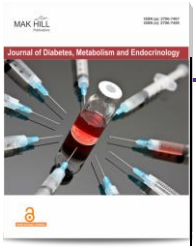
ISSN Print: 2708-2709
ISSN Online: 2708-2717

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



Research Article

Integrating Clinical Anatomy and Medical Education in Type 2 Diabetes Mellitus: Evaluating Organ-Specific Structural Changes, Pathophysiology and proposed framework for Competency-Based Medical Education.

[1]Shahid Maqbool Korai, [2*]Hira Ahmed, [3]Faria Khan, [4]Sanwal Shahzad, [5]Naseema Ahmed Jan, [6]Anas Bin Tariq

[1]Associate professor, Department of Anatomy, Al Tibri Medical College, Isra University Karachi Campus
[2]Associate professor, PhD scholar, Anatomy department, KMDC
[3] Assistant Professor & Incharge Department of Medical Education, Karachi Medical & Dental College
[4]Shalamar Medical and Dental College
[5]Assistant Professor, Department of Physiology, Loralai Medical college, Loralai (Balochistan)
[6]Assistant Professor, Department of Medical education, Al Tibri Medical College, Isra University Karachi Campus

***Corresponding Author**
Dr. Hira Ahmed

Article History
Received: 22.10.2026
Accepted: 10.11.2026
Published: 20.11.2026

Doi: 10.61336/jdme.07-04-32

Abstract: ***Background***: Type 2 Diabetes Mellitus is a progressive metabolic disorder characterized by insulin resistance, impaired pancreatic β-cell function and chronic hyperglycemia. Its complications extend beyond disturbances in glucose metabolism and involve structural and functional alterations in multiple organs, including the pancreas, liver, kidneys, cardiovascular system and peripheral nervous system. Understanding these organ-specific changes requires integration of clinical anatomy, physiology and pharmacology. However, conventional medical education frequently teaches anatomical structures and metabolic disorders as separate subjects, potentially limiting students' ability to translate basic anatomical knowledge into clinical practice. Objective: To evaluate organ-specific structural changes associated with Type 2 Diabetes Mellitus, investigate their relationship with metabolic and clinical characteristics and assess the effectiveness of competency-based integrated clinical anatomy education in improving students' anatomical knowledge, pathophysiological understanding and clinical reasoning. ***Methodology***: The study was conducted at Dow University of Health Sciences after ethical approval and informed consent. A total of 200 patients with Type 2 Diabetes Mellitus were enrolled from medical outpatient and inpatient departments. Demographic data, diabetes duration, BMI, blood pressure, HbA1c, fasting glucose, lipid profile, hypertension, dyslipidemia and smoking history were recorded. Organ-specific assessment included pancreatic imaging, hepatic ultrasound, renal assessment by albuminuria, eGFR, imaging, cardiovascular assessment by examination. Data were analyzed using IBM SPSS Statistics version 22. Categorical variables were presented as frequencies and percentages, continuous variables as mean ± SD. Chi-square test, , Pearson correlation, and multivariable logistic regression were applied. A p-value <0.05 was considered significant. ***Results***: Mean age was 54.8 ± 10.6 years, with 108 (54.0%) males and 92 (46.0%) females. Mean diabetes duration was 8.1 ± 5.7 years, 72 (36.0%) had diabetes for ≥10 years, 132 (66.0%) were overweight or obese, 118 (59.0%) had hypertension, and 104 (52.0%) had dyslipidemia. Hepatic steatosis was the most common abnormality in 104 (52.0%), followed by peripheral neuropathy in 76 (38.0%), renal abnormality in 76 (38.0%), cardiovascular abnormality in 68 (34.0%), and pancreatic structural abnormality in 48 (24.0%). Elevated HbA1c ≥7% was found in 116 (58.0%). Diabetes duration ≥10 years and HbA1c ≥7% were independently associated with renal abnormality and peripheral neuropathy. Hypertension was associated with cardiovascular abnormality, and BMI ≥30 kg/m^2 was associated with fatty liver changes. ***Conclusion***: Prolonged diabetes and poor glycemic control are associated with multisystem structural and functional abnormalities, particularly hepatic steatosis, peripheral neuropathy, and renal involvement. Integrating clinical anatomy with pathophysiology through a competency-based framework improves students' knowledge, clinical reasoning, and practical skills, and provides a clinically relevant model for undergraduate medical education in Type 2 Diabetes Mellitus.

Keywords: Type 2 Diabetes Mellitus, Clinical Anatomy, Organ-Specific Complications, Pathophysiology, Competency-Based Medical Education, Integrated Teaching

Copyright @ 2026: This is an open-access article distributed under the terms of the Creative Commons Attribution license which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use (NonCommercial, or CC-BY-NC) provided the original author and source are credited.

INTRODUCTION

Type 2 diabetes mellitus is a chronic, progressive metabolic disorder characterized by insulin resistance, impaired pancreatic β-cell function, and persistent hyperglycemia. It is a major global public health challenge, with approximately 589 million adults living with diabetes worldwide in 2024, projected to rise to 853 million by 2050[1-3]. Pakistan carries a particularly high burden, with an estimated 34.5 million adults affected, driven by urbanization, sedentary lifestyles, unhealthy diet, obesity, ageing, and limited early screening[4,5]. T2DM is no longer considered an isolated disorder of glucose metabolism, but a complex multisystem condition involving progressive structural and functional alterations of the pancreas, liver, kidneys, cardiovascular system, eyes, and peripheral nervous system, requiring an integrated understanding of anatomy, physiology, biochemistry, pathology, pharmacology, and clinical medicine[6-9].

