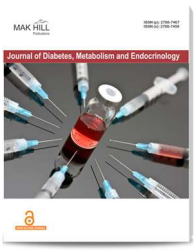

ISSN Print: 2708-2709
ISSN Online: 2708-2717

Journal of Diabetes, Metabolism and Endocrinology
https://jdmeonline.com/

Research Article

An Observational Study of Pancreatic Elasticity on the Basis of Shear Wave Velocity by Elastography in Patients of Type 2 Diabetes Mellitus and Its Relation with Diabetic Microangiopathies at SMS Medical College, Jaipur

Namra Tak[1*], Abdullah Sheikh[2], Mukesh Mittal[3] and Anu Bhandari[4]

[1]Postgraduate Junior Resident, Department of Radiodiagnosis and Modern Imaging, S.M.S. Medical College and Attached Group of Hospitals, Jaipur, Rajasthan, India

[2]Senior Resident, Department of Internal Medicine, R.U.H.S. Medical College and Attached Group of Hospitals, Jaipur, Rajasthan, India

[3]Professor, Department of Radiodiagnosis and Modern Imaging, S.M.S. Medical College and Attached Group of Hospitals, Jaipur, Rajasthan, India

[4]Senior Professor, Head of Department of Radiodiagnosis and Modern Imaging, S.M.S. Medical College and Attached Group of Hospitals, Jaipur, Rajasthan, India

***Corresponding Author**
Namra Tak

Article History
Received: 08.06.2026
Accepted: 23.07.2026
Published: 19.08.2026

doi: 10.61336/jdme.0703.04

Abstract: Background: Diabetic microangiopathy is a major cause of morbidity in type 2 diabetes mellitus (T2DM), yet the exocrine pancreas is rarely evaluated as a target of the same fibrotic and microvascular processes. Shear Wave Elastography (SWE) enables non-invasive quantification of pancreatic stiffness through Shear Wave Velocity (SWV), a validated surrogate of tissue fibrosis. **Objective:** To assess pancreatic elasticity on the basis of SWV in patients with T2DM and to determine its relationship with the presence and burden of diabetic microangiopathy. **Methods:** In this hospital-based cross-sectional study, 150 patients with T2DM were enrolled and divided into two groups of 75 each: those with microangiopathy (retinopathy, nephropathy and/or neuropathy) and those without. All patients underwent conventional B-mode ultrasonography and pancreatic SWE using a 2–5 MHz convex transducer. SWV (m/s) and elasticity modulus (kPa) were measured at the head, body and tail. Group comparisons, correlation analysis, logistic regression and receiver operating characteristic (ROC) analysis were performed; $p<0.05$ was considered significant. **Results:** Mean pancreatic SWV (1.9±0.3 vs. 1.3±0.2 m/s) and elasticity modulus (10.9±3.2 vs. 5.2±1.7 kPa) were significantly higher in the microangiopathy group ($p<0.001$). Patients with microangiopathy were older, had longer disease duration, poorer glycaemic control, shorter pancreatic length, wider main pancreatic duct and more coarse echotexture (all $p<0.001$). Pancreatic body SWV correlated most strongly with the number of microangiopathic complications ($r = 0.72$) and inversely with fasting C-peptide ($r = −0.46$). Age, duration of T2DM and HbA1c were independent predictors of microangiopathy. ROC analysis showed excellent discrimination, with AUCs of 0.947 (head), 0.941 (body) and 0.934 (tail) at cut-offs of ≥1.63–1.66 m/s. **Conclusion:** Pancreatic stiffness measured by SWE is significantly increased in T2DM patients with microangiopathy and tracks with complication burden, disease duration and glycaemic control. Pancreatic SWV is a promising non-invasive imaging biomarker for identifying diabetic microvascular disease.

Keywords: Type 2 Diabetes Mellitus; Shear Wave Elastography; Shear Wave Velocity; Pancreatic Stiffness; Diabetic Microangiopathy; Acoustic Radiation Force Impulse

INTRODUCTION

Diabetes Mellitus (DM) is a chronic metabolic disorder characterised by persistent hyperglycaemia resulting from defects in insulin secretion, insulin action, or both [1]. It is a global health problem and its incidence has risen steadily over recent decades [2]. The International Diabetes Federation estimates that nearly 500 million people currently live with diabetes worldwide, a figure projected to rise by approximately 30% by 2045 and diabetes accounts for close to 10% of global all-cause mortality among adults aged 20–99 years [3]. India carries the second-largest diabetic population globally, with an estimated 77 million affected individuals and an adult prevalence of about 8.9% [4,5].

