Sudan University of Science and Technology

Collage of Graduate Studies

Assessment of Pancreas in Diabetic Patients using Ultrasound

تقييم البنكرياس لدى مرضى السكري باستخدام الموجات فوق الصوتيه

Thesis Submitted for Partial Fulfillment of the Requirement of MSc Degree in Medical Diagnostic Ultrasound

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بسم الله الرحمن الرحيم

قَالَ تَعَالَىٰ:

﴿ وَقُلِ ٱلۡحَمۡدُ لِلَّهِ ٱلَّذِي لَمۡ يَتَّخِذۡ وَلَدٗا وَلَمۡ يَكُن لَّهُۥ شَرِيكٞ فِي ٱلۡمُلۡكِ وَلَمۡ يَكُن لَّهُۥ وَلِيّٞ مِّنَ ٱلذُّلِّۖ وَكَبِّرۡهُ تَكۡبِيرَۢا ١١١ ﴾

صدق الله العظيم

سورة الاسراء

الآية(111)

Dedication

To my father and mother whom encouraged me join in M.sc of ultrasound. They supplied me with all my needs and followed me step by step. They never show unwillingness or annoyance to my needs.

To my brothers and sisters who helped me a lot.

To my friends whom always support and helped me all the time.

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First I am grateful to Allah as he helped me to gain knowledge to finish this research. He also gave me health and patience to overcome the difficulties. So I am thankful to him all my life.

Secondly my thanks also to my supervisor Dr. Asma Ibrahim who devoted her time and generously gave his knowledge and experience to me without limits, I would like to express my gratitude to

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My thanks extended to Ultrasound departments which is the place where I took all my samples. And my colleagues who helped me to finished my research.

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ملخص الدراسه

هذه الدراسة وصفية مستقبلية أجريت فى مستشفى الشرطه ومركز المتكامل الصحي فى الفترة من ديسمبر2016 الى مارس 2017 .

الهدف من هذه الدراسة هو تقييم البنكرياس للمرضى المصابين بالسكرى النوع الثاني بواسطة جهازالموجات فوق الصوتية .استبعدت الدراسة المرضى المصابين بالانواع الاخرى من السكرى . اتبعت الدراسة البرتوكول العالمى لانجاز فحص البنكرياس بواسطة الموجات فوق الصوتية .تم جمع البيانات وتصنيفها وتحليلها بواسطة برنامج التحليل الاحصائى .ووجدت الدراسة أن المرضى الاناث اكثر من الّذكور بواقع 52 % و 48 % بالترتيب ,واكثر الفئات العمرية اصابة هم 61- 68سنه ويمثلون 26%.

اظهرت نتائج الدراسة ان مرضى السكرى من النوع الثاني يتعرضون الى تغيرات فى راس وجسم واظهرت ان هناك علاقه طرديه بين جسم البنكرياس وكل من عمر حيث يزيد 0.0099 سم مع كل سنه و 0.00380سم عند زيادة واحد كيلو جرام وعكسيه مع فترة احتضان المرض حيث ينقص مقدار 0.005سم مع كل تقدم سنه في عمر المرض . وبهذه النتائج توصلت الدراسة الى ان الجسم اكثر تأثراً من الراس بالمرض وايضا توصلت الدراسه الى ان البنكرياس ينقص فى حجمه عند المرضى ويزيد النقصان بزيادة فترة احتضان المرض .ايضا اظهرت نتائج الدراسة الى ان البنكرياس يصبح اكثر بياضا بالتصوير بالموجات فوق الصوتية مع زيادة فترة احتضان المرض مقارنه بالبنكرياس الطبيعى.

أوصت الدراسة باستخدام الموجات فوق الصوتية بصورة روتينية فى وحدات معالجة مرضى السكرى.

Abstract

A prospective and descriptive study was performed at Alribat hospital and Almutakamil Health during the period from December to march 2017. It has been done on 50 patients with type II diabetes and compared with 25 non diabetic patients. Aloka SSD- 500 (Japan) and ESAOTE Pie Medical Aquila – Japanese company – (3.5 -5 Megahertz) curve linear probe are used. The data was analyzed by using SPSS.

The study was aimed to determine the echogenicity changes in the pancreas and to evaluate changes in the size of the head and the body of the pancreas in type2 diabetic patients by measuring the antero posterior diameter of head and body of the pancreas.

The data was collected, classified, analyzed by using SPSS. The analysis of the results found that the male patients (48%) and (52%) females, most affected age group was in age from 61-68 years (26%).

Pancreas ultrasound of sample showed that most of the patients developed abnormal pancreas findings in head and body of pancreas. Direct relations noted between body measurement and patient weight 0.0038cm for any one Kilogram increased, increased by 0.0099cm for any one year increased in age and inverse with duration of diabetes which decreased by 0.005cm for any one year increased in duration. The result represent that body more affected than the head of pancreas, also the pancreas become more hyperechoic than normal and all these findings increase with increase duration of illness.

The study recommended utilization of ultrasound in diabetic patients' management and follow up.

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LIST OF ABBREVIATIONS

DM	:	Diabetes mellitus
US	:	Ultra Sound
IVC	:	Inferior Vana Cava
SMA	:	Superior Mesenteric Artery
IDDM	:	Insulin Dependent Diabetes mellitus
NIDDM	:	Non Insulin Dependent Diabetes mellitus

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Chapter One

Introduction

1-1 Introduction

Diabetes mellitus (DM) comprises a group of common metabolic disorders that share the phenotype of hyperglycemia. (Mealey and Ocampo, (2007))

There are two types of DM: type I which is an autoimmune disorder with infiltration of inflammatory cells in Islets of Langerhans and distraction of pancreatic beta cells and type II which is characterized with disturbance in insulin secretion, peripheral resistance to insulin and overproduction of glucose by liver. Pancreas as the insulin-producing gland is changed and destroyed in the process that leads to diabetes. (Hekmatnia et al. 2007)

The pancreas lies retroperitoneally in roughly the transpyloric plane. For descriptive purposes it is divided into head, neck, body and tail, It's macroscopically is lobulated and is contained within a finecapsule; these lobules are made up of alveoli of serous secretory cells draining via their ductinto the principal ducts. Between these alveoli lie the insulin-secretin.The main duct of the pancreas (Wirsung) runs the length of the gland and usually opens at the ampulla of Vater in common with the common bile duct; occasionally it drains separately into the duodenum. An islet of Langerhans.The accessory duct (of Santorini) passes from the lower part of the head in front of the main duct, communicates with it, and then opens into the duodenum above it. Occasionally it is absent. (Snell 2003)

The pancreas is one of the juices and enzymes production organ, this enzymes help in digesting fat, protein, and carbohydrates before being absorbed by the intestine. The pancreas also produces insulin, which is important in regulating the
Glucose concentration in the blood. Any system dysfunction or irregularity occurs

to the pancreas may lead to several diseases such as diabetes mellitus, acute Pancreatitis, chronic pancreatitis, pancreatic enzyme deficiency as well as pancreas tumor. (Daniel et al. (2014)).

Ultrasound imaging plays an effective role in the evaluation of normal as well as abnormal pancreas. Many researchers suggest ultrasonography technique as the primary method of assessment of pancreatic diseases, as it is reasonably accurate, cost effective and lack of side effect. (Beckett et al. (1985)

1-2 Problem of study

DM is disease that can affect many organs in the body, one of the major organ is pancreas because it's closely related to the change induced by DM in its cellular component so its lead to change both cellular behavior and morphology. Also advanced age and lead to pancreas change .This study to identifying the etiology of change.

1-3 Objectives:

1-3-1 General objective:

To assess the pancreas in diabetic patients using abdominal ultrasonography.

1-3-2 Specific objective:

- To assess the echogenicity changes in pancreas
- To measure the head and body of pancreas using U/S.
- To test the difference in pancreatic size between the normal and the diabetic Patient.
- To evaluate the disease relative to is related variables e.g. duration of the disease.

1-4 Overview of study:

This study consist of five chapters, chapter one was an introduction introduce briefly this thesis and contained (problem of study also contain general, specific objectives, significant of the study and overview of the study). Chapter two literature review in which contain anatomy, physiology, pathology, sonographic of normal pancreas. Chapter three was describe the methodology (material, method) used in this study. Chapter four was included result of presentation of final finding of study; chapter five included discussion, conclusion and recommendation for future scope in addition to references and appendices.

Chapter Two

Literature Review

2-1 Anatomy

The pancreas is a non-encapsulated, retroperitoneal structure that lies in the anterior pararenal space between the duodenal loop and the splenic hilum. The total length from head to tail is 12.5 to 15 cm. The pancreas is draped transversely over the spine and great vessels. For descriptive purposes it is divided into head, neck, body and tail. The head lies to the right of the spine within the "C" loop formed by the superior, descending and transverse portions of the duodenum. The uncinate process is the medial tapered projection of the head that extends posterior to the superior mesenteric vein and may be large enough to extend posterior to the superior mesenteric artery. The neck is the portion of the gland that lies anterior to the superior mesenteric vein and the origin of the main portal vein. The body extends to the left of the neck and runs anterior to the splenic vein. The neck and body of the pancreas are often the most anteriorly located portions of the gland. It exhibits a wide head adjacent to the curvature of the duodenum, a central, elongated body projecting toward the left lateral abdominal wall, and a tail that tapers as it approaches the spleen. The left border of the vertebral column is an arbitrary plane for dividing the body from the tail. The tail extends across the left adrenal gland and upper pole of the left kidney to end near the hilum of the spleen. (Michael (2007))

2-1-1Structure*:* The pancreas contains modified simple cuboidal epithelial cells called acinar cells. These cells, which are organized into large clusters termed acini or lobules, secrete the mucin and digestive enzymes of the pancreatic juice. The simple cuboidal epithelial cells lining the pancreatic ducts secrete bicarbonate (alkaline fluid) to help neutralize the acidic chyme arriving in the duodenum from the stomach. Most of the pancreatic juice travels through ducts that merge to form

the main pancreatic duct, which drains into the major duodenal papilla in the duodenum. A smaller accessory pancreatic duct drains a small amount of pancreatic juiceinto a minor duodenal papilla in the duodenum. (Michael (2007)).

