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ORIGINAL ARTICLE

Evaluation of Correlation between Plasma Glucose, Lipid Profile and Serum Amylase Among Obese Type 2 Diabetes Mellitus Patients

T. Rajalakshmi,[1,*] K. V. Vijayakumar[2] and K. Ashok[3]
[1]*Associate Professor, Department of Biochemistry, Govt Mohan Kumaramangalam Medical College, Salem, Tamilnadu, India*
[2]*Associate Professor, Department of Biochemistry, Government Medical College, Krishnagiri, Tamilnadu, India*
[3]*Senior Resident, Department of General Medicine, Government Mohan Kumaramangalam Medical College, Salem, Tamilnadu, India*

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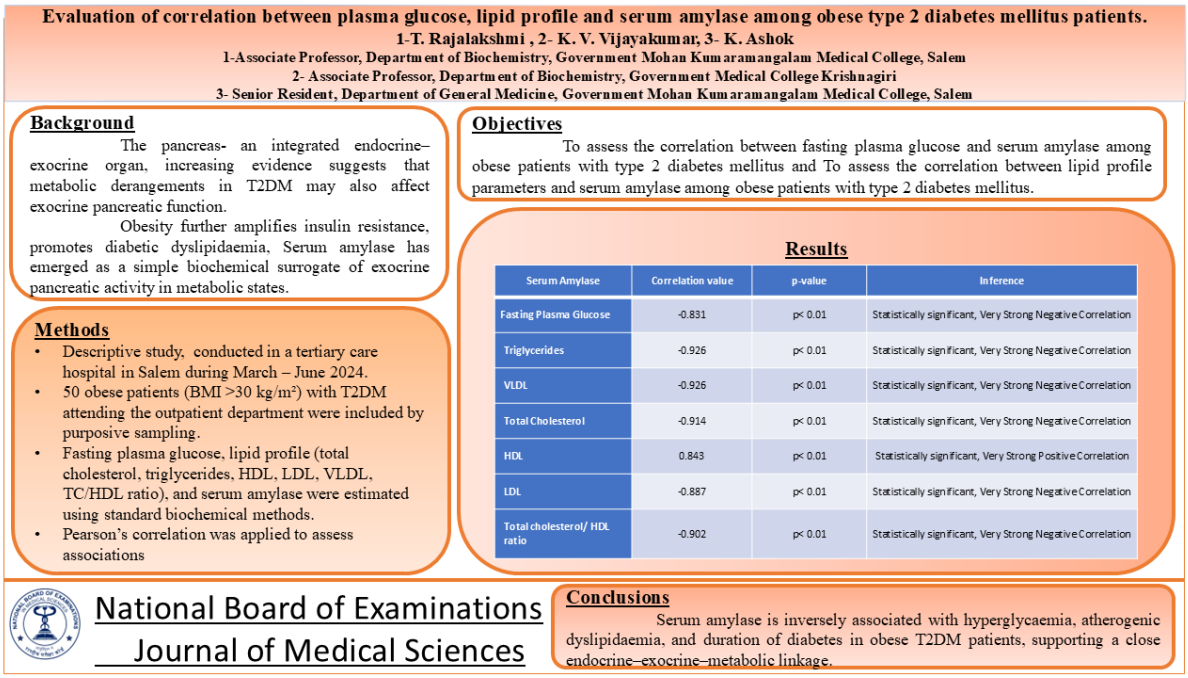
Abstract

