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Original Research Article

The association of non-alcoholic fatty pancreas disease and glycemic status

Pashaura Singh[1], Kamalpreet Singh[1], Avtar Singh Dhanju[1], Gurinder Bir Singh[2], Pranjali Batra[1]*, Alaipreet Kaur[3]

[1]Department of Medicine, [2]Department of Radiodiagnosis and Imaging, Government Medical College, Amritsar, Punjab, India
[3]Department of Radiodiagnosis, Dr. Rajendra Prasad Government Medical College, Tanda, Himachal Pradesh, India

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***Correspondence:**
Dr. Pranjali Batra,
E-mail: pranjali926@gmail.com

ABSTRACT

Background: Non-alcoholic fatty pancreas disease (NAFPD) is an emerging clinical entity. NAFPD is characterised by excessive fat deposition in the pancreas in the absence of alcohol consumption. Recent studies suggest that NAFPD might be associated with beta cell dysfunction, insulin resistance and inflammation which might lead to development of diabetes. NAFPD might be used as an initial indicator of glucometabolic disturbances and identify the patients with prediabetes.
Methods: This was a cross sectional study in which the glycemic status of 50 patients with NAFPD with ultrasonographic evidence of increased echogenicity of pancreas was assessed and association between glycemic variability and NAFPD was determined. The patients were also assessed for the ultrasonographic evidence of fatty liver.
Results: Pre-diabetes was noted in 32% subjects while diabetes was noted in 20% subjects. Thus, 52% patients with NAFPD had abnormal glycemic status. The 48% subjects i.e., 24 patients had normoglycemia. The presence of fatty liver was statistically significant in normoglycemia and diabetes mellitus with p=0.001 and 0.045 respectively. No statistically significant association was noted between fatty liver and prediabetes with p=0.175. No causal relationship was seen between fatty liver and glycemic variability in patients with NAFPD.
Conclusions: NAFPD is associated with impaired glycemic status. It is also seen frequently with fatty liver. Its early detection may help to identify the patients with prediabetes who may benefit from timely introduction of interventions to reduce the rising morbidity and mortality due to diabetes mellitus.

Keywords: NAFPD, Prediabetes, Diabetes mellitus, Fatty liver

INTRODUCTION

Non-alcoholic fatty pancreas disease (NAFPD) is an emerging clinical entity, despite the parallelism with NAFLD, which has been extensively studied, our knowledge about NAFPD is still in infancy. NAFPD describes fat deposition in pancreas in association with obesity.[1] NAFPD is also used to represent a phenotypic entity ranging from deposition of fat in the pancreas to pancreatic inflammation, and resultant fibrosis, similar to NAFLD. NAFPD may represent a manifestation of metabolic syndrome.[2] The potential sources of lipids in the liver or pancreas might come from circulating free fatty acids, de novo lipogenesis and dietary fat intake. Visceral adipose tissue appears to be pathogenic factor in the development of hepatic and pancreatic steatosis. It can release greater amount of adipokines and pro inflammatory cytokines, promoting insulin resistance, enhancing triglyceride lipolysis and thus releasing free fatty acids into the circulation and promoting deposition in liver and pancreas.[3] Intracellular accumulation of triglycerides and its metabolites in pancreas promotes

apoptosis and dysfunction of β-cells.[4] NAFPD is independently related to prediabetes and pancreatic fat content is negatively associated with insulin secretion in prediabetes.[5] Insulin resistance causes peripheral lipolysis, thereby increasing the influx of fatty acids to the liver.[6] Lipolysis in the pancreatic adipocytes further increases the portal influx of fatty acids. As a consequence, hepatic insulin resistance and NAFLD develop.[7]