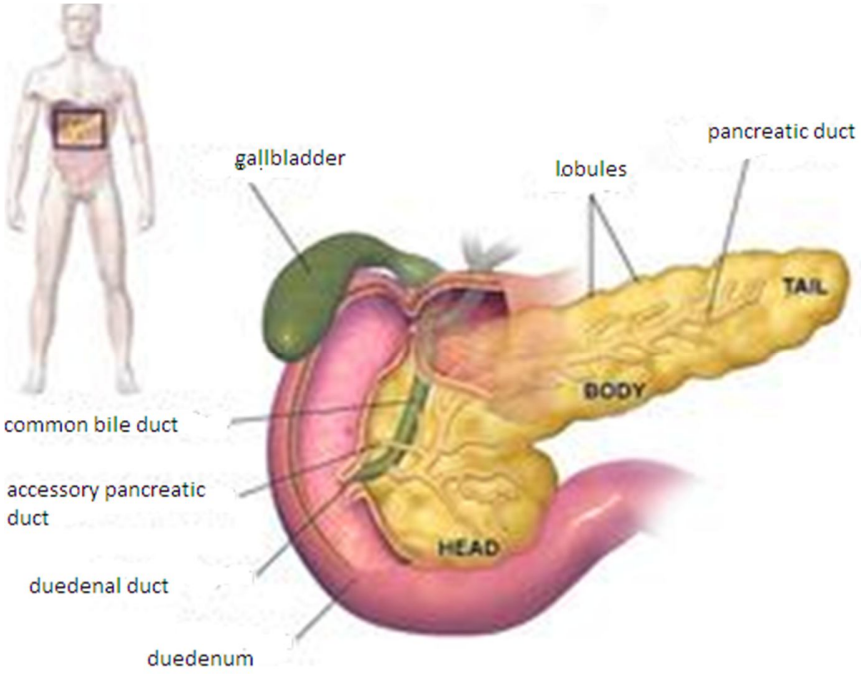

Figure (2-1) demonstrates the normal segmentations of pancreas (wikipedia.org)

2-1-2Relations*:* The head lies in the C-curve of the duodenum and sends out the uncinate process which hooks posteriorly to the superior mesenteric vessels as these travels from behind the pancreas into the root of the mesentery. Posteriorly lie the inferior vena cava, the commencement of the portal vein, aorta, and superior mesenteric vessels, the crura of diaphragm, celiac plexus, the left kidney and suprarenal gland. The tortuous splenic artery runs along the upper border of the pancreas. The splenic vein runs behind the gland, receives the inferior mesenteric vein and joins the superior mesenteric to form the portal vein behind the pancreatic neck.The important posterior relationships, the common bile duct lies either in a groove in the right extremity of the gland or embedded in its substance, as it passes

to open into the second part of the duodenum. Anteriorly lies the stomach separated by the lesser sac. To the left, the pancreatic tail lies against the hilum of the spleen. (Michael (2007))

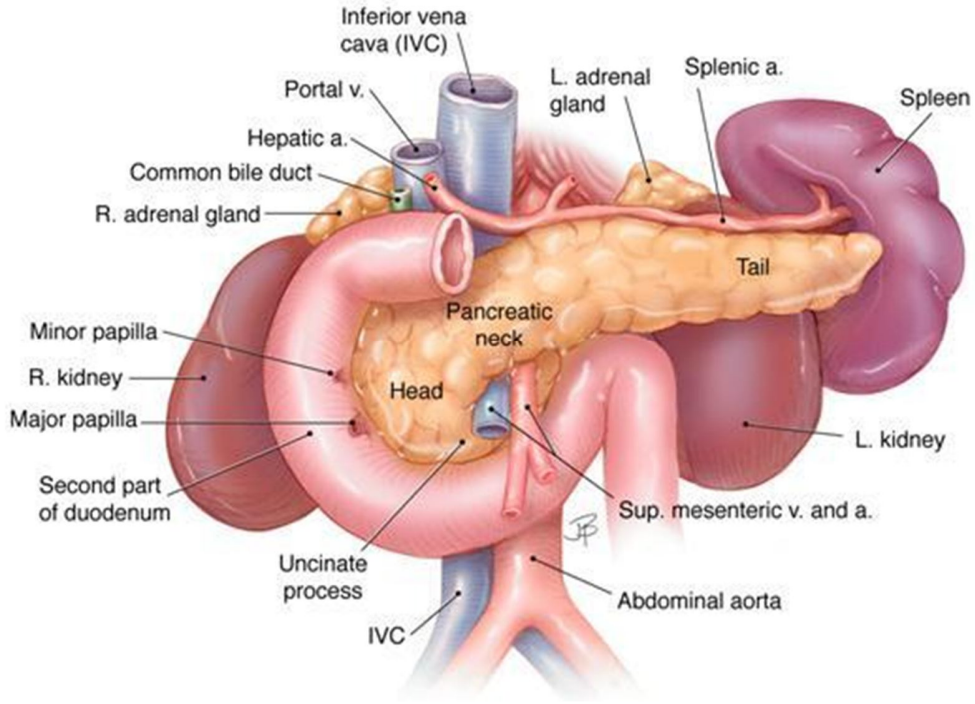

Figure (2-2): demonstrate pancreas relation to adjacent (wikipedia.org)

2-1-3The blood supply*:* Blood is supplied from the splenic and the pancreaticoduodenal arteries; the corresponding veins drain into the portal system. (Ellis, 2006)

2-1-4The lymphatics*:* drain into nodes which lie along its upper border, in the groove between its head and the duodenum, and along the root of the superior mesenteric vessels. (Ellis, 2006)

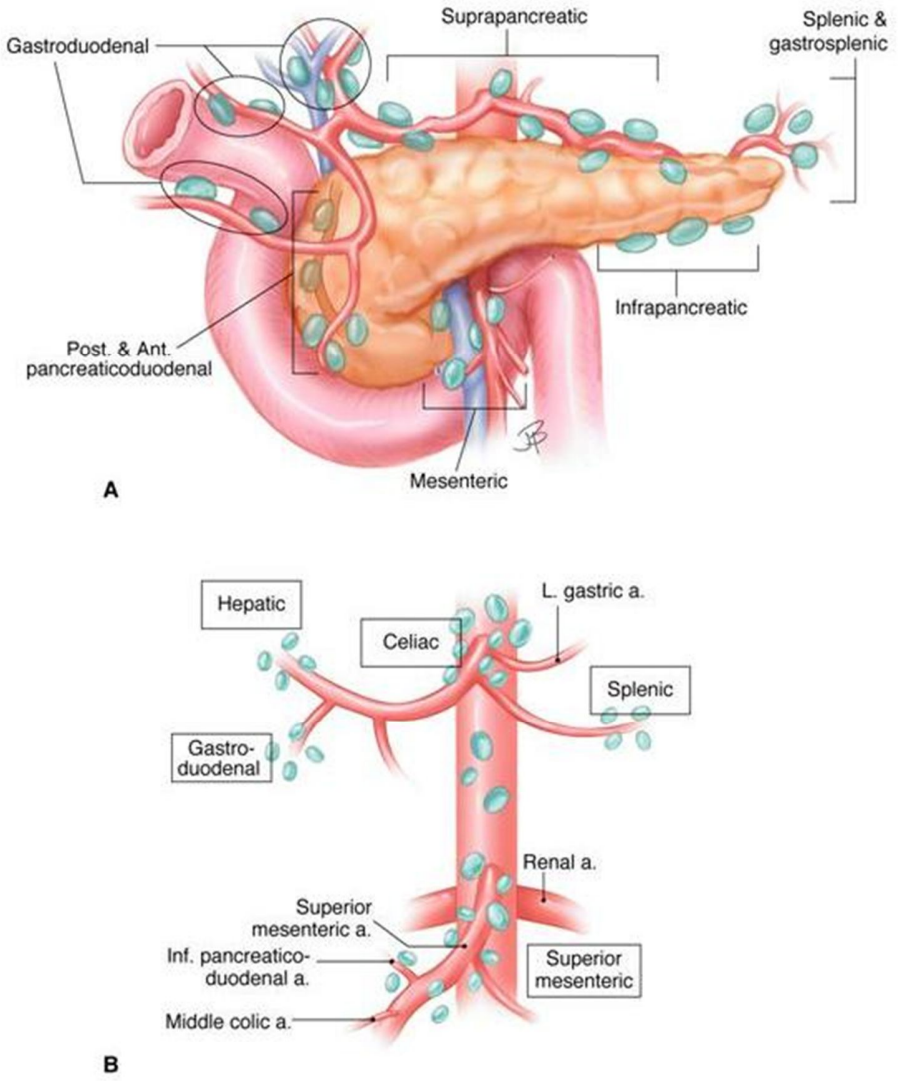

Figure (2-3) demonstrate blood supply and lymphatic drainage of pancreas (wikipedia.org)

2-1-5Variations*:* The size of the pancreas varies considerably. Several anatomical variations exist, relating to the embryological development of the two pancreatic buds. The pancreas develops from these buds on either side of the duodenum. The ventral bud eventually rotates to lie next to the dorsal bud, eventually fusing. If the two buds each having a duct, do not fuse, a pancreas may exist with two separate ducts, a condition known as a pancreas divisum. This condition has no physiologic consequence. If the ventral bud does not fully rotate, an annular pancreas may

exist. This is where sections of the pancreas completely encircle the duodenum, and may even lead to duodenal atresia. An accessory pancreatic duct may exist if the main duct of pancreas does not regress. (Richard et al. (2007))

2-2 Development:

The pancreas starts to form at week 4 from two separate outgrowths of the bile duct, called the ventral pancreatic bud and the dorsal pancreatic bud. By week 6, the ventral pancreatic bud and the biliary apparatus rotate behind the duodenum. The ventral and dorsal pancreatic buds fuse to form the pancreas. The pancreas develops from a larger dorsal diverticulum from the duodenum and a smaller ventral outpouching from the side of the common bile duct. The ventral pouch swings round posteriorly to fuse with the lower aspect of the dorsal diverticulum, trapping the superior mesenteric vessels between the two parts. The ducts of the two formative segments of the pancreas then communicate; that of the smaller takes over the main pancreatic flow to form the main duct, leaving the original duct of the larger portion of the gland as the accessory duct. (Schoenwolf 2009)

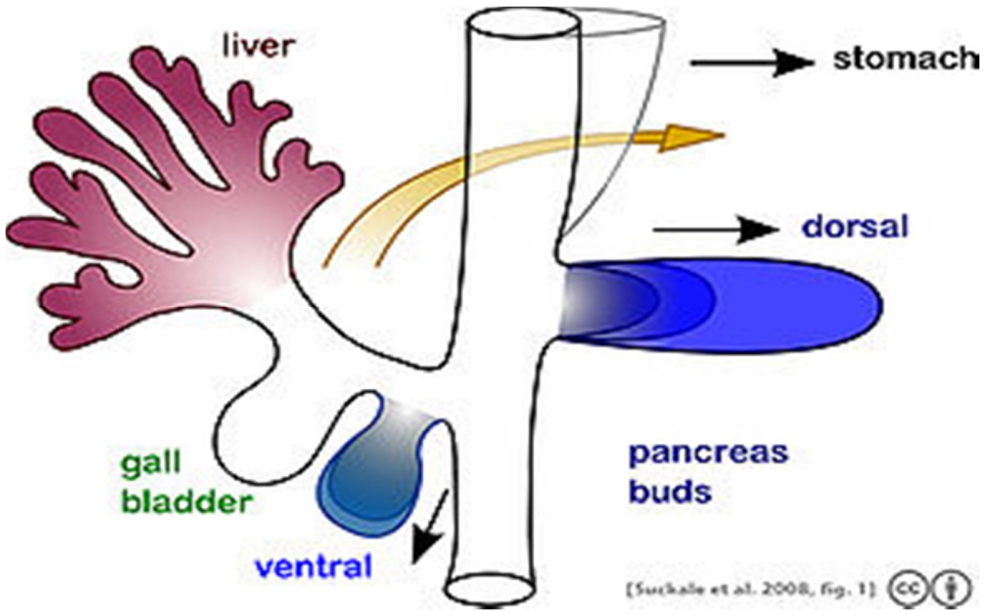

Figure (2-4): demonstrate pancreas development (wikipedia.org)

2-3 Physiology:

The pancreas is located in the upper left quadrant of the abdominal cavity, extending from the curve of the duodenum to the spleen. Although the pancreas is both an exocrine (digestive) gland as well as an endocrine gland, only its endocrine functions. The hormone-producing cells of the pancreas are called islets of Langerhans (pancreatic islets they contain alpha cells that produce glucagon and beta cells that produce insulin. (Richard et al. (2007)).

