Original Article

Exploring the Differences in Serum Amylase Levels among Obese and Non-obese Individuals with Type 2 Diabetes Mellitus

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ABSTRACT

Background

The pancreas is a double-entity organ, and previous studies have shown that a significant proportion of patients with diabetes mellitus and obesity have pancreatic exocrine insufficiency (PEI). Increased risks of metabolic disorders, metabolic syndrome, and diabetes were linked to low serum amylase levels.

Methodology

Study population was be taken from patients visiting to K.R Hospital, Mysore with type-2 diabetes mellitus for follow up and they are grouped into two, based on their BMI as obese diabetics & non-obese Diabetics.

Result

The present study showed statistically decreased Serum Amylase in obese diabetics when compared with non-obese type 2 diabetic and increased Total Cholesterol/ HDL ratio. Present study also shows increased serum Triglyceride, Total cholesterol, LDL, VLDL in obese diabetic when compared with non-obese type 2 diabetes but not statistically significant. The study showed negative correlation between Serum Amylase with blood glucose & serum HDL but not statistically significant.

Conclusion

The current study findings suggest that lower serum amylase levels in obese people with type 2 diabetes may be linked to an exocrine-endocrine axis disruption, which impairs exocrine pancreatic function. This could then be utilized as a potential marker to evaluate the exocrine functions of the pancreas.

Keywords

Type 2 diabetes; Obese; Serum amylase; Lipid profile

INTRODUCTION

Diabetes mellitus is a chronic metabolic disorder due to derangement in beta cells of pancreas leading to hyperglycemia. The pancreas being a dual function organ involving endocrine and exocrine actions it is involved also in the digestion, absorption, and metabolism of nutrients [1].

When a person's Body Mass Index (BMI) is over 30 kg/m2, they are considered obese. Of the more than 1.9 billion people who were overweight in 2014, over 650 million adults were obese. Currently regarded as a pandemic, it can impact 10–30% of the global adult population [2,3].

About 50% of individuals with insulin-dependent diabetes (IDDM) and 30–50% of

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patients with non-insulin-dependent diabetes (NIDDM or Type 2 DM) have pancreatic insufficiency [4]. The research done in both animal models and humans with type 2 diabetes, the endocrine and exocrine pancreas loses its continuous interstitial matrix link, resulting in a malfunctioning insulin-acinar-ductal-incretin gut hormonal axis [5]. In addition, the production and release of enzymes are impacted by abnormalities in insulin secretion and signaling pathways [6].

Pancreas Exocrine Insufficiency [PEI] leads to difficulty in digestion due to decreased levels of certain enzymes such as lipases, amylase etc. This can also lead to malabsorption and deficiencies, especially of fat-soluble vitamins A, D, E & K[7]. The raised serum amylase levels were used for detection of acute pancreatitis [8,9] along with diabetic ketoacidosis [10,11] and renal insufficiency [12,13]. In other studies, the decreased amylase levels seen in patients with chronic pancreatitis [14]. An elevated risk of metabolic disorders, abnormal glucose metabolism, insulin resistance, and decreased insulin secretion may be indicated by low serum amylase levels. Serum amylase levels could also be a promising diagnostic that is strongly linked to the general activity of islet β cells [15].

One of the main causes of the pathophysiology of insulin resistance (IR) is abnormalities in lipid metabolism. Numerous studies indicate that insulin resistance, which is mediated by hyperinsulinemia, increases the production of VLDL in the liver and is responsible for the higher levels of plasma triglycerides seen in healthy individuals of normal weight, obese individuals without diabetes, and individuals with type 2 diabetes [16,17,18].

Only few studies have examined the association between Serum Amylase in type 2 DM patients in relation to body mass index. Thus, this study was undertaken to estimate the levels of serum amylase in type 2 diabetes and to correlate it with glucose & lipid profile.

MATERIALS AND METHODS

The study was a cross-sectional study conducted on patients visiting to K.R Hospital, Mysore with type-2 diabetes mellitus for follow up and they are grouped into two, based on their BMI as obese diabetics & non-obese Diabetics. The study was conducted with approval of Institutional Time Bound Research Committee (ECR/134/Inst/KA/2013/RR-19) and all participants include with the written informed consent. Total 100 subjects included in the study with an age group of 30-60 years of both genders. The study excluded following subjects from the study patients with type-1 diabetes, gestational diabetes, liver dysfunction, renal dysfunction and systemic diseases.

