Introduction

1.1 Introduction

The pancreas lies in the epigastric and left hypochondriac region .the anterior surface is covered by the lesser sac of (mental bursa) .the Posterior surface is adherent to the paraneal peritoneum of the dorsal body Wall. Anatomically is divided into head measured 2.5cm (width) body Measured 2cm (width), and tell measured 2 cm (width) (April Ernest, 1997).

The panaceas is both exocrine and endocrine glands, it's exocrine Function is to produce pancreatic juice. The endocrine panaceas consist of the 1 million microscopic clusters of endocrine cells, the islets of the langerhans of the pancreas threes cells synthesize insulin and alpha cells Elaborate glucagon (Green 1978).

Diabetes mellitus is a chronic disorder of carbohydrate, fat, and protein Metabolism, relative or absolute deficiency in the insulin secretary response is characteristic features of the diabetes mellitus , as is the Resulting in hyperglycemia (vinay Kumar et al 2002).

Diabetes mellitus has been classified tow major categories; these differ in There pattern of inheritance, insulin responses and origin. Type 1 Diabetes, insulin dependent diabetes mellitus or juvenile onset Diabetes, caused by autoimmune destruction of the beta cells, and type II diabetes, non insulin dependent is a more complex than diabetes type I because there is combination of resistance to the actions of insulin in liver and muscle together with impaired pancreatic .B –cell Function leading to relative insulin deficiency. Insulin resistance appears to come first, and leads to elevated insulin secretion in order to maintain Normal blood glucose levels. The primary cause of insulin resistance Remain unclear. Intra abdominal

(central) adipose tissue is metabolically active and release large quantities of FFAs which may induce insulin Resistance. Physical activity is another important determinant of insulin Sensitivity (NickiR.et al 2010).

Pre-diabetic pancreas in type 1diabetes are include insulates (infiltration Of the islets with momo nuclear cells containing activated macrophages, Helper cytotoxic and suppressor T lymphocyte B lymphocytes), initial patchiness of this lesion with ,until a very late stage ,lobules Containing heavily infiltrated islets seen adjacent to unaffected lobules And B-cell specificity of the destructive process. In the early stage of type2 diabetes, reduction in the total mass of pancreatic islet tissue is modest . At the time of diagnosis, around 50% of B-cell function has been lost and this declines progressively with time. Deposition of fat in the liver is a common association with central obesity and is exacerbated by insulin resistance and/or deficiency (NickiR.et al 2010).

Pancreas could be investigated by different modalities e.g.: computed Tomography magnetic resonance imaging and ultrasound. CT and MRI are good informative type of investigation but they are quite expensive. Ultrasound is becoming widely used in investigation an abdominal Organs including pancreas.

1.2 Problem

There was Lack of represents in field of diabetic type-II changes using ultrasonography in Sudan

1.3 Objectives

1.3.1 General Objectives

Evaluation of pancreas in diabetic patient's type-II using ultrasonography.

1.3.2 Specific Objectives

To detect the morphology variation of pancreas in diabetic patient using ultrasonography in relation with disease duration.

To measure head, neck, body and tail of pancreas.

To correlate between pancreas in non diabetic patient and the diabetic patient type-II.

1.4 Study Overview

The research fall on five chapters

Chapter one which include introduction, problem and study, objectives and overview of the study.

Chapter two included an anatomy, physiology, pathology and previous studies.

Chapter three deals with the methodology, where it provides an outline of material and methods used to acquire the data in this study as well as the method of analysis approach.

Chapter four were presented in results.

Chapter five include discussion of results, conclusion and recommendation followed by references and appendices.

Literature review

2.1 Anatomy of pancreas

The pancreas is a comma-shaped organ that functions as both an exocrine and endocrine gland. It is located within the epigastrium between the C-loop of the duodenum and the splenic hilum, some of the pancreatic may be covered by peritoneum, the pancreas is considered to be retroperitoneal organ (Steven M. Penny et al 2011).

The pancreas is 12 to 18 cm long. Panaceas divided into the tail, body, neck, head, and uncinate process, Tail is most superior portion of the pancreas lying anterior and parallel with the splenic vein and anterior to the upper pole of the left kidney, posterior to the stomach, and lateral to the spine, generally extends toward the splenic hilum (occasionally left renal hilum). Body Largest and most anterior aspect of the pancreas Lies anterior to the aorta, superior mesenteric artery, superior mesenteric vein, splenic vein, left renal vein, and spine. and posterior to the antrum of the stomach. Neck Lies directly anterior to the superior mesenteric vein and portosplenic confluence. And posterior to the pylorus of the stomach. (Susanna Ovel et al 2009).

Pancreatic Head is the key pancreatic structure; common bile duct stones, periampullary neoplasm's, and pancreatic/ extra hepatic duct obstructions occur here. Failure to adequately visualize the pancreatic head is a common but usually avoidable technical failure. Supine compression imaging is useful but rarely allows demonstration of the periampullary region. Vascular landmarks for the pancreatic head are the inferior vena cava (IVC) dorsally, the SMA and (SMV) medially, and the gastroduodenal artery

(GDA) and the pancreatic duodenal arcade anterolaterally. The pancreatic head is usually directly ventral to the IVC. Cephalic to the pancreas, the IVC is adjacent to the portal vein; this location is the entrance into the lesser peritoneal sac, the epiploic foramen(foramen of Winslow). The uncinate process (or uncinate) is a portion of the caudal pancreatic head that wraps around behind the SMA and SMV, ending in a point oriented medially. The uncinate process is medial and dorsal to the SMA and SMV (Carol M. Rumack et al1998).

Neck of pancreas lies directly anterior to the superior mesenteric vein and portosplenic confluence and posterior to the pylorus of the stomach. Body of pancreas is Largest and most anterior aspect of the pancreas it Lies anterior to the aorta, superior mesenteric artery, superior mesenteric vein, Splenic vein, left renal vein, and spine and Lies posterior to the antrum of the stomach.(Susanna Ovel et al 2009).

Tail of pancreas Is narrow usually reaching the inferior part of the ga stric surface of the spleen ,lying in the splenorenl (lienorenal) ligament ,together with the spleenic vessels (Williams L .Peter et al 1995).

Pancreas size in adult head 3cm, neck 2.5cm, body2.5cm and tail 2 cm (Susanna Ovel et al 2009).
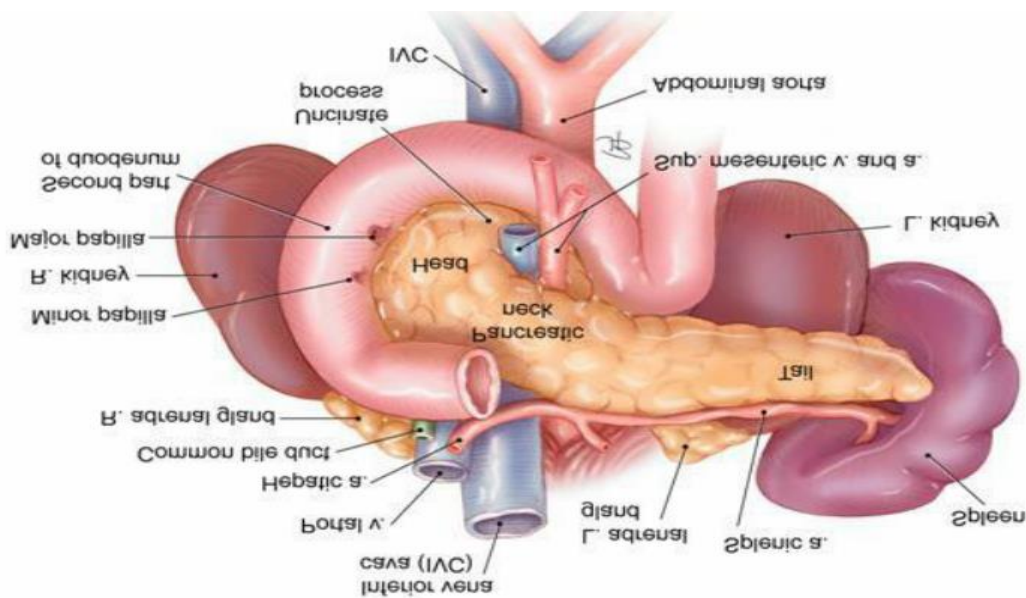
Figure.2.1 Anatomic relationships of the pancreas with surrounding organs and structures (Daniel S, et al, 2014).

Ducts of the pancreas contain smooth muscles that aid in transportation of the pancreatic enzymes. Duct of Wirsung is Primary secretoary duct extending the entire length of the pancreas and Joins the distal common bile duct entering the descending portion of the duodenum through the ampulla of vater. Frequently visualized in the body of the pancreas. Duct of Santorini Secondary secretary duct draining the upper anterior portion of the pancreas. Enters the duodenum at the minor papilla approximately 2 cm proximal to the ampulla of Vate (Susanna Ovel et al 2009).

