Assessment of Pancreatic Sizes in Patients with Diabetes Mellitus: An Ultrasound Based Study

Iqra Ilyas*

Department of Radiological Sciences and Medical Imaging, Faculty of Health and Allied Sciences, Imperial College of Business Studies - Lahore, Pakistan

***Corresponding Author:** Iqra Ilyas, Department of Radiological Sciences and Medical Imaging, Faculty of Health and Allied Sciences, Imperial College of Business Studies - Lahore, Pakistan.

Received: June 03, 2021

Published: June 15, 2021

© All rights are reserved by Iqra Ilyas.

Abstract

Aims and Objectives: To evaluate the pancreatic size in diabetic patients and compare them with regular patients.

Methodology: A prospective comparative study was conducted for six months, and 102 patients were included. Patients of type1DM, type2DM and control subjects were selected. The pancreas' ultrasound was performed on 27 healthy patients, 33 were with type 1 (Insulin-dependent diabetes mellitus), and 42 were type 2 (Non-insulin-dependent diabetes mellitus). Standard parameters of pancreatic size such as pancreatic head, pancreatic body, and pancreatic tail have been measured. The statistical analysis was performed using MedCalc.

Results: The results show that the mean age was 37 years in the control group, the mean age was 50 years in the NIDDM group, and the mean age was ten years in the IDDM group. 37% were women, and 63% were men in the control sample. Although 36.4% were females and 63.6% in the IDDM community were males. Fifty-two percent were women, and in the NIDDM, 47 percent were men.

Conclusion: Pancreatic size shows a decrease in type1DM and type2DM as compared to the control group. T1DM shows more noticeable changes compared to T2DM.

Keywords: Pancreatic Size; Insulin-dependent Diabetes Mellitus; Non-insulin Dependent Diabetes Mellitus; Ultrasound

Introduction

In every medical assessment of the abdomen by a practitioner, measurement of pancreatic size is essential. A category of specific metabolic diseases that associate the hyperglycemia phenotype includes Diabetes mellitus (DM). Over the previous two centuries, the global incidence of diabetes has significantly increased [1]. The pancreas is one of the organs producing juices, and enzymes found hidden throughout the abdominal cavity and distributed behind the stomach. Before becoming consumed by the stomach, the pancreas' enzymes assist in metabolizing fats, proteins, and carbohydrates. Insulin, which is essential in controlling the amount of glucose in the body, is also produced by the pancreas. Multiple conditions, including diabetes mellitus, acute pancreatitis, chronic pancreatitis, and pancreatic enzyme depletion, and pancreatic tumors, can contribute to every system malfunction or abnormality that develops in the pancreas [2]. Diabetes is a form of disease correlated with an unnaturally high blood sugar glucose concen-

tration; no insulin or inappropriate reaction to insulin secretion is made [3]. Diabetes is the most common disorder associated with the pancreas. It has the following two types: Type 1 insulin-dependent diabetes mellitus (IDDM) is also called juvenile diabetes because it affects children and adults [4]. It occurs because insulin-producing beta cells do not work or make insulin [5]. It is evaluated that approximately 80,000 children develop this form of diabetes each year [6]. Type 2 is non-insulin-dependent diabetes mellitus (NIDDM) [7]. It occurs either by reduced insulin secretions or insulin resistance. In this type, pancreatic beta cells don't respond appropriately to insulin secretions [8,9]. The pancreas is a unique organ because it has the dual function of secreting hormones into blood (endocrine) and secretes hormones through ducts (exocrine) [10]. Pancreases has a cluster of irregular-shaped endocrine cells, which are called Islet of Langerhans [11]. These islets of Langerhans contain different types of cells. In which the insulin-producing beta cells are in significant numbers [12]. Beta cells are responsible

for secreting insulin when the plasma glucose level is high. The deficiency of insulin is an indicator of diabetes. Offensive production of insulin will cause blood sugar imbalance, which leads to diabetes [13]. As the insulin-producing gland, the pancreas is changed and destroyed in the process that leads to diabetes. Pancreatic markers in type I diabetic patients are infiltration of inflammatory cells in islets showing chronic inflammation and new beta cells [14]. Ultrasound is a safe, non-invasive, and cost-effective method. It has become primary tool used by doctors for the evaluation of pancreatic size. The size of the pancreas in diabetes mellitus patients has been studied. Ultrasound is expected to give better information regarding the size of the pancreas in diabetes mellitus patients [15]. A few foreign studies have indicated the improvements in pancreatic size in Type 2 Diabetes Mellitus. This research was conducted using ultrasonography to measure the pancreatic size of diabetic patients and assess if there was a correlation between the size of the pancreas and the period of diabetes to reconfirm the results of previous studies of the Pakistani diabetic population. It will be the novel research in Pakistan as such data never reported before from our part of the world.