Since pancreatic fat is associated with metabolic syndrome, type 2 diabetes mellitus, pancreatitis, pancreatic cancer and NAFLD, it is indispensable to diagnose fatty pancreas. Currently non-invasive imaging techniques form the cornerstone of diagnosis of NAFPD. These are abdominal ultrasonography, endoscopic ultrasound, computed tomography (CT), magnetic resonance imaging (MRI). Pancreatic fat accumulation appears hyperechoic as compared with liver or kidney, a reference point for diagnosis.[8] The increase in echogenicity of the pancreatic body over the kidney echogenicity is described as fatty pancreas. The pancreas and the kidney cannot be compared in the same acoustic window, the pancreatic echogenicity is compared with the kidney echogenicity using the difference between the pancreatic and liver echogenicity and liver and kidney echogenicity. Fatty pancreas is divided into four stages : non fatty pancreas group ,where pancreatic echogenicity is similar to the kidney parenchymal echogenicity; light fatty pancreas group, where pancreatic echogenicity is higher than the kidney echogenicity but very much lower than the retroperitoneal fat; severely fatty pancreas, where pancreatic echogenicity is higher than the kidney echogenicity but a little lower than retroperitoneal echogenicity; and highly fatty pancreas, where pancreatic echogenicity is similar to the retroperitoneal fat. Some studies suggest that retroperitoneal fat is better reference point for determining pancreatic echogenicity.[9] Figure 1 shows the increased echogenicity of the pancreas on the ultrasonography. Figure 2 shows increased echogenicity of liver on ultrasonography.

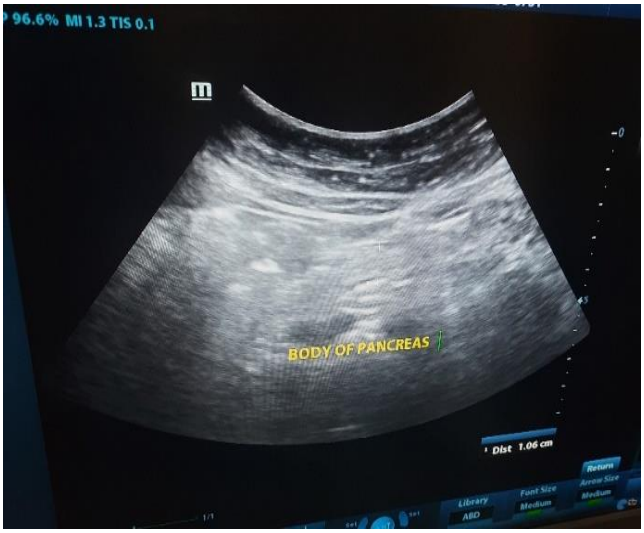

Figure 1: Increased echogenicity of pancreas.

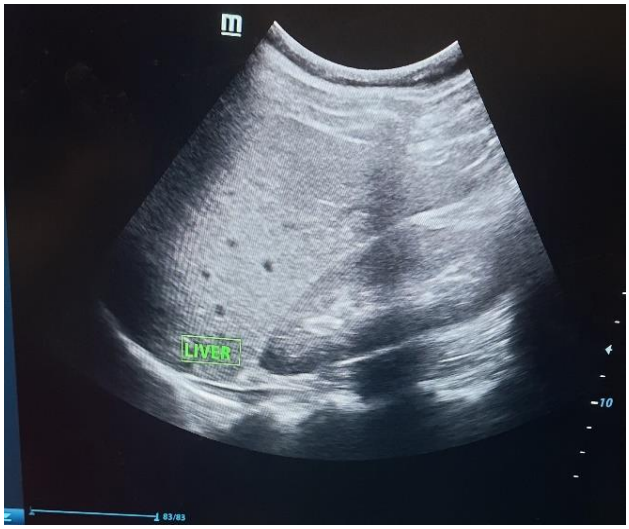

Figure 2: Increased echogenicity of liver.

Hounsfield units (HU) are used to measure the severity of pancreatic fat accumulation in the CT, showing lower value than spleen.[10] MRI signal arises from water and fat molecules within different organs, producing small differences in the resonance frequencies, thereby allowing quantification of fat.[11]

Detection of NAFPD may be an opportunity to advise patients regarding the risk of developing type 2 diabetes mellitus and regarding concomitant presence of NAFLD which may have important clinical implications such as progression to chronic liver disease.[12] The aim of the study was to determine the glycemic status in patients with NAFPD and to study the presence of NAFLD in NAFPD.