The classical microvascular complications of diabetes retinopathy, nephropathy and neuropathy together contribute substantially to diabetes-related morbidity, disability and mortality. Although diabetic microangiopathy has been extensively characterized in the retina, kidney and peripheral nerves, the corresponding structural and mechanical changes in the pancreas remain poorly understood, largely because pancreatic biopsy is invasive and ethically challenging [6]. Increasing evidence suggests that diabetes may itself be regarded as a form of chronic pancreatic injury, with progressive impairment of both endocrine and exocrine function [7–9].

Data In T2DM, sustained metabolic derangement injures both islet cells and exocrine acinar tissue, producing pancreatic fibrosis, increased tissue stiffness and gradual loss of acinar-parenchymal architecture [10].

These changes are thought to contribute not only to worsening glycaemic control but also to the development of microvascular complications. Recent imaging advances allow non-invasive assessment of these tissue characteristics. Acoustic Radiation Force Impulse (ARFI) and shear wave elastography exploit the principle that tissue elasticity is directly related to the square of the Shear Wave Velocity (SWV); higher SWV reflects greater stiffness and fibrosis [11,12]. Histological studies have confirmed a significant positive correlation between SWV and the severity of pancreatic fibrosis, validating elastography as a reliable surrogate of pancreatic structural change [13,14].

The mechanistic basis for pancreatic stiffening in diabetes is multifactorial. Chronic hyperglycaemia promotes the formation of advanced glycation end-products, oxidative stress and microvascular ischaemia, which together activate pancreatic stellate cells the principal effectors of pancreatic fibrogenesis.15,16 Activated stellate cells deposit collagen and extracellular matrix, progressively replacing acinar parenchyma with fibro-fatty tissue and increasing tissue stiffness. In parallel, the well-described beta-cell deficit and increased beta-cell apoptosis of T2DM reduce endocrine reserve, so that measures of pancreatic stiffness may indirectly reflect the loss of functioning islet mass [17,18]. Because the same microvascular and pro-fibrotic pathways drive retinopathy, nephropathy and neuropathy, it is biologically plausible that pancreatic stiffness rises in parallel with the accrual of microangiopathic complications [16,18].

Preliminary observations indicate that pancreatic SWV is higher in patients with diabetes than in healthy individuals, suggesting that alterations in pancreatic mechanical properties may parallel disease progression and its complications [19]. We therefore hypothesised that pancreatic stiffness is associated with the presence and severity of diabetic microangiopathy. The present study was designed to evaluate pancreatic morphology and stiffness, using conventional ultrasonography and shear wave elastography, in T2DM patients with and without microangiopathy and to correlate these imaging parameters with relevant demographic, clinical and biochemical variables.

MATERIALS AND METHODS

Study Design and Setting

This was an observational, hospital-based cross-sectional study conducted in the Department of Radiodiagnosis, Sawai Man Singh Medical College and attached hospitals, Jaipur, Rajasthan. The study was initiated after approval from the Institutional Research Review Board and Institutional Ethics Committee and written informed consent was obtained from every participant. Data collection continued until the desired sample size was achieved.

Study Population and Grouping

A total of 150 adult patients (aged>18 years) with an established clinical diagnosis of type 2 diabetes mellitus were enrolled by consecutive sampling. They were divided into two equal groups: Group I comprised 75 patients with T2DM and diabetic microangiopathy and Group II comprised 75 patients with T2DM without microangiopathy. Diabetic microangiopathy was defined as the presence of one or more diabetes-related microvascular complications diabetic retinopathy, nephropathy and/or neuropathy documented from clinical records and relevant investigations.

Eligibility Criteria

Patients aged over 18 years, of either sex, with T2DM (with or without microangiopathy) who provided written informed consent were included. Patients were excluded if they had chronic liver disease, significant renal disease, cardiac disease or pancreatitis; pancreatic masses or prior pancreatic surgery; a history of significant alcohol abuse; sonographic features suggestive of chronic pancreatitis (dilated or beaded main duct, intraductal calculi, parenchymal calcification or pseudocyst); or if they were unable to undergo an adequate ultrasonographic or elastographic examination.