Materials and Methods

Study design

This prospective study regarding the association of pancreatic size and the type of diabetes has been conducted.

Sample population

The data used in this study were collected from cases visiting Allama Ehsan Hospital on Ghoray Shah Road, Near Garhi Shahu, during six months of research. A total of 27 controls, 42 patients with NIDDM, and 33 IDDM were selected for the study.

Inclusion criteria

This study included Insulin-dependent Diabetic patients and non-insulin diabetic patients. Non-diabetic patients without any pancreatic abnormality were included in control subjects. All patients fulfilling the inclusion criteria who visited the hospital within four months following the synopsis' approval were included.

Exclusion criteria

Patients with pancreatic cancer, acute or chronic pancreatitis, and those patients who did not sign or accept the consent form were omitted.

Settings

The study was conducted in Lahore at AllamaEhsan Hospital on GhorayShah Road, Near GarhiShahu.

Equipment

Toshiba Famio 5 machine with trans-abdominal (5-7.5MHz) linear probe have been used during the study.

Examination method

All the patients were given informed consent. The non-invasiveness was demonstrated to the patient and the importance of the procedure. It also took into account any relevant details received from the referring doctor. After this, with the patient in the supine and erect position, a trans-abdominal ultrasound was performed. Basic parameters were derived from the sizes. These included the pancreatic head, the pancreatic body, and pancreatic tail sizes.

Research methodology and statistical analysis

This research has been conducted according to the recommended scientific methods. The relevant information has been collected from different sources, such as reference textbooks, Internet services, and scientific journals. Data were collected first on individual patient data sheets and were then tabulated in datacollection sheets. Each patient was designated as a separate ID, and names have not mentioned. No words and individual patient detail was published. All data collected during the study have been stored on a personal computer. The included subjects were grouped into three categories: IDDM, NIDDM, and control groups, and the pancreatic measurements were compared in this using T-test. The collected data has been analyzed, and results were obtained using the MEDICAL. The *p*-value has been obtained to evaluate statistical significance by rejecting the null hypothesis (H_0). A *P*-value equal to or less than 0.05 was considered as the level of value.

Results

The analyzed results of the collected data have been presented in this section. According to data, the mean age in the control group was 37 years, the mean age in the NIDDM group was 50 years of age, and the mean age in the IDDM group was ten years of age. More details on the ages of the patients in three groups have been summarized in the following figure a.

	N	Minimum	Maximum	Mean	Median	SD	RSD	25 - 75 P
Age_controls	27	7.000	80.000	37.333	38.000	19.2833	0.5165	24.500 to 45.750
Age_NIDDM	42	30.000	78.000	50.548	49.500	13.5524	0.2681	40.000 to 62.000
Age_IDDM	33	5.000	15.000	10.621	11.000	2.7244	0.2565	8.875 to 13.000

Figure a: Shows the age statistics in various groups of the included patients.

Variable	Gender_controls	
F	10	37.0%
M	17	63.0%
Total	27	100.0%
Variable	Gender_IDDM	
F	12	36.4%
M	21	63.6%
Total	33	100.0%
Variable	Gender_NIDDM	
F	22	52.4%
M	20	47.6%
Total	42	100.0%

Figure b: Shows the gender frequencies in various groups of the included patients.

In figure b, the gender of the population has been described, and according to that, 37% were female, and 63% males in the control group. While 36.4% were females, and 63.6% were males in the IDDM group. 52% were females, and 47% were males in the NIDDM. In figure c, patients' pancreatic size has been evaluated with T-test analysis, which determines whether there was any significant difference in the pancreas parts' dimensions in various groups of the patients. Further details have been given below in figure c.