Vascular anatomy of the pancreas arterial blood supply to the head of the pancreas is via the gastroduodenal artery. The body and tail of the pancreas receive their blood supply from the splenic and superior mesenteric arteries. Venous drainage is achieved by means of the splenic vein, superior mesenteric vein, inferior mesenteric vein, and portal veins (Steven M. Penny et al 2011).
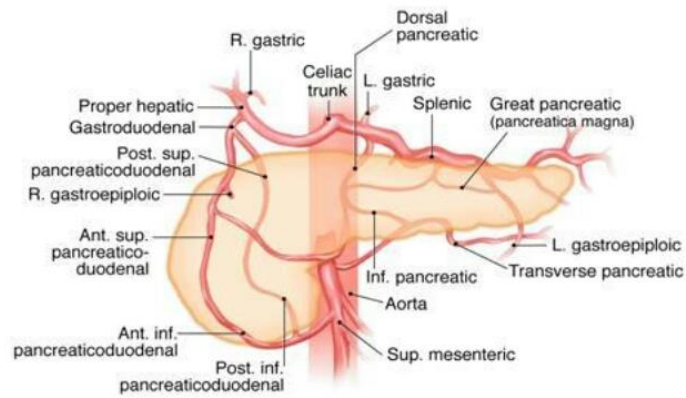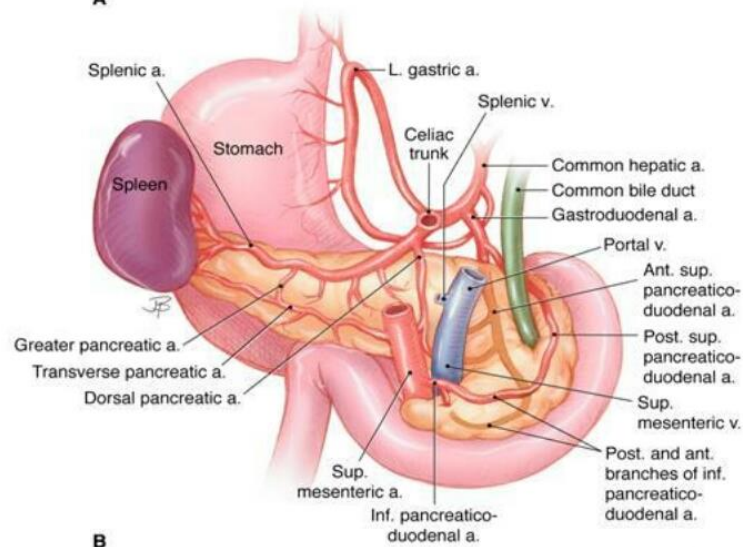
Figure 2.2 The arterial blood supply of the pancreas (Daniel S, et al, 2014).

2.2 physiology of pancreas

The pancreas is a comma-shaped organ that functions as both an exocrine and endocrine gland. Primarily, the pancreas is an exocrine gland that aids in digestion .The acinar cells of the pancreas carry out the exocrine function, as they produce vital digestive enzymes such as amylase, lipase, and sodium bicarbonate. The pancreas also produces trypsin, chymotrypsin, and carboxy polypeptidase as part of its exocrine function. These pancreatic enzymes drain from the pancreas into the main pancreatic duct, or duct of Wirsung, into the ampulla of Vater. There, they are mixed with bile from the liver and released into the duodenum through the sphincter of Oddi. The resultant fluid is mixed with chime in the duodenum to instigate the breakdown of various food components (Steven M. Penny, 2011).

Endocrine component of the pancreas consists of islet cells that create and release important hormones directly into the bloodstream. Two of the main pancreatic hormones are insulin, which acts to lower blood sugar, and glucagon, which acts to raise blood sugar. Maintaining proper blood sugar levels is crucial to the functioning of key organs including the brain, liver, and kidneys(Foulis AK, etal,2005).

The pancreas secretes enzymes that act on all the major types of foodstuff. All pancreatic enzymes are protein include ,trypsin, chymotrypsin, elastase and carboxy peptidase they are all secreted in an inactive precursor zygomen form, which is converted to the active enzyme in the intestinal lumen(SukarM.Y.et al 2000).

2.3 Pathology of the Pancreas

2.3.1 Congenital Anomaly

Congenital anomalies can present in pancreas like Pancreatic Divisum its Failure of the normal fusion of the ducts of Wirsung and Santorini. Duct of Wirsung is small and only drains the inferior portion of the pancreatic head. Duct of Santorini drains the majority of the pancreas. Associated with a higher incidence of pancreatitis, Annular Pancreas its rare anomaly caused by the failure of a normal regression of the left ventral bud. The head of the pancreas surrounds the duodenum, resulting in obstruction of the biliary tree or duodenum. Male prevalence, Ectopic Pancreatic Tissue Ectopic tissue located in the stomach, duodenum, and small or large intestines. Small, polypoid-appearing mass, male prevalence. And Cystic Fibrosis Autosomal recessive exocrine gland disorder in which organs become clogged with mucus secreted by the exocrine glands (Susanna Ovel et al 2009).

2.3.2 Pancreatitis

Pancreatitis is inflammation of the pancreas. The pancreas is a large organ behind the stomach that produces digestive enzymes. There are two main types, acute pancreatitis and chronic pancreatitis. Symptoms of pancreatitis include pain in the upper abdomen, nausea and vomiting. The pain often goes into the back and is usually severe. In acute pancreatitis a fever may occur and symptoms typically resolve in a few days. In chronic pancreatitis weight loss, fatty stool, and diarrhea occur. Complications may include infection, bleeding, diabetes, or problems with other organs (D. Whitcomb 2006)

The most common causes of acute pancreatitis are gallstones and heavy alcohol use. Other causes include direct trauma, certain medications, infections such as mumps, and tumors among others. Chronic pancreatitis

may develop as a result of acute pancreatitis. It is most commonly due to many years of heavy alcohol use. Other causes include high levels of blood fats, high blood calcium, some 24 medications, and certain genetic disorders such as cystic fibrosis among others.(D.Whitcomb 2006).

Smoking increases the risk of both acute and chronic pancreatitis, Diagnosis of acute pancreatitis is based on a threefold increase in the blood of either amylase or lipase. In chronic pancreatitis these tests may be normal. Medical imaging such as ultrasound and CT scan may also be useful. Acute pancreatitis is usually treated with intravenous fluids, pain medication, and sometimes antibiotics. Typically no eating or drinking is allowed and a tube may be placed into the stomach. A procedure known as a endoscopic retrograde cholangiopancreatography (ERCP)

may be done to open the pancreatic duct if blocked. In those with gallstones the gallbladder is often also removed. In chronic pancreatitis, in addition to the above, temporary feeding through a nasogastric tube may be used to provide adequate nutrition. Long-term dietary changes and pancreatic enzyme replacement may be required. And occasionally surgery is done to remove parts of the pancreas.(D. Whitcomb 2006).

Acute pancreatitis occurs in about 30 per 100,000 people a year. New cases of chronic pancreatitis develop in about 8 per 100,000 people a year and currently affect about 50 per 100,000 people in the United States. Globally, in 2013 pancreatitis resulted in 123,000 deaths up from 83,000 deaths in 1990.It is more common in men than women. Often chronic pancreatitis starts between the ages of 30 and 40while it is rare in children. Acute pancreatitis was first described on autopsy in 1882 while chronic pancreatitis was first described in 1946.(D. Whitcomb 2006).
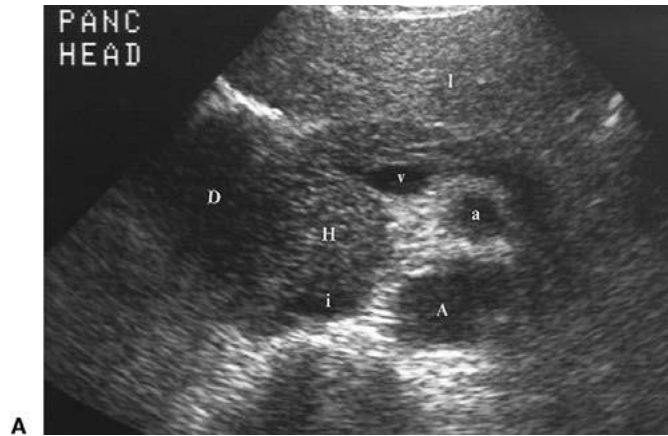
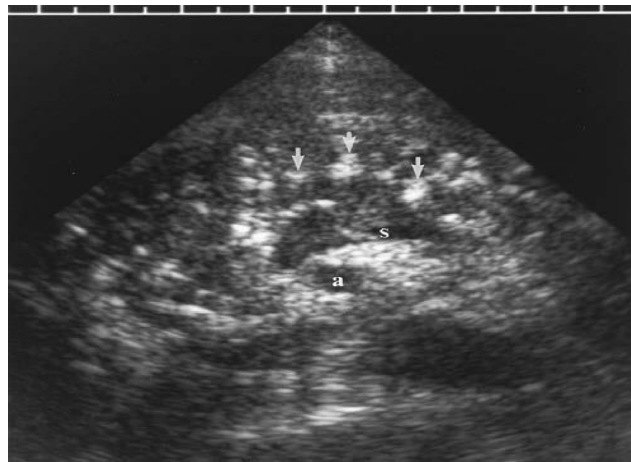**Figure 2.3**Acute pancreatitis (Steven M. Penny et al 2011)

Figure 2.4 Chronic pancreatitis (Steven M. Penny et al 2011)

