid pathology, and musculoskeletal disorders. Its primary clinical advantage lies in its ability to provide biomechanical information that cannot be obtained through conventional B-mode ultrasound. By quantifying how tissues deform under stress, elastography offers an objective measure of stiffness that reflects underlying histological characteristics, including fibrosis, edema, or changes in extracellular matrix composition. These attributes make the technique particularly appealing for assessing early neuropathic changes in diabetes, where structural alterations may be subtle or absent in early stages. [35–37]

Two major categories of elastography exist: strain elastography and shear wave elastography (SWE). Strain elastography measures tissue deformation caused by manual or physiologic compression, displaying relative stiffness using a qualitative or semi-quantitative color map. However, strain elastography has limitations, including operator dependency, limited repeatability, and lack of absolute stiffness quantification. In contrast, SWE generates mechanical shear waves within tissues using an acoustic radiation force produced by the ultrasound transducer. These shear waves propagate perpendicularly through tissue, and their velocity is directly proportional to tissue stiffness—faster velocities indicate stiffer tissues. Because SWE quantifies stiffness in meters per second or kilopascals, it provides more reproducible and comparable results across studies and operators, making it particularly valuable for evaluating peripheral nerves. [38–40]

The physics of shear wave propagation is central to understanding its application in neuropathy. Peripheral nerves are anisotropic structures composed of fascicles surrounded by connective tissue layers, meaning stiffness varies depending on orientation and composition. SWE captures these differences by measuring how shear waves move through intraneural tissues, which are altered by edema, axonal loss, or fibrosis—key pathological features of diabetic neuropathy. Studies demonstrate that diabetic nerves, particularly the tibial nerve, exhibit increased stiffness long before gross structural changes appear, reflecting early endoneurial compromise. The ability of SWE to capture such biomechanical changes enhances its utility for detecting subclinical DPN. [41–42]

In peripheral nerve imaging, SWE is typically performed by placing the transducer gently over the nerve without exerting excessive pressure, ensuring that tissue stiffness measurements reflect intrinsic properties rather than artificial compression. The region of interest is positioned over the nerve on either long- or short-axis images, and multiple measurements are taken to improve reproducibility. Careful attention to probe orientation, limb positioning, and avoidance of anisotropy is essential to obtaining reliable stiffness values. These technical considerations are particularly important when evaluating the tibial nerve at the ankle, where surrounding tendons and vessels may introduce artifacts if not carefully excluded during acquisition. [43–44]

The clinical value of elastography arises from its ability to detect changes that are not visible on standard ultrasound. While B-mode imaging identifies morphological alterations such as nerve enlargement or fascicular irregularity, elastography quantifies internal mechanical properties that reflect the physiologic and histologic progression of neuropathy. Increased nerve stiffness correlates with endoneurial fibrosis, chronic inflammation, and increased intraneural pressure, all of which are hallmark processes in diabetic neuropathy. Several studies have demonstrated that SWE can distinguish between healthy nerves, diabetic nerves without clinical neuropathy, and nerves in patients with established DPN, highlighting

its ability to identify early pathologic change. [45–46]

Beyond diagnosis, elastography holds promise for monitoring disease progression and treatment response. Because nerve stiffness increases with neuropathy severity, SWE may allow clinicians to track changes longitudinally and evaluate the impact of glycemic control or disease-modifying therapies. Although longitudinal data remain limited, emerging evidence suggests that SWE may serve as a quantitative biomarker for neuropathy progression, enabling earlier interventions aimed at preventing irreversible nerve damage. As research expands, elastography may become an important companion tool in the comprehensive radiologic evaluation of peripheral neuropathies. [47–48]