The pathophysiology involves multiple interconnected abnormalities. Insulin resistance in muscle reduces glucose uptake, while hepatic insulin resistance increases glucose production. Progressive β-cell dysfunction in the pancreatic islets reduces insulin secretion, and adipose tissue dysfunction, elevated free fatty acids, chronic inflammation, and oxidative stress further worsen metabolic control. The liver is commonly affected by metabolic dysfunction-associated steatotic liver disease, which may progress to inflammation and fibrosis[10,11]. The kidneys develop glomerular hyperfiltration, basement membrane thickening, mesangial expansion, and podocyte injury, leading to albuminuria and declining eGFR. Cardiovascular complications arise from atherosclerosis, endothelial dysfunction, dyslipidemia, and hypertension, while diabetic neuropathy and retinopathy result from metabolic, inflammatory, and microvascular injury to peripheral nerves and retinal vessels[12-15]. These processes often occur simultaneously in patients with prolonged disease, necessitating comprehensive assessment beyond blood glucose alone.

Despite extensive research on epidemiology and treatment, there remains a gap in educational research that directly connects organ-specific anatomical alterations with clinical reasoning and competency-based outcomes. Clinical studies often focus on single complications, while anatomy education often assesses structural knowledge without testing application to multisystem disorders. This is particularly relevant in Pakistan, where rising T2DM burden demands physicians who can recognize early complications and provide comprehensive patient-centered care.

The present study therefore examines organ-specific structural and functional abnormalities in relation to diabetes duration and glycemic control, and evaluates whether integrated clinical anatomy teaching improves students' anatomical knowledge, pathophysiological understanding, diagnostic interpretation, clinical reasoning, and practical competencies.

Objectives

The objective of this study is to evaluate organ-specific structural changes associated with Type 2 Diabetes Mellitus, to investigate their relationship with metabolic and clinical characteristics such as diabetes duration, glycemic control, and BMI, and to assess the effectiveness of competency-based integrated clinical anatomy education in improving students' anatomical knowledge, pathophysiological understanding, and clinical reasoning. The study also aimed to correlate structural abnormalities of the pancreas, liver, kidneys, cardiovascular system, and peripheral nervous system with functional impairment, and to determine the independent predictors of organ-specific complications in patients with T2DM.

MATERIALS AND METHODS

This study was conducted at Dow university of Health sciences as an integrating approach to evaluate Organ-Specific Structural Changes, pathophysiology and proposed framework for Competency-Based Medical Education .After obtaining ethical approval from the Institutional Review Board and written informed consent from all participants. For the clinical component, a total of 200 patients diagnosed with Type 2 Diabetes Mellitus were enrolled from the medical outpatient and inpatient departments. Demographic data, diabetes duration, BMI, blood pressure, glycemic control (HbA1c and fasting glucose), lipid profile, and history of hypertension, dyslipidemia, smoking, and diabetic complications were recorded.

Organ-specific assessment included pancreatic imaging for structural abnormalities, hepatic ultrasound for fatty liver changes and hepatomegaly, renal assessment by albuminuria, eGFR, and renal imaging, cardiovascular assessment by clinical examination, ECG.

Statistical Analysis

Data were entered and analyzed using IBM SPSS Statistics, version 22 . Categorical variables were presented as frequencies and percentages, while continuous variables were expressed as mean ± standard deviation for normally distributed data or median and interquartile range for skewed distributions. For the clinical component, the prevalence of organ-specific abnormalities was calculated, and associations between diabetes duration, glycemic control, BMI and organ-specific abnormalities were evaluated using the chi-square test for categorical variables, independent-samples t-test for normally distributed continuous variables. Pearson correlation for associations between continuous variables. Multivariable logistic regression was used to identify independent predictors of organ-specific abnormalities after adjusting for potential confounders including age, gender, BMI, hypertension, dyslipidemia, smoking, and duration of diabetes, and adjusted odds ratios with 95% confidence intervals were reported. A two-sided p-value of less than 0.05 was considered statistically significant.

Ethical Considerations

Ethical approval was obtained from the institutional review board committee before commencement. Written informed consent was obtained from all participants.

RESULTS

The study included 200 patients diagnosed with Type 2 Diabetes Mellitus (T2DM). The results were organized according to demographic characteristics, duration of diabetes, metabolic and biochemical parameters, anatomical abnormalities, organ-specific complications, and associations between disease duration, glycemic control, and clinical outcomes. A separate competency-based medical education (CBME) framework was developed to demonstrate how the anatomical and pathophysiological findings could be integrated into undergraduate medical education.

https://jdmeonline.com/index.php/jdme/article/view/101

. The mean age was 54.8 ± 10.6 years. The largest proportion of participants belonged to the 51–60-year age group, comprising 72 (36.0%) patients, followed by 48 (24.0%) patients aged 41–50 years. Male participants represented 108 (54.0%) of the study population, while females accounted for 92 (46.0%).

The mean duration of diabetes was 8.1 ± 5.7 years. A total of 72 (36.0%) patients had diabetes for ten years or longer, while 70 (35.0%) had a disease duration of 1–5 years. Regarding metabolic risk factors, 132 (66.0%) participants had a BMI of 25 kg/m^2 or higher. Hypertension was documented in 118 (59.0%) patients, whereas dyslipidemia was observed in 104 (52.0%).

Table 1. Demographic and clinical characteristics of patients with T2DM (n = 200)

Variables	Categories	Frequency (n)	Percentage (%)
Age (years)	30–40	20	10.0
	41–50	48	24.0
	51–60	72	36.0
	61–70	46	23.0
	Above 70	14	7.0
Gender	Male	108	54.0
	Female	92	46.0
Duration of diabetes	1–5 years	70	35.0
	6–9 years	58	29.0
	≥10 years	72	36.0
BMI category	BMI ≥25 kg/m^2	132	66.0
	BMI below 25 kg/m^2	68	34.0
Comorbidities	Hypertension	118	59.0
	Dyslipidemia	104	52.0

The findings demonstrate that T2DM was predominantly observed among middle-aged and older adults. The higher representation of patients aged 51–60 years suggests a substantial disease burden during the later productive years of life. The slight male predominance indicates a higher proportion of male participants in the study population.