2.3.3 Pancreatic Neoplasm's

Neoplasm's are difficult to differentiate from one another and are generally managed identically—by pancreaticoduodenectomy (Whipple resection). Jaundice is the most common presentation of these tumors (85%). Tumors in this group include pancreatic ductal adenocarcinoma (about two thirds of Periampullary neoplasms), ampullary carcinoma (15%-25%), duodenal carcinoma (10%), and distal cholangiocarcinoma (10%).Survival is best for duodenal and ampullary carcinoma, but only in comparison to the

poor survival rates for cholangiocarcinoma and especially pancreatic cancer Pancreatic ductal adenocarcinoma is the most common primary pancreatic neoplasm, accounting for 85% to 95% of all pancreatic malignancies. Ductal adenocarcinoma has a slight male predominance, most frequently affecting patients 60 to 80 years of age(Carol M. Rumack et al 2011).
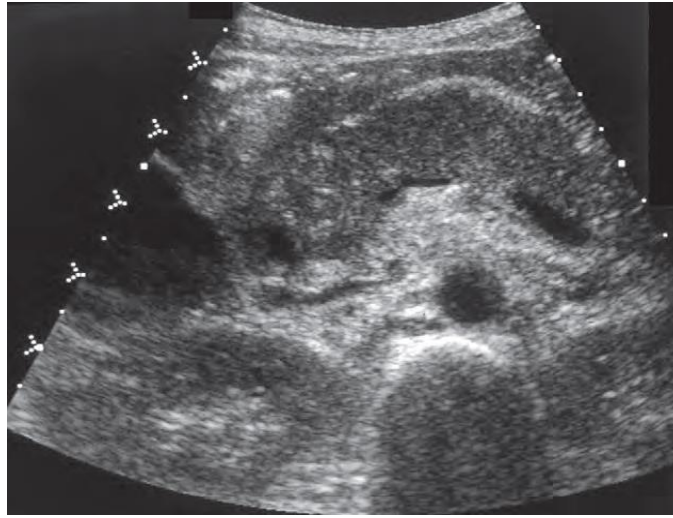

Figure 2.5 Diffuse pancreatic carcinoma (Carol M. Rumack et al 2011)

2.3.4 Cystic Pancreatic Lesions

 Cystic pancreatic lesions is increasingly important because improved imaging techniques result in the detection of progressively more of these lesions, Although the number of cystic lesions of the pancreas detected is increasing because of improved imaging techniques, pancreatic pseudocyst remains the most common, accounting for 75% or more of all cystic lesions .A careful history must be obtained to rule out previous acute or chronic pancreatitis, so as to minimize the chance of confusing pseudocyst with other cystic masses. Non pseudo cyst lesions include simple cysts and cystic neoplasm's Simple pancreatic cysts are rare in the general population, with a prevalence of 0.2%to 1.2%.These low percentages may

be underestimated because imaging and autopsy studies have recorded a substantially higher prevalence, about 20% and 24.3%, respectively. Our experience suggests that the lower prevalence rates are closer to the actual experience in clinical imaging. Detecting a simple pancreatic cyst should raise suspicion of an inherited disease that has a high prevalence of cysts, such as autosomal dominant polycystic kidney disease (ADPKD) or von Hippel–Lindau (VHL) disease. Multiple pancreatic cysts can also occur in cystic fibrosis. Multiple pancreatic cysts are more common in VHL disease than in ADPKD. Prevalence of pancreatic cysts in VHL patients ranges from 50% to 90%, making pancreatic cysts the most common lesion in VHL disease. Thus, multiple simple pancreatic cysts should suggest the diagnosis of VHL. In addition to simple cysts, other pancreatic lesions associated with VHL include serous cystic neoplasm and pancreatic endocrine tumors, with a slightly increased risk of ductal adenocarcinoma (Carol M. Rumack et al 2011).
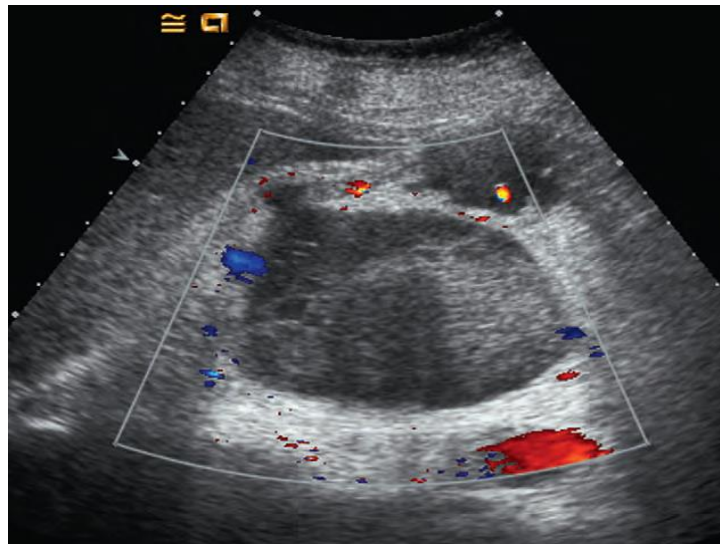

Figure 2.6 Hemorrhagic pseudocysts in pancreatic body (Carol M. Rumack et al 2011)

2.3.5 Diabetic Mellitus

Diabetes mellitus is group of metabolic disorders manifested by abnormally high level of glucose in the blood (Mealy et al 2000).

About 10 % of Caucasian patients with presumably type II diabetes have glutamate acid decarboxylase (GAD) antibodies or so-called latent auto-immune diabetes in adults (LADA). In addition, among patients with early onset of familial diabetes younger than 40 years, about 15 % carry mutations in MODY (Maturity Onset Diabetes of the Youth) or mitochondrial genes. It has been demonstrated that carriers of MODY mutations (even glucose-tolerant individuals) are characterized by a severe impairment of insulin secretion .In patients with newly diagnosed type II diabetes in the UKPDS, autoantibody detection (identifying patients with LADA) has been shown more powerful to predict subsequent insulin dependency than other classical predictive factors using stepwise logistic regression analysis, and this observation has been confirmed in other Scandinavian studies.(Fonseca V et al 2010).

These data clearly emphasize the heterogeneous nature of "type II" diabetes and stress the importance of defining the underlying subtypes when studying the pathogenesis of the disease, and especially the respective contributions of defects in insulin secretion and insulin sensitivity Controversy exists regarding the primacy of the contribution of defects in insulin sensitivity versus insulin secretion to the development of type II diabetes. Basal insulin concentrations in patients with type II diabetes have been reported as either elevated, reduced or normal. In addition, cross-sectional studies examining plasma insulin responses during have found

an inverted U relationship between the 2-h plasma glucose level (a generally recognized index of glucose tolerance) and the 2-h plasma insulin level. This pattern has been interpreted to indicate that early on, as glucose tolerance decreases, there is increased insulin secretion and, therefore, that insulin resistance, rather than insulin deficiency, is responsible for the development of impaired glucose tolerance. (Fonseca V et al 2010).

However, it is difficult to compare fasting insulin levels of different individuals unless they are matched for both the steady state glucose concentration and body adiposity (as a marker of insulin resistance) .When such matching is performed, it is evident that there is a marked insulin secretary defect in all patients with type II diabetes. Thus hyperinsulinaemia, even if appropriate for the prevailing hyperglycemia, does not necessarily indicate normal B-cell function. In addition, the interpretation of OGTT data may be questioned as it does not take into consideration the importance of the kinetics of insulin release and the dependence of insulin secretion upon the prevailing plasma glucose concentrations. Clearly there is a progressive decrease in the early (30 min) plasma insulin response as glucose tolerance deteriorates.(Fonseca V et al 2010).

These and other observations provide evidence that there is impaired pancreatic B cell function before the onset of impaired glucose tolerance (IGT) and that late hyperinsulinaemia may actually be the result of an inadequate B-cell response to the hyperglycaemia due to impaired early insulin release and may not necessarily indicate the presence of insulin resistance. Thus, the misleading dichotomy established between insulin deficiency (versus impaired insulin release) and insulin resistance has led to a general under emphasis of the issue of the appropriateness of B-cell

function. It has become clear that B-cell function should be interpreted in the context of the degree of insulin sensitivity. (Fonseca V et al 2010).

Failure to take into account the hyperbolic relationship between B-cell function and insulin sensitivity has been misleading. By accounting for this interaction, it has been clearly demonstrated that subjects at high risk for developing type II diabetes (older individuals, women with a history of gestational diabetes or polycystic ovary syndrome, subjects with impaired glucose tolerance) have impaired B-cell function. Despite the fact that it has long]been the prevalent view that insulin resistance is the main genetic factor predisposing to development of type II diabetes, the recent literature better supports the case of impaired insulin secretion being the initial and main genetic factor predisposing to type II diabetes. Evidence to support this view can be found especially in studies in people at high risk to subsequently develop type II diabetes as discordant monozygotic twins or first-degree relatives of patients with type II diabetes. (Fonseca V et al 2010).

In patients with IGT or in the early stages of type II diabetes, first phase insulin releases almost invariably lost. This defect results in an impaired inhibition of endogenous (liver) glucose production and plays a pathogenic role in post meal hyperglycaemia. Therefore, the restoration of the dynamics of insulin secretion following a meal should be seen as a rational therapeutic approach in the treatment of type II diabetes because it induces an overall improvement in glucose tolerance with the advantage of possibly lowering chronic hyper insulinaemia. Whereas type II diabetes is characterized by an absent first phase insulin release to intravenous glucose, second phase insulin release is still sensitive to

glucose and therefore partially maintained in patients with compensated diabetes. These responses are maintained by hyperglycaemia until plasma glucose can no longer rise sufficiently to compensated for impaired insulin secretion. Generally decompensation occurs in patients with fasting plasma glucose levels greater than 200-250 mg/dl]. (Fonseca V et al 2010).