Background: Type 2 diabetes mellitus (T2DM) is characterized by chronic hyperglycaemia, insulin resistance, and progressive β-cell dysfunction. The pancreas functions as an integrated endocrine–exocrine organ, and increasing evidence suggests that metabolic derangements in T2DM may also affect exocrine pancreatic function. Obesity further amplifies insulin resistance, promotes diabetic dyslipidaemia, and contributes to pancreatic fat deposition, potentially influencing pancreatic enzyme secretion. Serum amylase has emerged as a simple biochemical surrogate of exocrine pancreatic activity in metabolic states. **Objectives**: To assess the correlation between fasting plasma glucose and serum amylase among obese patients with type 2 diabetes mellitus; To assess the correlation between lipid profile parameters and serum amylase among obese patients with type 2 diabetes mellitus. **Methods**: This descriptive cross-sectional study was conducted in a tertiary care teaching hospital in Salem during March – June 2024. 50 obese patients (BMI >30 kg/m²) with T2DM attending the outpatient department were included by purposive sampling. Fasting plasma glucose, lipid profile (total cholesterol, triglycerides, HDL, LDL, VLDL, TC/HDL ratio), and serum amylase were estimated using standard biochemical methods. Pearson's correlation was applied to assess associations. **Results**: The mean age of participants was 54.2 ± 6.2 years, with male predominance (68%). Mean fasting plasma glucose was 146.82 ± 23.02 mg/dl. The lipid profile demonstrated an atherogenic pattern, and mean serum amylase levels were relatively low (28.30 ± 7.64 IU/L). Serum amylase showed a strong inverse correlation with fasting plasma glucose ($r = -0.831$, $p < 0.01$), triglycerides, VLDL, total cholesterol, LDL, and TC/HDL ratio, and a strong positive correlation with HDL cholesterol ($p < 0.01$). **Conclusion**: Serum amylase is inversely associated with hyperglycaemia, atherogenic dyslipidaemia, and duration of diabetes in obese T2DM patients, supporting a close endocrine–exocrine–metabolic linkage.

Keywords: Type 2 diabetes mellitus, Obesity, Serum amylase, Diabetic dyslipidaemia, Fasting plasma glucose

*Corresponding Author: T. Rajalakshmi
Email: drrajipop@gmail.com

Graphical Abstract



Introduction

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by persistent hyperglycaemia due to impaired insulin secretion, impaired insulin action, or both. In type 2 diabetes mellitus (T2DM), progressive β-cell dysfunction occurs on a background of insulin resistance, and fasting plasma glucose (FPG) is widely used as a pragmatic biochemical index of basal glycaemic status and hepatic glucose output [1,2].

The pancreas is a mixed exocrine–endocrine gland with a strong anatomical and functional interdependence between compartments. The exocrine pancreas constitutes approximately 85% of pancreatic mass, while endocrine islet tissue accounts for about 2%, with the remainder comprising ducts, vasculature and extracellular matrix [3]. Beyond simple co-localisation, contemporary evidence supports bidirectional endocrine–exocrine "crosstalk" mediated by shared developmental origins, vascular–paracrine signalling, neural inputs and gut–pancreas hormonal pathways, such that perturbations in one compartment can influence the other [4]. This biological linkage provides a clear biochemical rationale for examining endocrine markers (plasma glucose) alongside exocrine markers (serum pancreatic enzymes) within the same patient.

Serum amylase, produced predominantly by pancreatic acinar cells (with contribution from salivary isoenzymes), is an accessible, low-cost biochemical surrogate that may reflect aspects of exocrine pancreatic function in population studies. Importantly, "low" serum amylase has gained attention as a metabolic signal rather than only a pancreatitis marker; recent clinical reviews highlight that reduced serum amylase can be seen in metabolic states and may carry diagnostic or prognostic implications depending on context [5].

Obesity is a key modifier in this relationship and is central to the present

study focus. Obesity amplifies insulin resistance, increases inflammatory adipokines, and promotes ectopic fat deposition, including in the pancreas. Non-alcoholic fatty pancreatic disease (NAFPD)/pancreatic steatosis is increasingly described as a metabolic phenotype associated with obesity, insulin resistance and metabolic syndrome, with potential progression to pancreatic inflammation and fibrosis [6]. Pancreatic fat and low-grade inflammation may plausibly impair both β-cell function (worsening hyperglycaemia) and acinar/ductal function (altering enzyme output), thereby strengthening the biological rationale to examine glucose–amylase correlations specifically among obese T2DM patients [7].

In addition to abnormalities in glucose metabolism, T2DM is characteristically associated with diabetic dyslipidaemia, primarily due to insulin resistance–mediated alterations in hepatic lipid synthesis, impaired lipoprotein lipase activity [8].

Against this background, the present study evaluates the correlation between fasting plasma glucose and serum amylase as representative biochemical indices of endocrine and exocrine pancreatic function, respectively, in obese patients with T2DM and additionally their correlation with Lipid profile.

Objectives

- To assess the correlation between Fasting Plasma Glucose and serum amylase among Obese Type 2 Diabetes Mellitus.
- To assess the correlation between lipid profile and serum amylase among Obese Type 2 Diabetes Mellitus.