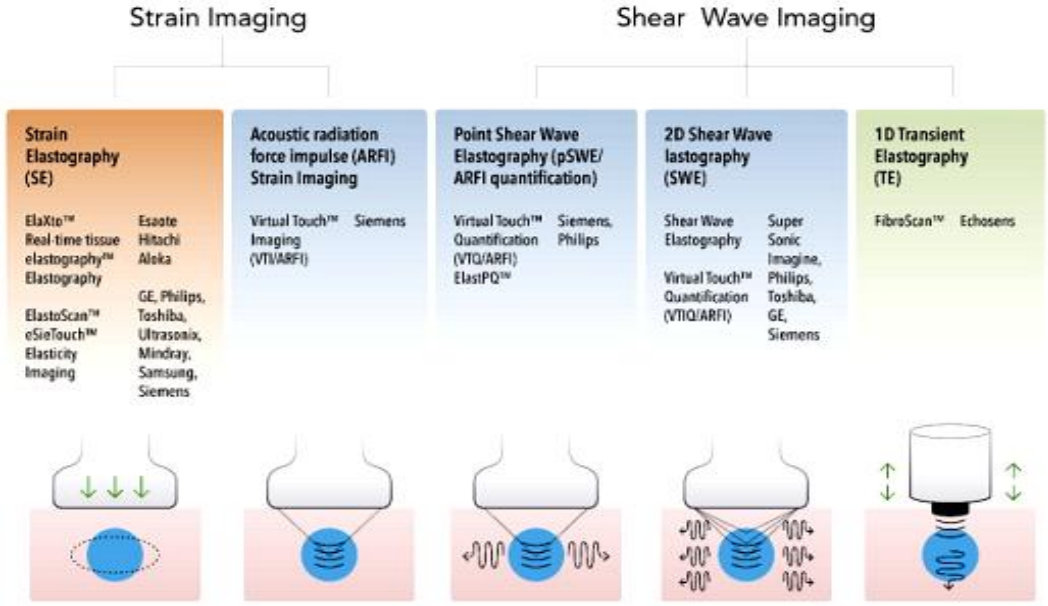

Figure (1): Ultrasound Elastography Techniques [49]

Shear Wave Elastography of the Tibial Nerve in Diabetic Peripheral Neuropathy

Shear wave elastography (SWE) has emerged as a promising quantitative imaging tool for assessing peripheral nerve stiffness in diabetic peripheral neuropathy (DPN). The tibial nerve, in particular, has been extensively studied due to its accessibility at the medial ankle and its involvement in the characteristic length-dependent pattern of DPN. SWE measures the velocity of shear waves generated within the nerve, providing a direct estimate of intraneural stiffness. Increased stiffness reflects pathological changes such as endoneurial fibrosis, axonal degeneration, edema, and increased intraneural pressure. Studies consistently demonstrate that tibial nerve stiffness is significantly higher in patients with established DPN compared to healthy individuals and diabetic patients without neuropathy, suggesting that SWE captures early physiological alterations that precede morphological abnormalities detected by conventional ultrasound. [49–51]

One of the most clinically significant advantages of tibial nerve SWE is its ability to detect subclinical neuropathy, a stage in which symptoms and nerve conduction studies may still be normal. Several studies have shown that diabetic patients without clinical or electrophysiologic evidence of neuropathy already exhibit increased tibial nerve stiffness, indicating that SWE may identify early nerve dysfunction before traditional diagnostic markers become abnormal. This early detection capability is vital, as timely initiation of glycemic control and risk factor modification may prevent progression to irreversible nerve damage. In this regard, SWE holds potential as a screening tool for high-risk diabetic populations, especially in primary care or endocrinology settings where electrophysiologic testing may not be readily accessible. [52–54]

Comparative studies between SWE and nerve conduction studies (NCS) further highlight the diagnostic value of elastography. While NCS remains the gold standard for large-fiber neuropathy, it has limited ability to detect early or small-fiber abnormalities. In contrast, SWE is capable of detecting mechanical changes related to small-fiber pathology and early endoneurial remodeling. Research shows that tibial

nerve stiffness correlates moderately with electrophysiologic parameters such as conduction velocity and amplitude, yet SWE often reveals abnormalities in cases where NCS remains normal. This suggests that elastography provides complementary diagnostic information and may bridge the gap between clinical findings and electrophysiologic testing. [55–56]

Standardized SWE techniques are critical for reliable nerve stiffness measurement. Proper probe positioning, minimal transducer pressure, and correct limb alignment help avoid artifacts and ensure reproducibility. Most studies perform measurements in both long- and short-axis planes, ensuring that anisotropic nerve properties are accounted for. Consistent measurement protocols are particularly important for the tibial nerve at the ankle due to the close proximity of tendons, vessels, and fat tissue, which can interfere with shear wave propagation. Despite these considerations, SWE has shown high intra- and inter-observer reliability, further supporting its use in both clinical practice and research. [57–58]