The United Kingdom Prospective Diabetes Study (UKPDS) clearly showed that type II diabetes is a progressive disorder, and that the relentless decline in glycaemic control over years is undoubtly related to a decrease in B-cell function. We previously reported that insulin secretion decompensation (as assessed during an intravenous glucagon test) in face of insulin resistance (as assessed during a euglycaemichyerinsulinaemic clamp) and further progressive failure explain much of the natural type II diabetes and the progression towards insulin requirement. Indices of B-cell function that are independent of plasma glucose level or that account for the effect of hyperglycaemia reveal a marked impairment of insulin secretion early in type II diabetes. Alterations in pulsatile insulin release and ultradian oscillatory insulin secretion can be observed .The B cell is also unable to oscillate in concert with the fluctuations in plasma glucose induced by an oscillating glucose infusion. Inefficient pro insulin processing to insulin leads to increased pro insulin to insulin ratio. (Fonseca V et al 2010).

This abnormality does not appear to be secondary to the increased secretory demand but rather reflects a yet-undefined abnormality of B-cell function in the insulin secretory process. A reduction in the release of islet amyloid polypeptide (IAPP-, known also as amylin) has been observed in established

type II diabetes. Finally, the slope of glycaemic potentiating and AIR max are both markedly reduced whereas PG-50 is normal. Thus, there is a major reduction in the capacity of type II diabetic patients to secrete insulin despite normal sensitivity of the B cells to the potentiating effects of glucose .The mechanism for the defect in glucose regulation of insulin secretion in subjects with type II diabetes is largely unknown .Some data suggest that the islet mass is reduced and therefore the capacity of the pancreas to secrete insulin is diminished in type II diabetes. Recent observations showed that the major defect leading to a decrease in B-cell mass in type II diabetes is increased apoptosis. However, the reduction is not sufficient to explain the marked defect in insulin secretion. (Fonseca V et al 2010).

The functional B-cell loss exceeds the expected impact of a 20-50 % loss of B cells reported at autopsy. This loss may be explained by the simultaneous deposition of amyloid, a product of human IAPP normally produced in the B cell and secreted along with insulin. It has been hypothesized that the process of amyloid fibril formation impairs function early (as evidenced by disproportionate hyperproinsulinaemia) and leads to late B-cell failure and eventual death (Fonseca V et al 1985).

Studies to impair such fibril formation offer the possibility of developing preventive means for the relentless downhill course of the disease, which is one of the most important current clinical problems in type II diabetes management. Recent animal findings with B-cell-specific disruption of the insulin receptor suggest that impaired insulin secretion might be a result of insulin resistance in the B cells themselves.(Fonseca V et al 2010).

The loss of first-phase insulin secretion after glucose challenge seen in these BIRKO (Beta cell Insulin Receptor Knock Out) mice resembles what is seen in human type II diabetes. These data suggest that signals mediated by the insulin receptor are essential for the release of insulin secretary vesicles Finally, once diabetes is established, chronic hyperglycaemia and hyperlipidaemia can exert deleterious effects on B-cell function, respectively referred to as glucotoxicity and lipotoxicity. It is conceivable that glucotoxicity and lipotoxicity interdependently converge toward the generation of damaging effectors on B-cell function (Fonseca V et al 2010)

2.4 Ultrasound Technique of Pancreas

Patient preparation nothing by mouth (NPO) 6 to 8 hour before examination for adults, 6 hours for children, and 4hours for infants. Emergency examinations may be performed without preparation Use the highest-frequency abdominal transducer possible to obtain optimal resolution For penetration depth. Place gain settings to display the normal liver parenchyma as amedium shade of gray with adjustments to reduce echoes within the vessels. Focal zone(s) at or below the place of interest. Sufficient imaging depth to visualize structures immediately posterior to the region of interest (Susanna Ovel et al 2009).

Harmonic imaging and decreasing the compression(dynamic range) can be used to reduce artifactual echoes within anechoic structures and to improve prominence of posterior acoustic shadowing. Spatial compounding can be used to improve visualization of structures posterior to a highly attenuating structure. Begin with the patient in thesupine position. The pancreas lies obliquely in the abdomenand may be difficult to visualize. Use the liver as an acoustical window. The entire pancreas and surrounding vascular landmarks must be examined and documented in two scanning planes from the level of the celiac axis to below the renal veins. Varying patient position and imaging windows should aid in visualization Suspended inspiration, expiration, or Valsalva maneuver may optimize visualization distending the stomach with water may aid in outlining the pancreas. Documentation and measurement of any abnormality in two scanning planes with and without color Doppler should be included (Susanna Ovel et al 2009).

Normal Pancreas Smooth or coarse homogeneous parenchyma, Adult pancreas is either isoechoic or hyperechoic when compared to the normal

liver, May appear hypoechoic in young children and hyperechoic in older adults. May demonstrate a cobblestone appearance. Abnormal Pancreas Irregular or heterogeneous parenchyma and Calcifications (Susanna Ovel et al 2009).

Normal Pancreatic Duct Anechoic nonvascular tubular structure, Smooth parallelhy perechoic walls measuring 3mm in the head/neck and 2 mm in the body. Most commonly visualized in the body of the pancreas. Abnormal Pancreatic Duct Anechoic nonvascular tubular structure, Irregular or nonparallel hyperechoic walls. Measurement exceeding 3mm in the head/neck or 2 mm in the body (Susanna Ovel et al 2009).

Figure 2.7 Normal pancreatic ultrasound (Berthold Block et al 2004)

2.5 Previous Studies

In Mohammad Reza Kolahriz (2006) study Astatistically significant difference in mean pancreas head anterior- posterior diameter was observed between case (all diabetic patients) and control groups (P = 0.001). Likewise, comparing mean pancreatic body size of diabetic patients group (type I and II) to that of control group demonstrated a significant difference (P =0.001). Mean pancreatic head and body size were 17.2 ± 2.8 mm and 7.9 ± 1.6 mm, respectively, in insulin-dependent diabetic patients, whereas these measurements were 20.9 ± 3.6 mm and 9.4 ± 2.1 mm, respectively, in noninsulin dependent diabetic patients, and 24.2 ± 4mm and 13.5 ± 2.1 mm, respectively, in the control group. There was a statistically significant difference among the three groups (P<0.001). A direct correlation was observed between the pancreas head and body size (r = 0.69, P<0.001). An inverse correlation was observed between the size of pancreas head and duration of disease (r = -0.45, P<0.001). Likewise, an inverse correlation was seen between the size of pancreas body and duration of disease. Pancreas head and body size were different among the three groups. Of the three groups, the control group had the largest, and type I diabetic patients had the smallest pancreas head and body size. No significant difference in pancreas echogenicity was observed among the three groups.

M Gouldet al; 1985studied size of pancreas in diabetic patient based ultrasound. and this study in UK using a real time linear array system (Picker LS 3000), they tested Sixty adult diabetic patients: 22 had insulin dependent diabetes (group1) and 19 non-insulin dependent diabetes (group 2) and 19were non-ketotic patients who had to be given insulin because of inadequacy of diabetic control with oral hypoglycaemic

agents (group 3). Nineteen healthy controls were also studied. in this study measure the head(area medially to SMA) and body(area anterior to SV) of pancreas, These results show that the pancreas is significantly smaller in diabetic patients than in healthy controls. Furthermore, patients with insulin dependent diabetes have significantly smaller pancreases than patients with non-insulin dependent disease. Non-ketotic patients whose diabetes was not controlled with maximum doses of oral hypoglycaemic agents and who required insulin had pancreases intermediate in size between those in the other two groups. This is the first study to document these changes systematically. The pattern of diminution in the size of the pancreas in diabetes parallels the impairment of exocrine function

Previously described by us. Thus patients with insulin dependent diabetes have the lowest serum concentrations of pancreatic enzymes. Patients with non-insulin dependent disease have marginally reduced serum pancreatic enzyme values, while patients with non-ketotic disease who require insulin because of inadequate control with oral hypoglycaemic agents have serum pancreatic enzyme values between those of the other two groups. In group 3 the sizes of the head and body of the pancreas were intermediate between those in groups 1 and 2.There was no correlation between size of the pancreas and body weight or duration of diabetes.

Another study by Alzaid A etal (1993) they studied evaluation 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 II

(non-insulin-13 dependent) diabetes, and 19 non-diabetic subjects. in this study measure the head (area medially to SMA) and body(area anterior to SV) of pancreas. The result of their 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 p atients 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 insulin-deficient 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 II diabetic patients. Pancreatic size did not correlate with age, body mass index or the duration of diabetes. We conclude that the pancreas is a smaller organ in patients with diabetes mellitus and that the decrement in size is maxima in insulin dependent/insulin-deficient subjects. Ultrasonography, therefore, can potentially serve to discriminate between the different types of diabetes.(Alzaid A etal 1993). And A. Alzaidetal 1993 studied The Size Of the pancreas in diabetes by ultrasound ,there study was in Sudia Arabia and tested on diabeticpatients:14with Type1(insulin-dependent) diabetes,10insulintreated and 33tablet-treatedpatients with Type2(non-insulin-dependent) diabetes, and 19non-diabetic subjects. The result of their styde ,the pancreas of patients with Type1diabetes was markedly smaller ($p<0.0001$)than the pancreas in non-diabetic subjects. The result of this study The pancreas of patients with Type II diabetes was more moderate in size : larger ($p<0.001$) than that of Type1diabetic patients but smaller($p<0.5$) than the pancreas of the control. Pancreatic size did not

correlate with age, body mass index or the duration of diabetes. We conclude 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.(Alzaid A etal 1993)

Materials and Methods

3.1 Material

3.1.1 Subjects

Retroscrpective crosssectional study which carried out in Khartoum hospitals from February up to May 2017. Included 50 cases male and female (25 normal control group and 25 had diabetic mullets) all patient has aged 25 to 68 years old normal and had diabetic mellitus type-II and exclude diabetic type-I, will select to undergo the transabdomial ultrasound scanning. The subject was randomly selected with different age, duration of disease, gender and measuring head, neck, body and tail to find the correlation of these parameters to pancreas.