Methodology

This descriptive cross-sectional study was conducted in a tertiary care teaching hospital in Salem during March – June 2024. Sample size for this study was calculated to estimate the mean serum amylase with 95% confidence using $n=(Z\alpha*\sigma/d)^2$. The SD of serum amylase among T2DM patients was taken as 29.56 U/L from a published open-access study [9]. With Z=1.96 and allowable error (d) as 8 U/L, the required sample size arrived as 50. Hence, 50 patients of type 2 Diabetes mellitus with BMI > 30 (Obese as per WHO BMI categories) attending OPD, were included in this study by purposive sampling technique. After obtaining informed consent from the study recruits, Fasting Plasma Glucose, Lipid profile and serum Amylase were estimated. Patients with acute illness, those who were not on fasting and those who denied consent were excluded. Fasting Plasma Glucose was estimated by GOD-POD method. The serum Triglycerides and Total Cholesterol was measured by enzymatic GPO-PAP method. Serum HDL was measured by Phosphotungstic acid method and Serum VLDL & LDL were calculated by Friedwalds formula. Serum Amylase was estimated using a chromogenic substrate CNPG3(2-Chloro 4-NitroPhenyl linked with Galactomaltoside) which acts upon amylase to release more than 95% of 2-chloro 4-Nitrophenyl and forms 2- chloro, 4-Nitrophenyl D-Maltoside, Malotriose & Glucose. Amylase activity was measured at 405nm using Semi Auto Analyzer.

Results

Demographic and Clinical characteristics

This study included 50 participants, the mean age of them was 54.2 $\pm$ 6.2 years, ranging from 44 to 65 years, with a Male

predominance (68%)., indicating that the cohort predominantly represented middle-aged to elderly male individuals. The Mean BMI of the study participants was 31.92 ± 1.29 kg/m^2, ranging from 30.1 to 35.5 kg/m^2. Of the 50 study participants, 16 (32%) were with DM for < 5 years, 16 (32%) were with DM for 5-10 years and the rest 18 (36%) had DM for >10 years. Table 1 shows the demographic and clinical characteristics of the study population.

Table 1. Demographic and Clinical characteristics of study participants

Variable		**Descriptive Statistics**			
		Mean	**S.D.**	**Min.**	**Max.**
Age in yrs		54.2	6.15	44	65
BMI (kg/m^2)		31.92	1.29	30.07	35.55
		Frequency		**Percentage**	
Gender	**Male**	34		68	
	Female	16		32	
Duration of DM	**<5 years**	16		32	
	5-10 years	16		32	
	>10 years	18		36	

Fasting Plasma Glucose, Lipid Profile and Serum Amylase levels

With respect to glycaemic status, fasting plasma glucose levels demonstrated poor glycaemic control, with a mean value of 146.82 ± 23.02 mg/dl and a wide range from 114 to 192 mg/dl. Lipid profile analysis revealed dyslipidaemia typical of type 2 diabetes mellitus. Mean serum triglyceride (TGL) levels were 155.32 ± 29.83 mg/dl (range: 110–213 mg/dl), while very-low-density lipoprotein (VLDL) levels averaged 31.06 ± 5.96 mg/dl. Total cholesterol (TCHO) showed a mean of 167.54 ± 24.34 mg/dl, with values ranging from 131 to 212 mg/dl.

High-density lipoprotein (HDL) cholesterol levels were relatively low, with a mean of 33.98 ± 4.33 mg/dl, whereas low-density lipoprotein (LDL) cholesterol demonstrated a mean value of 102.49 ± 22.97 mg/dl. The total cholesterol to HDL ratio (TC/HDL), an established atherogenic index, was elevated with a mean of 5.08 ± 1.32, indicating increased cardiovascular risk within the study population.

Serum amylase levels, representing exocrine pancreatic function, were notably

on the lower side, with a mean value of 28.30 ± 7.64 IU/L and a range of 17.43 to 43.03 IU/L. Overall, the descriptive analysis highlights a cohort of obese T2DM patients with suboptimal glycaemic control, characteristic diabetic dyslipidaemia, and relatively reduced serum amylase levels, providing the basis for subsequent correlation analysis between fasting plasma glucose and serum amylase.

