ORIGINAL ARTICLE

Sonographic Evaluation of Pancreatic Morphology in Patients with Diabetes Mellitus

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ABSTRACT

Introduction: Diabetes mellitus is a chronic metabolic disorder linked to pancreatic structural changes detectable by imaging. This study evaluates pancreatic size and echogenicity in Type 1 and Type 2 diabetes mellitus compared to healthy controls, emphasizing sonography's utility in resource-limited settings where advanced imaging is scarce. **Aims:** To assess pancreatic morphological changes via ultrasound in Type 1 and Type 2 diabetes patients, correlating findings with clinical parameters such as disease duration, HbA1c, and body mass index. **Methods:** A prospective cross-sectional study included 300 subjects: 100 Type 1, 100 Type 2 diabetes mellitus and 100 age- and sex-matched healthy controls. Standardized fasting transabdominal ultrasound measured anteroposterior diameters of the pancreatic head, body, and tail were taken. Echogenicity was graded (0–3) relative to liver echogenicity. Clinical data disease duration, HbA1c, and body mass index were collected. Statistical analyses compared groups and assessed correlations. **Results:** Type 1 diabetes mellitus patients exhibited marked pancreatic atrophy across all segments, with the most severe reduction in the tail. Type 2 diabetes patients showed mild reduction in pancreatic head diameter but relatively preserved body and tail dimensions. Pancreatic size inversely correlated with disease duration in Type 1 ($r=-0.45$, $p<0.001$) and Type 2 Diabetes ($r=-0.28$, $p<0.001$). Increased echogenicity was observed in 68% of Type 1 and 72% of Type 2 patients, versus 12% of controls. In Type 2 patients, echogenicity positively correlated with body mass index ($r=0.34$, $p<0.001$). **Conclusion:** Sonography is a valuable, non-invasive, and cost-effective tool for assessing pancreatic changes in diabetes. Type 1 diabetes mellitus is associated with marked pancreatic atrophy correlating with disease duration, while Type 2 diabetes mellitus demonstrates primarily increased echogenicity related to fatty infiltration and body mass index, with only mild size reduction. These findings highlight sonography's potential as a primary diagnostic and monitoring tool in resource-limited settings.

Keywords: Body mass index, Diabetes, Magnetic resonance imaging, Pancreas, Ultrasonography

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INTRODUCTION

Diabetes mellitus (DM) is a global pandemic, with prevalence rising sharply, especially in low- and middle-income countries.[1] Characterized by chronic hyperglycemia due to impaired insulin secretion or action, DM's systemic complications are well-documented, but morphological changes in the pancreas remain understudied.[2] Advanced imaging like CT and MRI has revealed reduced pancreatic volume in Type 1 diabetes, while Type 2 diabetes shows increased pancreatic fat content with variable size changes.[3–5] However, these modalities are limited by cost, radiation exposure, and availability, particularly in resource-constrained regions. In South Asia, including Nepal, the diabetes burden is escalating, yet access to advanced diagnostic tools is restricted.[6] Transabdominal ultrasonography offers a practical solution as a non-invasive, radiation-free, and widely accessible tool that can reliably assess pancreatic dimensions and echotexture when standardized protocols are followed. [7,8]

While prior studies suggest reduced pancreatic size in diabetes, comprehensive evaluations differentiating Type 1 (T1DM) and Type 2 diabetes (T2DM) and correlating findings with clinical parameters are lacking. This study evaluates the utility of sonography in assessing pancreatic morphological changes in T1DM and T2DM patients compared to healthy controls. We correlate sonographic findings with disease duration, glycemic control (HbA1c), and body mass index (BMI), hypothesizing distinct morphological patterns between diabetes types.

METHODS

This prospective, cross-sectional study was conducted from September 2023 to August 2024 at Nepalgunj Medical College and Teaching Hospital, Nepal. The study population included 300 subjects, evenly divided into three groups: 100 patients with T1DM, 100 patients with T2DM, and 100 age- and sex-matched healthy controls (HC). Ethical approval for the study was obtained from the Institutional Review Committee (IRC) of Nepalgunj Medical College and Teaching Hospital, with the approval number 05/080-081. Written informed consent was obtained from all participants prior to their enrollment in the study.