Pancreas ultrasongraphic scanning protocols over all ultrasonographic scanning will conducted by using ultrasound machine.

Patient well came Fasting for 8 to 12 hrs, if patient was eaten, still do the exam. Stomach contents can displace gas and act as a window for the sound beam. For this reason, 2 to 4 cups of water or noncarbonated drink can be given to the patient to evaluation if gas is obscuring the pancreas then ask patient to supine and Different position should be used whenever the suggested position does not give the desired result, using 3.0 or 3.5 MHz transducer or 5.0 MHz for thin patient and anterior lying pancreas. The measurement will conduct by placing the transducer on the superficial of the upper abdominal area with subjects laid in supine position during scanning

Figure (2-6).Shows us machines

3.1.2 Data Collection Equipments:

The device well set to a 2Dand convex probe with the frequency of 3,5MHz to 5 MHz will use for imaging from longitudinal and transverse planes.

3.2 Method

3.2.1 Scanning Patient with Ultrasound Using the Protocol of the Pancreas as Flow:

3.2.1.1Transabdominal Technique:

The patient lies supine and should have Fasting for 8 to 12 hrs, If the Pt has eaten, still do the exam. Stomach contents can displace gas and act as a window for the sound beam, for this reason, 2 to 4 cups of water or noncarbonated drink can be given to the patient to evaluation if gas is obscuring the pancreas. Can use breathing technique which is deep, held inspiration. Different breathing techniques should be used whenever the suggested breathing technique does not give the desired results

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Transverse and longitudinal scanning is preformed to assess the entire pan crease in many planes.

3.2.1.2 Measurement of the pancreas parts

Was taken by measuring longitudinal head, neck, body and tail

Which the normal measurement of head is 3.0 cm, normal measurement of neck is 2.5 cm normal measurement of body is 2.5 cm and normal measurement of tail is 2.0cm.

3.2.2 Data collection

Data collecting sheet was use to collect the date from number of patient,

Four regions were selected the head, neck , body and tail of pancreas, wall mass, echogensity, duration, age and gender.

3.2.3 Data analysis

the data of this study well analyzed by using Microsoft and statistical package for social science, using frequency tables bar plot and histogram to display the variable use .

3.2.4 Ethical considerations

There is no patient name or individual details all patient informed by the procedure and process of data acquisition.

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Results

4.1 Result and analysis

Table (4-1) Mean ± Std. Deviation for patient related variables.

variable	Mean ± std deviation
Age of diabetic patient(yrs)	45,92 ±9.165
Duration of diabetic disease (yrs).	9,36±6.96
Age of control group (yrs)	44.28± 10.532

Table (4-1) Mean ± Std. Deviation for patient related variables.

Variables	Mean± STD normal control group	Mean ± STD diabetic patient
Head of pancreas	2.3840±.07461	1.7788±0.038223
Neck of pancreas	1.816±0.1885	1.7352±0.05173
Body of pancreas	2.2760±0.07234	1.7280±0.03969
Tail of pancreas	2.288±0.072572	1.7196±0.0.03372

Table(4-3) frequency distribution of gender of diabetic patient

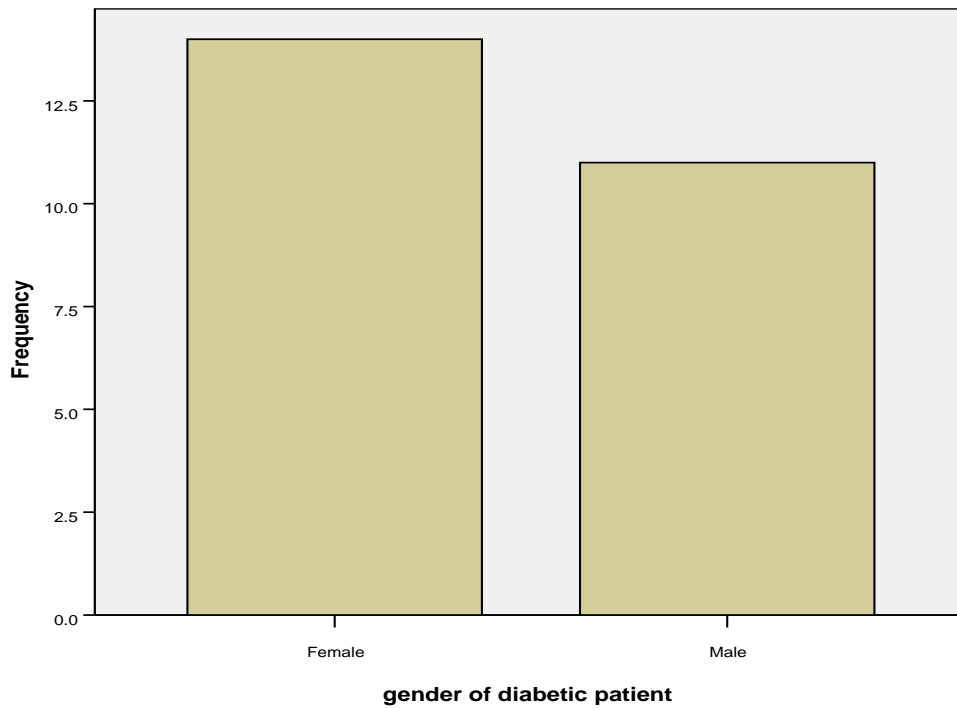|	Frequency	Percent	Valid Percent	Cumulative Percent
vaid				
Female	14	56.0	56.0	56.0
Male	11	44.0	44.0	100.0
Total	25	100.0	100.0	

Figure (4.1) Distribution of diabetic patient gender

Table (4-4) Descriptive Statistics for head of pancreas in diabetic patient

	Number	Minimum	Maximum	Mean	Std. Deviation
head in diabetic pt	25	1.68	1.86	1.7788	.03822
Valid N (list wise)	25				

Table (4-5) Descriptive Statistics for head of pancreas in control group

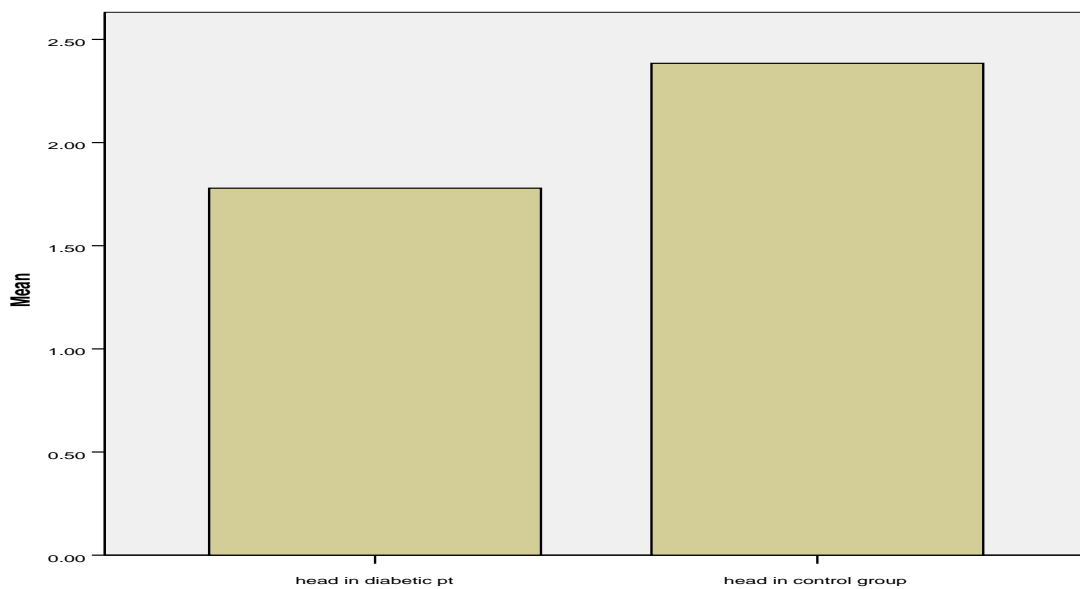|	N	Minimum	Maximum	Mean	Std. Deviation
head in control group	25	2.20	2.50	2.3840	.07461
Valid N (list wise)	25				

Figure (4.2) Relation between head of pancreases in diabetic and control group

Table (4-6) Descriptive Statistics for Neck of pancreas in diabetic patient

	N	Minimum	Maximum	Mean	Std. Deviation
neck in diabetic patient	25	1.66	1.80	1.7352	.05173
Valid N (listwise)	25				