METHODS

The study was conducted in the medicine department in collaboration with the department of radiodiagnosis and Imaging, Guru Nanak Dev hospital attached with Government Medical College, Amritsar from period of April 2021 to December 2021 after taking permission from institutional ethics committee.

This was a cross sectional study. A total of 50 patients with NAFPD were enrolled in this study. The patients were explained in their vernacular language about the procedures to be adopted in the study and their informed written consent was taken. A detailed history, clinical examination and necessary laboratory investigations were performed.

Inclusion criteria

Subjects willing to comply with study requirements and had signed an informed consent form. All patients with either of the following were investigated for ultrasonographic evidence of NAFPD: waist circumference >90 cm in men and >80 cm in women, BMI (Body mass index) >22.9 kg/m^2, fasting triglycerides >150

mg/dl, HDL (high density lipoprotein) <40 mg/dl in men and <50 mg/dl in women were included in the study.

Exclusion criteria

Patients with alcohol intake >20 g/day, history of diabetes mellitus, history of pancreatitis, recent (3 months) changes in weight (5%) were excluded from the study.

The 50 patients fulfilling inclusion criteria with ultrasonographic evidence of fatty pancreas were included in the study. Transabdominal ultrasonography was done with SAMSUNG RS80A, Philips CLEAR VIEW 350 and Mindray DC-8 machines, using convex probes of frequency ranging from 3 to 5 MHz. Echogenicity of pancreas was assessed comparing it to that of liver, kidney and retroperitoneal fat. A standard questionnaire including a detailed history of present and past medical conditions; family history of medical diseases; previous history of medications, alcohol, drug addiction and blood or blood product transfusion was taken. Physical examination including general physical examination, per abdominal examination, cardiovascular examination, respiratory examination and central nervous system examination were done. Random blood sugar (RBS)/ fasting blood sugar (FBS) and oral glucose tolerance test (GTT) were checked for all study participants. A 2-hour OGTT (75 g) was performed after an overnight fast. Blood sugar levels were measured after 2 hours. According to the results of FBS and 2-hour post oral GTT RBS, patients were categorized as normoglycemia, prediabetes and diabetes mellitus.

Criteria used for prediabetes as per FBS and 2 hour post oral GTT RBS values were: FBS of 100-125 mg/dl, fasting is defined as no caloric intake for at least 8 hours. An oral GTT (75 g) 2-hour blood glucose of 140-199 mg/dl. Criteria used for diabetes mellitus as per FBS and 2 hour post oral GTT RBS values were: FBS ≥126 mg/dl, fasting is defined as no caloric intake for at least 8 hours. An oral GTT (75 g) 2-hour blood glucose of ≥200 mg/dl.[13]

Statistical analysis

The data was collected, tabulated, subjected to quantitative statistical analysis using Stata 16.0 (Stata Corp LLC, Texas, and USA) software and relevant conclusions were drawn.

RESULTS

Among 50 patients studied, 27 (54%) patients were males, 23 (46%) patients were females. The mean age of the studied patients was 50.14 years. The mean age was 49.07 years in males and 51.39 years in females.

Pre-diabetes was noted in 32% subjects i.e., 16 patients, while diabetes was noted in 20% subjects i.e., 10 patients. Thus, 52% patients with NAFPD had abnormal glycemic status. 48% subjects i.e., 24 patients had normoglycemia. Out of 27 males with NAFPD that 44.45% had normoglycemia, 33.33% had prediabetes and 22.22% had diabetes mellitus. Of 23 females included in study population, 52.17% had normoglycemia, 30.43% had prediabetes and diabetes mellitus was noted in 17.4%. Out of 26 patients with prediabetes and diabetes mellitus,15 were males. However, it was not statistically significant (normoglycemia p=0.586; prediabetes p=0.535; diabetes mellitus p=0.736).

The 36 patients (72%) out of total 50 patients of NAFPD had fatty liver on ultrasonographic examination. The presence of fatty liver was statistically significant in normoglycemia and diabetes mellitus with p=0.001 and 0.045 respectively. No statistically significant association was noted between fatty liver and prediabetes with p=0.175. Figure 3 shows presence of fatty liver according to glycemic status.