Participants with T1DM and T2DM were diagnosed according to the criteria established by the American Diabetes Association (ADA). Healthy controls were selected based on normal fasting glucose levels and the absence of any known chronic diseases. Exclusion criteria for all groups included a history of acute or chronic pancreatitis, pancreatic surgery, pancreatic malignancy, cystic fibrosis, alcoholism, or any condition known to affect pancreatic morphology. A consecutive sampling technique was employed to recruit participants from the outpatient and inpatient departments of Nepalgunj Medical College and Teaching Hospital. Healthy controls were selected from the general population and matched for age and sex to the diabetic groups. Sample size was estimated for the primary outcome, the anteroposterior (AP) diameter of the pancreatic head, using pilot standard deviations (T1DM = 2.9 mm, T2DM = 1.7 mm, controls = 3.8 mm). For a two-sided two-sample t-test to detect a clinically meaningful difference of 3.0 mm with an alpha of 0.05 and 80% power, the minimum required per-group sample sizes were approximately 20 for T1DM versus controls, 16 for T2DM versus controls, and 10 for T1DM versus T2DM. To allow for multiple comparisons, subgroup analyses, and potential missing data, we enrolled 100 participants per group, providing over 99% power to detect the prespecified 3.0 mm difference given the pilot variability.

For all subjects, demographic data (age, sex), anthropometric measurements (height, weight, and body mass index [BMI]), and clinical parameters, including fasting plasma glucose (FPG), glycosylated hemoglobin (HbA1c), and disease duration (for the diabetic groups), were collected.

All ultrasound examinations were performed by a single experienced radiologist using a General Electronics P6 ultrasound machine with a 3.5 MHz curvilinear transducer. To ensure standardization and minimize operator dependence, a consistent protocol was followed. All subjects were required to fast for at least 8 hours to minimize bowel gas interference. Patients were scanned in the supine position and, if necessary, in a right posterior oblique position to optimize visualization of the pancreas. The pancreas was visualized in both transverse and longitudinal planes, and the AP diameters of the pancreatic head, body, and tail were measured at standardized anatomical landmarks. The head was measured at the level of the gastroduodenal artery, the body at the level of the superior mesenteric artery, and the tail just anterior to the splenic vein.

Pancreatic echogenicity was qualitatively assessed and graded on a scale of 0 to 3 relative to liver echogenicity. Grade 0 indicated echogenicity isoechoic to the liver, Grade 1 indicated mildly hyperechoic compared to the liver, Grade 2 indicated moderately hyperechoic compared to the liver, and Grade 3 indicated markedly hyperechoic echogenicity, similar to retroperitoneal fat. The radiologist was blinded to the subjects' clinical status (T1DM, T2DM, or HC) to minimize bias.

STATISTICAL ANALYSIS

Descriptive statistics were used to summarize the data. Group comparisons were performed using one-way analysis of variance (ANOVA) with post-hoc Tukey's Honestly Significant Difference (HSD) test for continuous variables and the Chi-square test for categorical variables. Pearson's correlation coefficient was used to assess the relationship between pancreatic dimensions, echogenicity, and clinical parameters. A p-value of <0.05 was considered statistically significant. All analyses were performed using SPSS version 25.0.

RESULTS

The clinical characteristics of the study participants are summarized in Table I. The mean age was comparable across all three groups. The T2DM group had a significantly higher mean BMI than the T1DM and HC groups. As expected, HbA1c and FPG levels were significantly higher in both diabetic groups compared to the healthy control group.

Variable	T1DM Group (n=100)	T2DM Group (n=100)	Control Group (n=100)	P-value
Age (years, mean ± SD)	45.2 ± 11.5	52.1 ± 10.8	48.8±12.6	-
Male Gender (n, %)	52 (52.0%)	55 (55.0%)	47 (47.0%)	-
Diabetes Duration (years, mean ± SD)	14.2 ± 6.1	9.8 ± 4.5	N/A	<0.001
HbA1c (%, mean ± SD)	8.1 ± 1.5	7.5 ± 1.2	5.4 ± 0.5	<0.001
BMI (kg/m2, mean ± SD)	25.1 ± 3.8	31.4 ± 5.2	24.8 ± 3.5	<0.001

Table I: Study Population Characteristics and Demographics

The sonographic measurements of pancreatic AP diameters are presented in Table II. The T1DM group showed marked atrophy with significantly smaller mean AP diameters across all three pancreatic segments compared to controls ($p < 0.001$ for all). The T2DM group showed significant reduction only in the pancreatic head diameter ($p < 0.001$), while the body and tail dimensions were relatively preserved with only mild, non-significant reductions compared to controls. Significant inverse correlation was found between pancreatic size and disease duration. Specifically, the combined pancreatic diameter (head + body + tail) was negatively correlated with the duration of illness in both T1DM ($r=-0.45$, $p<0.001$) and T2DM ($r=-0.28$, $p<0.001$) groups.