Table (4-7) Descriptive Statistics for Neck of pancreas in control group

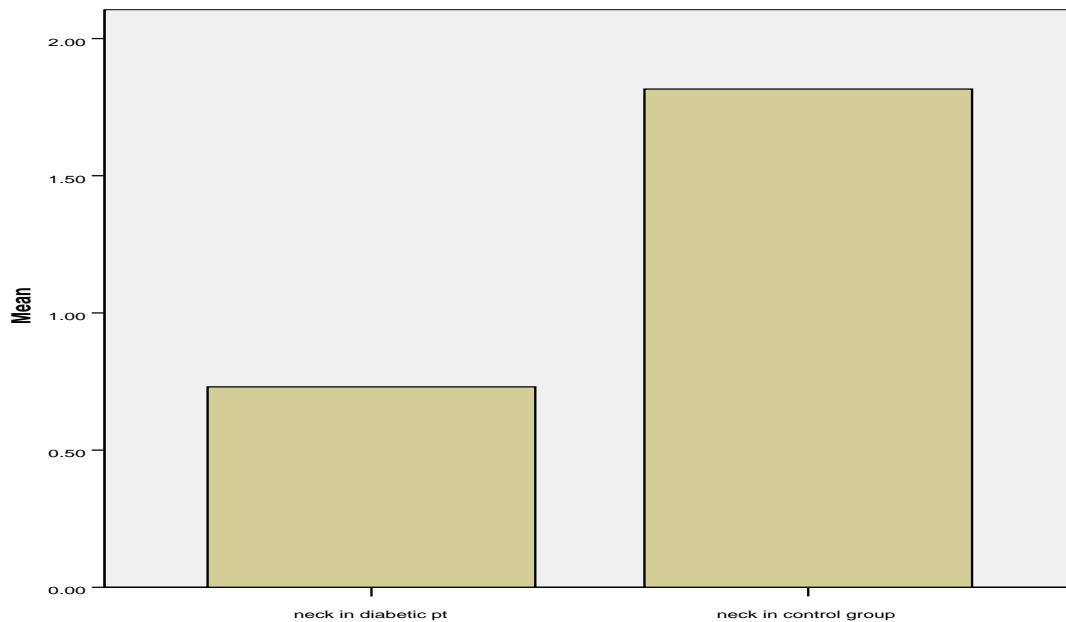|	N	Minimum	Maximum	Mean	Std. Deviation
neck in control group	25	1.400	2.000	1.81600	.188591
Valid N (list wise)	25				

Figure (4.3) Relation between neck of pancreases in diabetic and control group

Table (4-8) Descriptive Statistics for body of pancreas in diabetic patient

	Number	Minimum	Maximum	Mean	Std. Deviation
body in diabetic pt	25	1.64	1.80	1.7280	.03969
Valid N (list wise)	25				

Table (4-9) Descriptive Statistics for body of pancreas in control group

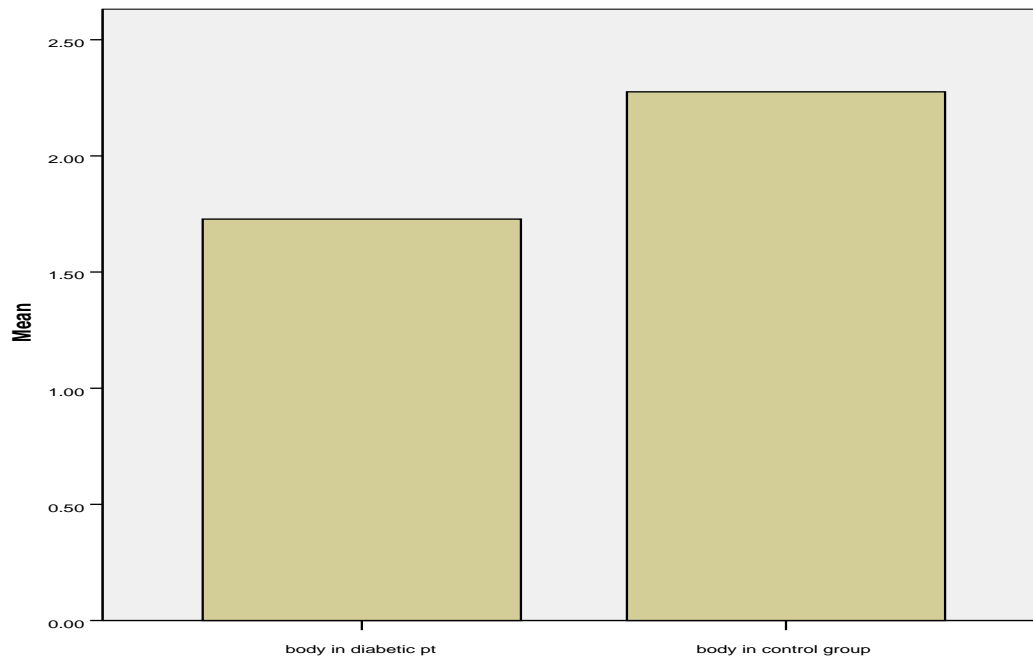|	N	Minimum	Maximum	Mean	Std. Deviation
body in control group	25	2.20	2.40	2.2760	.07234
Valid N (list wise)	25				

Figure (4.4) Relation between body of pancreases in diabetic and control group

Table (4-10) Descriptive Statistics for tail of pancreas in diabetic patient

	Number	Minimum	Maximum	Mean	Std. Deviation
tail in diabetic pt	25	1.64	1.78	1.7196	.03372
Valid N (list wise)	25				

Table (4-11) Descriptive Statistics for tail of pancreas in control group

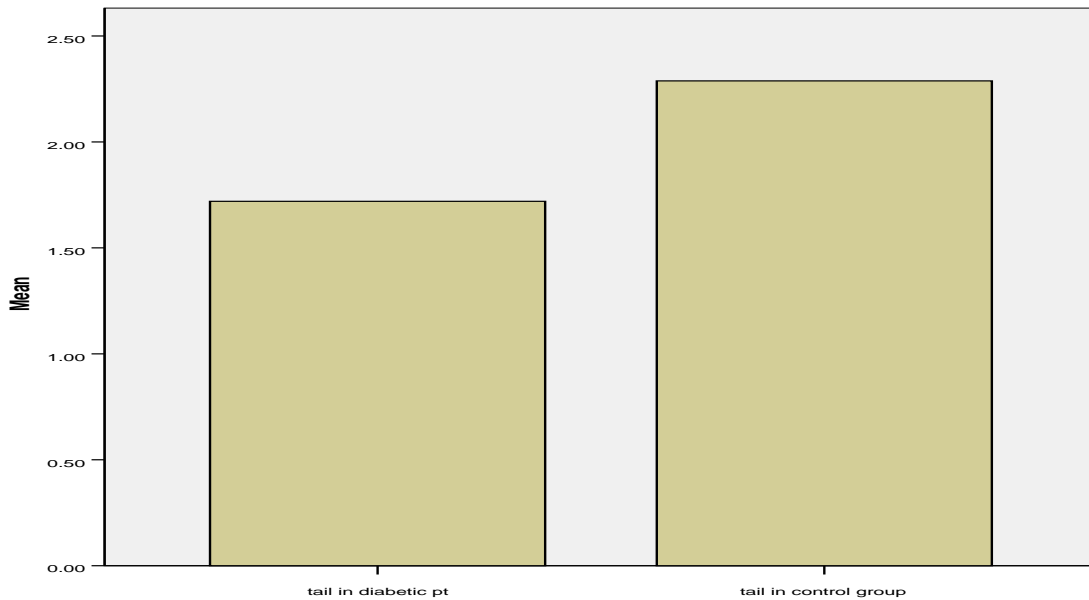|	N	Minimum	Maximum	Mean	Std. Deviation
tail in control group	25	2.20	2.40	2.2880	.07257
Valid N (listwise)	25				