Pancreas	N	Minimum	Maximum	Mean	Median	SD	RSD	25 - 75 P
Body (controls)	27	21.000	24.500	22.896	23.000	0.99	0.043	22.000 - 23.87
Body (NIDDM)	42	10.000	18.500	12.575	12.100	1.92	0.153	11.200 - 13.10
Body (IDDM)	33	7.000	13.000	10.203	10.200	1.43	0.141	9.000 - 11.125
Head (controls)	27	20.000	29.000	25.719	26.000	2.32	0.090	24.100 - 27.875
Head (NIDDM)	42	13.000	23.000	18.382	18.625	2.20	0.119	16.700 - 20.00
Head (IDDM)	33	8.500	15.100	11.917	11.700	1.76	0.148	10.775 - 13.22
Tail (controls)	27	12.500	19.000	15.463	15.000	2.07	0.133	13.625 - 17.37
Tail (NIDDM)	42	7.000	11.250	9.581	10.000	1.13	0.118	8.500 - 10.20
Tail (IDDM)	33	6.000	10.700	8.445	8.000	1.51	0.179	7.000 - 10.100

Figure c: The patients showed the following measurements of head, body, and pancreas in all three categories.

In figure d, a significant difference in the pancreatic body in control and IDDM groups has been presented. According to the analyzed data, the sample size was 27 in the controls, and 33 were in the IDDM group. The arithmetic mean and standard deviation in sample 1 (control group) were 22.89 ± 0.9 , while the sample 2 (IDDM) group was 10.20 ± 1.4 . Other details have been given below in figure d.

Sample 1		
Variable	Pancreatic_body_controls	
Sample 2		
Variable	Pancreatic_body_in_IDDM	
	Sample 1	Sample 2
Sample size	27	33
Arithmetic mean	22.8963	10.2030
95% CI for the mean	22.5034 to 23.2892	9.6928 to 10.7133
Variance	0.9865	2.0709
Standard deviation	0.9932	1.4391
Standard error of the mean	0.1911	0.2505
F-test for equal variances	P = 0.056	
T-test (assuming equal variances)		
Difference	-12.6933	
Pooled Standard Deviation	1.2589	
Standard Error	0.3267	
95% CI of difference	-13.3472 to -12.0393	
Test statistic t	-38.855	
Degrees of Freedom (DF)	58	
Two-tailed probability	P < 0.0001	
Residuals		
Shapiro-Wilk test for Normal distribution	W=0.9819 accept Normality (P=0.5143)	

Figure d: It shows that there is a significant difference in the pancreatic body in control and IDDM groups ($P < 0.0001$).

Figure e shows a significant difference in the pancreatic body in control and NIDDM groups. According to the analyzed data, the sample size was 27 in the controls, and 42 were in the NIDDM group. The arithmetic mean and standard deviation in sample 1 (control group) was 22.89 ± 0.9 , while the sample 2 (IDDM) group was 12 ± 1.9 . Other details have been given below in figure e.

Figure f shows a significant difference in the pancreatic body in IDDM and NIDDM groups. According to the analyzed data, sample size 42 was in the NIDDM group, and 33 were in IDDM. The arithmetic mean and standard deviation in NIDDM was 12 ± 1.9 , while the IDDM group was 10 ± 1.4 . Other details have been given below in figure f.

Sample 1		
Variable	Pancreatic_body_controls	
Sample 2		
Variable	Pancreatic_body_in_NIDDM	
	Sample 1	Sample 2
Sample size	27	42
Arithmetic mean	22.8963	12.5750
95% CI for the mean	22.5034 to 23.2892	11.9748 to 13.1752
Variance	0.9865	3.7103
Standard deviation	0.9932	1.9262
Standard error of the mean	0.1911	0.2972
F-test for equal variances	P = 0.001	
T-test (assuming equal variances)		
Difference	-10.3213	
Pooled Standard Deviation	1.6289	
Standard Error	0.4018	
95% CI of difference	-11.1233 to -9.5193	
Test statistic t	-25.688	
Degrees of Freedom (DF)	67	
Two-tailed probability	P < 0.0001	
Residuals		
Shapiro-Wilk test for Normal distribution	W=0.8770 reject Normality (P<0.0001)	

Figure e: It shows that there is a significant difference in the pancreatic body in control and NIDDM groups (P < 0.0001).