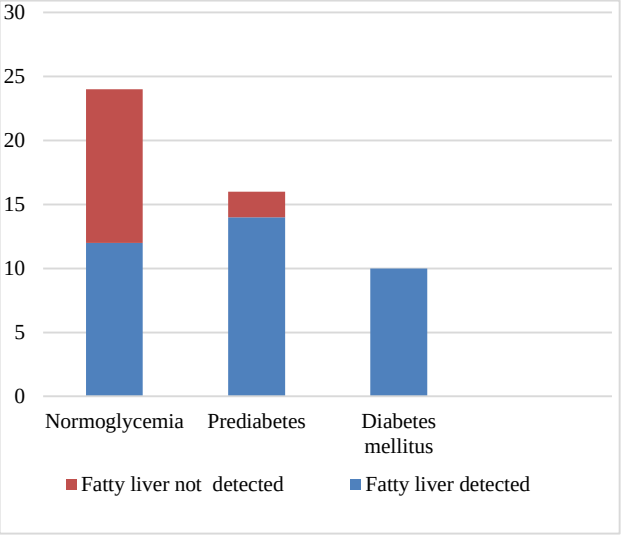

Figure 3: Presence of fatty liver according to glycemic status.

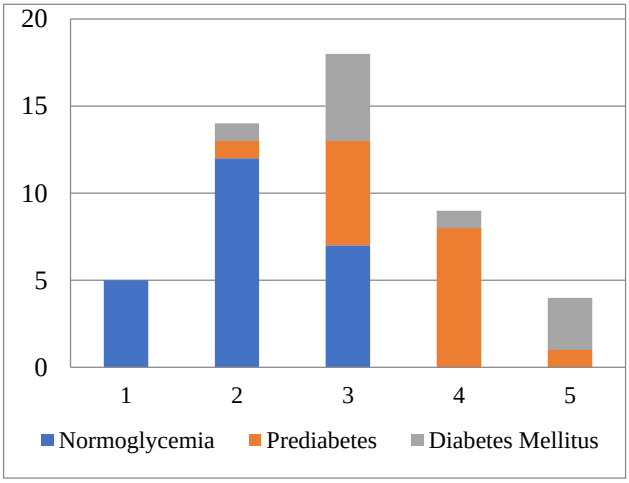

Figure 4: Variation of number of metabolic syndrome parameters according to glycemic status.

Table 1: The clinical characteristics of the study subjects with normoglycemia, prediabetes and diabetes mellitus.

Clinical characteristics	Normoglycemia	Prediabetes	Diabetes mellitus
FBS (mg/dl)	92.33±7.30, p<0.0001	107.69±5.73, p<0.0001	132.20±3.42, p<0.0001
2-Hour blood sugar post oral GTT (mg/dl)	133.42±4.07, p<0.0001	169±16.82, p<0.0001	216.60±7.18, p<0.0001
Total cholesterol (mg/dl)	185.12±9.47, p<0.0001	199.81±16.49, p=0.38	213.10±11.23, p<0.0001
LDL (mg/dl), (low density lipoprotein)	112.33±9.98, p<0.0001	123.69±15.12, p=0.46	132.70±13.78, p=0.004
Triglyceride (mg/dl)	133.17±19.43, p<0.0001	156.88±27.04, p=0.14	164.80±25.31, p=0.034
HDL (mg/dl)	42.25±4.15, p=0.9	40.44±5.68, p=0.18	45.50±4.12, p=0.036
VLDL (mg/dl), (very low-density lipoprotein)	26.42±3.63, p<0.0001	31.00±5.15, p=0.16	32.80±5.18, p=0.022
BMI (kg/m^2)	26.55±1.99, p<0.0001	29.36±2.11, p=0.004	28.43±2.45, p=0.768
Waist circumference (cm)	88.92±6.82, p=0.022	94±7.25, p=0.188	93.9±5.8, p=0.384

Among 50 patients of NAFPD, 31 patients (62%) had metabolic syndrome. The percentages of the NAFPD patients with metabolic syndrome in normoglycemia, prediabetes and diabetes mellitus were 29.17%, 93.375% and 90% respectively. The number of parameters of metabolic syndrome increased with impairment in glycemic status as shown in Figure 4. The presence of fatty liver in the studied patients increased the number of parameters of metabolic syndrome. It was statistically significant (p=0.043).