Table 1: Showing the various parameters estimated among non-obese and obese diabetic subjects.

Parameters	Diabetic patients with obesity (50)	Diabetic patients without obesity (50)	p value
Serum amylase (U/L)	58.85±20.03	78.49±30.9	**0.002**
Total cholesterol (mg/dl)	186.68±44.21	172.70±49.97	0.110
Triglycerides(mg/dl)	223.85±127.14	187.81±183.30	**0.019**
HDL Cholesterol (mg/dl)	41.86±14.74	46.14±14.15	0.137
LDL Cholesterol (mg/dl)	100.11±40.69	92.70±44.24	0.462
VLDL Cholesterol (mg/dl)	44.71±25.42	38.86±39.74	**0.027**
Total Cholesterol/ HDL	7.40±15.09	3.92±1.62	**0.005**
FBS (mg/dl)	136.86±67.62	163.43±84.01	0.330
PPBS (mg/dl)	232.07±103.95	247.92±125.96	0.796
Waist (CM)	111.36±20.51	86.57±15.44	**<.001**
BMI	32.35±3.60	21.21±2.23	**<.001**

The study population was divided into 2 groups based on BMI as per WHO Criteria. Groups-1with BMI18-24.9 as non-obese diabetics &Group 2 with BMI >30 as obese Diabetic subjects.

The demographic details were collected from the subjects which includes age, gender, occupation, diet, physical activity, BMI, Blood pressure and others was extracted from questionnaire.

A 3ml of fasting venous sample was collected from patients in a plain vaccutainer under aseptic precautions & serum was analyzed immediately for serum glucose, lipid profile and serum Amylase by enzymatic method using fully automated chemistry analyzer Cobas 6000. The obtained data entered in the excel and analyzed using Epi info software.

Ethical clearance

ECR/134/Inst/KA/2013/RR-19

RESULTS

The study results showed serum amylase is significantly lower in diabetic patients with obesity compared to those without obesity (p = 0.002). Whereas the triglycerides (p = 0.002), VLDL Cholesterol (p = 0.027), Total Cholesterol/HDL ratio (p = 0.005), Waist circumference (p < 0.001) and BMI (p < 0.001) significantly higher in diabetic patients with obesity compared to those without obesity as shown in table 1.

Table 2 shows the statistically significant changes in waist and BMI among obese and Non-obese male subjects but all other parameters were non-significant. In females we found statistically significant changes in waist, BMI, Serum amylase, TG, VLDL cholesterol levels.

Table 2: Showing parameters among obese and non-obese male and female subjects.

	Non-Obese male (25)	Obese male (24)		Non-Obese Female (25)	Obese Female (26)	
	Mean ±SD	Mean± SD	P Value	Mean ±SD	Mean± SD	P Value
Age (years)	48.47±12.84	48.00±10.05	.926	40.75 ± 20.03	48.11 ± 9.00	0.208
Waist(cm)	86.42±20.06	103.20±18.28	**.016**	86.75 ± 7.58	116.80 ± 20.57	**< 0.001**
BMI	21.64±2.47	31.10±1.25	**<.001**	20.69 ± 1.84	33.17 ± 4.40	**< 0.001**
FBS (mg/dl)	174.00±85.70	128.70±71.65	.182	145.48 ± 73.44	149.99 ± 77.77	0.852
PPBS (mmg/dl)	271.69±122.71	238.85±133.09	.590	219.83 ± 124.87	229.60 ± 92.78	0.77
Serum amylase (U/L)	86.68±38.29	65.94±22.54	**0.04**	72.31 ± 15.42	52.45 ± 15.00	**0.006**
Total cholestrol (mg/dl)	170.29±41.22	188.27±42.35	.237	172.14 ± 57.79	187.11 ± 49.12	0.423
Triglycerides (mg/dl)	245.11±236.12	282.58±170.20	.836	124.45 ± 70.88	176.56 ± 53.27	**0.019**
HDL Cholestrol (mg/dl)	43.11±15.53	34.16±12.95	.089	48.80 ± 12.17	48.01 ± 13.55	0.749
TC/HDL ratio	4.13±0.98	11.77±22.69	.268	3.83 ± 2.07	4.13 ± 1.50	0.613
LDL Cholestrol (mg/dl)	85.32±41.02	97.60±36.44	.724	98.45 ± 47.11	103.79 ± 45.09	0.832
VLDL Cholestrol (mg/dl)	52.25±51.86	56.52±34.04	.913	24.89 ± 14.18	35.31 ± 10.65	**0.019**
LDL/HDL ratio	2.06±0.96	7.25±15.83	.296	2.26 ± 1.70	2.37 ± 1.29	0.827
Chol/HDL	4.13±0.98	11.77±22.69	.268	3.83 ± 2.07	4.14 ± 1.49	0.609
TG/HDL	6.22±5.40	17.63±34.66	.284	2.86 ± 2.08	3.85 ± 1.37	0.098
FPG/HDL	4.28±2.10	8.33±17.34	.455	3.45 ± 2.80	3.22 ± 1.42	0.647