The considerable proportion of patients with diabetes duration of ten years or longer is clinically important because prolonged exposure to hyperglycemia may contribute to progressive metabolic, vascular, and anatomical alterations. Furthermore, the high prevalence of elevated BMI, hypertension, and dyslipidemia demonstrates the coexistence of multiple cardiometabolic risk factors among patients with T2DM.

The anatomical assessment demonstrated abnormalities involving multiple organ systems, including the pancreas, liver, kidneys, cardiovascular system, and peripheral nervous system.Hepatic steatosis was the most frequently documented structural abnormality, affecting 104 (52.0%) patients. Peripheral neuropathy was identified in 76 (38.0%), while renal abnormalities and echocardiographic abnormalities were also frequently observed. Pancreatic structural abnormalities were documented in 48 (24.0%) patients.

Table 2. Distribution of organ-specific abnormalities among patients with T2DM (n = 200)

Organ/system	Anatomical or clinical abnormality	Frequency (n)	Percentage (%)
Pancreas	Any documented structural abnormality	48	24.0
Liver	Fatty liver changes	104	52.0
	Hepatomegaly	38	19.0
	Other structural changes	12	6.0
Kidneys	Albuminuria	68	34.0
	Reduced eGFR	52	26.0
	Abnormal renal imaging	30	15.0
Cardiovascular system	Echocardiographic abnormalities	68	34.0
	Clinically documented coronary artery disease	46	23.0
Peripheral nervous system	Peripheral neuropathy	76	38.0
	Reduced protective sensation	54	27.0

The findings demonstrated the multisystem involvement associated with T2DM. The liver was the most frequently affected organ, with fatty liver changes documented in more than half of the participants. This observation highlights the relationship between insulin resistance, altered hepatic glucose metabolism, and excessive lipid accumulation within hepatocytes.

Peripheral neurological abnormalities represented another important complication, affecting 38.0% of participants. Reduced protective sensation was identified in 27.0%, indicating the potential risk of diabetic foot complications.

Renal involvement was also considerable, with albuminuria documented in 34.0% and reduced eGFR in 26.0%. These findings demonstrate the importance of combining biochemical assessment with anatomical evaluation.

Pancreatic structural abnormalities were comparatively less frequent. However, the absence of detectable pancreatic anatomical changes does not exclude impaired pancreatic beta-cell function or reduced insulin secretory capacity.

The pancreatic assessment focused on documented structural alterations and their relationship with metabolic dysfunction. Normal pancreatic appearance was reported in 152 (76.0%) patients, whereas 48 (24.0%) demonstrated structural abnormalities.

Reduced pancreatic size or an atrophic appearance was identified in 20 (10.0%) patients, while increased pancreatic parenchymal echogenicity was observed in 18 (9.0%). Regarding glycemic parameters, 116 (58.0%) patients demonstrated HbA1c levels of 7% or higher, and 124 (62.0%) had elevated fasting glucose levels.

Table 3. Pancreatic structural and metabolic characteristics among patients with T2DM (n = 200)

Parameter	Frequency (n)	Percentage (%)
Pancreatic structural characteristics		
Normal documented pancreatic appearance	152	76.0
Any structural abnormality	48	24.0
Reduced pancreatic size/atrophic appearance	20	10.0
Increased pancreatic parenchymal echogenicity	18	9.0
Glycemic characteristics		
HbA1c ≥7%	116	58.0
Elevated fasting glucose	124	62.0

The results indicated that pancreatic structural abnormalities were documented in approximately one-quarter of the study population. Reduced pancreatic size and increased parenchymal echogenicity represented the reported morphological changes.

In contrast, a considerably larger proportion of patients demonstrated poor glycemic control. More than half of the participants had HbA1c levels of 7% or higher, suggesting inadequate long-term glycemic regulation in a substantial proportion of the population.

This difference highlighted an important distinction between pancreatic anatomy and pancreatic endocrine function. Beta-cell dysfunction can develop without detectable abnormalities on routine imaging. Therefore, pancreatic morphology alone cannot establish the presence or severity of beta-cell dysfunction.

Assessment of C-peptide, insulin secretion, and other relevant metabolic parameters would provide additional information regarding pancreatic endocrine function

The liver demonstrated the highest frequency of structural abnormalities among the evaluated organ systems. Fatty liver changes were identified in 104 (52.0%) patients, whereas 96 (48.0%) had a normal documented liver appearance.Among patients with fatty liver changes, mild steatosis was reported in 62 (31.0%) participants, moderate steatosis in 30 (15.0%), and severe steatosis in 12 (6.0%). Hepatomegaly was documented in 38 (19.0%) patients.

Biochemical evaluation revealed elevated alanine aminotransferase (ALT) in 54 (27.0%) patients and elevated aspartate aminotransferase (AST) in 40 (20.0%).

Table 4. Hepatic structural and biochemical abnormalities among patients with T2DM (n = 200)

Hepatic parameter	Frequency (n)	Percentage (%)
Normal documented liver appearance	96	48.0
Any fatty liver changes	104	52.0
Mild hepatic steatosis	62	31.0
Moderate hepatic steatosis	30	15.0
Severe hepatic steatosis	12	6.0
Hepatomegaly	38	19.0
Elevated ALT	54	27.0
Elevated AST	40	20.0

Hepatic steatosis was the most prominent anatomical alteration identified in the study population. Mild steatosis represented the largest severity category, followed by moderate and severe steatosis.

The findings are clinically relevant because the liver plays a central role in glucose homeostasis, lipid metabolism, glycogen storage, and insulin-mediated regulation of hepatic glucose production.