Insulin: increases the transport of glucose from the blood into cells by increasing the permeability of cell membranes to glucose. (Brain, liver, and kidney cells, however, are not dependent on insulin for glucose intake.) Insulin is also important in the metabolism of other food types; it enables cells to take in fatty acids and amino acids to use in the synthesis of lipids and proteins (notenergy production). Without insulin, blood levels of lipids tend to rise and cells accumulate excess fatty acids. With respect to blood glucose, insulin decreases its level by promoting the use of glucose for energy production. Insulin is a vital hormone; we cannot survive for very long without it. A deficiency of insulin or in its functioning is called diabetes mellitus. (Ramzi et al. (2003)).

Secretion of insulin is stimulated by hyperglycemia**,** a high blood glucose level. This state occurs after eating, especially of meals high in carbohydrates. As glucose is absorbed from the small intestine into the blood, insulin is secreted to enable cells to use the glucose for immediate energy. At the same time, any excess glucose will be stored in the liver and muscles as glycogen. You will also notice the cells called delta cells. These produce the hormone somatostatin, which is identical to growth hormone inhibiting hormone from the hypothalamus. Pancreatic somatostatin acts locally to inhibit the secretion of insulin and glucagon, and it seems to slow the absorption of the end products of digestion in the small intestine. (Richard et al. (2007))

Glucagon: stimulates the liver to change glycogen to glucose (this process is called glycogenolysis, which literally means "glycogen breakdown") and to increase the use of fats and excess amino acids for energy production. The process of gluconeogenesis (literally, "making new glucose") is the conversion of excess amino acids into simple carbohydrates that may enter the reactions of cell respiration. The overall effect of glucagon, therefore, is to raise the blood glucose level and to make all types of food available for energy production. The secretion of glucagon is stimulated by hypoglycemia, a low blood glucose level. Such a state may occur between meals or during physiological stress situations such as exercise. (Richard et al. (2007))

2-4 Pathology:

2-4-1Diabetes Mellitus: is a clinically and genetically heterogeneous group of metabolic disorders manifested by abnormally high levels of glucose in the blood. The hyperglycemia is the result of a deficiency of insulin secretion caused by pancreatic b-cell dysfunction or of resistance to the action of insulin in liver and muscle, or a combination of these. Frequently this metabolic disarrangement is associated with alterations in adipocyte metabolism. Diabetes is a syndrome and it is now recognized that chronic hyperglycemia leads to long-term damage to different organs including the heart, eyes, kidneys, nerves, and vascular system. (Ramzi et al. (2003))

Diabetes mellitus type 1: is a chronic autoimmune disorder in which the immune system attacks the insulin-secreting cells of the pancreas. Insulin is needed to keep blood sugar levels within optimal ranges, and its lack can lead to high blood sugar. As an untreated chronic condition, diabetic neuropathy can result. Type 1 diabetes can develop at any age but is most often diagnosed before adulthood. For type 1 diabetics, insulin injections are critical for survival. An experimental procedure to

treat type 1 diabetes is the transplantation of pancreatic islet cells from a donor into the patient's liver so that the cells can produce the deficient insulin. (*Fauci et al (2012))*

Diabetes mellitus type 2: is the most common form of diabetes. The causes for high blood sugar in this form of diabetes usually are a combination of insulin resistance and impaired insulin secretion, with both genetic and environmental factors playing an important role in the development of the disease. The management of type 2 diabetes relies on a series of changes in diet and physical activity with the purpose of reducing blood sugar levels to normal ranges and increasing insulin sensitivity. Biguanides such as metformin are also used as part of the treatment along with insulin therapy. (*Fauci et al. (2012))*

2-4-2Cystic Fibrosis*:* This common cause of pancreatic disease in childhood is inherited as an autosomal recessive. A specific gene mutation ΔF508 is present in 70% of cases. The gene(s) code for a membrane protein in epithelial cells which regulates chloride transport (the cystic fibrosis transmembrane regular, CFTR). Defective chloride channel transport secondarily leads to a failure to hydrate pancreatic secretion. The increased viscosity of such secretions then leads to ductular obstruction and secondary pancreatic damage. Ninety per cent of patients with cystic fibrosis will have pancreatic failure, and in the majority of these this will be present from the perinatal period. (Ramzi et al. (2003))

2-4-3Pancreatitis*:* Pancreatitis is inflammation of the pancreas. There are two forms of pancreatitis, which are different in causes and symptoms, and require different treatment.Pancreatitis is divided into acute and chronic. By definition acute pancreatitis is a process that occurs on the background of a previously normal pancreas and can return to normal after resolution of the episode. In

chronic pancreatitis there is continuing inflammation with irreversible structural changes. In practice the differentiation between acute and chronic pancreatitis may be extremely difficult, particularly in the setting of recurrent acute episodes which may represent true acute pancreatitis or may be an acute manifestation of underlying chronic disease.(Ramzi et al .(2003))

Acute pancreatitis: In the western world gallstones and alcohol account for the vast majority of episodes. Alcohol also causes chronic pancreatitis. The severity of the pancreatitis may range from mild and self-limiting to extremely severe with extensive pancreatic and peripancreatic necrosis as well as hemorrhage. In its most severe form the mortality raises to between 40-50 %.(Ramzi et al. (2003))

Chronic pancreatitis: In developed countries by far the most common cause of chronic pancreatitis is alcohol, accounting for 60-80% of cases.In developing countries malnutrition and associated dietary factors have been implicated. In a small group of patients chronic pancreatitis has been shown to be hereditary, inherited as an autosomal dominant condition with variable penetrance. Almost all patients with cystic fibrosis have established chronic pancreatitis, usually from birth. Cystic fibrosis gene mutations have also been identified in patients with chronic pancreatitis but in whom there were no other manifestations of cystic fibrosis. (Ramzi et al. (2003))

2-4-4Pancreatic Neoplasms *:*

Adenocarcinoma: It is the most common type of pancreatic cancer. Almost all adenocarcinomas originate in the ductal epithelium rather than the acini. "Approximately 70% of the pancreatic cancers arise in the region of the head, 15% to 20% in the body, and 5% in the tail. In 20% of cases the tumor is distributed diffusely throughout the gland". Most patients are males over the age of 60. The prognosis is poor with a one year survival rate of 8 %.(Dean 2005)

Cystic Neoplasms of the Pancreas: These are not uncommon. Seventy-five per cent of these lesions will be pseudocysts but of the remainder the majority is true cystic neoplasms. These neoplastic lesions can be divided into the serous and mucinous cyst adenomata. Serous cyst adenomata are composed of multiple small cystic cavities lined by cuboidal glycogen-rich, mucin-poor cells. These lesions tend to occur in an elderly age group and are often an asymptomatic finding. Malignant transformation in a serous cystadenoma is extremely rare. Mucinous cyst adenomata are almost exclusively found in women in the 5th and 6th decade and are sited in the pancreatic body and tail. Multiloculor cysts are lined by tall mucin-synthesizing cells. Twenty per cent of these lesions are malignant at the time of presentation and the majority appears to have a malignant potential. As a consequence they are much more likely to produce symptoms. (Dean 2005)

Neuroendocrine Tumors: The islets of Langerhans have the capacity to synthesize more than one hormone. They also synthesize ectopic hormones that are not usually found in the pancreas such as gastrin, adrenocorticotrophin, vasoactive intestinal peptide and growth hormone. Whilst many pancreatic endocrine tumors are multihormonal, one peptide tends to predominate and is responsible for the clinical syndrome. Other tumors, whilst containing peptide hormone, are functionally inactive.The majority of the endocrine pancreatic tumors are malignant in their behavior. (Dean 2005)

2-5Pancreatic Sonographic Appearances

The pancreatic parenchyma has a homogeneous texture, coarser than that of the liver. Its' echogenicity should be at least as greater slightly greater than that of the liver (assuming, of course, that the patient does not have fatty infiltration of the liver. The echogenicity varies with age and obesity. Slim young adults often have pancreases isodense to the adjacent normal livers. Older slim adults have pancreases slightly more echogenic than the adjacent normal liver.Since the

pancreas has no true capsule, the echogenic retroperitoneal fat may infiltrate the gland of an obese patient resulting in a more echogenic pancreas.

Fat also makes the borders of the pancreas difficult to define. The increased echogenicity resulting from excessive body fat is reversible.Retroperitoneal fat can also influence the position and sonographic appearance of the gland. Increasing amounts of retroperitoneal fat push the pancreatic head and tail anteriorly so all parts of the pancreas will be at the same depth. The entire pancreas may also be pushed more anteriorly. (Dean, 2005)

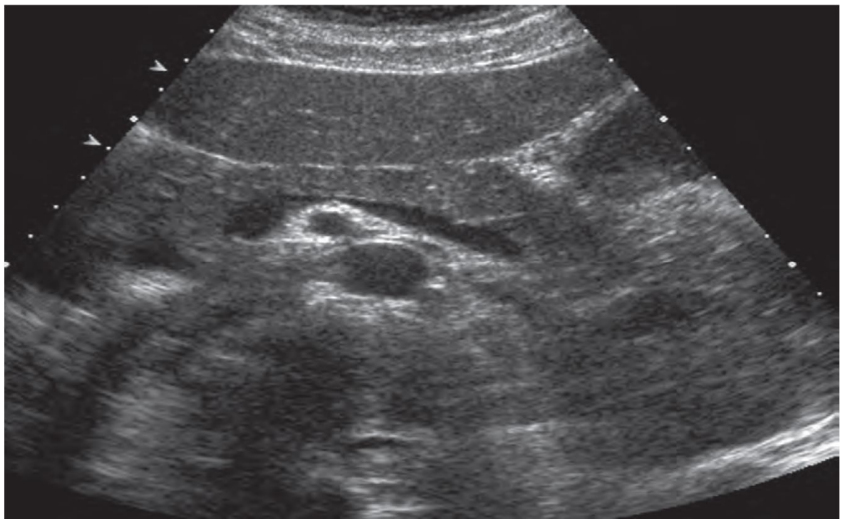

Figure (2-5): sonogrsm transverse scan of the pancreas (Rumack, 2011)

Dimensions*:* The size has been measured by various authors but due to variable shapes and a range of normal sizes, absolute measurements may be misleading if measurements are used as the only indicator of disease. The maximal anteroposterior dimension is 3 cm for the head and 2.5 cm for the body. The tail is measured in a plane perpendicular to the anterior wall of the tail and should be 2.0 cm or less. The AP dimensions of the neck range from 1.0 to 2.0 cm.