Sample Size

The sample size was calculated at a 95% confidence level and 80% power, with an alpha error of 0.05, based on an expected area under the ROC curve of 0.747 for pancreatic body SWV and an expected microangiopathy proportion of 42.72% from a reference study. The minimum required size was 140 (70 per group); this was rounded up to 150 (75 per group) to compensate for incomplete data and technically inadequate examinations and to improve statistical power.

Equipment and Imaging Technique

All examinations were performed using an ultrasound scanner equipped with a 2–5 MHz convex transducer and shear wave elastography capability (Hitachi Aplio-500 or equivalent). With the patient supine and the epigastrium exposed, the transducer was placed below the xiphoid process; deep inspiration and breath-holding were used to optimise the acoustic window and minimise motion artefact. Grey-scaleultrasonography was first performed in transverse and longitudinal planes to assess pancreatic size, contour, echogenicity and to measure the thickness of the head, body and tail, together with the main pancreatic duct diameter.

Shear wave elastography was then performed without excessive transducer pressure. A region of interest measuring approximately 10 × 5 mm was placed within homogeneous parenchyma of the head, body and tail, avoiding vessels, ducts, focal lesions, calcifications and bowel gas. Up to ten SWV measurements were attempted from each region during breath-holding, with a technical success rate of at least 60% considered acceptable. The median eight technically adequate values from each region were used to calculate the mean SWV (m/s), from which the elasticity modulus (kPa) was derived.

RESULTS

Baseline Demographic, Clinical and Biochemical Profile

Patients with microangiopathy were significantly older than those without (mean 58.1±8.8 vs. 48.3±9.0 years; p<0.001) and had a markedly longer duration of diabetes (10.5±3.6 vs. 5.6±2.8 years; p<0.001). Glycaemic control was significantly poorer in the microangiopathy group, with higher HbA1c (8.6±1.2% vs. 7.7±1.1%) and fasting blood glucose (198.3±24.1 vs. 182.2±20.9 mg/dL; p<0.001 for both). Gender distribution (p = 0.624) and body mass index (26.3±2.5 vs. 26.5±3.3 kg/m^2; p = 0.67) were comparable between groups (Table 1).

Pancreatic Morphology and Stiffness

B-mode ultrasonography showed a significantly shorter pancreatic length (129.3±14.2 vs. 146.1±12.0 mm) and a wider main pancreatic duct (1.5±0.3 vs. 1.2±0.2 mm) in the microangiopathy group (p<0.001 for both). A coarse or heterogeneous echotexture was present in 69.3% of patients with microangiopathy versus 20.0% of those without (p<0.001).

Table 1: Baseline Demographic, Clinical and Biochemical Characteristics of the Two Groups

Parameter	Microangiopathy (n = 75)	No microangiopathy (n = 75)	p-value
Age (years)	58.1±8.8	48.3±9.0	< 0.001
Female / Male (%)	54.7 / 45.3	49.3 / 50.7	0.624
BMI (kg/m^2)	26.3±2.5	26.5±3.3	0.67
HbA1c (%)	8.6±1.2	7.7±1.1	< 0.001
Fasting blood glucose (mg/dL)	198.3±24.1	182.2±20.9	< 0.001
Duration of T2DM (years)	10.5±3.6	5.6±2.8	< 0.001

Values are mean±SD unless otherwise stated. T2DM, type 2 diabetes mellitus; BMI, body mass index

Table 2: Comparison of B-Mode Ultrasonographic and Shear Wave Elastography Parameters

Parameter	Microangiopathy	No microangiopathy	p-value
Pancreatic length (mm)	129.3±14.2	146.1±12.0	< 0.001
Main pancreatic duct (mm)	1.5±0.3	1.2±0.2	< 0.001
Coarse/heterogeneous echotexture (%)	69.3	20.0	< 0.001
Mean shear wave velocity (m/s)	1.9±0.3	1.3±0.2	< 0.001
Elasticity modulus (kPa)	10.9±3.2	5.2±1.7	< 0.001

SWV was measured separately at the pancreatic head, body and tail; all regions showed the same direction and significance of difference