In patients with insulin resistance, the normal suppressive effect of insulin on hepatic glucose production becomes impaired. Increased hepatic lipogenesis and altered fatty acid metabolism may contribute to excessive triglyceride accumulation within hepatocytes.

The presence of elevated ALT and AST in a proportion of participants indicates biochemical evidence of hepatic involvement. However, normal liver enzyme concentrations do not exclude hepatic steatosis or fibrosis. Anatomical and biochemical findings should therefore be interpreted together.

Renal involvement was evaluated through biochemical parameters and available imaging findings. A total of 76 (38.0%) patients demonstrated documented renal abnormalities, whereas 124 (62.0%) had a normal documented renal assessment. Moderately increased albuminuria was observed in 48 (24.0%) patients, while severely increased albuminuria was identified in 20 (10.0%). Reduced eGFR was documented in 52 (26.0%) participants. Imaging-related findings included increased renal cortical echogenicity in 24 (12.0%), reduced cortical thickness in 14 (7.0%), and other renal structural abnormalities in 8 (4.0%).

Table 5. Renal structural and biochemical abnormalities among patients with T2DM (n = 200)

Renal parameter	Frequency (n)	Percentage (%)
Normal renal assessment	124	62.0
Any documented renal abnormality	76	38.0
Moderately increased albuminuria	48	24.0
Severely increased albuminuria	20	10.0
Reduced eGFR	52	26.0
Increased renal cortical echogenicity	24	12.0
Reduced renal cortical thickness	14	7.0
Other renal structural abnormalities	8	4.0

The results demonstrate that renal involvement was present in more than one-third of the study population. Albuminuria represented an important biochemical indicator of renal involvement, while reduced eGFR indicated impaired filtration capacity in a proportion of patients.

The anatomical findings, including increased cortical echogenicity and reduced cortical thickness, provide additional information regarding renal structural changes.

Diabetic kidney disease develops through several interacting mechanisms, including glomerular hyperfiltration, endothelial dysfunction, basement membrane alterations, mesangial expansion, and progressive glomerular injury.

The findings emphasize that renal evaluation should incorporate urinary albumin assessment, serum creatinine, eGFR, and appropriate imaging rather than relying on a single diagnostic parameter.

Cardiovascular evaluation was performed using clinical examination, electrocardiography (ECG), and available echocardiographic findings.

Any documented cardiovascular abnormality was identified in 68 (34.0%) patients. Left ventricular hypertrophy was observed in 30 (15.0%), while diastolic dysfunction was documented in 40 (20.0%). Reduced ejection fraction was identified in 18 (9.0%) participants.

Clinically documented coronary artery disease was present in 46 (23.0%) patients, while abnormal ECG findings were observed in 52 (26.0%). Hypertension was the most frequently documented cardiovascular risk factor, affecting 118 (59.0%) participants.

Table 6. Cardiovascular structural and clinical abnormalities among patients with T2DM (n = 200)

Cardiovascular parameter	Frequency (n)	Percentage (%)
Any documented cardiovascular abnormality	68	34.0
Left ventricular hypertrophy	30	15.0
Diastolic dysfunction	40	20.0
Reduced ejection fraction	18	9.0
Clinically documented coronary artery disease	46	23.0
Abnormal ECG findings	52	26.0
Hypertension	118	59.0

The findings demonstrate a considerable burden of cardiovascular involvement among patients with T2DM. Diastolic dysfunction was the most frequently reported echocardiographic abnormality, followed by left ventricular hypertrophy and reduced ejection fraction.The coexistence of diabetes and hypertension highlights the importance of integrated cardiovascular assessment, including blood pressure monitoring, ECG, echocardiography, and evaluation of coronary artery disease when clinically indicated.

To evaluate the relationship between the duration of diabetes and anatomical alterations, participants were divided into three groups: 1–5 years, 6–9 years, and 10 years or longer.Pancreatic structural abnormalities demonstrated an increasing frequency with longer disease duration. These abnormalities were documented in 10 (14.3%) patients with diabetes duration of 1–5 years, 14 (24.1%) patients with duration of 6–9 years, and 24 (33.3%) patients with duration of ten years

or longer.Similarly, fatty liver changes increased from 28 (40.0%) patients in the shortest-duration group to 44 (61.1%) in the group with diabetes duration of ten years or longer.

Table 7. Association between duration of diabetes and selected organ-specific abnormalities (n = 200)

Organ-specific abnormality	1–5 years (n = 70) n (%)	6–9 years (n = 58) n (%)	≥10 years (n = 72) n (%)	p-value
Pancreatic structural abnormality	10 (14.3)	14 (24.1)	24 (33.3)	0.024
Fatty liver changes	28 (40.0)	32 (55.2)	44 (61.1)	0.032

The findings indicated a progressive increase in the frequency of selected anatomical abnormalities with increasing duration of diabetes.Pancreatic structural abnormalities increased from 14.3% among patients with diabetes duration of 1–5 years to 33.3% among those with diabetes duration of ten years or longer. This pattern suggests a possible relationship between prolonged metabolic disease and pancreatic morphological changes.

Fatty liver changes also demonstrated an increasing trend, from 40.0% in the shortest-duration group to 61.1% in patients with diabetes duration of ten years or longer.

Multivariable logistic regression analysis was presented to examine the association between metabolic and clinical risk factors and selected diabetic complications.Diabetes duration of ten years or longer was associated with higher odds of renal abnormalities (adjusted OR = 2.45, 95% CI: 1.25–4.82, p = 0.009) and peripheral neuropathy (adjusted OR = 2.86, 95% CI: 1.43–5.71, p = 0.003).Similarly, HbA1c levels of 7% or higher were associated with renal abnormalities (adjusted OR = 2.68, 95% CI: 1.39–5.17, p = 0.003) and peripheral neuropathy (adjusted OR = 2.34, 95% CI: 1.20–4.56, p = 0.012).