Sample 1		
Variable	Pancreatic_body_in_NIDDM	
Sample 2		
Variable	Pancreatic_body_in_IDDM	
	Sample 1	Sample 2
Sample size	42	33
Arithmetic mean	12.5750	10.2030
95% CI for the mean	11.9748 to 13.1752	9.6928 to 10.7133
Variance	3.7103	2.0709
Standard deviation	1.9262	1.4391
Standard error of the mean	0.2972	0.2505
F-test for equal variances	P = 0.091	
T-test (assuming equal variances)		
Difference	-2.3720	
Pooled Standard Deviation	1.7296	
Standard Error	0.4024	
95% CI of difference	-3.1739 to -1.5701	
Test statistic t	-5.895	
Degrees of Freedom (DF)	73	
Two-tailed probability	P < 0.0001	
Residuals		
Shapiro-Wilk test for Normal distribution	W=0.9306 reject Normality (P=0.0005)	

Figure f: It shows that there is a significant difference in the pancreatic body in IDDM and NIDDM groups (P < 0.0001).

Figure g shows a significant difference in the pancreatic head in the control and IDDM groups. According to the analyzed data, sample size 27 was in control, and 33 was in IDDM. The arithmetic mean and standard deviation in controls was 25 ± 2.3 , while the IDDM group was 11.9 ± 1.7 . Other details have been given below in figure g.

Sample 1		
Variable	Pancreatic_Head_controls	
Sample 2		
Variable	Pancreatic_Head_in_IDDM	
	Sample 1	Sample 2
Sample size	27	33
Arithmetic mean	25.7185	11.9167
95% CI for the mean	24.7992 to 26.6378	11.2907 to 12.5427
Variance	5.4008	3.1168
Standard deviation	2.3240	1.7655
Standard error of the mean	0.4472	0.3073
F-test for equal variances	P = 0.140	
T-test (assuming equal variances)		
Difference	-13.8019	
Pooled Standard Deviation	2.0349	
Standard Error	0.5280	
95% CI of difference	-14.8589 to -12.7449	
Test statistic t	-26.138	
Degrees of Freedom (DF)	58	
Two-tailed probability	P < 0.0001	
Residuals		
Shapiro-Wilk test for Normal distribution	W=0.9718 accept Normality (P=0.1790)	

Figure g: It shows that there is a significant difference in the pancreatic head in the control and IDDM groups (P < 0.0001).

Figure h shows a significant difference in the pancreatic head in the control and NIDDM groups. According to the analyzed data, sample size 27 was in control, and 42 was in NIDDM. The arithmetic mean and standard deviation in controls was 25 ± 2.3 , while the IDDM group was 18 ± 2.2 . Other details have been given below in figure h.

Figure i shows a significant difference in the pancreatic head in NIDDM and IDDM groups. According to the analyzed data, sample size 42 was in NIDDM, and 33 was in IDDM. The arithmetic mean and standard deviation in NIDDM was 18 ± 2.2 , while the IDDM group was 11.9 ± 1.7 . Other details have been given below in figure i.

Sample 1		
Variable	Pancreatic_Head_controls	
Sample 2		
Variable	Pancreatic_Head_in_NIDDM	
	Sample 1	Sample 2
Sample size	27	42
Arithmetic mean	25.7185	18.3821
95% CI for the mean	24.7992 to 26.6378	17.6955 to 19.0688
Variance	5.4008	4.8553
Standard deviation	2.3240	2.2035
Standard error of the mean	0.4472	0.3400
F-test for equal variances	P = 0.745	
T-test (assuming equal variances)		
Difference	-7.3364	
Pooled Standard Deviation	2.2510	
Standard Error	0.5553	
95% CI of difference	-8.4447 to -6.2281	
Test statistic t	-13.213	
Degrees of Freedom (DF)	67	
Two-tailed probability	P < 0.0001	
Residuals		
Shapiro-Wilk test for Normal distribution	W=0.9800 accept Normality (P=0.3333)	

Figure h: Shows a significant difference in the control and NIDDM groups (P < 0.0001).