The size and echogenicity varies with age. The pancreas normally atrophies with age. In children the pancreas may be significantly larger than the normal adult

measurements, whereas, in the elderly, it may be reduced to a thin strip of tissue which is heavily fibrosed. Fibrosis is fibrous tissue replacement of cells. Fibrous tissue is highly echogenic. Therefore, it is reasonable to expect children to have less echogenic pancreases than adults and young adults to have less echogenic pancreases than those of the elderly. Regardless of echogenicity differences due to age, the outline of the pancreas should always be smooth. In a few patients, particularly the obese, there are focal areas of decreased echogenicity as compared with the rest of the gland, which is highly echogenic. This is often in the posterior aspect of the pancreatic head. These are thought to represent areas of focalsparing in a gland diffusely infiltrated by fat. (Dean, 2005)

2-6Background studies

Akhigbe et al.(2016) was aimed to determine pancreatic anteroposterior (AP) dimensions in diabetics by sonography and compare with nondiabetics. The study was involved 150 diabetics with 150 sexes and age matched healthy normoglycemic group used as controls. Sonographic measurements of the AP dimensions of the pancreatic head, body, and tail of both study groups were performed with the use of 3.5 MHz curvilinear array transducer of a SonoAce X4 ultrasound machine. The results show Pancreas AP dimensions were significantly smaller in diabetics compared to those of the controls. The mean dimensions were 1.91 ± 0.26 cm, 0.95 ± 0.12 cm, and 0.91 ± 0.11 cm for the head, body, and tail, respectively, in diabetics and 2.32 ± 0.22 cm, 1.43 ± 0.19 cm, and 1.34 ± 0.20 cm in the control ($P < 0.001$ in all cases). The dimensions were also significantly smaller in the Type 1 diabetics compared to Type 2 ($P < 0.001$ in all cases). The mean duration of illness for the Types 1 and 2 diabetics were 3.09 ± 1.38 and 3.78 ± 3.12 years, respectively. Longer duration of illness was associated with smaller

pancreas body and tail dimensions, while pancreas head dimension was not significantly affected by the duration of illness. His study concludes that the diabetics have smaller pancreas AP dimensions compared to the normal population.

Silva et al (1993) was aimed to evaluate the relationship between the type and duration of diabetes and pancreas size by ultrasonography .The study was envolved pancreas images of 40IDDM and 36 NIDDM patients with b0.3-34 yrs of disease were compared with those of 60 normal healthy control subjects. The result was show the diameters +/-SD of the head , body , and tail of the pancreas in IDDM patients (1.9+/-0.3; 0.9+/- 0.2; and 1.4+/-0.2cm , respectively) were smaller than in NIDDM patients (2.7 +/- 0.4 ; 1.2 +/- 0.3 ; and 1.8 +/- 0.4 cm ,respectively) and control group subjects (2.4 +/- 0.4 ; 1.1 +/- 0.3 and 1.8 +/- 0.4 cm respectively). The pancreatic shrinking in IDDM patients was clearly evident after 10 years of the disease. NIDDM patients and control subjects had similar pancreatic dimensions, except for a greater body thickness in NIDDM patients with >10years of disease (1.2 +/- 0.4 vs. 1.1 +/- 0.3 cm). Pancreas image was hypoechogenic in 72.5%of IDDM patients and hyperechogenic in 83.3% of NIDDM patients. Her study concludes that the smaller pancreas in IDDM patients in comparison with NIDDM patients and control subjects were clearly demonstrated only after 10years of disease.

Reza Basiratnia et al (2005) was aimed to evaluate pancreatic diameter and echogenicity by sonography and to examine the correlation of these two factors with duration of disease in diabetes types I and II in comparison with controls. And this study was conducted on 60 patients with DM (type I and II) presenting to Alzahra and Kashani Hospitals. The Results show Diameter of pancreas was significantly different in diabetic patients and correlated with duration of disease.

And study was conclude that In type I diabetes, decrease in the size of pancreas was more prevalent than in type II diabetes and these changes become more prominent over time.

Hardt et al about Exocrine Pancreatic Insufficiency in Diabetes Mellitus and the result when use the ultrasound to detect the morphological change which was reduced size of the pancreas in patients with diabetes mellitus. Compared to sex- and age-matched controls the pancreas of children and adolescents with type 1 diabetes mellitus appeared clearly smaller.

Alzaid et al (1993) was aimed to evaluate of pancreas in diabetic by ultrasound, this study was in USA and the method was tested on 57 diabetic patients: 14 with Type 1 (insulin-dependent) diabetes, 10 insulin-treated and 33 tablet-treated patients with Type 2 (non-insulin-dependent) diabetes, and 19 nondiabetic subjects. In this study measure the head (area medially to SMA) and body (area anterior to SV) of pancreas .The result of there study, the pancreas of patients with Type 1 diabetes was markedly smaller ($p < 0.0001$) than the pancreas in non-diabetic subjects. The pancreas of patients with Type 2 diabetes was more moderate in size: larger ($p < 0.001$) than that of Type 1 diabetic patients but smaller ($p < 0.5$) than the pancreas of the control group. Pancreatic size of patients with Type 2 diabetes was also related to basal insulin secretion with insulindeficient patients (low or undetectable C-peptide) having smaller ($p < 0.05$) pancreases than those with normal insulin secretion. There was no difference in the size of the pancreas in the different treatment groups of Type 2 diabetic patients.
Pancreatic size did not correlate with age, body mass index or the duration of diabetes. His studyconclude that the pancreas is a smaller organ in patients with diabetes mellitus and that the decrement in size is maximal in insulin

dependent/insulin deficient subjects. Ultrasonography, therefore, can potentially serve to discriminate between the different types of diabetes.

Chapter Three

Materials and Methods

3-1 Materials:

3-1-1 Study Group:

A prospective, descriptive study deal with ultrasound findings in patients with diabetes among Sudanese population. The study was conducted in some ultrasound departments in Khartoum state Alribat hospital, diagnostic radiology department (U/S section), And Almutakamil Health Center , from December 2016 up to march 2017, Sample frame was comprised of 50 diabetes patients and 25 non diabetic one in different Age, gender, weight, duration of diabetes .

3-1-2 Ultrasound units used:

Used Aloka SSD- 500 (Japan) ultrasound machine with multi-frequency curvilinear probe (3.5 – 5 MHZ) which has variable focal zone and frequency capability, and CHISON 600 M (China) with multi-frequency curvilinear probe (2.5 – 5 MHZ) , and ESAOTE Pie Medical Aquila – Japanese company – (3.5 -5 MHz) ,Which also has variable focal zone and frequency capability. Proper setting of the overall gain (system) gain and time gain or depth gain compensation (TGC/ DGC) was adjusted to optimally visualize each organ.

3-2 **Methods:**

3-2-1Pancreatic Scanning Techniques*:*

The patients and controls were scanned in the morning after an overnight fast. Scans were performed with the patients supine and erect.

Amore complete and clearer visualization of the pancreas was achieved by scanning with the patient oblique. The head (defined as the area medial to the superiormesenteric vein) and the body of the pancreas was measured separately, since these were often visualized to best advantage in different views. As the head

is often oriented in the longitudinal plane parallel to the inferiorvena cava, measurements were made in this plane below the portal veins well as in the transverse or oblique plane (taking the midpoint of the confluenceof the superior mesenteric and splenic veins as the marker point). The scans were recorded on photographic paper.

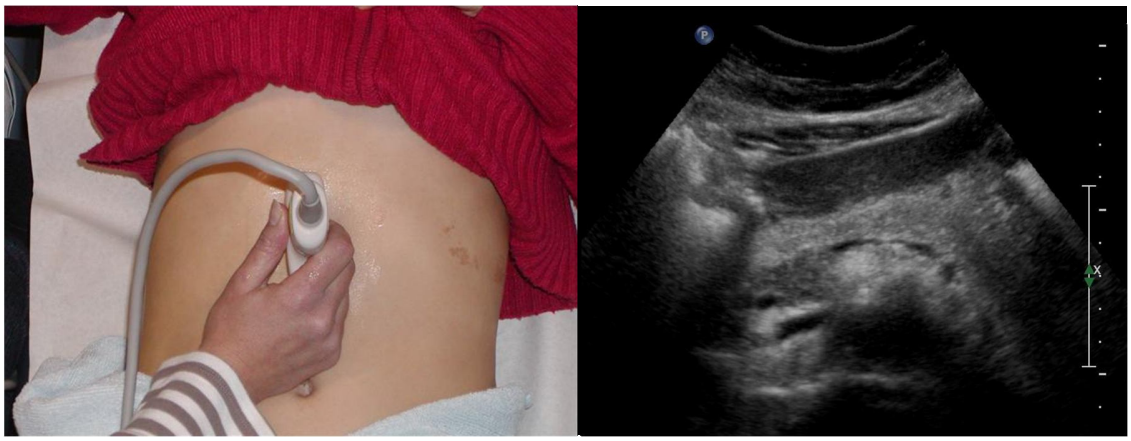

Figure (3-1) *Transverse planeof pancreas*

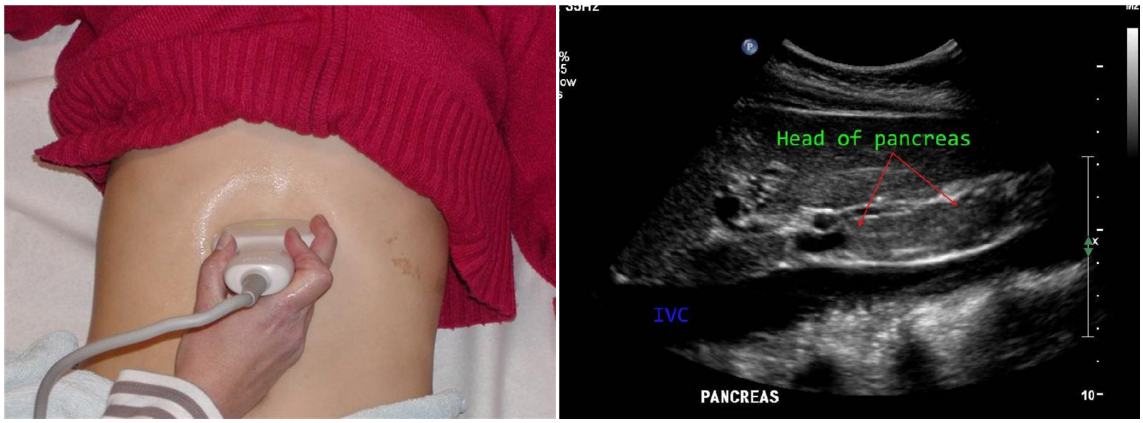

Figure (3-2): sagital *plane of pancreas*

3-2-2 Data analysis:

The data have been analyzed by SPSS by using the various statistic computerize methods.

3-2-3 Data presentation:

For data presentation dummy tables and figures has been used.

Chapter Four

Result

Table (4-1): shows the gender (sex) frequency distribution

Gender	Frequency	Percent
Male	24	48.0
Female	26	52.0
Total	50	100.0

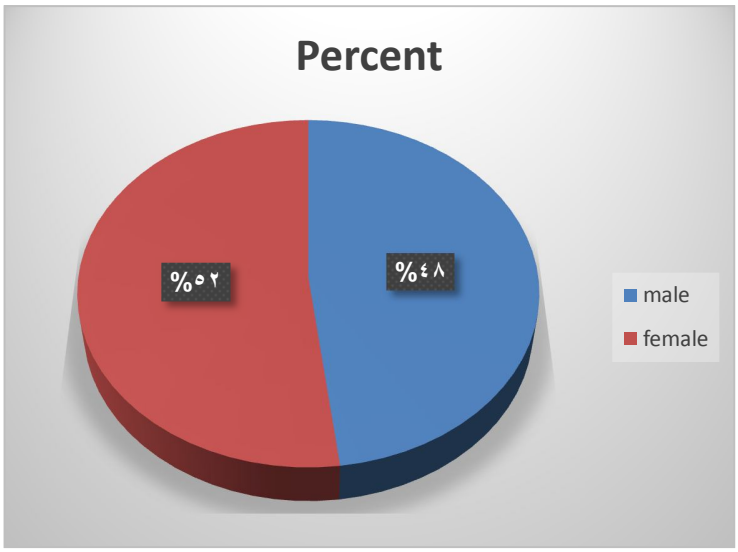

Figure (4-1): Gender distribution

Table (4-2): Frequency distribution of patients according to age.