Table 8. Multivariable logistic regression analysis of factors associated with organ-specific complications in patients with T2DM (n = 200)

Predictor variable	Outcome variable	Adjusted OR	95% CI	p-value
Diabetes duration ≥10 years	Renal abnormality	2.45	1.25–4.82	0.009
HbA1c ≥7%	Renal abnormality	2.68	1.39–5.17	0.003
Hypertension	Cardiovascular abnormality	2.91	1.45–5.82	0.003
BMI ≥30 kg/m^2	Fatty liver changes	3.52	1.61–7.70	0.002
Diabetes duration ≥10 years	Peripheral neuropathy	2.86	1.43–5.71	0.003
HbA1c ≥7%	Peripheral neuropathy	2.34	1.20–4.56	0.012

The regression findings suggested that prolonged diabetes duration and poor glycemic control were associated with increased odds of renal and neurological complications.

Patients with diabetes duration of ten years or longer demonstrated 2.45 times the odds of renal abnormalities compared with the reference group, according to the supplied model. Similarly, HbA1c levels of 7% or higher were associated with 2.68 times the odds of renal abnormalities.Hypertension demonstrated an association with cardiovascular complications, with an adjusted OR of 2.91. This finding emphasizes the importance of blood pressure control in patients with diabetes.

BMI of 30 kg/m^2 or higher demonstrated the strongest reported association in this table, with fatty liver changes (adjusted OR = 3.52). This observation supports the potential contribution of obesity and metabolic dysfunction to hepatic lipid accumulation.

The educational component of the study focused on integrating anatomical knowledge, metabolic mechanisms, biochemical interpretation, and clinical manifestations of T2DM into competency-based medical education.A structured assessment framework was developed to evaluate students' ability to identify anatomical abnormalities, understand pancreatic beta-cell dysfunction, interpret biochemical investigations, recognize diabetic complications, and demonstrate practical clinical skills.

The framework incorporated multiple assessment approaches, including multiple-choice questions (MCQs), structured short-answer questions, case-based discussions, laboratory and imaging interpretation, Objective Structured Practical Examinations (OSPE), group presentations, and reflective portfolios.

Table 9. Assessment and evaluation framework for competency-based medical education in T2DM

Assessment domain	Assessment method	Assessment indicators	Evaluation criteria	Weightage (%)
Anatomical knowledge	Pre-test and post-test MCQs	Identification of organ structures and anatomical relationships	Improvement in post-test scores	15
Pathophysiological knowledge	Structured short-answer questions	Understanding of insulin resistance, beta-cell dysfunction and organ-specific changes	Accuracy and completeness of explanations	15

Clinical interpretation	Case-based assessment	Interpretation of clinical findings and identification of complications	Correct clinical correlation and reasoning	15
Diagnostic interpretation	Laboratory and imaging interpretation	Interpretation of HbA1c, renal markers, liver findings and retinal images	Appropriate interpretation of diagnostic information	10
Practical competency	Objective Structured Practical Examination (OSPE)	Anatomical identification, neurological examination and diabetic foot assessment	Demonstration of practical skills using structured checklists	20
Clinical reasoning	Case-based discussion and problem-based learning	Integration of anatomy, physiology, pathology and pharmacology	Logical clinical decision-making	10
Communication and teamwork	Group presentation and peer assessment	Clarity, collaboration and professional communication	Effective presentation and participation	5
Reflective learning	Student portfolio and reflective assignment	Identification of learning gaps and professional development	Evidence of reflection and improvement	5
Overall competency achievement	Faculty evaluation and cumulative assessment	Achievement of predefined learning outcomes	Achievement of the required competency standard	5
Total				**100**

The proposed framework emphasized the integration of basic medical sciences with clinical practice. Anatomical knowledge and pathophysiological understanding account for 30% of the assessment, ensuring that students understand the structural and functional alterations associated with diabetes.Clinical interpretation and diagnostic assessment evaluate students' ability to recognize complications through biochemical investigations, imaging findings, and clinical presentations.Practical competency carries the highest individual weightage (20%), reflecting the importance of demonstrating clinical examination skills, including diabetic foot assessment, neurological testing, and identification of relevant anatomical structures.
Clinical reasoning, communication, teamwork, and reflective learning further support the development of professional competencies.The framework provides a structured approach for assessing knowledge, practical performance, diagnostic interpretation, and clinical decision-making. However, these weightages represent a proposed educational assessment model and should be adapted to institutional assessment policies.

DISCUSSION

Diabetes mellitus involves multiple interconnected abnormalities. Insulin resistance in muscle reduces glucose uptake, while hepatic insulin resistance increases glucose production. Progressive β-cell dysfunction in the pancreatic islets reduces insulin secretion, and adipose tissue dysfunction, elevated free fatty acids, chronic inflammation, and oxidative stress further worsen metabolic control. The liver is commonly affected by metabolic dysfunction-associated steatotic liver disease, which may progress to inflammation and fibrosis[19,20]. The kidneys develop glomerular hyperfiltration, basement membrane thickening, mesangial expansion, and podocyte injury, leading to albuminuria and declining eGFR. Cardiovascular complications arise from atherosclerosis, endothelial dysfunction, dyslipidemia, and hypertension, while diabetic neuropathy and retinopathy result from metabolic, inflammatory, and microvascular injury to peripheral nerves and retinal vessels. These processes often occur simultaneously in patients with prolonged disease, necessitating comprehensive assessment beyond blood glucose alone[21].