The 18 patients (36%) out of total 50 patients of NAFPD were hypertensive no statistically significant association was seen between hypertension and glycemic progression in patients with NAFPD (p>0.05). Ten patients (20%) out of total 50 patients of NAFPD were smokers There was no statistical significant association observed between smoking and glycemic status (p>0.05). ALT (Alanine transaminase), AST (Aspartate transaminase) and serum lipase levels were within normal limits in all patients and did not show any significant difference (p>0.05).

DISCUSSION

Pancreas is not just an another target organ of ectopic fat deposition but it has significant metabolic consequences.[14] Recently, it has been observed that NAFPD is associated with β-cell dysfunction, insulin resistance and inflammation, that cause the development of diabetes and metabolic syndrome.[3] Pancreas is considered as an early site of ectopic fat deposition and fatty pancreas is regarded as an early marker of insulin resistance.[9] The increase in pancreatic fat may cause the loss of β-cell mass and function and may promote the development of diabetes mellitus.[15] Fatty infiltration of the pancreas is observed as ectopic adipocytes infiltrating the pancreatic tissue, causing initially pancreatic hypertrophy and hyperplasia[16] resulting in insulin resistance and dysfunction of pancreatic β-cells, with the risk of type 2 diabetes mellitus (T2DM). Thus, NAFPD occurs due to lipid metabolism disorder and the resulting abnormal secretion of adipokines and ectopic fat deposition in other organs interact to cause insulin resistance and glucose metabolism disorder, which leads to type 2 diabetes mellitus.[17]

A total of 50 patients with NAFPD were enrolled in the study. There were 27 males and 23 females in this study. The prevalence of NAFPD was higher in men ≤40 years. The prevalence of NAFPD was higher in females >40 years. Weng et al in their study showed that the prevalence of NAFPD was higher in male subjects than females less than 55 years. There was no difference in the prevalence between males and females after 55 years of age. This can be attributed to the role of estrogen in energy regulation and lipid metabolism. Dysfunction of lipid metabolism, caused by decreased estrogen in postmenopausal women, might be the reason of the significant increase in the prevalence of NAFPD in women after 55 years of age.[17]

Prediabetes was noted in 32% subjects i.e., 16 patients, while diabetes mellitus was noted in 20% subjects i.e., 10 patients. Thus, 52% patients with NAFPD had abnormal glycemic status. 48% subjects i.e., 24 patients had normoglycemia. Hung et al observed in their follow up analysis of 8856 participants that during the 29819.2 person-years of follow-up, 1217 (13.7%) and 449 (5.1%) of the 8856 participants developed glycemic progression and new diabetes, respectively.

Fatty pancreas was associated with more glycemic progression and incident diabetes after adjustment for confounders, HbA1c (glycated hemoglobin) concentration and NAFLD.[18] We found in 27 males with NAFPD that 44.45% had normoglycemia, 33.33% had prediabetes and 22.22% had diabetes mellitus. Of 23 females included in study population, 52.17% had normoglycemia, 30.43% had prediabetes and diabetes mellitus was noted in 17.4%. 15 out of 26 patients with prediabetes and diabetes mellitus were males. However, there was no statistically significant difference (normoglycemia p=0.586; prediabetes p=0.535; diabetes mellitus p=0.736).

The mean total cholesterol, LDL levels, triglycerides levels, HDL levels and VLDL levels were significantly higher in diabetes mellitus (p<0.05). In subjects with prediabetes, the mean total cholesterol, LDL levels, triglycerides levels and VLDL levels were higher than normoglycemia and lower than diabetes mellitus.