Age groups	Frequency	Percent
37-44	4	8.0
45-52	9	18.0
53-60	11	22.0
61-68	13	26.0
69-76	11	22.0
77-85	2	4.0
Total	50	100.0

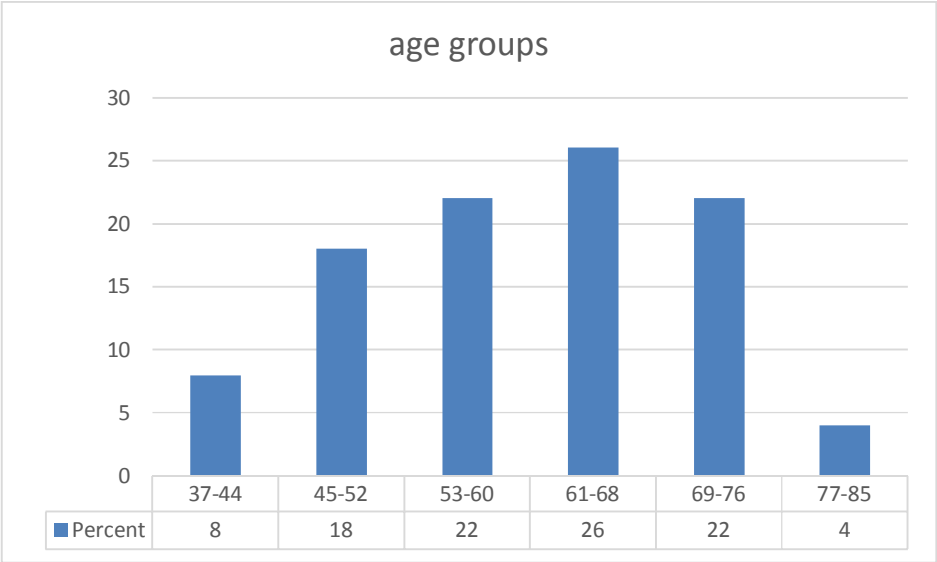

Figure (4-2): Distribution of patients according to age

Table (4-3): Frequency distribution of patient according to pancreas echogenicity

Echogenicity	Frequency	Percent
Hyper echogenic	32	64.0
Hypo echogenic	4	8.0
Normal	14	28.0
Total	50	100.0

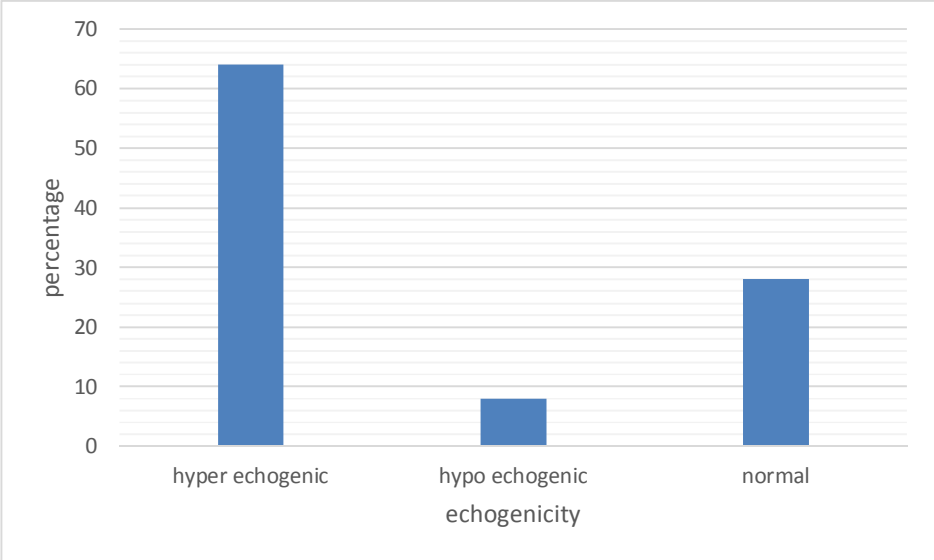

Figure (4-3): Distribution of patient according to pancreas echogenicity

Tables (4-4) shows the Mean ± Std. Deviation for patient related variables

Variables	Minimum	Maximum	Mean	Std. Deviation
age	37.0	85.0	60.200	11.7143
weight	60.0	82.0	71.240	5.9059
Head (cm)	0.8	3.3	2.084	0.4859
Body (cm)	0.7	3.2	1.644	0.4990
Durations (yrs.)	1.0	30.0	8.720	7.3291

Table (4-5): Relation between patient gender and size of head and body of pancreas

Group Statistics				
gender		N	Mean	Std. Deviation
Head (cm)	Male	24	2.113	0.4416
	Female	26	2.058	0.5308
Body (cm)	Male	24	1.658	0.5149
	Female	26	1.631	0.4938

Table (4-6): shows sample t-test for Equality of Means between head and body of pancreas

Independent Samples Test					
		t-test for Equality of Means			
		T	df	Sig. (2-tailed)	Mean Difference
Head (cm)	Equal variances assumed	0.395	48	0.695	0.0548
Body (cm)	Equal variances assumed	0.193	48	0.848	0.0276

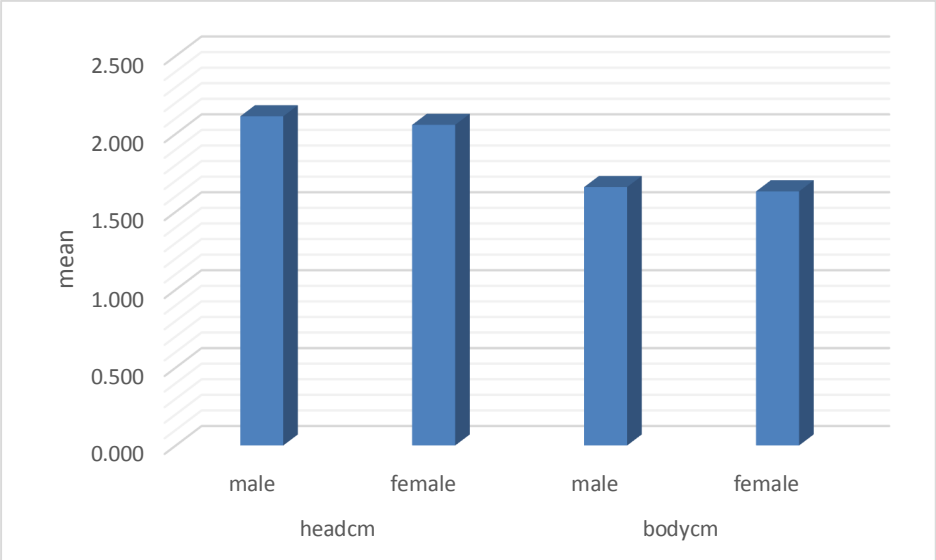

Figure (4-4): shows diagram of mean between head and body of pancreas in both male and female diabetic patient

Table (4-7) show dimensions of the head of pancreas as function of age

	age	N	Mean	Std. Deviation
Head (cm)	37-44	4	1.600	0.2449
	45-52	9	2.211	0.4807
	53-60	11	1.945	0.5538
	61-68	13	2.069	0.2810
	69-76	11	2.264	0.5767
	77-85	2	2.350	0.6364

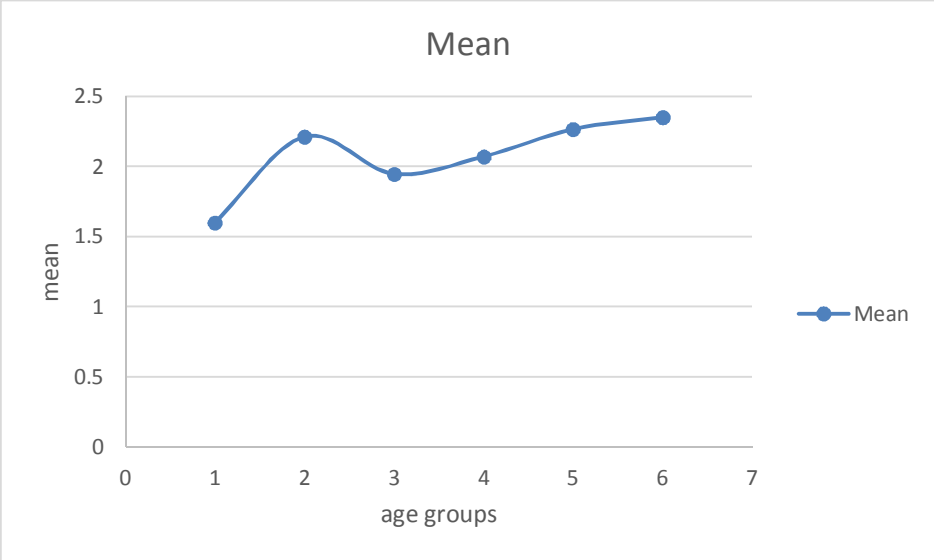

Figure (4-5) scatter plot show dimensions of the head of pancreas as function of age

Table (4-8) show dimensions of the body of pancreas as function of age

Group Statistics				
	age	N	Mean	Std. Deviation
Body(cm)	37-44	4	1.225	0.1893
	45-52	9	1.633	0.4690
	53-60	11	1.582	0.5400
	61-68	13	1.585	0.3236
	69-76	11	1.585	0.3236
	77-85	2	1.927	0.5350