Figure (4.5) Relation between tail of pancreases in diabetic and control group

Table (4-12) Descriptive frequency of pancreases echogensity in diabetic patient

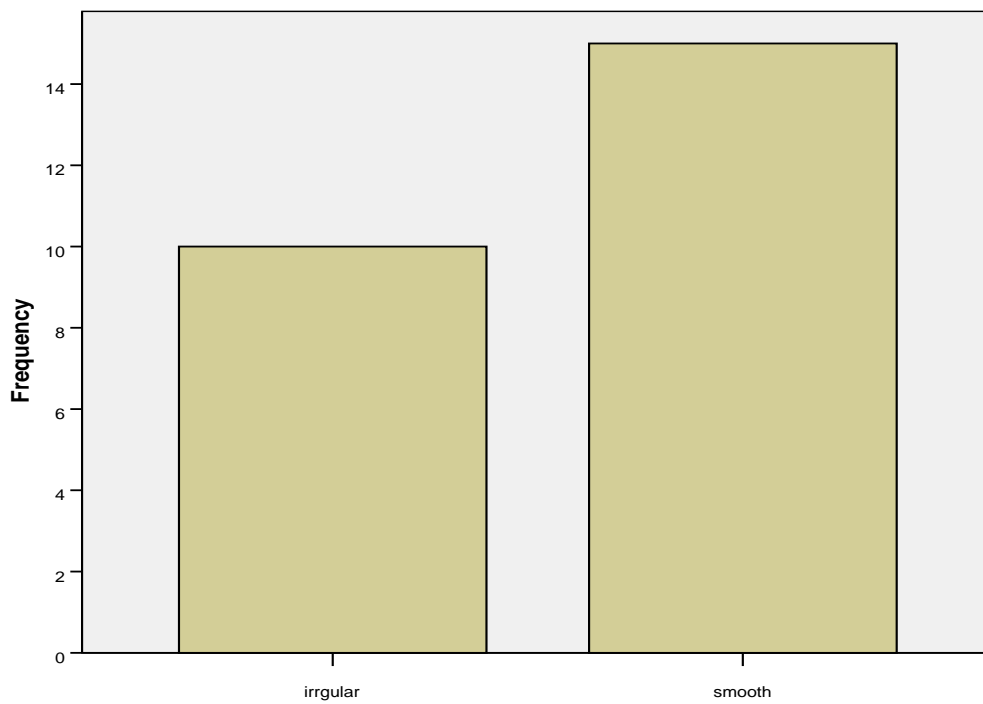|		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	hyper	13	52.0	52.0	52.0
	normal	12	48.0	48.0	100.0
	Total	25	100.0	100.0	
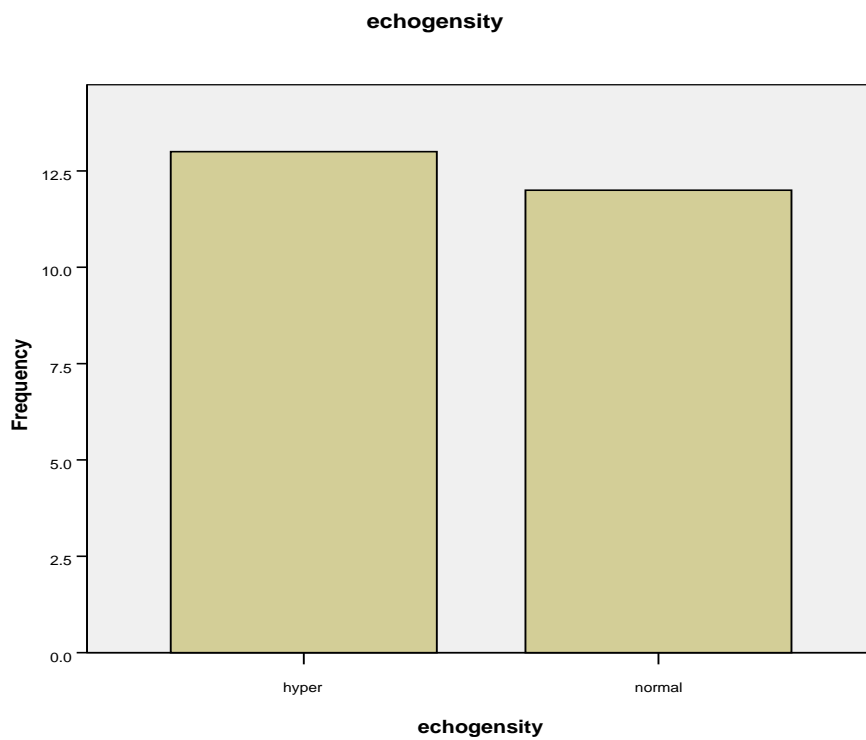
echogensity

Figure(4.6) Descriptive frequency of pancreases echogensity in diabetic pt

Table (4-13) Descriptive frequency of pancreases wall in diabetic patient

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	irrgular	10	40.0	40.0	40.0
	smooth	15	60.0	60.0	100.0
	Total	25	100.0	100.0	

wall of pancrease in diabetic patient

Figure (4.7) Descriptive frequency for pancreases wall in diabetic pt

Table (4-14) Descriptive frequency of pancreases mass in diabetic patient

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	no	22	88.0	88.0	88.0
	yes	3	12.0	12.0	100.0
	Total	25	100.0	100.0	

mas of pancrease in diabetic patient
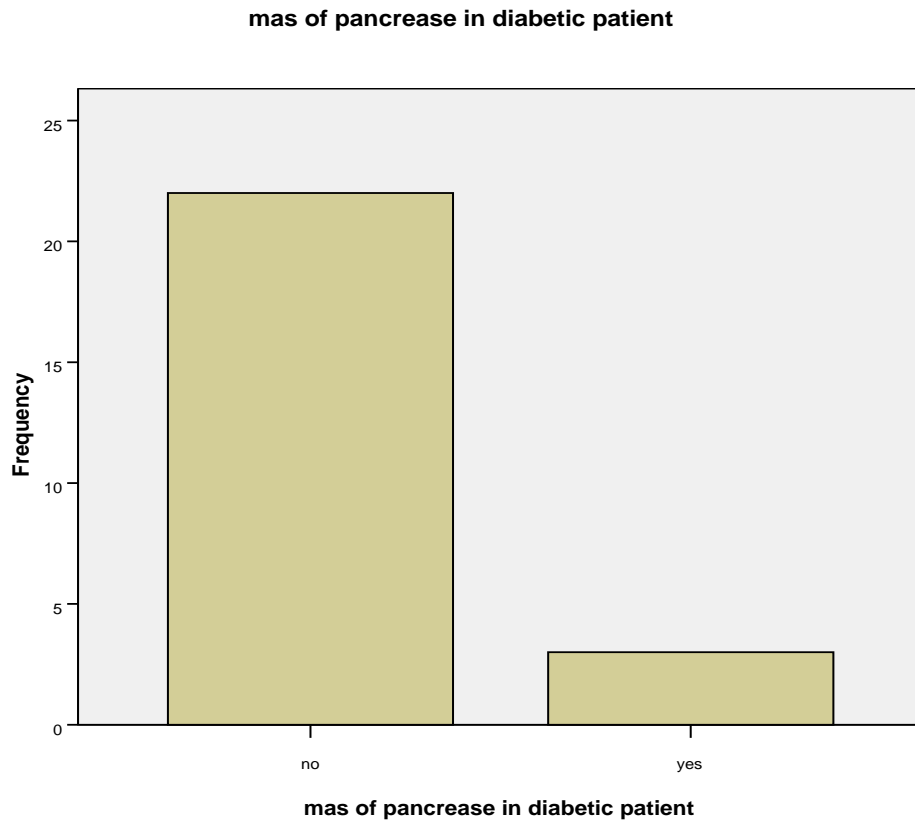
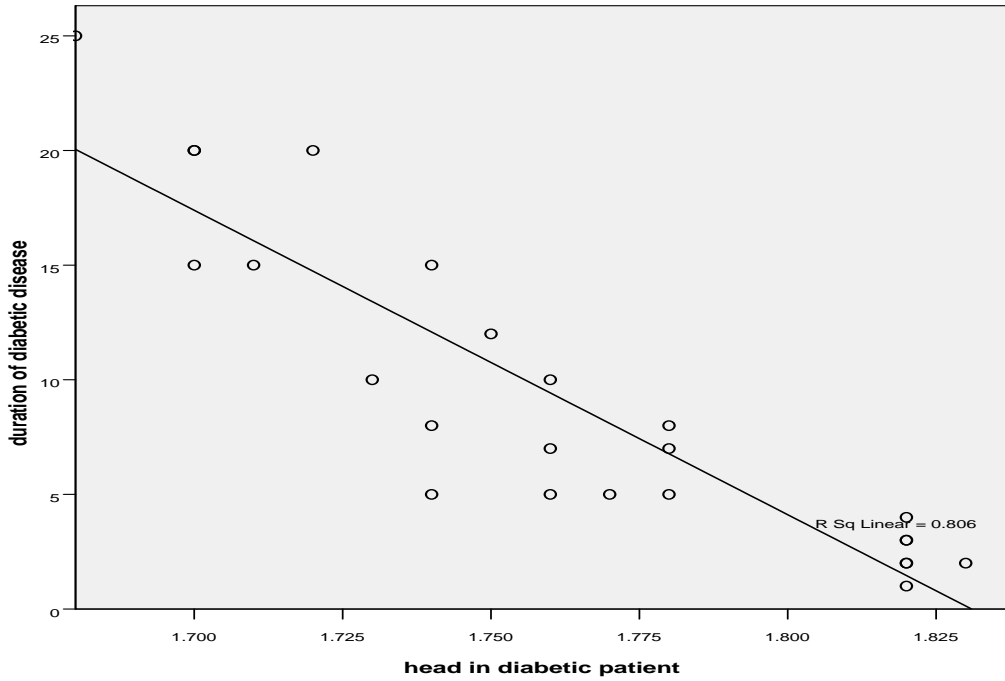**Figure (4.8)** Descriptive frequency for pancreases mass in diabetic pt