The integration of SWE into clinical practice for DPN assessment holds substantial potential but currently remains limited by several factors. Variability in cutoff values across different ultrasound systems, patient populations, and measurement techniques poses challenges for universal adoption. Additionally, most published studies involve relatively small sample sizes, and few have evaluated long-term outcomes or established normative databases for diverse age groups and ethnicities. Large-scale multicenter studies are needed to determine standardized stiffness thresholds that can be incorporated into diagnostic algorithms. Nevertheless, the existing evidence strongly supports the clinical utility of tibial nerve SWE as an adjunctive tool for early diagnosis, monitoring progression, and potentially assessing treatment response in diabetic peripheral neuropathy. [59]

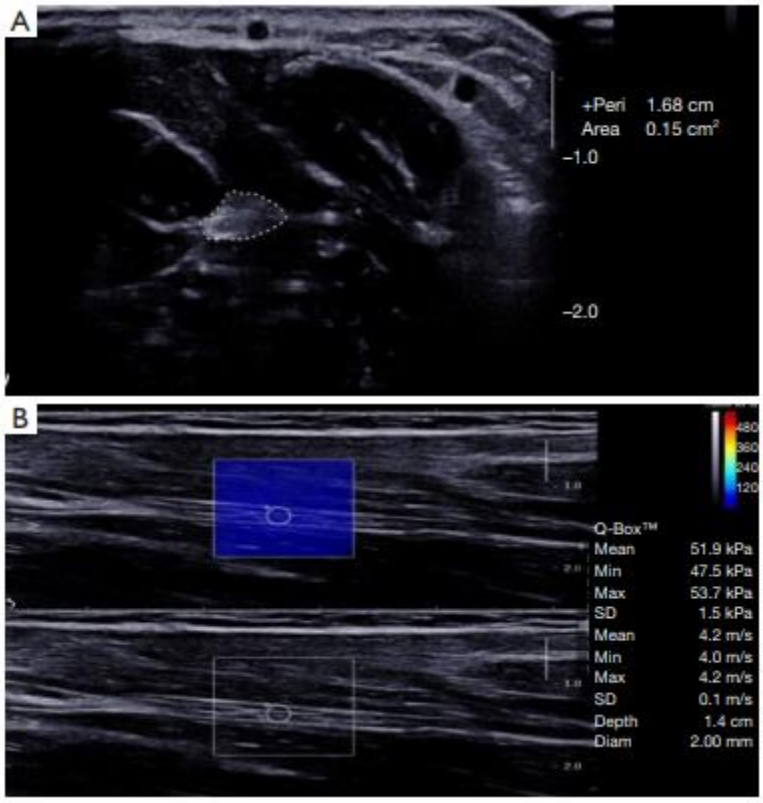

Figure (1): Sonographic findings of the median nerve in a patient with diabetic peripheral neuropathy (DPN) [58]

(A) The median nerve is observed at the middle forearm. Cross-sectional area (CSA) measurement and shear

wave elastography (SWE) scan of the median nerve; demonstration of the continuous boundary tracing technique used to measure nerve CSA (CSA =0.15 cm2).
(B) Two split ultrasound and shear wave elastography images at the same longitudinal level. Quantitative SWE measurement showed mean nerve stiffness was 4.2 m/s**.**

Diagnostic Performance of Tibial Nerve SWE: Accuracy, Cutoff Values, and Comparison With Other Modalities

The diagnostic performance of tibial nerve shear wave elastography (SWE) has been extensively evaluated in recent years, with multiple studies demonstrating its strong capability to differentiate between healthy individuals, diabetic patients without neuropathy, and those with clinically or electrophysiologically confirmed diabetic peripheral neuropathy (DPN). Research consistently shows that tibial nerve stiffness increases progressively with neuropathy severity, reflecting the underlying pathological changes of fibrosis, axonal degeneration, and altered intraneural microenvironment. Early work by Dikici et al. demonstrated significantly higher stiffness values in DPN patients compared to both diabetic controls and healthy subjects, establishing SWE as a sensitive discriminator of neuropathic involvement. Subsequent studies by Jiang et al. and Dong et al. confirmed these findings, reinforcing the potential role of SWE as an adjunct to conventional diagnostic tools. [54–56]