Table 2 shows the descriptive details of the Fasting plasma glucose, Lipid profile and Serum Amylase levels among the study participants.

Table 2. Descriptive data of Fasting Plasma Glucose, Lipid Profile and Serum Amylase

Variables	**Descriptive Data**			
	Mean	**S.D.**	**Min.**	**Max.**
Fasting Plasma Glucose (mg/dl)	146.82	23.02	114	192
TGL (mg/dl)	155.32	29.83	110	213
VLDL (mg/dl)	31.06	5.96	22	42.6
T.CHO (mg/dl)	167.54	24.34	131	212
HDL (mg/dl)	33.98	4.33	28	43
LDL (mg/dl)	102.49	22.97	68.8	148.2
TC/HDL ratio	5.08	1.32	3.3	7.32
Serum Amylase (IU/L)	28.3	7.64	17.43	43.03

Correlation of serum amylase with glycaemic and lipid parameters

Pearson's correlation analysis demonstrated a very strong and statistically significant association between serum amylase levels and multiple metabolic parameters in the study population (Table 3). Serum amylase showed a very strong negative correlation with fasting plasma glucose ($r = -0.831$, $p < 0.01$), indicating that higher glycaemic levels were associated with progressively lower serum amylase concentrations.

Table 3. Correlation of serum amylase with glycaemic and lipid parameters

Serum Amylase	Correlation value	p-value	Inference
Fasting Plasma Glucose (mg/dl)	-0.831	p< 0.01	Statistically significant, Very Strong Negative Correlation
Triglycerides (mg/dl)	-0.926	p< 0.01	Statistically significant, Very Strong Negative Correlation
VLDL (mg/dl)	-0.926	p< 0.01	Statistically significant, Very Strong Negative Correlation
Total Cholesterol (mg/dl)	-0.914	p< 0.01	Statistically significant, Very Strong Negative Correlation
HDL(mg/dl)	0.843	p< 0.01	Statistically significant, Very Strong Positive Correlation
LDL (mg/dl)	-0.887	p< 0.01	Statistically significant, Very Strong Negative Correlation
Total cholesterol/ HDL ratio	-0.902	p< 0.01	Statistically significant, Very Strong Negative Correlation

Similarly, serum amylase exhibited a very strong inverse correlation with serum triglycerides ($r = -0.926$, $p < 0.01$) and very-low-density lipoprotein (VLDL) levels ($r = -0.926$, $p < 0.01$), suggesting a close relationship between exocrine pancreatic enzyme levels and atherogenic lipid fractions. A significant negative correlation was also observed between serum amylase and total cholesterol ($r = -0.914$, $p < 0.01$) as well as low-density lipoprotein (LDL) cholesterol ($r = -0.887$, $p < 0.01$).

In contrast, high-density lipoprotein (HDL) cholesterol demonstrated a very strong positive correlation with serum amylase ($r = 0.843$, $p < 0.01$), indicating that higher protective lipid levels were associated with higher serum amylase concentrations. Furthermore, the total cholesterol to HDL ratio, an established marker of cardiovascular risk, showed a

strong inverse relationship with serum amylase ($r = -0.902$, $p < 0.01$).

Overall, these findings indicate that lower serum amylase levels are consistently associated with poorer glycaemic control and an adverse lipid profile among obese patients with type 2 diabetes mellitus, supporting a close functional relationship between exocrine pancreatic activity, glucose metabolism, and lipid abnormalities.

Discussion

The present study comprised predominantly middle-aged type 2 diabetes mellitus, with a mean age of 54.2 ± 6.15 years, and a mean BMI of 31.92 ± 1.29 kg/m^2. This is consistent with epidemiological studies indicating that T2DM commonly manifests in the fifth and sixth decades of life, where cumulative insulin resistance and β-cell dysfunction increase with age and obesity [7,10].

In this cohort of obese T2DM patients, had a suboptimal fasting glycaemic control. The lipid profile showed an atherogenic pattern suggesting increased cardiometabolic risk. Serum amylase levels were relatively low in this cohort supporting the rationale to evaluate exocrine–metabolic correlations in obese T2DM. This biochemical pattern is consistent with the Indian description of diabetic (atherogenic) dyslipidaemia, where high triglycerides and low HDL-C are emphasized as typical and clinically important in T2DM; this is explicitly highlighted in the RSSDI consensus recommendations for dyslipidaemia management in diabetes [11].