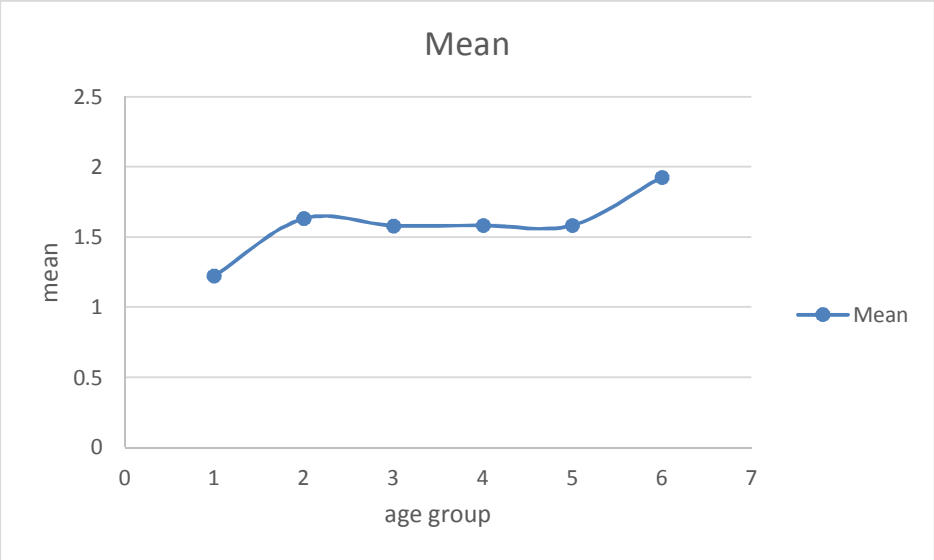

Figure (4-6) show dimensions of the body of pancreas as function of age

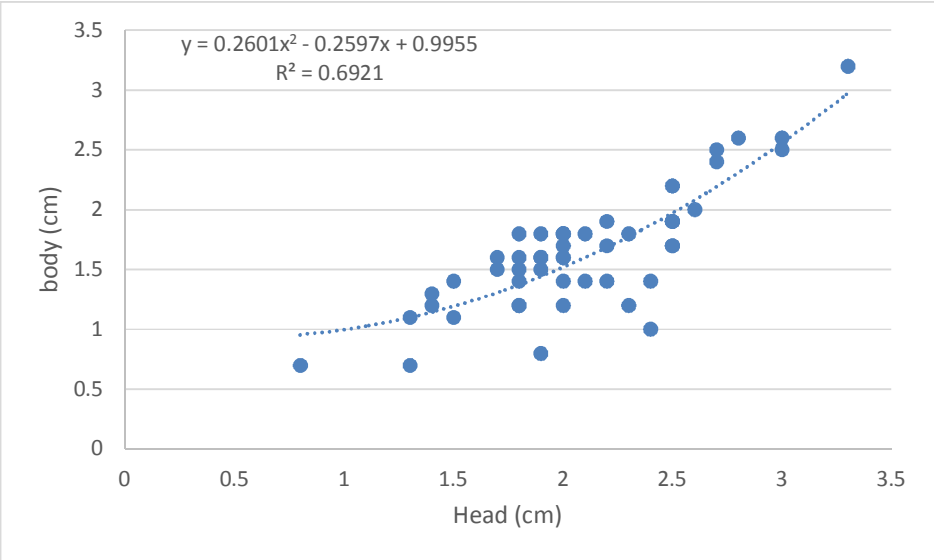

Figure (4-7) show mean between head and body of diabetic pancreas

$y = 0.8338x - 0.0937$, $R^2 = 0.6591$

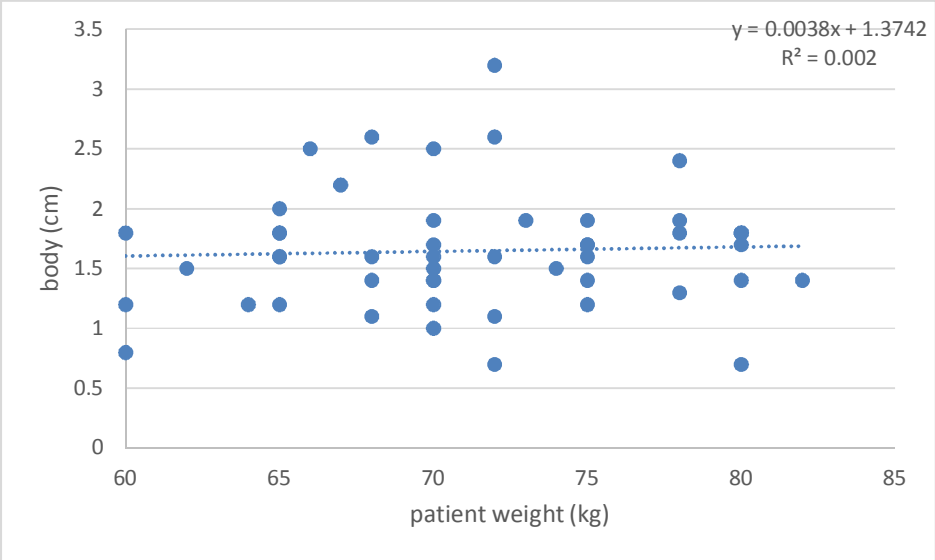

Figure (4-8) show relation between body of diabetic pancreas and patient weight

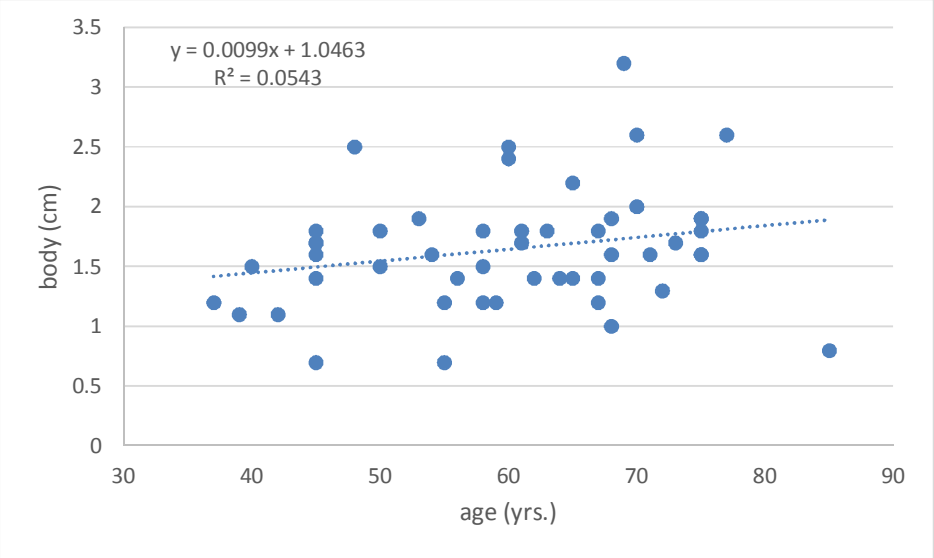

Figure (4-9) show relation between body of diabetic pancreas and patient age

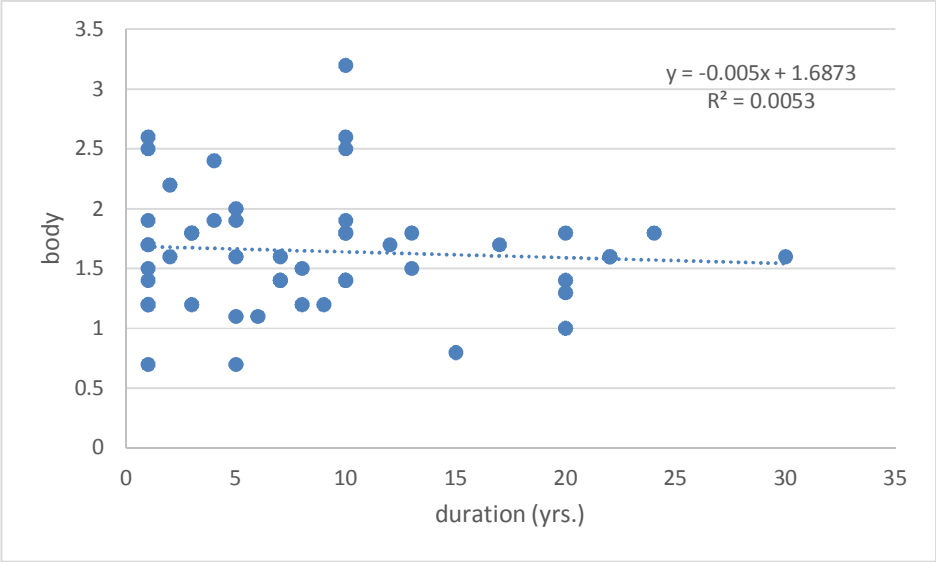

Figure (4-10) show relation between body of diabetic pancreas and duration of diabetes

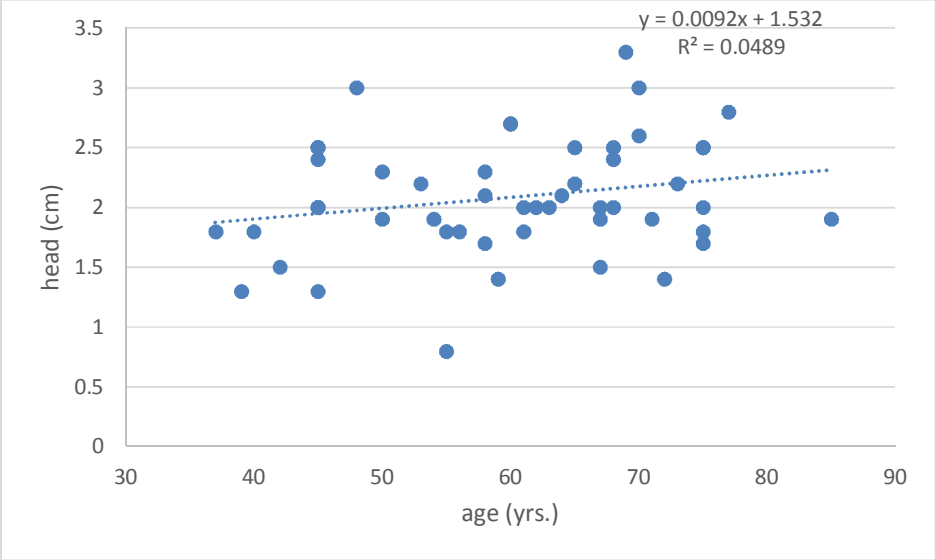

Figure (4-11) show relation between head of diabetic pancreas and patient age

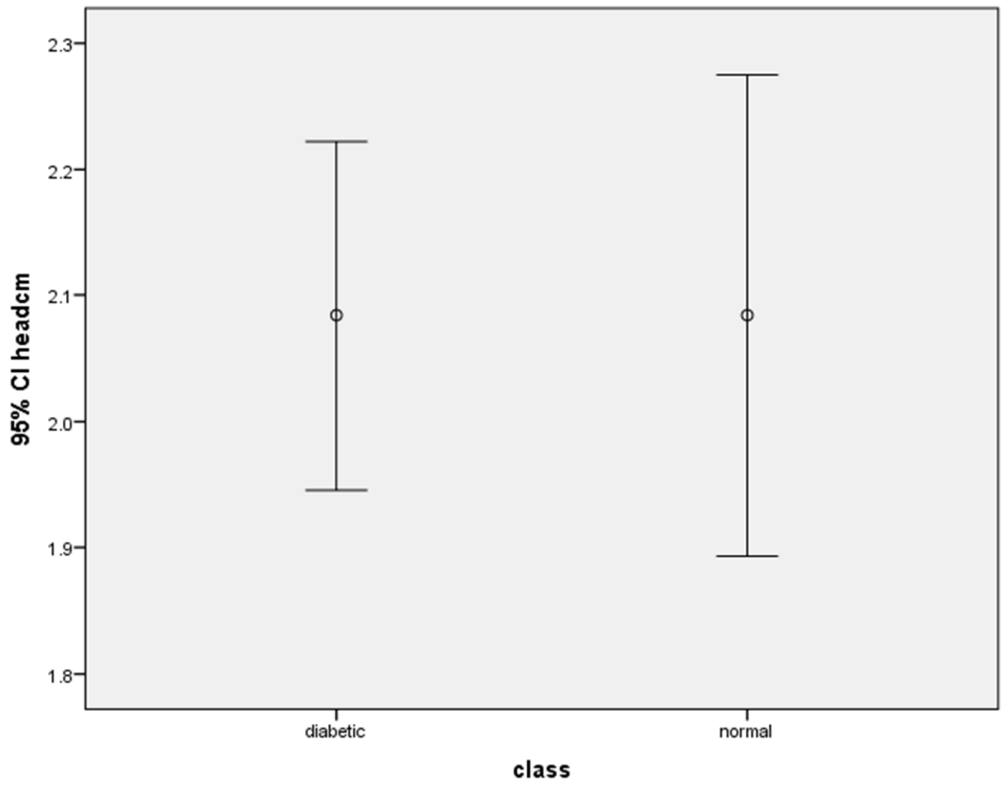

Figure (4-12) box plot shows measure the head of pancreas in control and diabetic group.