Furthermore, comparison among gender-wise as shown in Table 3 found a statistically significant values in HDL cholesterol levels whereas all other parameters showed no significance.

Table 3: Gender wise comparison among obese subjects

Parameter	Obese Female (26)	Obese Male (24)	p Value
Age (years)	48.11 ± 9.00	48.00 ± 10.05	0.78
Waist (cm)	116.80 ± 20.57	103.20 ± 18.28	0.535
BMI	33.17 ± 4.40	31.10 ± 1.25	0.195
FBS (mg/dl)	149.99 ± 77.77	128.70 ± 71.65	0.461
PPBS (mg/dl)	229.60 ± 92.78	238.85 ± 133.09	0.947
Serum Amylase (U/L)	52.45 ± 15.00	65.94 ± 22.54	0.112
Total Cholesterol (mg/dl)	187.11 ± 49.12	188.27 ± 42.35	0.569
Triglycerides (mg/dl)	176.56 ± 53.27	282.58 ± 170.20	0.069
HDL Cholesterol (mg/dl)	48.01 ± 13.55	34.16 ± 12.95	0.029
TC/HDL Ratio	4.13 ± 1.50	11.77 ± 22.69	0.286
LDL Cholesterol (mg/dl)	103.79 ± 45.09	97.60 ± 36.44	0.922
VLDL Cholesterol (mg/dl)	35.31 ± 10.65	56.52 ± 34.04	0.069
LDL/HDL Ratio	2.37 ± 1.29	7.25 ± 15.83	0.325
Chol/HDL Ratio	4.14 ± 1.49	11.77 ± 22.69	0.286
TG/HDL Ratio	3.85 ± 1.37	17.63 ± 34.66	0.213
FPG/HDL Ratio	3.22 ± 1.42	8.33 ± 17.34	0.354

Table 4 shows, that age significantly positively correlated with TC/HDL ratio, LDL cholesterol, LDL/ HDL ratio, and Cholesterol/HDL ratio among female subjects whereas in males it was negatively correlated but was not significant.

In males, BMI was significantly negatively correlated with LDL cholesterol whereas in females negative but non-significant.

In females, FBS significantly positively correlated with PPBS, TC, LDL and FPG/HDL ratio, whereas in males shown significantly positively correlated with TG, and VLDL cholesterol levels.

Serum amylase levels showed a positive significant correlation with TC/HDL ratio, LDL/ HDL ratio, Cholesterol/HDL ratio and TG/HDL ratio, but in males negatively correlated with no significance.

Table 4: Shows the correlation of age, waist, BMI, FBS, and PPBS with different parameters among male and female obese subjects

