Print ISSN: 2321-6379
Online ISSN: 2321-595X
DOI: 10.17354/ijss/2017/379

Case Report

A Case Report of Pancreatic Lipomatosis

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Abstract

The pancreas is an exocrine and endocrine organ that is related to the stomach, duodenum, colon, and spleen. Fatty degeneration of the pancreas is common with aging; the entire pancreas may be replaced by fat, and the patient may have no clinical symptoms. We here present a case of an 18-year-old male known case of Type 1 diabetes who was admitted to our hospital for diffuse abdominal pain for the past 8 days, which was non-colicky in nature associated with nausea, anorexia and generalized weakness, no aggravating or relieving factors. With this scenario, he was investigated further for abdominal pain and found to have pancreatic lipomatosis.

Key words: Common bile duct, Computed tomography, Magnetic resonance cholangiopancreatography

INTRODUCTION

The pancreas is an exocrine and endocrine organ approximately 15-20 cm long that is related to the stomach, duodenum, colon, and spleen. Fatty degeneration of the pancreas is common with aging; the entire pancreas may be replaced by fat, and the patient may have no clinical symptoms. Fatty replacement of exocrine pancreas, also known as fatty infiltration, lipomatosis, adipose atrophy, or lipomatous pseudohypertrophy is a well-documented benign entity of speculative origin.[1] The exact etiopathogenesis behind fatty replacement is not known; however, several predisposing factors have been suggested. These include obesity, diabetes mellitus, chronic pancreatitis, hereditary pancreatitis, pancreatic duct obstruction by calculus or tumor, and cystic fibrosis.[2]

Fatty replacement may be focal or diffuse. Focal fatty replacement is the most common degenerative lesion of pancreas and has no major clinical significance. Total fat replacement is a rare condition and is associated with pancreatic enzyme deficiency and malabsorption. Following are few subtypes:

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Month of Submission : 05-2017
Month of Peer Review : 06-2017
Month of Acceptance : 07-2017
Month of Publishing : 07-2017

- Even pancreatic lipomatosis
- Uneven pancreatic lipomatosis.

There are four different types of uneven pancreatic lipomatosis. Type 1a (35% of cases) is characterized by replacement of the head with sparing of the uncinate process and peribiliary region; Type 1b (36%), by replacement of the head, neck, and body, with sparing of the uncinate process and peribiliary region; Type 2a (12%), by replacement of the head, including the uncinate process, and sparing of the peribiliary region; and Type 2b (18%), by total replacement of the pancreas with sparing of the peribiliary region.[3]

Progressive B-cell dysfunction, in the context of insulin resistance, is a hallmark of Type 2 diabetes.[4] Glucose toxicity, ensuing from diabetes-related hyperglycemia, has been regarded as a contributor to B-cell damage.[5] In contrast, chronic exposure of the pancreatic islets to non-esterified fatty acids (NEFAs) is considered as a potential primary cause of B-cell dysfunction.[6] In obese individuals, increased lipolysis contributes to high levels of circulating NEFAs, whereas liver insulin resistance leads to elevated hepatic output of triglyceride-rich particles.[7] When NEFA supply exceeds utilization, non-adipose tissues, including the pancreatic islets, start accumulating triglycerides,[6] which is aggravated by the simultaneous presence of hyperglycemia.[5,8,9] Experimental and autopsy data indicate that fatty infiltration of the pancreas may contribute to a decrease in B-cell mass and function.

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CASE REPORT

We here present a case of an 18-year-old male known case of Type 1 diabetes who was admitted to our hospital for diffuse abdominal pain for the past 8 days, which was non colicky in nature, radiating to back associated with nausea, anorexia, and generalized weakness, no aggravating or relieving factors. General examination revealed tachycardia, elevated blood pressure with a normal respiratory rate. Physical examination revealed mild generalized tenderness all over abdomen, respiratory, and cardiovascular examination showed no abnormality. Blood sugar level at the time of admission was 81 mg/dL. Hemoglobin A 1c was 13.9% serum ketones were 4+ and urinary ketones 1+. Routine blood tests, liver function test, renal function test, arterial blood gases, serum amylase and lipase, and levels were normal. Ultrasound of abdomen was normal. Computed tomography (CT) of abdomen revealed gross atrophy with near complete fatty replacement of pancreas. No intrapancreatic mass, calcification, or pancreatic ductal dilatation was seen. Magnetic resonance cholangiopancreatography (MRCP) using 2D fast spin echo (FSE) and spoiled gradient (SPGR) sequences revealed near complete fatty replacement pancreatic parenchyma, suggests pancreatic lipomatosis. Both hepatic ducts, common hepatic duct, cystic duct, their confluence and common ducts were normal. There was no evidence of dilatation of intra/extrahepatic biliary or portal radicals. On the basis of the above-mentioned findings, the diagnosis of diffuse pancreatic lipomatosis was made. Patients were managed with adequate hydration and insulin therapy and other supportive treatments (Figures 1-3).

