

# Is Non - Alcoholic Fatty Infiltration of Pancreas - A Precursor of Diabetes Mellitus?

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## Introduction

Fatty infiltration of pancreas (FIP) is defined as increased pancreatic parenchymal fat deposition which appears as echogenic pancreas on abdominal ultrasonography (USG) and hypodensity with fat-attenuation on computed tomography (CT) showing suppression of signals on fat-suppression techniques on magnetic resonance imaging (MRI). FIP has been described as a known factor affecting insulin secretion from pancreas as well as insulin sensitivity, both of which affect the pathogenesis of pre-diabetes & diabetes mellitus [1-3]. FIP is multifactorial with alcoholism and obesity being the commonest cause.

## Aims & Objectives

To determine relationship of non-alcoholic fatty infiltration of pancreas and pre-diabetes & diabetes mellitus.

## Material & Methods

This is a hospital-based, cross-sectional study of 100 subjects between 35 to 50 years of age who were evaluated for pre-diabetes & diabetes mellitus following sonographic confirmation of fatty infiltration of pancreas after exclusion of alcoholism. The study was performed on Siemens Acuson S2000 USG scanner.

## Inclusion criterion

Fatty infiltration of pancreas on ultrasonography

## Exclusion Criterion

Patients having fatty infiltration of liver were excluded

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from the study.

Transaxial images of neck and body region of pancreas was taken and its parenchymal echogenicity was compared to that adjacent left hepatic lobe. Fatty infiltration of pancreatic parenchyma was considered when its echogenicity was more than left hepatic lobe parenchyma. Before comparison of hepatic & pancreatic parenchymal echogenicity, fatty infiltration of liver was also noted. Patients with fatty infiltration of pancreas were subjected to fasting & postprandial blood sugar estimation along with HbA1c. Glucose tolerance test was also performed wherever required.

## Criteria used for pre-diabetes were:

- HbA1C of 5.7-6.4% or,
- Fasting blood glucose of 100-125 mg/dl, fasting is defined as no caloric intake for at least 8 hours or,
- An OGTT (75gm) 2 hour blood glucose of 140-199 mg/dl.

## Criteria used for diabetes are:

- HbA1C > 6.4% or,
- Fasting plasma glucose  $\geq$ 126 mg/dL (7mmol/L); fasting is defined as no caloric intake for at least 8 hours or,
- An OGTT (75gram) 2-hour blood glucose level  $\geq$  200 mg/dL.

## Results

Our study included 60 males and 40 females with median age 43 years.

Pre-diabetes was noted in 32% subjects with fatty infiltration of pancreas in our study i.e. 32 out of 100 while diabetes mellitus was noted in 18% subjects i.e. 18 out of 100.

Thus nearly, 50% subjects with non-alcoholic fatty infiltration of pancreas in our study revealed pre-diabetes and diabetes mellitus. Majority of the subjects with fatty infiltration of pancreas who were detected with pre-diabetes & diabetes were males (42 out of 50).

## Discussion

Multiple studies conducted in different parts of world have revealed a strong association of non-alcoholic fatty infiltration of pancreas with pre-diabetes and diabetes mellitus [1-3]. Ectopic fat deposition in the pancreas (fatty pancreas) is termed as nonalcoholic fatty pancreas disease when alcoholism is excluded.

Nonalcoholic fatty pancreas may promote development of chronic pancreatitis and pancreatic cancer, and may exacerbate the severity of acute pancreatitis [1-3]. In humans, pancreatic fat content is closely associated with BMI, insulin resistance, metabolic syndrome and hepatic parenchymal fatty infiltration.

Few studies indicate that pancreatic lipid content is negatively associated with insulin secretion in non-diabetic subjects and those with pre-diabetes [3]. Both pancreatic fat replacement with acinar cell death and pancreatic fat infiltration due to obesity contribute to pancreatic steatosis [1, 3]. Chronic exposure to high-fat diet in animal experiments induced both interlobular and intralobular fat accumulation, inflammatory cell infiltration and fibrosis in pancreas and thus damage to the normal pancreatic architecture and islets. Similarly, chronic exposure to high-fat diet caused development of insulin resistance and features of fatty infiltration of pancreas.

Fatty infiltration of pancreas can be easily detected on routine ultrasonography of abdomen in form of increased echogenicity of pancreatic parenchyma relative to the adjacent part of left hepatic lobe. CT demonstrates diffuse parenchymal fat attenuation on noncontrast studies with relative poor enhancement on postcontrast images. MRI has an advantage of revealing fatty infiltration of pancreatic parenchyma as diffuse hyperintensity on both T1 & T2 weighted images with suppression of signals on fat-suppressed images.

Since there is an increased prevalence of pre-diabetes & diabetes in patients showing fatty infiltration of pancreas, hence it should be reported in routine reporting as it may lead to early detection and management with subsequent decrease in co-morbidity.

## Conclusions

There is a high incidence of pre-diabetes and diabetes mellitus in patients with non-alcoholic fatty infiltration of pancreas especially where obesity has been excluded with a higher incidence in males. Rising incidence of pre-diabetes and diabetes mellitus in the community makes it imperative for radiologist to report non-alcoholic fatty infiltration of pancreas on routine ultrasonographic examination to facilitate early detection so that early institution of appropriate therapy can prevent related morbidity.

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