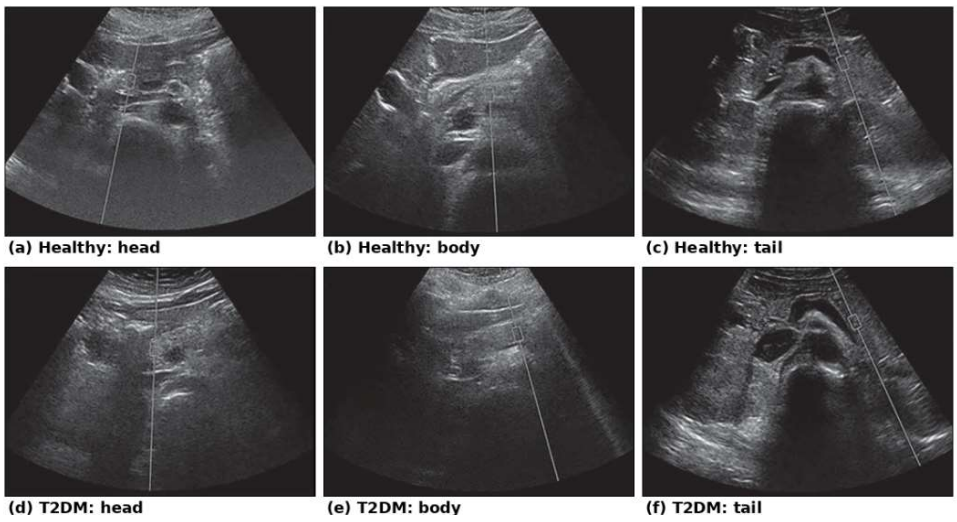

Figure 1: Representative Pancreatic Shear-Wave Elastography (ARFI) Acquisitions. Top Row: Healthy Pancreas at the (a) Head, (b) Body and (c) Tail, With Low Shear-Wave Velocity. Bottom Row: Pancreas in type 2 Diabetes Mellitus at the (d) Head, (e) Body and (f) Tail, Showing Higher Shear-Wave Velocity Consistent with Increased Parenchymal Stiffness. Rectangular Regions of Interest Were Placed within Homogeneous Parenchyma, Avoiding Vessels, Ducts and Artefacts

Table 3: Univariate and Multivariate Logistic Regression for Factors Associated with Diabetic Microangiopathy

Variable	Univariate OR (95% CI)	p	Adjusted OR (95% CI)	p
Age (per year)	1.13 (1.08–1.19)	<0.001	1.29 (1.15–1.44)	<0.001
T2DM duration (per year)	1.58 (1.37–1.82)	<0.001	2.03 (1.57–2.63)	<0.001
HbA1c (per 1%)	1.96 (1.43–2.70)	<0.001	3.59 (1.78–7.25)	<0.001
BMI (per kg/m^2)	0.98 (0.87–1.09)	0.668	1.08 (0.88–1.32)	0.462
Male sex (vs. female)	0.81 (0.43–1.53)	0.513	2.04 (0.58–7.17)	0.267

OR, odds ratio; CI, confidence interval

Table 4: Correlation of Pancreatic Body SWV and Thickness with Clinical and Biochemical Variables (correlation coefficient, r)

Variable	SWV – body (r)	Thickness – body (r)
Number of microangiopathic complications	+0.72	−0.63
Systolic blood pressure	+0.46	−(moderate)
Fasting C-peptide	−0.46	+0.42
Duration of diabetes	+0.42	−0.45
Microalbuminuria	+0.37	−(moderate)
Age	+(sig.)	−0.35

All listed correlations were statistically significant (p<0.05). BMI, lipids and urea showed no significant correlation

Table 5: ROC-Derived Cut-off Values of Pancreatic SWV for Diagnosing Diabetic Microangiopathy

Region	Cut-off (m/s)	Sensitivity (%)	Specificity (%)	AUC
Head	≥ 1.63	82.7	94.7	0.947
Body	≥ 1.63	82.7	93.3	0.941
Tail	≥ 1.66	80.0	96.0	0.934

AUC, area under the receiver operating characteristic curve (95% CI: head 0.909–0.984; body 0.902–0.980; tail 0.892–0.975)

Shear wave elastography demonstrated markedly increased stiffness in the microangiopathy group: mean SWV was 1.9±0.3 vs. 1.3±0.2 m/s and the elasticity modulus was 10.9±3.2 vs. 5.2±1.7 kPa (p<0.001 for all comparisons, at head, body and tail) (Table 2, Figure 1).