In Indian clinical cohorts, dyslipidaemia is reported to be highly prevalent among patients with T2DM and contributes substantially to atherosclerotic risk, aligning with the elevated TG and low HDL observed in the present study [11].

Regarding exocrine markers, Indian studies such as Patel et al. have also reported altered serum amylase in T2DM and described an inverse relationship with glycaemic measures, supporting the clinical relevance of including serum amylase in metabolic profiling [9].

The present study demonstrated a very strong and statistically significant inverse correlation between serum amylase and fasting plasma glucose indicating that worsening glycaemic status was associated with progressively lower serum amylase levels in obese patients with T2DM. This finding suggests a close functional link between endocrine pancreatic dysfunction and exocrine pancreatic activity in the diabetic state. Comparable findings have been reported by many Indian literatures as they observed significantly lower serum amylase levels in patients with type 2 diabetes mellitus and reported a negative association between serum amylase and glycaemic parameters, supporting the present observation that hyperglycaemia is associated with reduced exocrine enzyme levels [9,12-14].

In addition to glycaemia, serum amylase showed a very strong negative correlation with triglycerides, VLDL, total cholesterol, LDL cholesterol and TC/HDL ratio while demonstrating a strong positive correlation with HDL cholesterol all of which were statistically significant ($p < 0.01$). These findings indicate that lower serum amylase levels are closely associated with a more atherogenic lipid profile in obese T2DM patients, as emphasized in the RSSDI consensus recommendations (2022) [11].

Although Indian studies directly correlating serum amylase with lipid

fractions are limited, the observed associations are biologically plausible and align with reports suggesting that pancreatic fat accumulation, insulin resistance, and altered lipid metabolism may collectively impair exocrine pancreatic function.

A plausible interpretation is that obesity-driven insulin resistance may simultaneously worsen fasting glycaemia and promote atherogenic dyslipidaemia, while chronic metabolic stress and pancreatic fat/inflammation may contribute to lower exocrine enzyme output, reflected as reduced serum amylase in susceptible T2DM phenotypes. Concurrently, the same metabolic milieu promotes diabetic dyslipidaemia, thereby explaining the strong associations between serum amylase and adverse lipid parameters observed in this study [11,15].

Conclusion

Thus, Our study demonstrates an evident relationship between glycaemic status, lipid abnormalities, and exocrine pancreatic function among obese patients with type 2 diabetes mellitus. These findings support the concept of an integrated endocrine–exocrine–metabolic dysfunction in obesity-associated T2DM, wherein chronic hyperglycaemia, insulin resistance, and dyslipidaemia collectively contribute to pancreatic functional alterations. However, larger prospective studies incorporating direct measures of exocrine pancreatic function and pancreatic imaging are warranted to validate these observations and to clarify their clinical implications.

Limitations

The cross-sectional study design limits causal inference. Purposive sampling may introduce selection bias and restrict generalizability. Potential confounders including age, sex, duration of diabetes, glycaemic control, alcohol intake, and concurrent medications (insulin and lipid-lowering agents) may have influenced serum amylase and lipid parameters and could not be fully adjusted for. BMI categorization was based on WHO criteria; the use of Asian/Indian–specific cut-offs may have been more appropriate and could limit generalizability to the Indian population. These factors should be considered when interpreting the findings.

Clinical Implications

Serum amylase, a simple and easily available test, may serve as an adjunct marker of metabolic burden and possible exocrine pancreatic involvement in obese patients with type 2 diabetes mellitus, particularly in resource-limited settings. The observed associations warrant larger longitudinal studies incorporating direct exocrine function tests and pancreatic imaging to determine its predictive value and clinical utility.

Statements and Declarations

Conflicts of interest

The authors declare that they do not have conflict of interest.

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Use of AI tool: Authors declare to have used Grammarly software to enhance the grammar and readability of the article, but have rechecked its contents before submission.