Reported cutoff values for diagnosing DPN vary somewhat across studies due to differences in ultrasound equipment, frequency, region of interest, and patient demographics. However, most research reports tibial nerve stiffness thresholds ranging from approximately 3.0 to 4.0 m/s as indicative of neuropathic involvement. Jiang et al. identified an optimal cutoff of 3.76 m/s for distinguishing patients with DPN, with excellent sensitivity and specificity, while other studies reported similar thresholds within this range. Importantly, elevated stiffness values are also reported in diabetic patients without clinical symptoms or abnormal nerve conduction studies, suggesting that SWE may detect early or subclinical neuropathic changes. This ability to identify neuropathy before conventional markers become abnormal underscores the importance of SWE as part of an early detection strategy. [45,52,55]

When compared directly to nerve conduction studies (NCS), SWE demonstrates significant advantages in certain diagnostic contexts. NCS remains the gold standard for assessing large-fiber dysfunction, yet it has inherent limitations including its inability to assess small-fiber involvement, patient discomfort, operator dependency, and reduced feasibility for large-scale population screening. SWE, by contrast, is noninvasive, rapid, and free of electrical stimulation, making it more acceptable to patients. Studies show moderate correlation between tibial nerve stiffness and electrophysiologic parameters such as conduction velocity and compound muscle action potential amplitude, yet SWE often reveals abnormalities in individuals whose NCS results remain within normal limits. This suggests that SWE provides complementary information and may detect neuropathic changes earlier than NCS in specific patient groups. [49,53,56]

In comparison with high-resolution ultrasound (HRUS), SWE offers superior diagnostic value due to its ability to quantify biomechanical properties rather than relying solely on morphologic features. While HRUS has shown utility in identifying nerve enlargement and changes in echogenicity or vascularity, structural changes tend to appear late in the course of neuropathy. SWE, however, assesses stiffness changes that reflect early microstructural alterations in the endoneurial environment, including edema, fibrosis, and increased pressure. Combining HRUS with SWE can improve diagnostic accuracy further by integrating structural and mechanical information. Several studies affirm that adding SWE to HRUS significantly enhances sensitivity and specificity in detecting early DPN compared with HRUS alone. [28,33,38]

The specificity of SWE in distinguishing DPN from other neuropathic disorders represents another important clinical consideration. Conditions such as entrapment neuropathies, inflammatory neuropathies, and hereditary neuropathies can also alter nerve stiffness. However, the pattern, location, and magnitude of stiffness changes often differ between diabetic neuropathy and non-diabetic etiologies. For instance, inflammatory neuropathies may show more diffuse stiffness alterations, while entrapment

neuropathies typically demonstrate focal stiffness increases. Although more comparative studies are needed, early evidence suggests that SWE may aid in differential diagnosis when interpreted alongside clinical context and additional imaging findings. [34,35,44]

Clinical Applications of Tibial Nerve SWE in Early Detection, Severity Grading, and Follow-Up

Shear wave elastography (SWE) has important clinical applications in the early detection of diabetic peripheral neuropathy (DPN). Because increased tibial nerve stiffness can be identified before the development of symptoms or electrophysiologic abnormalities, SWE provides an opportunity to diagnose neuropathy at a stage when interventions such as improved glycemic control, lifestyle modification, or early pharmacologic therapy may still prevent progression. Studies demonstrate that diabetic patients without clinical neuropathy but with elevated stiffness values have a higher likelihood of developing DPN, making SWE a promising screening tool for high-risk individuals in endocrinology and primary-care settings. [52,54,55]

SWE also offers value in severity grading of neuropathy. Several trials have shown a stepwise increase in tibial nerve stiffness from healthy controls to diabetics without neuropathy, to mild, moderate, and severe DPN. This gradation reflects progressive intraneural fibrosis, axonal degeneration, and changes in endoneurial pressure. By quantifying these biomechanical changes, SWE may complement existing clinical scoring systems such as vibration threshold testing or symptom questionnaires, providing objective data that can enhance staging accuracy. Incorporating SWE into comprehensive neuropathy assessments may therefore improve consistency in evaluating disease severity across different clinical settings. [45,53,56]