	Age	Waist	BMI	FBS	PPBS	Serum amylase	TC	TG	HDL Cholesterol (mg/dl)	TC/HDL ratio	LDL Cholesterol (mg/dl)	VLDL Cholesterol (mg/dl)	LDL/HDL ratio	Chol/HDL Ratio	TG/HDL Ratio	FPG/HDL Ratio
Females Obese																
Age	1	-.373	-.164	.113	.233	.010	.450	.026	-.302	**.604****	**.574***	.026	**.632****	**.605****	.319	.272
		.127	.516	.654	.353	.971	.061	.918	.223	**.008**	**.013**	.918	**.005**	**.008**	.197	.274
Waist	-.373	1	.100	-.140	-.276	-.248	-.123	-.164	.001	-.180	-.096	-.164	-.162	-.181	-.225	-.231
	.127		.692	.579	.267	.354	.626	.516	.998	.476	.704	.516	.521	.471	.369	.357
BMI	-.164	.100	1	-.111	-.131	-.227	.045	.221	.212	-.195	-.067	.221	-.221	-.196	-.024	-.217
	.516	.692		.662	.605	.398	.860	.378	.399	.437	.791	.378	.379	.436	.925	.388
FBS	.113	-.140	-.111	1	**.918****	-.244	**.540***	.016	.316	.077	**.490***	.016	.123	.079	-.152	**.803****
	.654	.579	.662		**<.001**	.362	**.021**	.950	.202	.762	**.039**	.950	.628	.756	.546	**<.001**
	18	18	18	18	18	16	18	18	18	18	18	18	18	18	18	18
PPBS	.233	-.276	-.131	**.918****	1	-.214	**.514***	.132	.221	.189	.462	.132	.214	.192	.035	**.854****
	.353	.267	.605	**<.001**		.426	**.029**	.601	.379	.452	.054	.601	.394	.445	.891	**<.001**
Sr Amylase	.010	-.248	-.227	-.244	-.214	1	.227	.226	-.338	**.576***	.296	.226	**.535***	**.575***	**.627****	.040
	.971	.354	.398	.362	.426		.397	.399	.200	**.020**	.265	.399	**.033**	**.020**	**.009**	.883
Male Obese																
Age	1	.145	.327	.046	.524	.253	-.067	-.147	.493	-.270	-.116	-.147	-.263	-.270	-.285	-.257
		.653	.300	.887	.098	.428	.836	.648	.103	.395	.721	.648	.409	.395	.370	.420
Waist	.145	1	-.130	-.080	.071	.087	-.150	-.070	-.120	.100	-.067	-.070	.102	.100	.094	.092
	.653		.687	.805	.837	.789	.641	.829	.710	.758	.837	.829	.753	.758	.771	.775
BMI	.327	-.130	1	.431	.021	-.465	-.120	.293	.569	-.184	**-.615***	.293	-.208	-.184	-.127	-.118
	.300	.687		.162	.952	.127	.711	.355	.054	.568	**.033**	.355	.517	.568	.693	.714
FBS	.046	-.080	.431	1	.508	-.178	.331	**.727****	.132	.156	-.341	**.727****	.119	.156	.240	.254
	.887	.805	.162		.111	.579	.293	**.007**	.682	.628	.279	**.007**	.714	.628	.452	.426
PPBS	.524	.071	.021	.508	1	.273	.559	.569	.091	.040	.107	.569	.020	.040	.086	.066
	.098	.837	.952	.111		.416	.074	.068	.790	.906	.753	.068	.953	.906	.801	.847
Sr Amylase	.253	.087	-.465	-.178	.273	1	.297	-.130	.080	-.304	.438	-.130	-.286	-.304	-.343	-.348
	.428	.789	.127	.579	.416		.349	.686	.804	.336	.154	.686	.367	.336	.276	.268

The lipid profile correlation is shown in Table 5. It showed TC in females was positively significantly correlated with TG, TC/HDL ratio, LDL cholesterol, VLDL cholesterol, LDL/ HDL ratio and Cholesterol/ HDL ratio, but in males, it was correlated with TG, LDL cholesterol and VLDL cholesterol only.

The HDL cholesterol levels in the male and female groups showed a significant negative correlation TC/HDL ratio, LDL/HDL ratio, Cholesterol/HDL ratio and TG/HDL ratio.

The LDL cholesterol in females showed positive significant correlation with TC, TC/HDL ratio, LDL/HDL ratio, Cholesterol/ HDL ratio and FPG/ HDL ratio, whereas in males it was positively significant correlation with TC.