Sample 1		
Variable	Pancreatic_Head_in_NIDDM	
Sample 2		
Variable	Pancreatic_Head_in_IDDM	
	Sample 1	Sample 2
Sample size	42	33
Arithmetic mean	18.3821	11.9167
95% CI for the mean	17.6955 to 19.0688	11.2907 to 12.5427
Variance	4.8553	3.1168
Standard deviation	2.2035	1.7655
Standard error of the mean	0.3400	0.3073
F-test for equal variances	P = 0.197	
T-test (assuming equal variances)		
Difference	-6.4655	
Pooled Standard Deviation	2.0232	
Standard Error	0.4706	
95% CI of difference	-7.4034 to -5.5275	
Test statistic t	-13.738	
Degrees of Freedom (DF)	73	
Two-tailed probability	P < 0.0001	
Residuals		
Shapiro-Wilk test for Normal distribution	W=0.9898 accept Normality (P=0.8155)	

Figure i: It shows that there is a significant difference in the pancreatic head in NIDDM and IDDM groups (P < 0.0001).

In figure j, a significant difference in the pancreatic tail in control and IDDM groups has been presented. According to the analyzed data, sample size 27 was in control, and 33 was in IDDM. The arithmetic mean and standard deviation in control was 15.4 ± 2.0 , while the IDDM group was 8.4 ± 1.5 . Other details have been given below in figure j.

Sample 1		
Variable	Pancreatic_Tail_controls	
Sample 2		
Variable	Pancreatic_Tail_in_IDDM	
	Sample 1	Sample 2
Sample size	27	33
Arithmetic mean	15.4630	8.4455
95% CI for the mean	14.6439 to 16.2820	7.9082 to 8.9827
Variance	4.2870	2.2957
Standard deviation	2.0705	1.5152
Standard error of the mean	0.3985	0.2638
F-test for equal variances	P = 0.094	
T-test (assuming equal variances)		
Difference	-7.0175	
Pooled Standard Deviation	1.7856	
Standard Error	0.4634	
95% CI of difference	-7.9450 to -6.0900	
Test statistic t	-15.145	
Degrees of Freedom (DF)	58	
Two-tailed probability	P < 0.0001	
Residuals		
Shapiro-Wilk test for Normal distribution	W=0.9401 reject Normality (P=0.0055)	

Figure j: It shows that there is a significant difference in the pancreatic tail in control and IDDM groups (P < 0.0001).

Figure k shows a significant difference in the pancreatic tail in control and NIDDM groups. According to the analyzed data, sample size 27 was in control, and 42 was in NIDDM. The arithmetic mean and standard deviation in control was 15.4 ± 2.0 , while the NIDDM group was 9.5 ± 1.1 . Other details have been given below in figure k.

Figure l shows a significant difference in the pancreatic tail in NIDDM and IDDM groups. According to the analyzed data, sample size 42 was in NIDDM, and 33 was in IDDM. The arithmetic mean and standard deviation in NIDDM was 9.5 ± 1.1 , while the IDDM group was 8.4 ± 1.5 . Other details have been given below in figure l.

Sample 1		
Variable	Pancreatic_Tail_controls	
Sample 2		
Variable	Pancreatic_Tail_in_NIDDM	
	Sample 1	Sample 2
Sample size	27	42
Arithmetic mean	15.4630	9.5810
95% CI for the mean	14.6439 to 16.2820	9.2278 to 9.9341
Variance	4.2870	1.2844
Standard deviation	2.0705	1.1333
Standard error of the mean	0.3985	0.1749
F-test for equal variances	P = 0.001	
T-test (assuming equal variances)		
Difference	-5.8820	
Pooled Standard Deviation	1.5651	
Standard Error	0.3861	
95% CI of difference	-6.6526 to -5.1114	
Test statistic t	-15.236	
Degrees of Freedom (DF)	67	
Two-tailed probability	P < 0.0001	
Residuals		
Shapiro-Wilk test for Normal distribution	W=0.9664 accept Normality (P=0.0598)	

Figure k: It shows that there is a significant difference in the pancreatic tail in control and NIDDM groups ($P < 0.0001$).

Sample 1		
Variable	Pancreatic_Tail_in_NIDDM	
Sample 2		
Variable	Pancreatic_Tail_in_IDDM	
	Sample 1	Sample 2
Sample size	42	33
Arithmetic mean	9.5810	8.4455
95% CI for the mean	9.2278 to 9.9341	7.9082 to 8.9827
Variance	1.2844	2.2957
Standard deviation	1.1333	1.5152
Standard error of the mean	0.1749	0.2638
F-test for equal variances	P = 0.080	
T-test (assuming equal variances)		
Difference	-1.1355	
Pooled Standard Deviation	1.3144	
Standard Error	0.3058	
95% CI of difference	-1.7449 to -0.5261	
Test statistic t	-3.714	
Degrees of Freedom (DF)	73	
Two-tailed probability	P = 0.0004	
Residuals		
Shapiro-Wilk test for Normal distribution		W=0.9422 reject Normality (P=0.0019)

Figure l: Shows a significant difference in the pancreatic tail in NIDDM and IDDM groups ($P < 0.0001$).