In follow-up assessment, SWE offers a noninvasive method to monitor neuropathy progression or treatment response. Longitudinal observations indicate that tibial nerve stiffness tends to increase over time in patients with poorly controlled diabetes, while stabilization or improvement in stiffness may occur with optimized glycemic management or risk factor modification. Although longer-term data remain limited, early evidence suggests that SWE could serve as a quantitative biomarker to track neuropathic changes over months or years. Such monitoring may support decision-making regarding treatment intensification and help identify individuals at higher risk of complications. [47,48,57]

Finally, SWE may be useful when diagnostic uncertainty exists, such as in differentiating early DPN from mechanical or inflammatory neuropathies. While overlap in stiffness values can occur, the pattern of involvement—diffuse in DPN versus focal in entrapment neuropathies—can help guide diagnosis when interpreted alongside HRUS findings and clinical context. This integrative approach strengthens diagnostic confidence and provides a more comprehensive understanding of nerve health. [34,44,58]

Conclusion

Shear wave elastography (SWE) has emerged as a valuable imaging tool for the evaluation of diabetic peripheral neuropathy (DPN), offering quantitative insight into tibial nerve biomechanics that complements traditional clinical and electrophysiologic methods. By detecting stiffness changes associated with early endoneurial fibrosis, edema, and axonal degeneration, SWE provides diagnostic information that often precedes abnormalities on nerve conduction studies or conventional ultrasound. This makes it particularly useful in identifying subclinical neuropathy, a stage at which interventions may still prevent progression to irreversible nerve damage.

The technique also shows promise for severity grading, with stiffness values increasing in parallel with clinical and electrophysiologic impairment. Its noninvasive nature, reproducibility, and compatibility with routine ultrasound workflows support its potential use in monitoring disease progression and evaluating treatment response. While current evidence is compelling, broader adoption of SWE requires standardization of measurement protocols, establishment of universal cutoff values, and validation through large multicenter longitudinal studies.

Nevertheless, SWE represents an important advancement in neuromuscular imaging. When integrated with high-resolution ultrasound and clinical examination, it offers a more comprehensive assessment of nerve health and supports earlier, more precise diagnosis of DPN. As research continues, tibial nerve SWE may become a central component of modern diabetic neuropathy evaluation, helping clinicians

identify risk earlier, guide management more effectively, and improve long-term outcomes for patients living with diabetes.