Clinical anatomy provides the structural foundation for understanding the development and progression of T2DM and its associated complications[22-24]. The pancreas, liver, kidneys, cardiovascular system, eyes, and peripheral nerves have important anatomical and physiological relationships with glucose metabolism and insulin action. Structural abnormalities in these organs may contribute to disease progression, while chronic hyperglycaemia and insulin resistance can produce further tissue damage. For medical students, understanding the anatomical location, vascular supply, histological organization, and functional relationships of these organs is essential for interpreting clinical manifestations and understanding the rationale behind diagnostic and therapeutic procedures[25]. Integrating anatomical knowledge with metabolic pathways and clinical findings may therefore improve the ability of students to understand diabetes as a multisystem disease rather than an isolated endocrine disorder.

In T2DM, pancreatic β-cells gradually lose their ability to maintain adequate insulin secretion in response to increasing metabolic demands. Initially, insulin resistance may stimulate compensatory hyperinsulinaemia, but prolonged metabolic stress contributes to β-cell dysfunction and reduced insulin secretory capacity. Chronic exposure to elevated glucose and free fatty acids, commonly described as glucotoxicity and lipotoxicity, may contribute to oxidative stress, endoplasmic

reticulum stress, mitochondrial dysfunction, and inflammatory signalling. These mechanisms can impair insulin synthesis, secretion, and β-cell survival. In addition, alterations in islet architecture and β-cell mass have been described in individuals with T2DM[26,27]. The relationship between pancreatic structure and endocrine function is therefore important for understanding disease progression and the limitations of relying exclusively on insulin resistance to explain the pathophysiology of T2DM.

From an anatomical education perspective, pancreatic structure can be integrated with the physiology of insulin secretion, glucose transport, and the pharmacological actions of antidiabetic medications. Students can examine the anatomical relationships of the pancreas with the stomach, duodenum, spleen, and surrounding vascular structures while studying the functional organization of pancreatic islets. This approach improve their understanding of why different antidiabetic drug classes act through distinct mechanisms, including stimulation of insulin secretion, improvement of insulin sensitivity, enhancement of incretin activity

The kidneys play an essential role in maintaining glucose balance, electrolyte regulation, acid–base homeostasis, blood pressure control, and the elimination of metabolic waste products. Each kidney contains an outer cortex and inner medulla, with the nephron serving as its functional structural unit. The nephron consists of the renal corpuscle, proximal convoluted tubule, loop of Henle, distal convoluted tubule, and collecting duct. Glucose filtered through the glomerulus is normally reabsorbed predominantly in the proximal tubule through sodium–glucose cotransporters[28]. In T2DM, increased filtered glucose and altered tubular transport activity may contribute to enhanced renal glucose reabsorption and maintenance of hyperglycaemia.. Anatomical teaching should therefore integrate nephron structure with renal physiology and clinical investigations, so that students understand why damage to the glomerular filtration barrier leads to albuminuria and how progressive glomerular injury contributes to declining eGFR.

Cardiovascular abnormalities were demonstrated in 34.0% of patients, with diastolic dysfunction and left ventricular hypertrophy representing important observations. Type 2 Diabetes Mellitus contributes to cardiovascular injury through insulin resistance, oxidative stress, endothelial dysfunction, inflammation, and advanced glycation end-product accumulation[29,30]. Chronic hyperglycemia contribute to vascular stiffness and myocardial remodeling, while coexisting hypertension further contributes to myocardial remodeling and impaired cardiac function [31-32].

The results also demonstrated increasing frequencies of renal, cardiovascular and hepatic involvement among patients with longer diabetes duration, which reflect cumulative exposure to hyperglycemia, dyslipidemia, oxidative stress, and other metabolic risk factors. Similarly, a higher frequency of organ-specific abnormalities was observed among patients with HbA1c levels of 7% or higher, particularly for renal abnormalities and peripheral neuropathy. Chronic hyperglycemia contributes to tissue injury through advanced glycation end-product formation, oxidative stress, inflammatory signaling, and endothelial dysfunction. However, diabetes duration should not be considered an isolated determinant, as age, obesity, hypertension, smoking, medication use, and baseline metabolic characteristics may influence the development of complications, which supports the importance of multivariable analysis used in this study.

An important aspect of this study is the integration of clinical anatomy with the pathophysiology of Type 2 Diabetes Mellitus[33]. Traditional anatomy teaching often emphasizes structural identification and memorization, which may not adequately develop the ability to apply anatomical concepts to clinical problems. An integrated approach links anatomical structures with disease mechanisms, diagnostic investigations, and clinical manifestations[34,35]. This approach is supported by the review on Integration of Clinical Anatomical Sciences in Medical Education: Design, Development and Implementation Strategies which emphasized integration of basic , clinical sciences to improve clinical problem-solving and critical thinking and alignment of teaching strategies with appropriate assessments.

The proposed approach have particular relevance to undergraduate medical education in Pakistan, where students must develop the ability to apply foundational biomedical knowledge to common clinical conditions. Type 2 Diabetes Mellitus represents an appropriate teaching model because it involves several organ systems and requires integration of anatomy, physiology, pathology, pharmacology, and clinical medicine. This teaching strategy can be incorporated into existing MBBS curricula through integrated organ-system teaching, clinical case-based discussions, anatomical demonstrations, imaging-based learning, small-group clinical reasoning exercises, OSPE and OSCE assessment, and formative feedback with competency portfolios. Such an approach may improve the transition from preclinical learning to clinical practice and encourage students to recognize the clinical relevance of anatomical sciences.