Table 5 : Showing lipid status correlation in obese female and male subjects

Male Group-Lipid status										
	TC	TG	HDL Cholesterol (mg/dl)	TC/HDL ratio	LDL Cholesterol (mg/dl)	VLDL Cholesterol (mg/dl)	LDL/HDL ratio	Chol/HDL Ratio	TG/HDL Ratio	FPG/HDL Ratio
TC	1	**.482***	.225	**.592****	**.908****	**.482***	**.611****	**.592****	.344	.422
		.043	.370	**.010**	**<.001**	**.043**	**.007**	**.010**	.162	.081
TG	**.482***	1	.436	.166	.158	**1.000****	.072	.164	.553*	-.066
	.043		.070	.511	.531	**<.001**	.776	.515	.017	.795
HDL	.225	.436	1	**-.607****	-.159	.436	**-.602****	**-.608****	**-.484***	-.239
	.370	.070		**.008**	.529	.070	**.008**	**.007**	**.042**	.339
TC/HDL ratio	**.592****	.166	**-.607****	1	**.789****	.166	**.991****	**1.000****	**.781****	**.478***
	.010	.511	**.008**		**<.001**	.511	**<.001**	**<.001**	**<.001**	**.045**
LDL	**.908****	.158	-.159	**.789****	1	.158	**.829****	**.789****	.391	**.548***
	<.001	.531	.529	**<.001**		.531	**<.001**	**<.001**	.109	**.019**
VLDL	**.482***	**1.000****	.436	.166	.158	1	.072	.164	**.553***	-.066
	.043	**<.001**	.070	.511	.531		.776	.515	**.017**	.795
LDL/HDL ratio	**.611****	.072	**-.602****	**.991****	**.829****	.072	1	**.991****	**.691****	**.495***
	.007	.776	**.008**	**<.001**	**<.001**	.776		**<.001**	**.001**	**.037**
Chol/HDL ratio	**.592****	.164	**-.608****	**1.000****	**.789****	.164	**.991****	1	**.781****	**.481***
	.010	.515	**.007**	**<.001**	**<.001**	.515	**<.001**		**<.001**	**.044**
TG/HDL ratio	.344	**.553***	**-.484***	**.781****	.391	**.553***	**.691****	**.781****	1	.284
	.162	**.017**	**.042**	**<.001**	.109	**.017**	**.001**	**<.001**		.253
FPG/HDL ratio	.422	-.066	-.239	**.478***	**.548***	-.066	**.495***	**.481***	.284	1
	.081	.795	.339	**.045**	**.019**	.795	**.037**	**.044**	.253	
Male Group- Lipid status										
TC	1	**.691***	-.215	.289	**.593***	**.691***	.271	.289	.327	.267
		.013	.502	.362	**.042**	**.013**	.394	.362	.299	.402
TG	**.691***	1	-.040	.123	-.116	**1.000****	.077	.122	.226	.171
	.013		.902	.704	.719	**<.001**	.812	.705	.481	.595

Male Group-Lipid status										
	TC	TG	HDL Cholesterol (mg/dl)	TC/HDL ratio	LDL Cholesterol (mg/dl)	VLDL Cholesterol (mg/dl)	LDL/HDL ratio	Chol/HDL Ratio	TG/HDL Ratio	FPG/HDL Ratio
HDL	-.215	-.040	1	**-.794****	-.568	-.040	-.798**	**-.794****	**-.777****	**-.757****
	.502	.902		**.002**	.054	.902	.002	**.002**	**.003**	**.004**
TC/HDL ratio	.289	.123	**-.794****	1	.504	.123	**.999****	**1.000****	**.994****	**.993****
	.362	.704	**.002**		.095	.704	**<.001**	**<.001**	**<.001**	**<.001**
LDL	**.593***	-.116	-.568	.504	1	-.116	.527	.504	.446	.419
	.042	.719	.054	.095		.719	.078	.095	.146	.175
VLDL	**.691***	**1.000****	-.040	.123	-.116	1	.077	.122	.226	.171
	.013	**<.001**	.902	.704	.719		.812	.705	.481	.595
LDL/HDL ratio	.271	.077	**-.798****	**.999****	.527	.077	1	**.999****	**.987****	**.988****
	.394	.812	**.002**	**<.001**	.078	.812		**<.001**	**<.001**	**<.001**
Chol/HDL ratio	.289	.122	**-.794****	**1.000****	.504	.122	**.999****	1	**.994****	**.993****
	.362	.705	**.002**	**<.001**	.095	.705	**<.001**		**<.001**	**<.001**
TG/HDL ratio	.327	.226	**-.777****	**.994****	.446	.226	**.987****	**.994****	1	**.995****
	.299	.481	**.003**	**<.001**	.146	.481	**<.001**	**<.001**		**<.001**
FPG/HDL ratio	.267	.171	**-.757****	**.993****	.419	.171	**.988****	**.993****	**.995****	1
	.402	.595	**.004**	**<.001**	.175	.595	**<.001**	**<.001**	**<.001**	