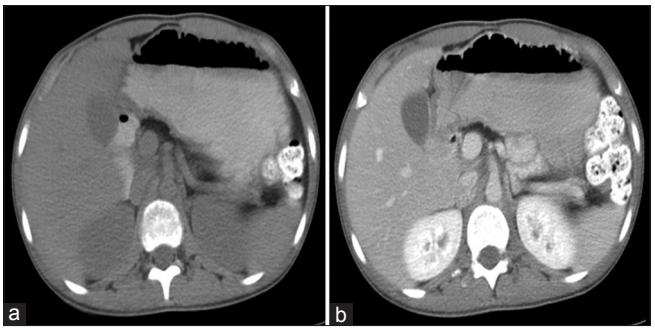

Figure 1: (a) Plain gross atrophy of pancreas is seen with parenchymal density uniformly decreased to the same level as that of the surrounding fatty tissue. (b) Contrast-enhanced: No pancreatic parenchyma enhancement seen

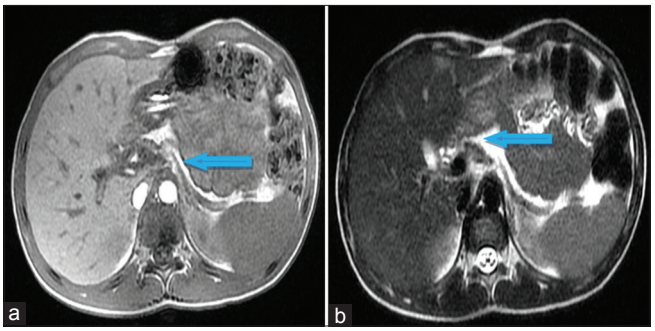

Figure 2: (a) T1 dual echo fast spoiled gradient echo, (b) axial T2 single-shot fast spin echo showing T1-weighted and T2-weighted hyperintense (fatty) signal of pancreas

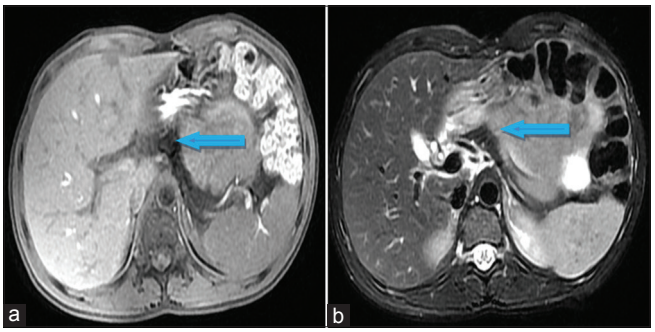

Figure 3: (a) Axial T1 FAT SATuration (FAT SAT) fast spoiled gradient echo, (b) axial T2 2D Fast Imaging Employing Steady state Acquisition (FIESTA) FAT SAT showing hypointense signal in pancreatic region s/o fat suppression

DISCUSSION

The accumulation of fat in the pancreatic gland has been referred to using various synonyms, such as pancreatic lipomatosis, fatty replacement, fatty infiltration, fatty pancreas, lipomatous pseudohypertrophy, non-alcoholic fatty pancreatic disease, and pancreatic steatosis.[10] Pancreatic lipomatosis is becoming an increasing problem worldwide due to the increasing incidence of obesity and diabetes mellitus. Fatty infiltration of the pancreas has been also reported in advanced cases of cystic fibrosis, Shwachman syndrome and Johanson-Blizzard syndrome. Other conditions related to diffuse fatty replacement of pancreas include steroid therapy, Cushing's syndrome, chronic pancreatitis, hemochromatosis, and malnutrition.

Fatty replacement may be focal or diffuse. Focal fatty replacement is the most common degenerative lesion of pancreas and has no major clinical significance. Total fat replacement is a rare condition and is associated with pancreatic enzyme deficiency and malabsorption. Fatty replacement may be uniform or unevenly distributed in the pancreas.

Patients are usually asymptomatic with small focal fatty replacement but may present with atypical abdominal pain, and/or steatorrhea, mass effect of pancreas on the duodenal loop, or malabsorption with severe fatty replacement.[11,12]

It is common in elderly and obese individuals and very rarely in youngs. Our patient was a young male who presented

with pain in abdomen and was diagnosed as pancreatic lipomatosis on CT abdomen. CT of abdomen revealed gross atrophy with near complete fatty replacement of pancreas. No intrapancreatic mass, calcification, or pancreatic ductal dilatation was seen. MRCP using 2D FSE and SPGR sequences revealed near complete fatty replacement pancreatic parenchyma, suggests pancreatic lipomatosis. Both hepatic ducts, common hepatic duct, cystic duct, their confluence and common ducts were normal. There was no evidence of dilatation of intra/extrahepatic biliary or portal radicals. Thus, probable cause of Type 1 diabetes in our patient was pancreatic lipomatosis. Considering the age of our patient it is a very rare entity in this age group.

Cross-sectional imaging, namely, CT has an important role in the evaluation of pancreatic disease. CT is particularly useful in detecting pancreatic duct obstruction by the calculus or tumor. Fatty infiltration of pancreas is seen on CT evidenced as pancreatic parenchymal soft tissue intermixed with fat. Associated atrophy is also seen in aged individual.

Fat accumulation in the pancreatic islets leads to a decreased insulin secretion and might explain why insulin resistant people cannot encounter the higher demands of insulin and then develop Type 2 diabetes mellitus.[13-17] In addition, a greater proportion of pancreatic fat was associated with increased insulin levels in obese non-diabetic participants. This may indicate that the toxic effect of pancreatic fat accumulation might require a long time before manifesting in impaired B-cell function and it has been assessed that pancreatic B-cell damage is present for more than a decade before diabetes is diagnosed.[18]

CONCLUSION

An 18-year-old male case of Type 1 diabetes found to have total pancreatic lipomatosis.

ACKNOWLEDGMENT

All the contributors would like to thank the entire medicine department which worked as a team in making the diagnosis and assisting the various procedures done for the patient.

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How to cite this article: Kashiva R, Agarwal R, Patil D. A Case Report of Pancreatic Lipomatosis. Int J Sci Stud 2017;5(4):280-282.

Source of Support: Nil, **Conflict of Interest:** None declared.