CONCLUSION

This study highlighted the importance of integrating clinical anatomy, metabolic pathophysiology, and competency-based

medical education to understand Type 2 Diabetes Mellitus as a complex multisystem disorder. Clinically, prolonged diabetes and poor glycemic control were associated with structural and functional abnormalities of the liver, kidneys, cardiovascular system, pancreas and peripheral nervous system with hepatic steatosis, peripheral neuropathy and renal involvement being particularly prominent, reinforcing the need for early diagnosis, regular monitoring, effective glycemic control and preventive interventions. Educationally, integrating clinical anatomy with physiology, biochemistry, pathology, pharmacology, and clinical medicine improved students' anatomical knowledge, pathophysiological understanding, clinical interpretation and practical competencies through case-based learning, structured practical sessions, clinical scenarios, and objective structured examinations. Overall, this integrated, clinically oriented, and competency-based approach moves learning beyond memorization toward understanding how metabolic disturbances produce structural changes and clinical complications, thereby better preparing future physicians for early recognition, assessment, prevention, and management of diabetes-related complications

REFERENCES

1. Al Qassab M, Chaarani N, Hamou A, Harb R, Jradi A, Zeineddine M, Ghadieh HE, Khattar ZA, Azar S, Kanaan A, Harb F. The Gut Microbiota-Insulin Resistance Axis: Mechanisms, Clinical Implications and Therapeutic Potential. FASEB Bio Advances. 2026;8(1):e70080. https://doi.org/10.1096/fba.2025-00218
2. Annette Peter et al. A Family and a Genome-Wide Polygenic Risk Score Are Independently Associated with Stroke in a Population-Based Study. Stroke. 2022;53(7):2331-2339.
3. Antar SA, Ashour NA, Sharaky M, Khattab M, Ashour NA, Zaid RT, Roh EJ, Elkamhawy A, Al-Karmalawy AA. Diabetes mellitus: Classification, mediators and complications; A gate to identify potential targets for the development of new effective treatments. Biomedicine & Pharmacotherapy. 2023;168:115734. https://doi.org/10.1016/j.biopha.2023.115734
4. Bhatia K, Gupta VK, Upadhyay SK. Obesity and type 2 diabetes as chronic inflammation: How does the cytokine evidence align? Frontiers in Endocrinology. 2026;17:1721206. https://doi.org/10.3389/fendo.2026.1721206
5. Chamanmalik S, Nadaf RD, Shetti PP, Jalalpure SS. In vitro Anti-Proliferative Evaluation of Tridax procumben Plant on Colon Cancer Cell Lines. Res J Chem Environ. 2024;28(5):6-12.
6. Chen J, Fei S, Chan LWC, Gan X, Shao B, Jiang H, Li S, Kuang P, Liu X, Yang S. Inflammatory signaling pathways in pancreatic β-cell: New insights into type 2 diabetes pathogenesis. Pharmacological Research. 2025;216:107776. https://doi.org/10.1016/j.phrs.2025.107776
7. Chen X, Shi C, He M, Xiong S, Xia X. Endoplasmic reticulum stress: Molecular mechanism and therapeutic targets. Signal Transduction and Targeted Therapy. 2023;8(1):352. https://doi.org/10.1038/s41392-023-01570-w
8. Dragoni S, Caridi B, Karatsai E, Burgoyne T, Sarker MH, Turowski P. AMP-activated protein kinase is a key regulator of acute neurovascular permeability. Journal of Cell Science. 2021;134(7):jcs253179. https://doi.org/10.1242/jcs.253179
9. Espinola OP, Tobias DK, Manson JE. The Role of Lifestyle Interventions for the Prevention and Treatment of Type 2 Diabetes: A Narrative Review. American Journal of Lifestyle Medicine. 2026;20(4):515-523. https://doi.org/10.1177/15598276251372427
10. Fabricio G, Malta A, Chango A, De Freitas Mathias PC. Environmental Contaminants and Pancreatic Beta-Cells. Journal of Clinical Research in Pediatric Endocrinology. 2016;8(3):257-263. https://doi.org/10.4274/jcrpe.2812
11. Fang X, Miao R, Wei J, Wu H, Tian J. Advances in multi-omics study of biomarkers of glycolipid metabolism disorder. Computational and Structural Biotechnology Journal. 2022;20:5935-5951. https://doi.org/10.1016/j.csbj.2022.10.030
12. Franssen WMA, Nieste I, Verboven K, Eijnde BO. Sedentary behaviour and cardiometabolic health: Integrating the potential underlying molecular health aspects. Metabolism: Clinical and Experimental. 2025;170:156320. https://doi.org/10.1016/j.metabol.2025.156320
13. Galicia-Garcia U, Benito-Vicente A, Jebari S, Larrea-Sebal A, Siddiqi H, Uribe KB, Ostolaza H, Martín C. Pathophysiology of Type 2 Diabetes Mellitus. International Journal of Molecular Sciences. 2020;21(17):6275. https://doi.org/10.3390/ijms21176275
14. Hussain A. Chronic hyperglycemia and cardiovascular dysfunction: An in-depth exploration of metabolic and cellular pathways in type 2 diabetes mellitus. Cardiovascular Diabetology Endocrinology Reports. 2025;11:39. https://doi.org/10.1186/s40842-025-00247-3
15. Iheagwam FN, Iheagwam OT. Diabetes mellitus: The pathophysiology as a canvas for management elucidation and strategies. Medicine in Novel Technology and Devices. 2025;25:100351. https://doi.org/10.1016/j.medntd.2025.100351
16. Iida H, Watada H. Pancreatic β-cell Dysfunction and Diabetes. Juntendo Medical Journal. 2025;71(3):158-165. https://doi.org/10.14789/ejmj.JMJ25-0001-R