Pancreas part	T1DM (Mean AP Diameter +/- SD in mm)	T2DM (Mean AP Diameter +/- SD in mm)	Controls (Mean AP Diameter +/- SD in mm)	P-value (T1DM vs Controls)	P-value (T2DM vs Controls)	P-value (T1DM vs T2 DM)
Head	17.8 ± 2.9	21.13 ± 1.68	23.31 ± 3.80	<0.001	<0.001	<0.001
Body	10.93 ± 2.0	20.17 ± 1.16	22.05 ± 2.06	<0.001	0.08	<0.001
Tail	11.2 ± 2.5	20.44 ± 1.17	23.31 ± 2.49	<0.001	0.12	<0.001

Table II: Pancreatic Morphometric Dimensions in Diabetes Mellitus vs. Controls

Pancreatic echogenicity was markedly different among the groups. Increased echogenicity (grades 1, 2, or 3) was observed in a large proportion of diabetic patients (68% in T1DM and 72% in T2DM), while only 12% of healthy controls exhibited this finding. In T2DM patients, there was a significant positive correlation between increased pancreatic echogenicity and BMI ($r=0.34$, $p<0.001$). This correlation was not observed in the T1DM group. No statistically significant correlation was observed between echogenicity and HbA1c in either diabetic group.

DISCUSSION

This study provides compelling evidence of significant pancreatic morphological changes in patients with both T1DM and T2DM compared to healthy controls, as detected by sonography. Conducted in a resource-limited setting like Nepal, our findings underscore the clinical utility of ultrasonography in environments where advanced imaging modalities are not widely available.[1] This research reinforces the value of radiology in understanding and monitoring pancreatic pathologies and provides a practical, real-world framework for using ultrasound to assess pancreatic health.

Our study revealed distinct morphological patterns between T1DM and T2DM. T1DM patients exhibited marked pancreatic atrophy across all segments, with the most pronounced reduction in the tail, which is anatomically rich in islet cells.[9] This observation strongly supports the prevailing understanding that T1DM, an autoimmune disease, leads to progressive destruction of insulin-producing β-cells and subsequent pancreatic volume loss.[10] The progressive nature of this atrophy is further supported by our finding of a strong inverse correlation between pancreatic size and disease duration in T1DM patients ($r=-0.45$, $p<0.001$). This is consistent with other studies that have shown a decline in pancreas volume during the first year after T1DM diagnosis.[11]

In contrast, T2DM patients showed only mild reduction in pancreatic head diameter, while body and tail dimensions remained relatively preserved. This finding aligns with the pathophysiology of T2DM, where the primary pancreatic change is fatty infiltration rather than atrophy, particularly in the context of obesity and metabolic syndrome. The weaker correlation between pancreatic size and disease duration in T2DM ($r=-0.28$, $p<0.001$) likely reflects the heterogeneous nature of T2DM and the competing effects of fatty infiltration (which may increase size) and long-term β-cell exhaustion (which may cause mild atrophy). These findings are especially relevant in resource-limited areas where patients often present with advanced disease due to delayed diagnosis and management.

Advanced imaging modalities like MRI have corroborated and quantified these atrophic changes with greater precision.[2] A comprehensive systematic review and meta-analysis of imaging studies, which included over 3,400 subjects, concluded that individuals with T1DM have significantly reduced pancreas size compared to controls, while findings in T2DM are more variable.[12] This meta-analysis highlighted that the volume reduction was substantially more significant in T1DM. These robust findings from large-scale analyses, combined with our sonographic data, confirm that pancreatic atrophy is a fundamental characteristic of T1DM, while T2DM shows heterogeneous size changes. The fact that ultrasound, a far more accessible tool, can detect these changes with statistical significance underscores its clinical utility as a screening and monitoring tool, particularly in settings where a full-body MRI is a distant reality. However, sonographic assessment may be limited in obese individuals due to acoustic attenuation and overlying bowel gas, which can obscure pancreatic visualization.