Predictors of Diabetic Microangiopathy

On univariate logistic regression, increasing age (OR = 1.13; 95% CI 1.08–1.19), longer duration of T2DM (OR = 1.58; 95% CI 1.37–1.82) and higher HbA1c (OR = 1.96; 95% CI 1.43–2.70) were each significantly associated with microangiopathy (p<0.001), whereas BMI (OR = 0.98; p = 0.668) and male sex (OR = 0.81; p = 0.513) were not. These three factors remained independent predictors after multivariable adjustment (Table 3).

Correlates of Pancreatic Body Stiffness

Pancreatic body SWV correlated most strongly with the number of microangiopathic complications (r = 0.72; p<0.001), followed by systolic blood pressure (r = 0.46), an inverse relationship with fasting C-peptide (r = −0.46), duration of diabetes (r = 0.42) and microalbuminuria (r = 0.37). Pancreatic body thickness showed the reciprocal, predominantly inverse pattern most strongly with the number of complications (r = −0.63) and correlated positively with fasting C-peptide (r = 0.42). The elasticity modulus correlated with age (r = 0.27) and disease duration (r = 0.43). No significant correlation was found with BMI, triglycerides, total or HDL cholesterol, or serum urea (Table 4).

Diagnostic Performance of Pancreatic SWV

ROC analysis showed excellent discrimination of microangiopathy by pancreatic SWV in all regions. The pancreatic head yielded an optimal cut-off of ≥1.63 m/s (sensitivity 82.7%, specificity 94.7%, AUC 0.947), the body ≥1.63 m/s (82.7%, 93.3%, AUC 0.941) and the tail ≥1.66 m/s (80.0%, 96.0%, AUC 0.934) (Table 5).

DISCUSSION

The central finding of this study is that pancreatic stiffness, quantified by shear wave elastography, is significantly higher in T2DM patients with microangiopathy and tracks closely with the number of microvascular complications, disease duration and glycaemic control. This positions the exocrine pancreas as a target organ of the same fibrotic and microvascular processes that damage the retina, kidney and nerves and is concordant with the growing literature on pancreatic elastography in diabetes [19,20].

Patients with microangiopathy were significantly older and age was independently predictive of complications and correlated with the elasticity modulus (r = 0.27). A positive age–stiffness relationship has been reported both in diabetic cohorts, where advancing age was independently associated with higher pancreatic body SWV [20] and in non-diabetic populations [21,22]. The age effect therefore reflects a genuine, reproducible relationship between aging and pancreatic

tissue stiffening rather than a diabetes-specific artefact. Gender was not associated with microangiopathy, consistent with reports that pancreatic SWE values are independent of sex [21].

Body mass index was comparable between groups and did not predict microangiopathy or correlate with SWV. This contrasts with reports linking pancreatic steatosis and metabolic syndrome to increased stiffness [22,23] and suggests that in a cohort with a comparably overweight distribution, glycaemic burden rather than adiposity dominates as the driver of pancreatic stiffening. Both HbA1c and fasting glucose were significantly higher in the microangiopathy group and HbA1c was the strongest metabolic predictor (adjusted OR = 3.59), mechanistically coherent with the established role of chronic hyperglycaemia in driving both microvascular injury and pancreatic fibrosis.

Disease duration was markedly longer in the microangiopathy group, showed strong predictive strength (adjusted OR = 2.03) and correlated with both the elasticity modulus (r = 0.43) and body SWV (r = 0.42). This dose-dependent relationship between exposure time and stiffness supports progressive fibro-fatty replacement of pancreatic parenchyma with cumulative disease duration. Correspondingly, B-mode imaging showed pancreatic atrophy, ductal dilatation and a coarse echotexture in the microangiopathy group recognised structural correlates of long-standing diabetes although the mechanical (SWE) parameters discriminated more powerfully than size alone [19].

Our absolute SWV values (1.9 vs. 1.3 m/s) and elasticity moduli (10.9 vs. 5.2 kPa) fall within the range described for the stiffened, diseased pancreas across previous elastography series.20,24 Pancreatic body SWV correlated most strongly with the number of microangiopathic complications (r = 0.72) and inversely with fasting C-peptide (r = −0.46), linking greater stiffness to poorer beta-cell reserve, while the microalbuminuria association echoes prior work identifying microalbuminuria among independent predictors of complications [20]. The absence of correlation with BMI, lipids and urea further localizes the stiffening process to glycaemic-, duration- and complication-related pathways rather than generalized dyslipidaemia.