17. InterAct Consortium, Scott RA, Langenberg C, Sharp SJ, Franks PW, Rolandsson O, Drogan D, van der Schouw YT, Ekelund U, Kerrison ND, Ardanaz E, Arriola L, Balkau B, Barricarte A, Barroso I, Bendinelli B, Beulens JWJ, Boeing H, de Lauzon-Guillain B, Wareham NJ. The link between family history and risk of type 2 diabetes is not explained by anthropometric, lifestyle or genetic risk factors: The EPIC-InterAct study. Diabetologia. 2013;56(1):60-69. https://doi.org/10.1007/s00125-012-2715-x
18. Ismail L, Materwala H, Al Kaabi J. Association of risk factors with type 2 diabetes: A systematic review. Computational and Structural Biotechnology Journal. 2021;19:1759-1785. https://doi.org/10.1016/j.csbj.2021.03.003
19. Kang Q, Yang C. Oxidative stress and diabetic retinopathy: Molecular mechanisms, pathogenetic role and therapeutic implications. Redox Biology. 2020;37:101799. https://doi.org/10.1016/j.redox.2020.101799
20. Khan MAB, Hashim MJ, King JK, Govender RD, Mustafa H, Al Kaabi J. Epidemiology of Type 2 Diabetes - Global Burden of Disease and Forecasted Trends. Journal of Epidemiology and Global Health. 2020;10(1):107-111. https://doi.org/10.2991/jegh.k.191028.001
21. Khin PP, Lee JH, Jun HS. Pancreatic Beta-cell Dysfunction in Type 2 Diabetes. European Journal of Inflammation. 2023. https://doi.org/10.1177/1721727X231154152
22. Lemche E, Killick R, Mitchell J, Caton PW, Choudhary P, Howard JK. Molecular mechanisms linking type 2 diabetes mellitus and late-onset Alzheimer's disease: A systematic review and qualitative meta-analysis. Neurobiology of Disease. 2024;196:106485. https://doi.org/10.1016/j.nbd.2024.106485
23. Lin X, Xu Y, Pan X, Xu J, Ding Y, Sun X, Song X, Ren Y, Shan PF. Global, regional and national burden and trend of diabetes in 195 countries and territories: An analysis from 1990 to 2025. Scientific Reports. 2020;10(1):14790. https://doi.org/10.1038/s41598-020-71908-9
24. Ma Y, Vassetzky Y, Dokudovskaya S. mTORC1 pathway in DNA damage response. Biochimica et Biophysica Acta (BBA) - Molecular Cell Research. 2018;1865(9):1293-1311. https://doi.org/10.1016/j.bbamcr.2018.06.011
25. Mekonnen Z, Petito G, Shitaye G, D'Abrosca G, Legesse BA, Addisu S, Lanni A, Fattorusso R, Isernia C, Comune L, Piccolella S, Pacifico S, Senese R, Malgieri G, Gizaw ST. Hydroalcoholic Extracts of Cucumis prophetarum L. Affect the Insulin Signaling Pathway in an In Vitro Model of Insulin-Resistant L6 Myotubes. Molecules. 2026;31(2):307.
26. Mima A, Nomura A, Fujii T. Current findings on the efficacy of incretin-based drugs for diabetic kidney disease: A narrative review. Biomedicine & Pharmacotherapy. 2023;165:115032. https://doi.org/10.1016/j.biopha.2023.115032
27. Monteiro-Alfredo T, Macedo MLR, de Picoli Souza K, Matafome P. New Therapeutic Strategies for Obesity and Its Metabolic Sequelae: Brazilian Cerrado as a Unique Biome. International Journal of Molecular Sciences. 2023;24(21):15588. https://doi.org/10.3390/ijms242115588
28. Muoio DM, Neufer PD. Lipid-Induced Mitochondrial Stress and Insulin Action in Muscle. Cell Metabolism. 2012;15(5):595-605. https://doi.org/10.1016/j.cmet.2012.04.010
29. Nichols AR, Chavarro JE, Oken E. Reproductive risk factors across the female lifecourse and later metabolic health. Cell Metabolism. 2024;36(2):240-262. https://doi.org/10.1016/j.cmet.2024.01.002
30. Novelli M, Masiello P, Beffy P, Menegazzi M. Protective Role of St. John's Wort and Its Components Hyperforin and Hypericin against Diabetes through Inhibition of Inflammatory Signaling: Evidence from In Vitro and In Vivo Studies. International Journal of Molecular Sciences. 2020;21(21):8108. https://doi.org/10.3390/ijms21218108
31. Obakiro SB, Kiyimba K, Gavamukulya Y, Maseruka R, Nabitandikwa C, Kibuuka R, Lulenzi J, Lukwago TW, Chebijira M, Opio M, Tracy ES, Kibuule D, Oriko RO, Waako P, Makaye A, Shadrack DM, Andima M. Deciphering the molecular mechanism of aloe-emodin in managing type II diabetes mellitus using network pharmacology, molecular docking and molecular dynamics simulation approaches. In Silico Pharmacology. 2025;13(1):45. https://doi.org/10.1007/s40203-025-00337-1
32. Passaro A, Sanz JM, Naumovski N, Sergi D. The complex interplay between oxinflammation, mitochondrial dysfunction and lipotoxicity: Focus on their role in the pathogenesis of skeletal muscle insulin resistance and modulation by dietary fatty acids. Advances in Redox Research. 2024;11:100100. https://doi.org/10.1016/j.arres.2024.100100
33. Patra JK, Das G, Fraceto LF, Campos EVR, Rodriguez-Torres MDP, Acosta-Torres LS, Diaz-Torres LA, Grillo R, Swamy MK, Sharma S, Habtemariam S, Shin HS. Nano based drug delivery systems: Recent developments and future prospects. Journal of Nanobiotechnology. 2018;16(1):71. https://doi.org/10.1186/s12951-018-0392-8
34. Peng M, Shen P, Kim KJ. Effects of continuous glucose monitoring on physical activity and diet in diabetes: A systematic review and meta-analysis. The International Journal of Behavioral Nutrition and Physical Activity. 2026;23:14. https://doi.org/10.1186/s12966-025-01870-0
35. Petersen MC, Shulman GI. Mechanisms of Insulin Action and Insulin Resistance. Physiological Reviews. 2018;98(4):2133-2223. https://doi.org/10.1152/physrev.00063.2017