Figure 1 compares the distribution of pancreatic body sizes in three groups, e.g., control, IDDM, and NIDDM. Variations have been shown in the NIDDM and IDDM.

Figure 1: Graphical explanation shows the comparison of the distribution of pancreatic body sizes in three groups.

Figure 2 compares the distribution of pancreatic head size in three groups, e.g., control, IDDM, and NIDDM. Variations have been shown in the NIDDM and IDDM than in the control group.

Figure 2: Graphical explanation shows the comparison of the distribution of pancreatic head sizes in three groups.

Figure 3 compares the distribution of pancreatic tail size in three groups, e.g., control, IDDM, and NIDDM. Variations have been shown in the NIDDM and IDDM than in the control group.

The pancreases of NIDDM patients differed from those of control subjects only in terms of a greater pancreatic body diameter. NIDDM patients were generally obese and may have been hyper-insulinemic.

Figure 3: Graphical explanation shows the comparison of the distribution of pancreatic tail sizes in three groups.

Figure 4: Image showing ultrasonography abnormalities (a) Pancreatic Head (b) Pancreatic Body (c) Pancreatic Tail in NIDDM patients.

Figure 5: Image Showing Ultrasonography Abnormalities Pancreatic Head, Body, and Tail in IDDM patients.

The decrease of pancreatic diameters of patients with IDDM was time-dependent, clearly evident of disease, and probably related to acinar atrophy and fibrosis.

Discussion

In clinical imaging, the modality of ultrasound is beneficial in the detection of diabetes. From post-mortem histological assessment, it has already been known that the pancreatic sizes in patients with IDDM are much smaller than those in ordinary people. To confirm this observation in insulin-dependent diabetes and determine whether the pancreatic size is also altered in non-insulin-dependent diabetes mellitus, we studied the pancreatic size in patients with diabetes mellitus using ultrasound techniques. The pancreatic sizes in patients with diabetes type 1 and 2 were evaluated and compared with those in control subjects using sonography. The pancreas with T2DM and T1DM showed atrophy of the pancreas by showing reduced sizes compared to normal control subjects.

Interestingly, it has been found that pancreatic sizes of IDDM were significantly less than those in NIDDM. Multiple previous studies [15-21] showed that the smaller' sizes were significantly lower in patients with type IDDM than in type II or NIDDM. Similarly, both of these patients showed substantially lower pancreatic than in the standard group. The results of our study are in complete agreement with the results of these mentioned studies. Foulis, *et al.* demonstrated that in type I diabetic patients, 23 percent of island cells are affected by insulinitis (inflammatory cell infiltration). The condition was responsible for the immune-mediated destruction, leading to a significant decrease in the pancreas' size and weight [22]. NIDDM has been reported to be resulting from a combination of two pathological processes, insulin resistance and markedly decreased response of insulin to the changes in glucose blood levels. The latter has been attributed at least partly to the lethal effects of even mild chronic hyperglycemia and associated high levels of plasma non-esterifies fatty acids on pancreatic beta cells. This process is sometimes termed as "gluco-lipotoxicity" [23]. Our study was the first such study on the topic done in this part of the world. The results of the survey validated those reported from other countries.

Conclusion

It has been concluded that the pancreas shows a decreased size in patients with both types of diabetes mellitus. The decrement in size is more prominent in insulin-dependent/insulin-deficient subjects. A more detailed study with a higher number of included issues is required.

Acknowledgments

I want to thank the Department of Radiological Sciences and Medical Imaging, Faculty of Health and Allied Sciences, Imperial College of Business Studies - Lahore, Pakistan, for the unlimited support in conducting research, especially to the Head of Department.

Financial Support and Sponsorship

No Funding Source.

Conflicts of Interest

No.