SWV showed excellent discrimination for microangiopathy across all pancreatic regions (AUC 0.934–0.947) at cut-offs of ≥1.63–1.66 m/s. These thresholds are consistent in magnitude with historical stiffness cut-offs derived for chronic pancreatitis and for postoperative fistula risk [12,13], reinforcing that SWV in this range reflects clinically meaningful fibrosis. The plausibility of SWV as a fibrosis surrogate is grounded in histological validation: SWV correlates significantly with the histological grade of pancreatic fibrosis and with collagen fibre content [13,14]. Together these findings support the interpretation that the elevated SWV in our microangiopathy group reflects accumulating pancreatic fibrosis driven by chronic hyperglycaemia and microvascular ischaemia.

Clinical Implications

These findings have several practical implications. First, because pancreatic SWE can be appended to a routine abdominal ultrasound without additional preparation, contrast or radiation, it offers a low-cost, repeatable and widely deployable adjunct for risk-stratifying patients with T2DM particularly relevant in resource-limited, high-burden settings such as India. Second, the strong, graded relationship between pancreatic body SWV and the number of microangiopathic complications (r = 0.72) suggests that stiffness could serve as an integrated marker of cumulative microvascular injury, complementing organ-specific screening for retinopathy, nephropathy and neuropathy. Third, the inverse relationship with fasting C-peptide links greater stiffness to diminished beta-cell reserve, raising the possibility that SWV might help identify patients drifting toward insulin dependence, consistent with recent work combining elastography with C-peptide indices [25]. Prospective, longitudinal and multicentre studies with histological or functional correlation are now needed to determine whether serial pancreatic SWV can predict incident complications and to establish validated, vendor-independent cut-offs for clinical use.

Limitations

This was a single-centre, cross-sectional study, so causality and temporal sequence cannot be established. Microangiopathy was defined from available clinical records and investigations, which may vary in sensitivity across complication types. Elastography is operator-dependent and influenced by depth, transducer pressure and the acoustic window; although a strict protocol and technical-success threshold were applied, residual measurement variability is possible. Finally, the absence of histological confirmation means that the fibrotic basis of increased stiffness is inferred from prior validation studies rather than directly demonstrated in this cohort.

CONCLUSION

Pancreatic stiffness assessed by shear wave elastography is significantly higher in patients with type 2 diabetes mellitus and microangiopathy than in those without, with a higher mean SWV (1.9±0.3 vs. 1.3±0.2 m/s) and elasticity modulus (10.9±3.2 vs. 5.2±1.7 kPa). Increased stiffness is accompanied by pancreatic atrophy, ductal dilatation and coarse echotexture and tracks with the burden of microvascular complications, disease duration and glycaemic control. With AUCs of 0.934–0.947 at cut-offs of ≥1.63–1.66 m/s, pancreatic SWV offers excellent diagnostic performance for identifying microangiopathy. Combining conventional ultrasonography with shear wave elastography thus provides a

promising, non-invasive means of assessing diabetes-related structural and mechanical changes in the pancreas and pancreatic SWV may serve as a useful imaging biomarker of diabetic microvascular disease.

Declarations

- **Ethical Approval:** The study was approved by the Institutional Research Review Board and Institutional Ethics Committee, SMS Medical College, Jaipur. Written informed consent was obtained from all participants
- **Conflicts of Interest:** The author declares no conflict of interest
- **Funding:** This research received no specific grant from any funding agency