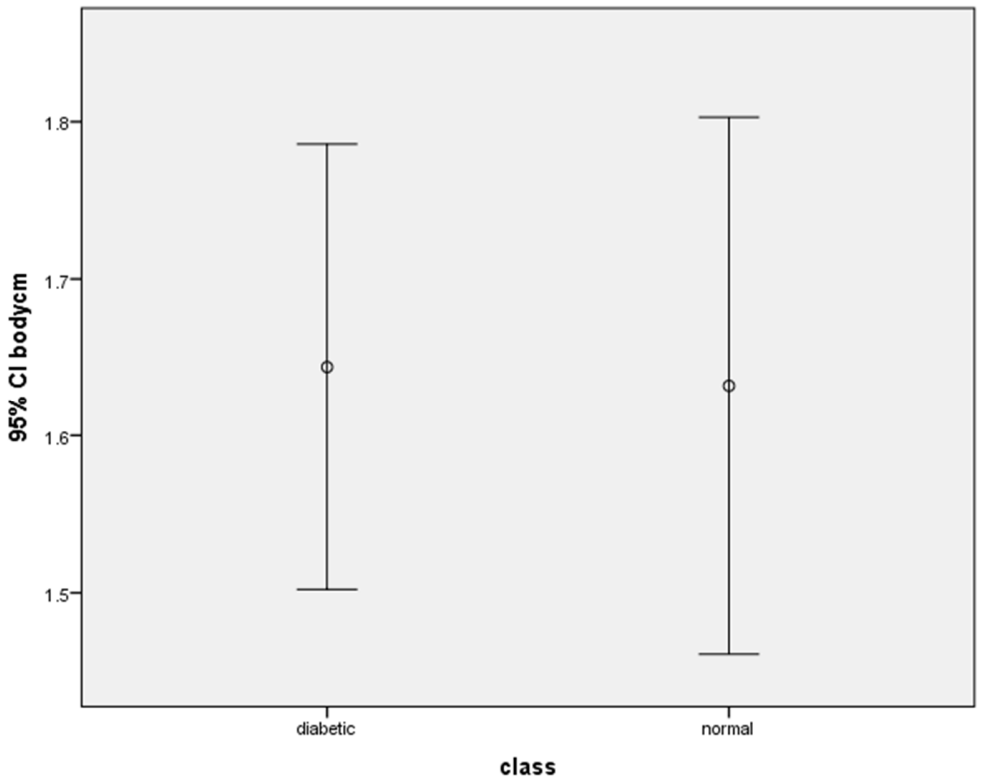

Figure (4-13) box plot shows measure the body of pancreas in control and diabetic group.

Chapter Five

Chapter Five

5-1 Discussion:

This study aimed to evaluate the change of pancreatic size and echogenicity in diabetic patients using abdominal ultrasound, 50 patients with diabetes underwent U/S examination of the abdomen and the pancreas was evaluated in all patients in Sagittal, and transverse section, also 25 of the healthy population was examined using the same technique and the head and body was measured using the US machine in order to test the difference between these groups, the data were collected from Khartoum state Alribat hospital, diagnostic radiology department (U/S section). And Almutakamil Health Center.

Table (4-1) and figure (4-1) show that 26 at the patients out of 50 (52%) are female while 24(48%) are male ,Table (4-2) and figure (4-2) shows Frequency distribution of patients according to age ,which the most diabetic age between 61-68 (26%) followed by age group between 53-60 and 69-76 (22%).

Table (4-3) and figure (4-3) show distribution of patients according to pancreas echogenicity the result according to this study which shows 32 patient out of 50 (64%)are hyperechogenic pancreas ,4 patients (8%) Hypoechogenic and 14 (28%) is normal echogenicity. The hypo echogenicity and hyper echogenicity was due to pancreatitis because the diabetes is the most cause of acute and chronic pancreatitis and this match the study of Silva et al (1993) about relationship between the type and duration of diabetes and pancreas size by ultrasonography .

One of the most important objectives was to investigate the relationship between the measurements and gender, where this study revealed that no significant difference was noted, so we identify that the gender was no co factor to affect such measurement. As in table (4-4) which show Mean ± Std. Deviation for patient related variable, Table (4-5): shows relation between patient gender and size of

head and body of which the head is measure (2.113±0.4416) for male, (2.058±0.5308) for female, and the body is (1.658±0.5149) for male, (1.631±0.4938) for female patient, use in independent sample T-test table (4-6) P value =0.695 for head and P=0.848 for body The normal measurement is 3 cm for the head and 2.5 cm for the body, in which the result show a significant difference noted between the diabetic patient condition and control group of healthy people.

Table and figure (4-4) shows sample t-test for Equality of Means between head and body of pancreas show significant relationship between head and body of diabetic pancreas, this result agree with Alzaid et al (1993) about evaluation of pancreas in diabetic by ultrasound.

Table (4-7) (4-8) and figure (4-5) (4-6) show dimensions of the head and body of pancreas as function of age, in this the age in five class group presented the largest size of the head and body within age class (77-85) represent in 2 patient with mean of (2.350 +/- 0.6364 cm) for head and (1.927 +/- 0.5350 cm) for body , and smallest size in age group (37-44) represent in 4 patients with mean of (1.600 +/- 0.2449 cm) for head , and (1.225+/- 0.1893cm) for the body .

Figure (4-7) show mean between head and body of diabetic pancreas, which represent direct relation in size.

Direct relations noted between body measurament and patient weight where the body increased by 0.0038cm for any one Kg increased in weight as in Figure (4-8) show relation between body of diabetic pancreas and patient weight, direct relation between head and body measurement with age where the head increased by 0.0092cm, body increased by 0.0099cm for any one year increased in age as in (4-9) (4-11) this result dis agree with (Dean 2005). And inverse relation between body with duration of diabetes where the body decreased by 0.005cm for any one year increased in duration as in (4-10).

This groups classified according to the age and the size was compared to the normal measurement which discuss in the table and found that the head and body of pancreas was reduced in size with in diabetic patients and this may due to destruction of the beta cells of pancreas increase with increase duration of illness, and this was match with all of previous studies in my research.

Figure (4-12) **,** (4-13) box plot shows measure the head and body of pancreas in control and diabetic group, this represent the decrease in size of head and body of pancreas in diabetic patients than the normal one. This agree with Akhigbe et al. (2016) about determine pancreatic anteroposterior (AP) dimensions in diabetics by sonography and compare with nondiabetics.

5-2Conclusion:

This study aimed to evaluate and measure the pancreatic size in type 2diabetic patient and healthy control group, and tested on fifty patients suffering from type 2 diabetes mellitus were evaluated and compared them with age and size of pancreas with normal measurement to access the morphology of the pancreas by ultrasonography which is non-invasive, economic, simple an easily available.

It was found that patients with type 2diabetes mellitus have a reduction in the head and body of the pancreas, and also found the echogenicity was increased and pancreas appear more hyperechoic than normal which were more pronounced in patients having longer duration of diabetes. There was also increased in echogenicity of pancreatic gland.

5-3 Recommendations

- The high incidences of abnormal pancreas findings in diabetic patients support the need for ultrasound screening in each diabetic centre.
- Pancreas scanning should be planned as one of the basic necessary exam in diabetic centers to aid in diagnosis, management and follow up.
- More training programmed should be planned for Sonographer and sonologist in the field of ultrasound especially in pancreas scanning to give accurate results.
- Use modern equipment with new soft ware program as segmentation of ultrasound pancreas image, is technique which treated the gases confusing the pancreas according to the location.
- Further studies should be done with expanding period of time and include more sample data for more precise and accurate results.

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Appendices

Appendices

- **Appendix1**

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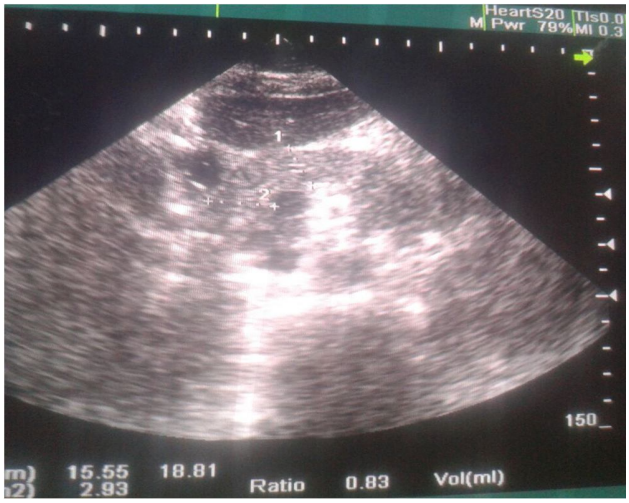

- Image(1): sonogram of pancreas female 58 years old and 13 years had diabetes

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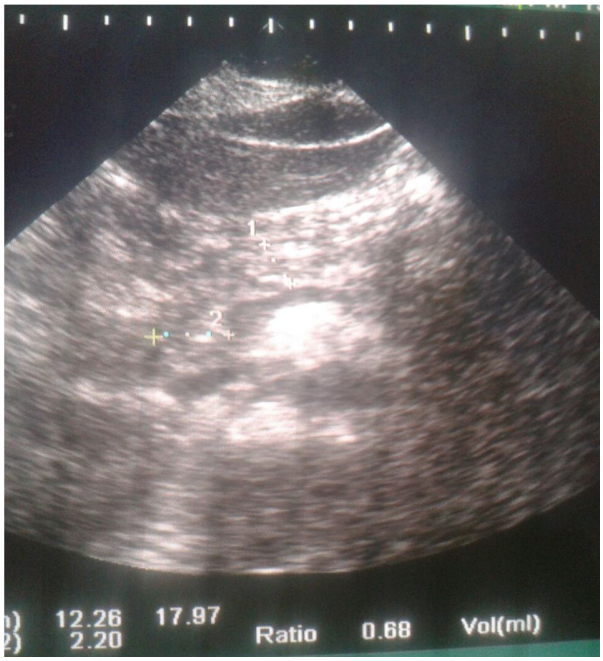
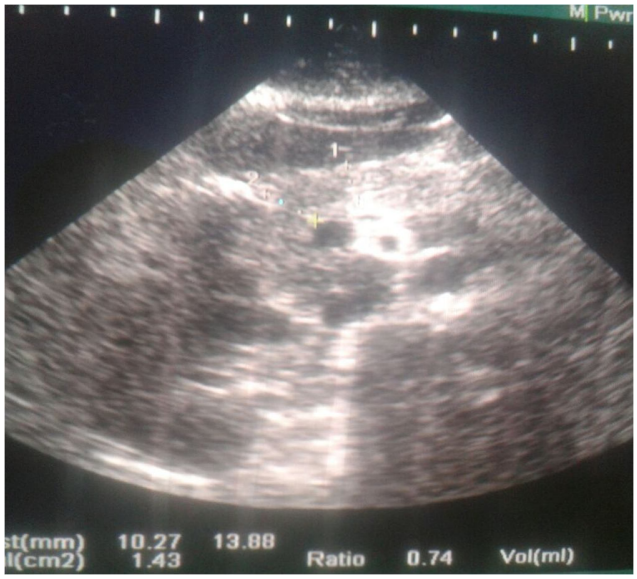
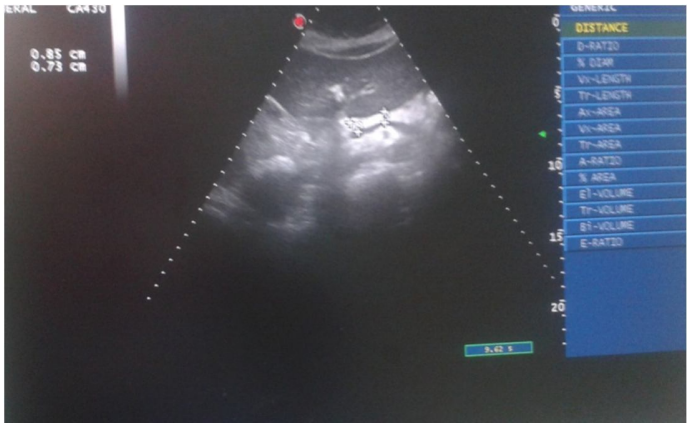