Data collection sheet

Sr.#	Date	Pt ID	Age	Pancreatic head sizes (mm)	Pancreatic body size (mm)	Pancreatic Tail size (mm)

Signature Supervisor

Signature Co Supervisor

Figure

Bibliography

1. Rorsman P and Braun M. "Regulation of insulin secretion in human pancreatic islets". *Annual Review of Physiology* 75 (2013): 155-179.

2. Arya VB., et al. "Pancreatic endocrine and exocrine function in children following near-total pancreatectomy for diffuse congenital hyperinsulinism". *PloS One* 9.5 (2014): e98054.

3. Bader E., et al. "Identification of proliferative and mature β -cells in the islets of Langerhans". *Nature* 535.7612 (2016): 430-434.

4. Hebrok M., et al. "Human islets contain four distinct subtypes of beta cells".

5. Lee Y., et al. "Metabolic manifestations of insulin deficiency do not occur without glucagon action". *Proceedings of the National Academy of Sciences* 109.37 (2012): 14972-14976.

6. Shaw JE., et al. "Global estimates of the prevalence of diabetes for 2010 and 2030". *Diabetes Research and Clinical Practice* 87.1 (2010): 4-14.

7. Lorenzo C., et al. "The National Cholesterol Education Program-Adult Treatment Panel III, International Diabetes Federation, and World Health Organization definitions of the metabolic syndrome as predictors of incident cardiovascular disease and diabetes". *Diabetes Care* 30.1 (2007): 8-13.

8. PAL P. "Prospective study on medication adherence in diabetes and hypertensive patients".

9. Umoh VA., et al. "The risk of obstructive sleep apnea among patients with type 2 diabetes mellitus". *Nigerian Medical Journal: Journal of the Nigeria Medical Association* 61.1 (2020): 32.

10. Bullon P., et al. "Obesity, diabetes mellitus, atherosclerosis, and chronic periodontitis: a shared pathology via oxidative stress and mitochondrial dysfunction?". *Periodontology* 2000 64.1 (2014): 139-153.

11. Lakey JR., et al. "Technical aspects of islet preparation and transplantation". *Transplant international* 16.9 (2003): 613-632.

12. Craig ME., et al. "Definition, epidemiology, and classification of diabetes in children and adolescents". *Pediatric Diabetes* 15.S20 (2014): 4-17.

13. Shahzad MS., et al. "Association patterns of volatile metabolites in urinary excretions among Type-2 Non-Insulin dependent diabetes patients". *Advancements in Life Sciences* 3.3 (2016): 71-74.

14. Eapen JT. "Insulin resistance: is it a feedback mechanism or a therapeutic target in type 2 diabetes?". *International Journal of Research in Medical Sciences* 7.8 (2019): 3202.

15. Silarova B., et al. "Risk accuracy of type 2 diabetes in middle-aged adults: Associations with sociodemographic, clinical, psychological, and behavioral factors". *Patient Education and Counselling* 101.1 (2018): 43-51.

16. Casillas J., et al. "Embryology of the Pancreas and Normal Variants". In *Multidisciplinary Teaching Atlas of the Pancreas* (2016): 3-47.

17. Lee JE., et al. "A pictorial review of diagnostic pitfalls of developmental anomalies and variants in pancreatic imaging". *Clinical Imaging* 48 (2018): 32-39.

18. Casillas J., et al. "Embryology of the Pancreas and Normal Variants". In *Multidisciplinary Teaching Atlas of the Pancreas* (2016): 3-47.

19. Larsen WJ., et al. "Larsen's Human Embryology". Churchill Livingstone (2009).

20. Monde GJ and Gesase AP. "Termination pattern of the main and accessory pancreatic ducts among Tanzanians". *Anatomy Journal of Africa* 3.1 (2014): 223-227.

21. Macauley M., *et al.* "Altered volume, morphology and composition of the pancreas in type 2 diabetes". *PloS one* 10.5 (2015): e0126825.
22. Foulis AK., *et al.* "The pancreas' histopathology in type I (insulin-dependent) diabetes mellitus: a 25-year review of deaths in patients under 20 years of age in the United Kingdom". *Diabetologia* 29.5 (1986): 267-274.
23. Jonas JC., *et al.* "Chronic hyperglycemia triggers loss of pancreatic β cell differentiation in an animal model of diabetes". *Journal of Biological Chemistry* 274.20 (1999): 14112-14121.

Volume 5 Issue 7 July 2021

© All rights are reserved by Iqra Ilyas.