REFERENCES

1. American Diabetes Association. "Diagnosis and classification of diabetes mellitus." *Diabetes Care*, vol. 32, suppl. 1, 2009, pp. S62–S67.
2. World Health Organization. *Diabetes Mellitus*. World Health Organization, 2010.
3. Cho, N.H. *et al*. "IDF diabetes atlas: Global estimates of diabetes prevalence for 2017 and projections for 2045." *Diabetes Research and Clinical Practice*, vol. 138, 2018, pp. 271–281.
4. International Diabetes Federation. *IDF Diabetes Atlas*. 9th ed., International Diabetes Federation, 2019.
5. Govindaswamy, S. *et al*. "Prevalence and complications of diabetes mellitus in India: A systematic review." *Indian Journal of Pharmaceutical Education and Research*, vol. 57, no. 2 Suppl., 2023, pp. S213–S225.
6. Saisho, Y. "Pancreas volume and fat deposition in diabetes and normal physiology: Consideration of the interplay between endocrine and exocrine pancreas." *Reviews in Diabetic Studies*, vol. 13, nos. 2–3, 2016, pp. 132–147.
7. Larger, E. *et al*. "Pancreatic exocrine function in patients with diabetes." *Diabetic Medicine*, vol. 29, no. 8, 2012, pp. 1047–1054.
8. Czako, L. *et al*. "Interactions between the endocrine and exocrine pancreas and their clinical relevance." *Pancreatology*, vol. 9, no. 4, 2009, pp. 351–359.
9. Hayden, M.R. *et al*. "Attenuation of endocrine–exocrine pancreatic communication in type 2 diabetes: pancreatic extracellular matrix ultrastructural abnormalities." *Journal of Cardiometabolic Syndrome*, vol. 3, no. 4, 2008, pp. 234–243.
10. Sakata, N. *et al*. "Pancreatic fibrosis and acinar cell atrophy in patients with type 2 diabetes mellitus." *Pancreas*, vol. 35, no. 4, 2007, pp. 369–376.
11. Kawada, N. and S. Tanaka. "Elastography for the pancreas: current status and future perspective." *World Journal of Gastroenterology*, vol. 22, no. 14, 2016, pp. 3712–3724.
12. Yashima, Y. *et al*. "Acoustic radiation force impulse elastography for noninvasive assessment of chronic pancreatitis." *Journal of Gastroenterology*, vol. 47, no. 4, 2012, pp. 427–432.
13. Harada, N. *et al*. "Acoustic radiation force impulse imaging of the pancreas for estimation of pathologic fibrosis and risk of postoperative pancreatic fistula." *Journal of the American College of Surgeons*, vol. 219, no. 5, 2014, pp. 887–894.e5.
14. Fujita, Y. *et al*. "Evaluation of pancreatic fibrosis with acoustic radiation force impulse imaging and automated quantification of pancreatic tissue components." *Pancreas*, vol. 47, no. 10, 2018, pp. 1277–1282.
15. Apte, M.V. *et al*. "Pancreatic stellate cells are activated by proinflammatory cytokines." *Gut*, vol. 44, no. 4, 1999, pp. 534–541.
16. Brownlee, M. "The pathobiology of diabetic complications: A unifying mechanism." *Diabetes*, vol. 54, no. 6, 2005, pp. 1615–1625.
17. Butler, A.E. *et al*. "Beta-cell deficit and increased beta-cell apoptosis in humans with type 2 diabetes." *Diabetes*, vol. 52, no. 1, 2003, pp. 102–110.
18. Forbes, J.M. and M.E. Cooper. "Mechanisms of diabetic complications." *Physiological Reviews*, vol. 93, no. 1, 2013, pp. 137–188.
19. He, Y. *et al*. "Pancreatic elastography from acoustic radiation force impulse imaging for evaluation of diabetic microangiopathy." *AJR American Journal of Roentgenology*, vol. 209, no. 4, 2017, pp. 775–780.
20. Yu, H. *et al*. "Pancreatic elastography from acoustic radiation force impulse imaging for evaluation of diabetic microangiopathy." *AJR American Journal of Roentgenology*, vol. 209, no. 4, 2017, pp. W244–W252.
21. Choudhury, S.R. *et al*. "Ultrasound shear wave elastography of normal pancreas in adult subjects." *Journal of Gastrointestinal and Abdominal Radiology,* vol. 6, no. 2, 2023, pp. 109–115.
22. Suzuki, H. *et al*. "What is the role of measuring shear wave dispersion using shear wave elastography in pancreatic parenchyma?" *Journal of Medical Ultrasonics*, vol. 47, no. 4, 2020, pp. 575–581.
23. Sezgin, O. *et al*. "Pancreatic steatosis is associated with both metabolic syndrome and pancreatic stiffness detected by ultrasound elastography." *Digestive Diseases and Sciences*, vol. 67, no. 1, 2022, pp. 293–304.
24. Sivaraju, L.S. *et al*. "Pancreatic 2D SWE: A potential tool for non-invasive evaluation of diabetes and diabetic microangiopathy?" *Journal of Ultrasound*, vol. 28, no. 4, 2025, pp. 959–967.
25. Sugita-Hamada, M. *et al*. "Simultaneous evaluation of shear wave elastography and c-peptide index for predicting need of insulin therapy in type 2 diabetes: A pilot study." *Journal of Personalized Medicine,* vol. 15, no. 7, 2025, pp. 277.