References

1. International Diabetes Federation. IDF Diabetes Atlas. 9th ed. Brussels, Belgium: International Diabetes Federation; 2019.
2. Selvarajah D, Kar D, Khunti K, et al. Diabetic peripheral neuropathy: advances in diagnosis and strategies for screening and early intervention. Lancet Diabetes Endocrinol. 2019;7(12):938-948.
3. Yu Y. Gold Standard for Diagnosis of DPN. Front Endocrinol (Lausanne). 2021;12:719356.
4. Callaghan BC, Price RS, Chen KS, et al. The importance of rare subtypes in diagnosis and treatment of peripheral neuropathy: a review. JAMA Neurol. 2015;72(12):1510-1518.
5. Elafros MA, Andersen H, Bennett DL, et al. Towards prevention of diabetic peripheral neuropathy: clinical presentation, pathogenesis, and new treatments. Lancet Neurol. 2022;21(10):922-936.
6. Reddy YM, Murthy JM, Suresh L, et al. Diagnosis and severity evaluation of ulnar neuropathy at the elbow by ultrasonography: a case-control study. J Med Ultrasound. 2022;30(3):189-195.
7. Ferraioli G. Review of liver elastography guidelines. J Ultrasound Med. 2019;38(1):9-14.
8. Dikici AS, Ustabasioglu FE, Delil S, et al. Evaluation of the tibial nerve with shear-wave elastography: a potential sonographic method for the diagnosis of diabetic peripheral neuropathy. Radiology. 2017;282(2):494-501.
9. Dong B, Lyu G, Yang X, et al. Shear wave elastography as a quantitative biomarker of diabetic peripheral neuropathy: a systematic review and meta-analysis. Front Public Health. 2022;10:915883.
10. Bodman MA, Dreyer MA, Varacallo M. Diabetic Peripheral Neuropathy. In: StatPearls [Internet]. Treasure Island, FL: StatPearls Publishing; 2024.
11. Hicks CW, Selvin E. Epidemiology of peripheral neuropathy and lower extremity disease in diabetes. Curr Diab Rep. 2019;19(10):86.
12. Pop-Busui R, Ang L, Boulton AJM, et al. Diabetic neuropathy: a position statement by the American Diabetes Association. Diabetes Care. 2017;40(1):136-154.
13. Selvarajah D, Kar D, Khunti K, et al. Diabetic peripheral neuropathy: advances in diagnosis and strategies for screening and early intervention. Lancet Diabetes Endocrinol. 2019;7:938-948.
14. Sloan G, Selvarajah D, Tesfaye S. Pathogenesis, diagnosis and clinical management of diabetic sensorimotor peripheral neuropathy. Nat Rev Endocrinol. 2021;17(7):400-420.
15. Baum P, Toyka KV, Blüher M, et al. Inflammatory mechanisms in the pathophysiology of diabetic peripheral neuropathy—new aspects. Int J Mol Sci. 2021;22(19):10325.
16. Feldman EL, Nave KA, Jensen TS, Bennett DL. New horizons in diabetic neuropathy: mechanisms, bioenergetics, and pain. Neuron. 2017;93(6):1296-1313.
17. Sloan G, Selvarajah D, Tesfaye S. Pathogenesis, diagnosis and clinical management of diabetic sensorimotor peripheral neuropathy. Nat Rev Endocrinol. 2021;17(7):400-420.
18. Elafros MA, Andersen H, Bennett DL, et al. Towards prevention of diabetic peripheral neuropathy: clinical presentation, pathogenesis, and new treatments. Lancet Neurol. 2022;21(10):922-936.
19. Albers JW, Pop-Busui R. Diabetic neuropathy: mechanisms, emerging treatments, and clinical testing. N Engl J Med. 2014;371(2):165-173.
20. Lee SS, Han HS, Kim H, et al. A review of the principles and methods of neuropathy testing in patients with diabetes mellitus. Diabetes Metab J. 2020;44(4):530-547.
21. Kakrani AL, Gokhale VS, Vohra KV, et al. Clinical and nerve conduction study correlation in patients of diabetic neuropathy. J Assoc Physicians India. 2014;62(1):24-27.
22. Hobson-Webb LD, Massey JM, Juel VC. Nerve ultrasound in diabetic polyneuropathy: correlation with clinical characteristics and electrodiagnostic testing. Muscle Nerve. 2013;47(3):379-384.
23. Chen R, Wang XL, Xue WL, et al. Application value of conventional ultrasound and real-time shear wave elastography in patients with type 2 diabetic polyneuropathy. Eur J Radiol. 2020;126:108965.