DISCUSSION

The studies have suggested that, two parts of the pancreas interact anatomically and functionally and impairment of one can impact the other. In diabetes mellitus, reduced pancreatic endocrine activity may impact its exocrine function, leading to malnutrition and poor digestion. Evidence indicated that the pancreatic endocrine hormones, particularly insulin, which has a trophic effect on the exocrine acinar cells, regulate the pancreatic exocrine function [19, 20]. Through a variety of methods, including the modulation of amylase gene transcription, activation of DNA, RNA, and acinar protein synthesis, and an increase in glucose uptake, insulin is thought to bind to its own receptor on the acinar cell, thereby stimulating and potentiating amylase production [21, 22]. A variety of enzymes, such as lipase and amylase, are produced by the pancreatic exocrine acinar cells to aid in the digestion of specific food particles. During digestion, amylase main function is to break down starch into maltose, maltotriose, and α-limit dextrins. Lipase is mostly produced by the pancreas and facilitates the conversion of triglycerides into monoglycerides and fatty acids [23]. The exocrine function, which involves effective food digestion, may also impact on glycemic control. Pancreatic exocrine insufficiency may be more common in people with obese diabetes and is less studied among Indian population. Effective and consistent digestion of meals will help to stabilize blood glucose levels and which will also affect insulin sensitivity and insulin requirements. Hence study was undertaken to understand the role of amylase in glycemic control, dyslipidemia among type 2 Diabetes Mellitus subjects. Hence we have studied the activities of serum amylase to understand the

effect on lipid metabolism by the exocrine pancreatic status. Furthermore, the autonomic nervous system and the naturally occurring gut chemicals secretin and cholecystokinin regulate the secretion of pancreatic juice. The well-known consequences of autonomic neuropathy and microvascular difficulties, which are so prevalent in diabetes mellitus, frequently cause this intricate balance to be disrupted [4]. The present study showed statistically decreased Serum Amylase in obese diabetes with mean standard deviation of 58.85±20.03 when compared with non-obese type 2 diabetic with mean standard deviation of 78.49±30.9, these results are in agreement with the studied done by Rakhee Yadav et al [1] As per studies done by Gupta et al decreased levels of serum amylase seen in patients with chronic pancreatitis [14], which suggests that in obese diabetic there is ongoing chronic pancreatic changes leading to chronic pancreatitis. According to a prior research done in middle-aged, asymptomatic adults by Muneyuki et al [24], showed low blood amylase levels were linked to higher insulin resistance and lower basal insulin levels and secretion.

Low serum amylase levels were linked to an increased risk of metabolic abnormalities, metabolic syndrome, and diabetes, according to recent research by Nakajima [15] and Lee JG [25]. Previous research has examined the fundamental processes via which insulin controls the production of amylase by pancreatic acinar cells. By attaching itself to its own receptor on acinar cells, insulin stimulates and amplifies the release of amylase through a variety of signaling pathways, such as controlling the transcription of the amylase gene and promoting the production of the matching protein in acinar cells [26].

In both healthy and diabetic rats, Patel et al. study demonstrated that insulin may increase the production of amylase from acinar cells; however, the impact was far less effective in diabetic rats. Additionally, research on diabetes mellitus patients revealed a decreased sensitivity to hormonal stimulation and pancreatic exocrine tissue fibrosis [27]. In another study the low serum amylase levels were linked to decreased insulin secretion and action in type 2 diabetes [28]. Reduced basal serum amylase secretion may be the result of the pathophysiological mechanism of type 2 diabetes, which involves both decreased insulin action and a lack in islet β cell secretion.

In the present study we also observed that VLDL, Total cholesterol/HDL ratio and serum triglyceride levels were significantly increased in obese subjects but other study showed changes in different parameters of lipid profile. A study by Babu et al showed increased cholesterol, LDL levels in high BMI subjects [29]. Another study done in Iraq showed significant increase in levels of serum cholesterol and serum LDL –C and a significant decrease HDL-C in obese people [30]. A study by Khan MN et al showed increased cholesterol, Triglycerides, LDL-c and also found decreased HDL-c in obese diabetic subjects [31].