However, the difference was statistically insignificant (p>0.05). The mean total cholesterol, LDL levels, triglycerides levels and VLDL levels were lower in normoglycemia and it was statistically significant (p<0.05). The mean HDL levels were higher in normoglycemia than prediabetes. However, there was no significant difference (p>0.05). Ou et al showed that with an increase in glycemia, a significantly greater proportion of subjects of both genders had NAFLD and fatty pancreas, low-HDL cholesterol, and hypertriglyceridemia. The total cholesterol levels and LDL levels were higher in prediabetes than normoglycemia and diabetes mellitus (p<0.05).[19]

The average BMI of the studied patients was 27.825 kg/m^2. It was higher in prediabetes than normoglycemia and diabetes. It was statistically significant in prediabetes and normoglycemia (p<0.05). The average waist circumference of the studied patients was 91.54 cm. The mean waist circumference was significantly lower in normoglycemia than prediabetes and diabetes mellitus (p=0.002). However, it was insignificant in prediabetes and diabetes mellitus (p>0.05). The 36% patients of NAFPD were hypertensive. No statistically significant association of hypertension was seen with glycemic progression (p>0.05). Ou et al showed that BMI was the highest in diabetes mellitus and the least in normoglycemia. It was highly significant (p<0.0001). Similar trends were noted for waist circumference and hypertension.[19]

It was found that 36 patients (72%) out of total 50 patients of NAFPD had fatty liver on ultrasonographic examination. Ulasoglu et al assessed the relationship between non-alcoholic pancreatic steatosis and non-alcoholic fatty liver disease. It was found that 15.1% had steatotic pancreas without NAFLD, and 50.7% had steatosis in both organs.[20] The presence of fatty liver was statistically significant in normoglycemia and diabetes mellitus with p=0.001 and 0.045 respectively. No statistically significant association was noted between fatty liver and prediabetes with p=0.175. No causal relationship was seen between fatty liver and glycemic variability. Hung et al showed that glycemic progression was the highest in patients with both NAFLD and fatty pancreas.[18]

In the present study, metabolic syndrome was detected in 62% of patients of NAFPD. Metabolic syndrome was noted in 29.17% of patients with normoglycemia. Metabolic syndrome was seen in 93.375% patients of prediabetes and 90% of diabetes mellitus. The number of parameters of metabolic syndrome increased with glycemic impairment. It was also observed that fatty liver was seen with patients with higher number of parameters of metabolic syndrome. It was statistically significant (p<0.05). These findings are in concordance with Lee et al. The incidence of metabolic syndrome in the fatty pancreas group was significantly higher than in the control group, and the numbers of metabolic syndrome parameters were significantly higher in the fatty pancreas group (p<0.05).[9] Similarly, Wu et al studied that the percentages of subjects displaying varying degrees of parameters for metabolic syndrome in fatty pancreas and non-fatty pancreas group. The percentages of subjects displaying 1, 2, 3, 4 or 5 parameters and the mean number of parameters were both higher for the fatty pancreas group (all p<0.01).[21]

In our study, it was observed that out of 50 patients of NAFPD, 10 were smokers. Among these, 3 had normoglycemia, 4 had prediabetes and 3 were found to have diabetes mellitus. No statistically significant association was observed between smoking and glycemic progression in patients with NAFPD (p>0.05). AST, ALT and lipase levels were all within normal range and did not show any significant difference in the present study Similarly, Kühn et al observed no significant association of pancreatic fat content with ALT/AST ratio and smoking but pancreatic fat content was related to lower lipase activity.[22]

There are some limitations in the present study. First, since the study is cross-sectional, it does not allow causal inferences between fatty pancreas and prediabetes or diabetes mellitus but the causal relationship is expected to link fatty pancreas and prediabetes or diabetes mellitus, not vice versa. Further, the sample size is too small to allow generalisation of results to the general population.

NAFPD is closely linked to dysregulation of glucose metabolism, fatty liver and the metabolic syndrome and its components. Therefore, it is imperative to identify it earlier to decrease the increasing menace of glucometabolic disorders.

CONCLUSIONS

NAFPD is an emerging clinical entity. NAFPD is associated with impaired glycemic status. It is also seen frequently with fatty liver. It is closely linked to metabolic syndrome. Its early detection may help to identify the patients with prediabetes who may benefit from timely introduction of interventions to reduce the rising morbidity and mortality due to diabetes mellitus.

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