Figure (4.9) Scatter diagram demonstrate the relationship between head measurement and the duration of diabetes
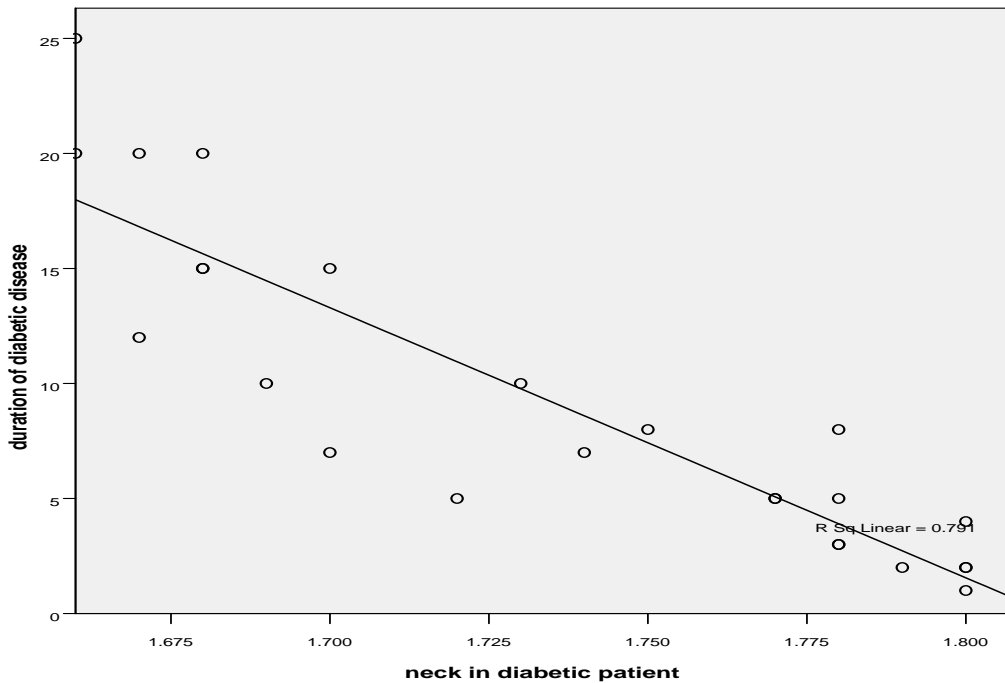

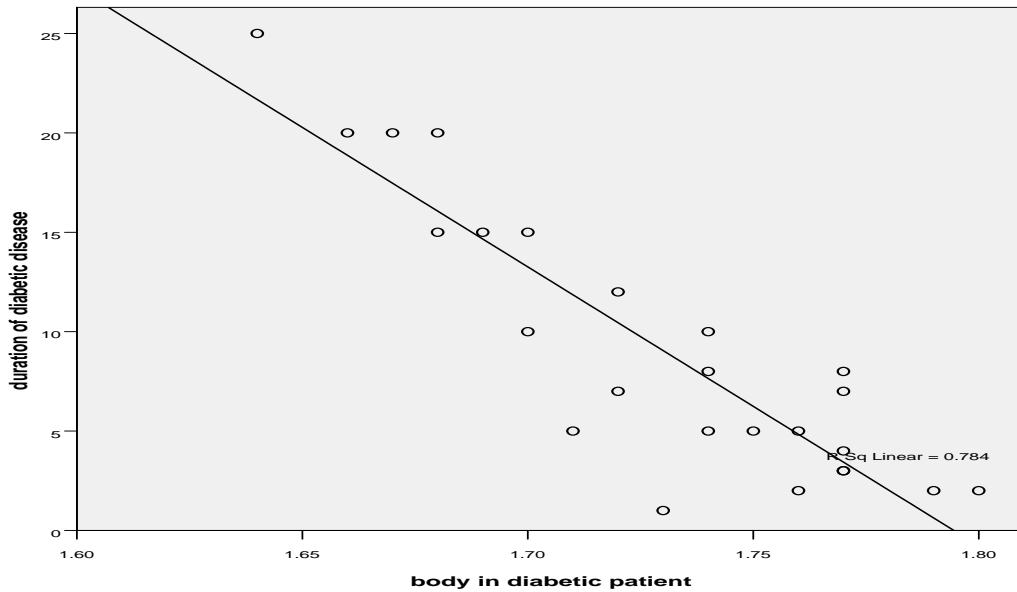**Figure (4.10)** scatter diagram demonstrate the relationship between neck measurement and duration of diabetes

Figure (4.11) scatter diagram demonstrate the relationship between body measurement and duration of diabetes
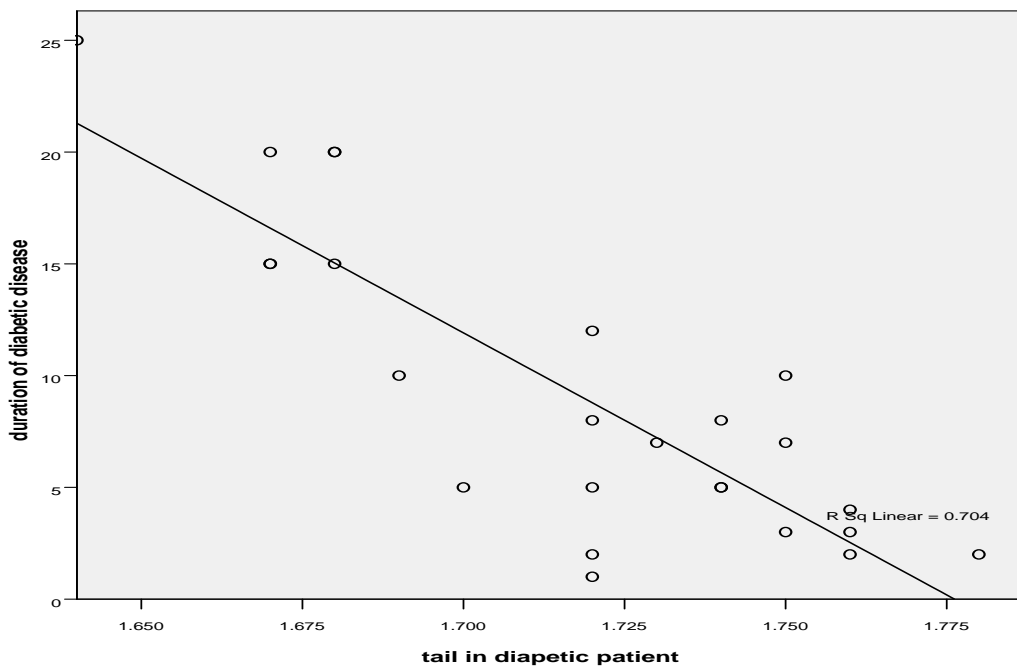

Figure (4.12) scatter diagram demonstrate the relationship between tail measurement and duration of diabetes

Discussion, Conclusions and Recommendations

5.1 Discussion

The result of this study showed that there was difference between the mean and S±TD of age of these diabetic patient which was 45,92 ±9.165 in diabetic and 44.28± 10.532 years for the control group in table (4-1).Table (4-2) showed that the head ,neck, body and tail mean ±STD are measurement:2.3840±0.07461,1.7788±0.038223/1.816±0.1885,1.7352±0.05 173/2.2760±0.07234,1.7280±0.03969 and 2.288±0.072572, 1.7196±0.03372 for control of healthy people and diabetic group. Table (4-3) showed descriptive statistics for head in diabetic pt witch the minimum measurement was 1.68 and maximum measurement was1.86 the mean was 1.7788 and stander deviation was.03822. Table (4-4) showed that the descriptive statistics for neck in diabetic pt witch the minimum measurement was 1.66 and maximum measurement is 1.80 the mean was 1.7304 and stander deviation was.05173.Table (4-5) showed that the descriptive statistics for body in diabetic pt witch the minimum measurement was 1.64 and maximum measurement is 1.80 the mean is 1.7280 and stander deviation was.03969. Table (4-6) showed that the descriptive statistics for tail in diabetic pt witch the minimum measurement was 1.64 and maximum measurement was 1.78 the mean is 1.7196 and stander deviation was.03372. Table (4-7) showed that the frequency distribution of gender of diabetic pt was 14 to female and 11 for the men whichwas the total of pt are 25 in diabetic group.

All these means that ultrasound finds in case of diabetic patient type-II usually include hyper echogensity, irregular out line, with or without presence of mass Mohammad Reza Kolahriz (2006), Alzaid (1993) agree with his study

The measuring of the head decreased by 0.0368 per year, the neck decreased by 0.0417 cm per year while the body decrease by 0.0366 cm per year, and the tail decrease by 0.0320 cm per year. head, neck, body and tail was decrees with disease duration Alzaid (1993) agree with this study.

5.2 Conclusions

This study aimed to evaluate and measure the pancreatic size in 50 diabetic patient and healthy control group by using ultrasound machine with curvilinear 3.5 MHz transducer. And tested in (Khartoum, Sudan) from Februry up May 2017.

All subjects were scanned for Abdominal Ultrasound in Transvers and longitudinal section, Measurements were obtained at areas including Head, neck, body and tail of pancreas this study include all diabetic patients with diabetic disease type II only and exclude all diabetic patients with type I, and analysis the collect data by Microsoft Excel and Statistical Package for Social Science program version.

The study concluded that there was significant invert relationship between the size of pancreas and diabetic duration compare with healthy control group, invert relationship between the size of pancreas and the age of patient, hyper echogensity of pancreases irregular out line, with or without presence of mass and gender was not influence.

5.3 Recommendations

Pancreas may be obscure by gases, so that make full descriptive preparations to patients give good result.

The measurements of pancreatic size for diabetic patient must be record to help the specialist to follow-up the patient and set the treatment plan.

Further studies should be done and increase number of patients to confirm the relation between diabetic and CBD and affect of diabetic in pancreatic duct.

Another study with more diabetes patients and correlate that study with different mathematic program may be use full.

The researcher notes that the size of the pancreas is different in normal Sudanese people so suggest study of that theory.

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