Beyond simple atrophy, our study demonstrates a significant increase in pancreatic echogenicity in diabetic patients, a finding present in a large majority of both T1DM (68%) and T2DM (72%) patients, compared to only 12% of controls. This sonographic finding, a key radiological indicator, reflects underlying histopathological changes. In T2DM, the positive correlation we found between increased echogenicity and BMI ($r=0.34$, $p<0.001$) strongly supports the concept of pancreatic steatosis, or fatty infiltration, as a primary driver of this appearance. This is a well-documented phenomenon in T2DM and is supported by MRI studies that have quantified pancreatic triglyceride content, finding it to be 23% greater in T2DM subjects than in controls.[3] In contrast, our study found no such correlation between echogenicity and BMI in the T1DM group. This suggests that while increased echogenicity is present in T1DM, its cause is fundamentally different and reflects the distinct pathophysiology of the two diabetes types. The histopathological hallmark of T1DM is insulitis, an inflammatory process that leads to fibrosis of the pancreas. Our sonographic findings, there-

fore, likely represent a combination of pancreatic atrophy and fibrosis in T1DM, distinct from the lipotoxic changes seen in T2DM. This differentiation is a crucial point for radiologists to consider when evaluating pancreatic sonograms in diabetic patients.

The radiological changes we identified are not merely morphological assessments but hold significant clinical implications. A recent MRI study highlighted the potential of using MRI-based pancreatic morphology and clinical characteristics to predict the risk of T2DM.[13] Their logistic regression analysis revealed complex relationships between pancreatic morphology and T2DM risk, achieving a predictive accuracy of 90.20%.[13] While these findings were based on MRI, they underscore the diagnostic and prognostic value of assessing pancreatic morphology. Such predictive models, once validated by simpler modalities, could be invaluable in a low-resource clinical setting.

Furthermore, another study found that serration of the pancreatic limbus, a morphological change seen in MRI, was more often observed in subjects with diabetes mellitus and was associated with older age and higher HbA1c values.[5] This corroborates our finding of correlations between pancreatic morphology and metabolic control, particularly the strong relationship observed in T1DM. The finding that these morphological changes, such as a smaller pancreatic volume and serrated changes, are associated with the progression of vascular complications elevates their importance from a simple observation to a potentially critical component of risk assessment, particularly in T1DM where atrophy is pronounced.[5,13] In a resource-limited setting such as this study site, where access to specialized vascular assessments may be limited, a simple sonographic finding could provide an early warning sign for clinicians to intensify metabolic control and screen for complications.

Our study has several limitations inherent to its design. The cross-sectional nature prevents us from drawing conclusions about causality and the temporal progression of these changes in individual patients. The single-center nature and a sample size of 300, while large for a sonography study, limit the generalizability of our findings. The inherent operator-dependent variability of ultrasound was however mitigated by the use of a single, experienced, and blinded radiologist.

CONCLUSION

In a disease as complex and multifaceted as diabetes, radiology, and specifically the accessible modality of ultrasound, has a vital role to play in enhancing our understanding and improving clinical management, especially in regions where resources are limited. Based on our findings, we recommend that ultrasound be integrated into routine diabetes care in resource-limited settings as a first-line tool for assessing pancreatic morphology. In T1DM, ultrasound can detect progressive atrophy and monitor disease progression, while in T2DM, it can identify fatty infiltration associated with metabolic syndrome. This approach could facilitate early detection of type-specific pancreatic changes, guide therapeutic decisions, and improve monitoring of disease progression. Future research should focus on multicenter, longitudinal studies to validate these sonographic markers over time. Furthermore, combining sonographic evaluation with quantitative imaging biomarkers from MRI (such as pancreatic fat fraction) and clinical markers (such as C-peptide and β-cell function indices) could provide a more comprehensive, integrated assessment of pancreatic health in diabetes. Ultrasound should therefore be considered a cost-effective first-line modality, supplemented by advanced imaging when clinically indicated. Such efforts could significantly enhance diabetes management and reduce the burden of complications in vulnerable populations.

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