The study showed negative correlation between serum amylase with serum glucose levels but not significant statistically. According to the research reviews above, hyperglycemia in diabetes mellitus may harm the exocrine pancreas' cells, decrease the production of pancreatic digesting enzymes, and ultimately impact lipid metabolism. Additionally, another research indicated that hyperglycemia may disrupt the cellular communication that governs the pancreatic transcription and protein metabolism, resulting in pancreatic exocrine insufficiency in individuals with obesity-related diabetes [32]. The table 2 showed the statistically significant changes in waist and BMI among obese and Non-obese male subjects but all other parameters were non-significant.

Table 3. Showed statistically significant changes in waist, BMI, Serum amylase, TG, VLDL cholesterol levels among females with mean SD 116.8±20cms,33.17±4.4, 52.45± 15IU/L, 176.56±53.mg/dl & 35.31±10.65mg/dl.

A study by Aljabri et al, shown that high HDL levels in men as well as in women with diabetes mellitus (25% and 27%, respectively) did not differ significantly (p 0.7) from those seen in non-diabetic men and women (23% and 25%, respectively). But we compared men and women who are obese and found significant changes in HDL levels with mean SD of 48±13.55& obese male of 34.16±12.95mg/dl whereas all other parameters showed no significance [33].

Table 4 shows, that age significantly positively correlated with TC/HDL ratio, LDL cholesterol, LDL/HDL ratio, and Cholesterol/HDL ratio among female subjects with r valve of 0.008, 0.013,0.005&0.008 respectively whereas in males it was negatively correlated but was not significant.

In female obese, FBS significantly positively correlated with PPBS, TC, LDL and FPG/HDL ratio, whereas in males shown significantly positively correlated with

TG, and VLDL cholesterol levels.

Studies have found a negative correlation between BMI, WC, and LDL-C in obese non-diabetic subjects [34-37]. In our study BMI was -vely correlated with LDL-c but significant only in male obese diabetics.

The lipid profile correlation is shown in the Table 5. It showed TC in females was positively significantly correlated with TG, TC/HDL ratio, LDL cholesterol, VLDL cholesterol, LDL/ HDL ratio and Cholesterol/ HDL ratio showed statistically significant positive correlation between Total cholesterol & LDL with r <0.001, but in males, total cholesterol was positively correlated with TG, LDL cholesterol ,VLDL cholesterol TC/HDL , LDL/ HDL ratio and Cholesterol/ HDL ratios but not statistically significant [38, 20,39]. The HDL cholesterol levels in the male and female groups showed a significant negative correlation TC/HDL ratio, LDL/HDL ratio, Cholesterol/HDL ratio and TG/ HDL ratio. The LDL cholesterol in females showed positive statistically significant correlation with TC, TC/HDL ratio, LDL/HDL ratio, Cholesterol/ HDL ratio and FPG/ HDL ratio, whereas in males it was positively correlation with TC, TC/HDL ratio, LDL/ HDL ratio, Cholesterol/ HDL ratio and FPG/ HDL ratio but statistically significant [40, 41].

CONCLUSION

The study delves into the profound decrease in serum amylase levels in obese type 2 diabetic individuals compared to their non-obese counterparts. This disparity hints at the impact of chronic hyperglycemia and insulin resistance on pancreatic exocrine dysfunction. This dysfunction, in turn, affects lipid metabolism, leading to notable elevations in VLDL, total cholesterol/HDL ratio, and serum triglycerides among obese subjects.

The observed negative correlation between serum amylase and glucose levels, suggests a possible association between impaired pancreatic enzyme activity and glycemic control. The role of insulin in stimulating amylase secretion highlights the intricate relationship between the endocrine and exocrine functions of the pancreas.

Furthermore, gender-specific variations in lipid profile correlations were detected, with significant positive correlations in females and similar trends in males, albeit not reaching statistical significance. These gender disparities underscore the necessity for a nuanced comprehension of metabolic interactions in diabetes.

In summary, this research accentuates the crucial role of pancreatic exocrine function in lipid metabolism and glycemic regulation in obese type 2 diabetic individuals. Future investigations should delve deeper into the mechanisms and potential therapeutic strategies aimed at enhancing exocrine function and improving metabolic outcomes in diabetic patients.

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