24. Bedewi MA, Elsifey AA, Alfaifi T, et al. Shear wave elastography of the tibial nerve in healthy subjects. Medicine (Baltimore). 2021;100(3):e23999.
25. Taljanovic MS, Gimber LH, Becker GW, et al. Shear-wave elastography: basic physics and musculoskeletal applications. Radiographics. 2017;37(3):855-870.
26. Sène D. Small fiber neuropathy: diagnosis, causes, and treatment. Joint Bone Spine. 2018;85(5):553-559.
27. Borire AA, Visser LH, Padua L, et al. Utility of maximum perfusion intensity as an ultrasonographic marker of intraneural blood flow. Muscle Nerve. 2017;55(1):77-83.
28. Noto YI, Shiga K, Tsuji Y, et al. Nerve ultrasound depicts peripheral nerve enlargement in patients with genetically distinct Charcot–Marie–Tooth disease. J Neurol Neurosurg Psychiatry. 2015;86(4):378-384.
29. Borire AA, Arnold R, Pussell BA, et al. Effects of hemodialysis on intraneural blood flow in end-stage kidney disease. Muscle Nerve. 2018;57(2):287-293.
30. Kyung-Min S, Seunghoon L, Eun Yong L, et al. Electroacupuncture for painful diabetic peripheral neuropathy: a randomized, assessor-blinded trial. Diabetes Care. 2018;41(5):e41-e42.
31. Çakici N, Fakkel TM, van Neck JW, et al. Systematic review of treatments for diabetic peripheral neuropathy. Diabet Med. 2016;33(11):1466-1476.
32. Wang Y, Li W, Peng W, et al. Acupuncture for postherpetic neuralgia: systematic review and meta-analysis. Medicine (Baltimore). 2018;97(34):e11986.
33. Yoshii Y, Tung WL, Ishii T. Strain and morphological changes of median nerve after carpal tunnel release. J Ultrasound Med. 2017;36(6):1153-1159.
34. Martin MJ, Cartwright MS. A pilot study of strain elastography in the diagnosis of carpal tunnel syndrome. J Clin Neurophysiol. 2017;34(2):114-118.
35. Sigrist RMS, Liau J, Kaffas AE, et al. Ultrasound elastography: review of techniques and clinical applications. Theranostics. 2017;7(5):1303-1329.
36. Wee TC, Simon NG. Ultrasound elastography for the evaluation of peripheral nerves: a systematic review. Muscle Nerve. 2019;60(5):501-512.
37. DeWall RJ. Ultrasound elastography: principles, techniques, and clinical applications. Crit Rev Biomed Eng. 2013;41(1):1-22.
38. Taljanovic MS, Gimber LH, Becker GW, et al. Shear-Wave Elastography: Basic Physics and Musculoskeletal Applications. Radiographics. 2017;37(3):855-870.
39. Gallardo E, Noto Y, Simon NG. Ultrasound in the diagnosis of peripheral neuropathy: structure meets function. J Neurol Neurosurg Psychiatry. 2015;86(10):1066-1074.
40. Jiang W, Huang S, Teng H, et al. Diagnostic performance of two-dimensional shear wave elastography for evaluating tibial nerve stiffness in patients with diabetic peripheral neuropathy. Eur Radiol. 2019;29(4):2167-2174.
41. Wei M, Ye X. Feasibility of point shear wave elastography for evaluating diabetic peripheral neuropathy. J Ultrasound Med. 2020;39(6):1135-1141.
42. Kaymaz S, Yigit M, Ufuk F, et al. Evaluation of the tibial nerve using shear-wave elastography in systemic sclerosis. Arch Rheumatol. 2023;38(4):556-565.
43. Ibrahim HR. Diagnostic value of shear wave ultrasound elastography of tibial nerve in diabetic neuropathy. Egypt J Radiol Nucl Med. 2022;53:102.
44. Barr RG, Cosgrove D, Brock M, et al. WFUMB guidelines on the clinical use of ultrasound elastography: Part 5. Prostate. Ultrasound Med Biol. 2017;43(1):27-48.
45. Dikici AS, Ustabasioglu FE, Delil S, et al. Tibial nerve SWE in DPN. Radiology. 2017;282:494-501.
46. Dong B, Lyu G, Yang X, et al. SWE as DPN biomarker. Front Public Health. 2022;10:915883.
47. Göya C, Daggulli M, Hamidi C, et al. ARFI in renal lesions. Radiol Med. 2015;120(3):296-303.
48. Dhyani M, Anvari A, Samir AE. Liver elastography. Abdom Imaging. 2015;40(4):698-708.
49. Kakrani AL, Gokhale VS, Vohra KV, et al. Clinical and NCS correlation in diabetic neuropathy. J Assoc Physicians India. 2014;62:24-27.
50. Sigrist RMS, Liau J, Kaffas AE, et al. (2017): Ultrasound elastography: review of techniques and clinical applications. Theranostics; 7(5): 1303-1329.
51. Kaymaz S et al. Tibial nerve SWE in systemic sclerosis. Arch Rheumatol. 2023;38:556-565.
52. Wei M, Ye X. SWE feasibility for DPN. J Ultrasound Med. 2020;39:1135-1141.

53. Bedewi MA et al. Tibial nerve SWE in healthy subjects. Medicine. 2021;100:e23999.
54. Dikici AS et al. SWE for DPN. Radiology. 2017;282:494-501.
55. Dong B et al. SWE meta-analysis. Front Public Health. 2022;10:915883.
56. Jiang W et al. Tibial nerve SWE accuracy. Eur Radiol. 2019;29:2167-2174.
57. Taljanovic MS et al. SWE physics. Radiographics. 2017;37:855-870.
58. Lee, S. S., Han, et al. (2020): A review of the principles and methods of neuropathy testing in patients with diabetes mellitus. Diabetes & Metabolism Journal; 44(4): 530–547
59. Gallardo E, Noto Y, Simon NG. Neuropathy US. JNNP. 2015;86:1066-1074.