- Image (2): sonogram of pancreas male 73 years old and 12 years had diabetes

- 

- Image (3): sonogram of pancreas male 72 years old and 20 years had diabetes

- 

- Image (4): sonogram of pancreas female 55 years old and 2 years had diabetes

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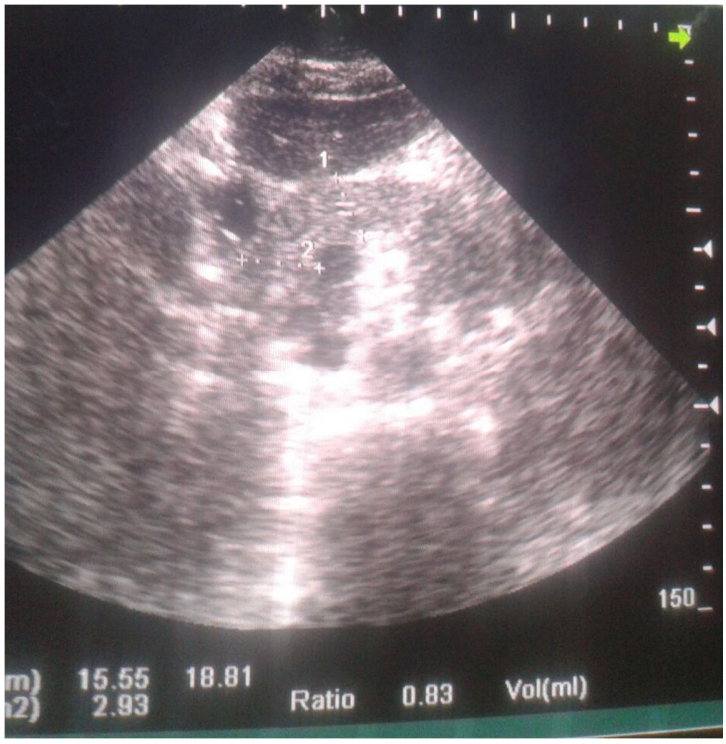
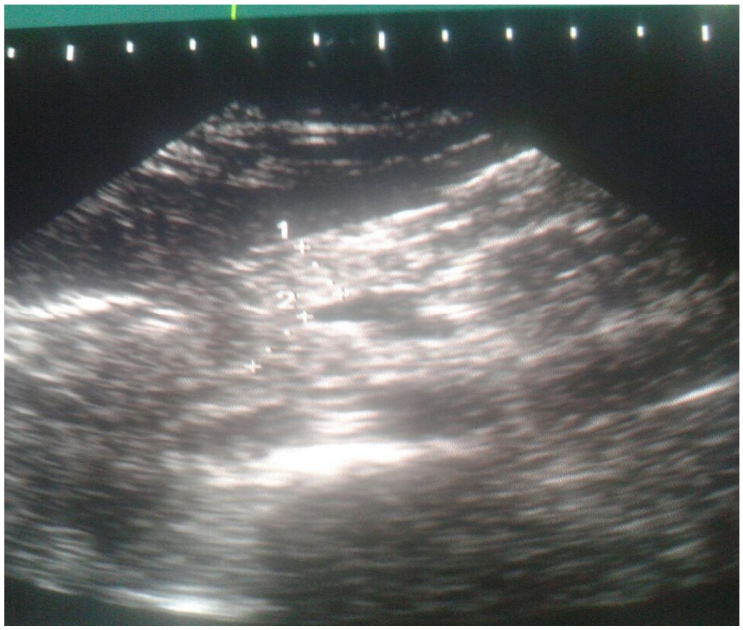
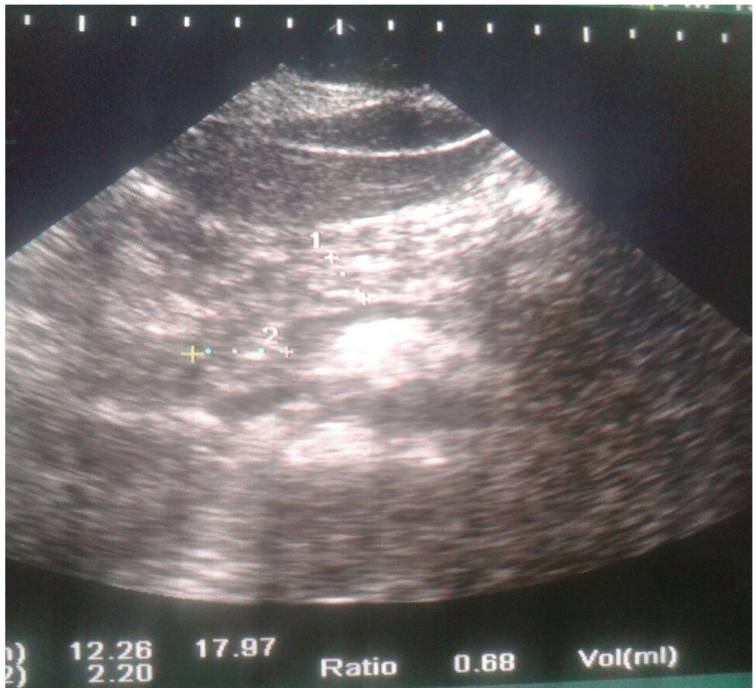
- 

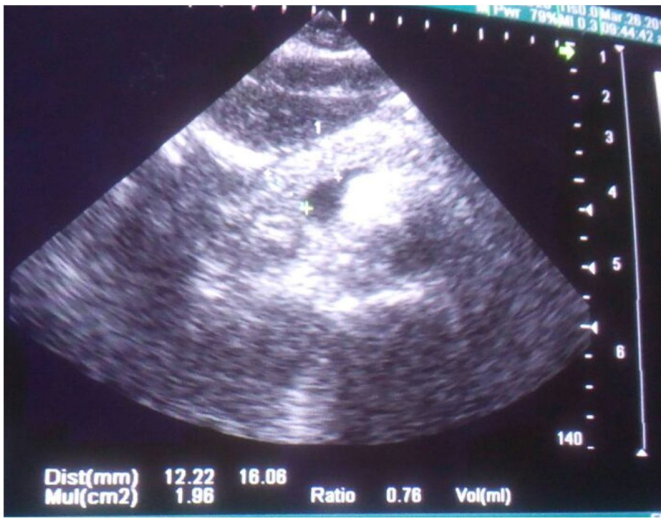
- Image (5): sonogram of pancreas female 50 years old and 3 years had diabetes

- 

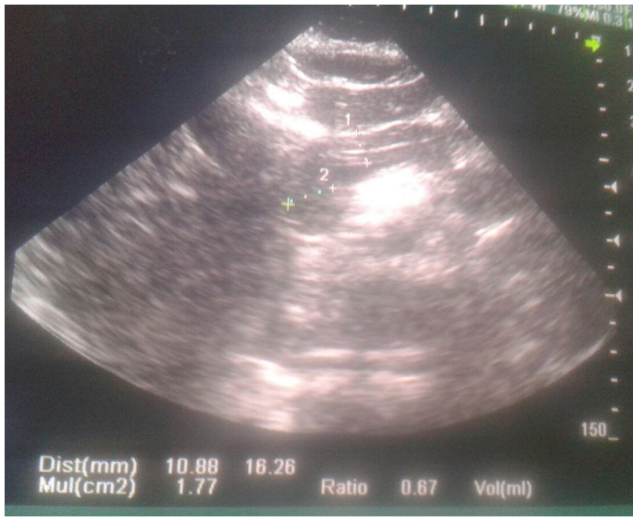
- Image (6): sonogram of pancreas male 67 years old and 8 years had diabetes

- 

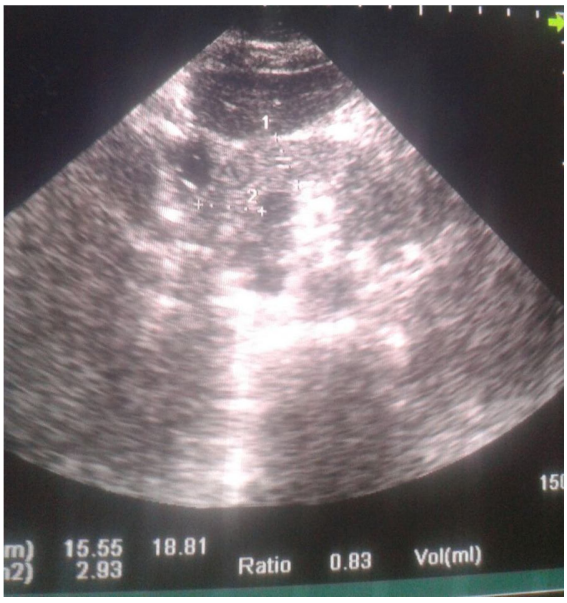
- Image (7): sonogram of pancreas male 73 years old and 12 years had diabetes



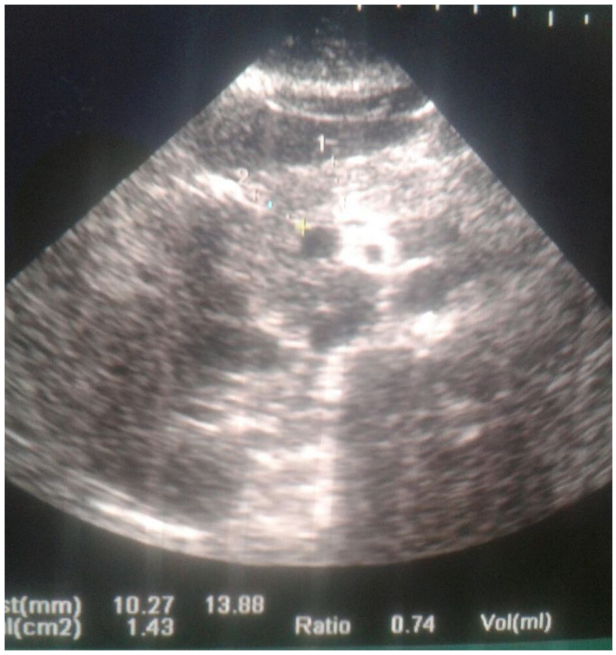
- Image (8): sonogram of pancreas female 54 years old and 2 years had diabetes

- 

- Image (9): sonogram of pancreas male 75 years old and 22 years had diabetes

- 

- Image (10): sonogram of pancreas female 50 years old and 3 years had diabetes

- 

- Image (11): sonogram of pancreas male 72 years old and 24 years had diabetes

Appendix 2

gender	Age	Weight(Kg)	Size of head	body	Echogenicity	Chorionicity (Year)