References

1. Dludla, P. V., Mabhida, S. E., Ziqubu, K., Nkambule, B. B., Mazibuko-Mbeje, S. E., Hanser, S., Basson, A. K., Pheiffer, C., & Kengne, A. P. Pancreatic β-cell dysfunction in type 2 diabetes: Implications of inflammation and oxidative stress. World journal of diabetes, 2023;14(3):130–146. https://doi.org/10.4239/wjd.v14.i3.130
2. Mittendorfer, B., van Vliet, S., Smith, G. I., Petersen, M. C., Patterson, B. W., & Klein, S. Impaired plasma glucose clearance is a key determinant of fasting hyperglycemia in people with obesity. Obesity (Silver Spring, Md.), 2024;32(3):540–546. https://doi.org/10.1002/oby.23963
3. Hopson P, Jayasinghe S, Donnan C, Pennock J, Shields MD, Sassano MF, et al. Assessment of exocrine pancreatic function in children and adolescents: a review of current methods and clinical applications. Front Pediatr. 2022;10:908542
4. Hu, C., Chen, Y., Yin, X. et al. Pancreatic endocrine and exocrine signaling and crosstalk in physiological and pathological status. Sig Transduct Target Ther 2025;10:39. https://doi.org/10.1038/s41392-024-02098-3
5. Jalal, M., Gbadegesin, S. A., Tehami, N., & Nakajima, K. What is the clinical significance of low serum amylase? Systematic review of the conditions associated with low serum amylase. Frontline gastroenterology, 2023;15(2):154–161. https://doi.org/10.1136/flgastro-2023-102405
6. Habas, E., Farfar, K., Habas, E., Rayani, A., & Elzouki, A. N. Extended Review and Updates of Nonalcoholic Fatty Pancreas Disease. Saudi Journal of Medicine & Medical Sciences, 2024;12(4):284–291. https://doi.org/10.4103/sjmms.sjmms_526_23
7. Singh P, Singh K, Dhanju AS, Singh GB, Batra P, Kaur A. The association of non-alcoholic fatty pancreas disease and glycemic status. Int J Adv Med. 2022;9(6):677–682. Available from: http://www.ijmedicine.com
8. Dilworth L, Facey A, Omoruyi F. Diabetes mellitus and its metabolic complications: the role of adipose tissue. Int J Mol Sci. 2021;22(14):7644
9. Patel AV, Daruwala AN, Kachhiya SM. The assessment of serum amylase in patients with type-II diabetes mellitus. Int J Res Med Sci. 2023;11(8):2961–2965. Available from: https://www.msjonline.org
10. Anjana RM, Mohan V. Diabetes in India: epidemiology and pathophysiology. Indian J Med Res. 2021;154(4):1–10. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8575896/
11. Saboo B, Agarwal S, Makkar BM, Chawla R, Ghosh S, Viswanathan V, et al. RSSDI consensus recommendations for dyslipidemia management in diabetes mellitus. Int J Diabetes Dev Ctries. 2022;42(1):3–28.
12. Kumar A, Itagi AS, Chetan TL, Neeralagi S. Study of serum amylase levels in patients with type 2 diabetes mellitus in a tertiary care hospital. Asian J Med Res. 2022;11(2):1–6. doi:10.47009/ajmr.2022.11.2.1.

Available from: https://aijournals.com/index.php/ajmr/article/download/2279/3094/

13. Madole, M. B., Iyer, C. M., Madivalar, M. T., Wadde, S. K., & Howale, D. S. Evaluation of Biochemical Markers Serum Amylase and Serum Lipase for the Assessment of Pancreatic Exocrine Function in Diabetes Mellitus. Journal of Clinical and Diagnostic Research JCDR, 2016;10(11):BC01–BC04. https://doi.org/10.7860/JCDR/2016/23787.8900
14. Sarathi, V., Tirupati, S., Sabinkar, G., & Mohan, R. Hyperamylasemia is not Associated with Dipeptidyl Peptidase 4 Inhibitors in South Indian Adults with Type 2 Diabetes Mellitus. International Journal of Applied & Basic Medical Research, 2023;13(2):113–116. https://doi.org/10.4103/ijabmr.ijabmr_503_22
15. Mehta, R. K., Koirala, P., Mallick, R. L., Parajuli, S., & Jha, R. Dyslipidemia in Patients with Type 2 Diabetes Mellitus in a Tertiary Care Centre: A Descriptive Cross-sectional Study. JNMA Journal of the Nepal Medical Association, 2021;59(236):305–309. https://doi.org/10.31729/jnma.6278