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CASE REPORT

# Nonalcoholic fatty pancreas disease in obese children: case reports

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## ABSTRACT

Nonalcoholic fatty pancreas disease (NAFPD) is defined as excessive lipid accumulation in the pancreas, which occurs in obese patients. Similarly to nonalcoholic fatty liver disease (NAFLD), it can progress towards more advanced stage – nonalcoholic steatopancreatitis (NASP). However, the epidemiology, pathogenesis, including its relation to metabolic syndrome (MetS), and treatment recommendations have not been fully established yet. The aim of our study was to present three cases of NAFPD in obese children. We present their examination, diagnostic process, laboratory and imaging study results to note that not only NAFLD but also NAFPD can coexist with childhood obesity.

KEY WORDS:
**metabolic syndrome, obesity, nonalcoholic fatty liver disease, nonalcoholic fatty pancreas disease.**

## INTRODUCTION

The problem of obesity in the paediatric population is becoming increasingly widespread due to sedentary lifestyle and overconsumption of calorie-dense food [1]. The prevalence of overweight and obesity among Polish children aged 7–18 years is estimated to be over 14% in girls and over 18% in boys [2]. The childhood obesity epidemic has resulted in an increased incidence of paediatric metabolic syndrome (MetS), which is reported to be 29.2% in a population of obese children and adolescents [3]. Over the past years the prevalence of nonalcoholic fatty liver disease (NAFLD) has escalated as well, making it the most frequent hepatopathy worldwide. Changes ranging from simple fat accumulation to steatohepatitis and liver fibrosis are included in the spectrum of this disease [4, 5]. In the past two decades the matter of fatty infiltration of another organ of the digestive system, the pancreas, has gained increasing attention. Therefore, the aim of the study was to present three cases of nonalcoholic fatty pancreas disease (NAFPD) in obese children.

## CASE REPORTS

### PATIENT 1

An eleven-year-old girl was admitted to hospital due to an intermittent abdominal pain with accompanying loose stools. There was no complaint of nausea, vomiting, fever or blood and mucus in stools. Her past medical history was remarkable for NAFLD, subclinical hypothyroidism, psoriasis, nocturnal enuresis, and attention-deficit hyperactivity disorder. A general physical examination revealed excessive subcutaneous adipose tis-

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**TABLE 1.** Patients' characteristics

| Characteristic | Patient 1 – female, 11 years old | Patient 2 – male, 18 years old | Patient 3 – female, 15 years old |
|---|---|---|---|
| Weight | 57.8 kg (> 97 c) | 107.7 kg (> 97 c) | 108 kg (> 97 c) |
| Height | 139 cm (25-50 c) | 185 cm (75-90 c) | 167 cm (50-75 c) |
| BMI | 29.9 kg/m$^2$ (> 97 c) | 31.5 kg/m$^2$ (> 97 c) | 38.72 kg/m$^2$ (> 97 c) |
| Waist circumference | 91 cm (> 95 c) | 112 cm (> 95 c) | 125 cm (> 95 c) |
| Hip circumference | 95 cm (> 90 c) | 117 cm (> 95 c) | 134 cm (> 95 c) |
| WHR | 0.96 | 0.96 | 0.93 |
| BP | 114/74 mm Hg (> 90 c) | 159/87 mm Hg (> 99 c) | 143/86 mm Hg (> 99 c) |
| ALT | 96 U/l | 55 U/l | 31 U/l |
| AST | 58 U/l | 36 U/l | 21 U/l |
| GGT | 36 IU/l | 39 IU/l | 27 IU/l |
| Lipase | 63 IU/l | 15 IU/l | 134 IU/l |
| α-amylase | 36 IU/l | 41 IU/l | 95 IU/l |
| Total cholesterol | 185 mg/dl | 162 mg/dl | 167 mg/dl |
| HDL – cholesterol | 54 mg/dl | 63 mg/dl | 88 mg/dl |
| LDL – cholesterol | 107 mg/dl | 95 mg/dl | 81 mg/dl |
| Triglycerides | 253 mg/dl | 110 mg/dl | 51 mg/dl |
| Glucose | 81 mg/dl | 101 mg/dl | 84 mg/dl |
| Insulin | 15.3 uIU/ml | 8.69 uIU/ml | 14.6 uIU/ml |
| HOMA – IR | 3.06 | 2.17 | 3.03 |

*BMI – body mass index, WHR – waist-hip ratio, BP – blood pressure, ALT – alanine transaminase, AST – aspartate transaminase, GGT – gamma-glutamyltransferase, HOMA-IR – homeostatic model assessment – insulin resistance*

sue, acanthosis nigricans on the neck, psoriasis plaques behind the ears and on the upper limbs, and tenderness of the abdomen. The patient underwent anthropometric measurements, her blood, urine, and stool samples were taken, and laboratory tests were performed. On admission, her BMI was 29.9 kg/m$^2$. Anthropometrics and significant laboratory test results are presented in Table 1. Viral hepatitis (HCV, HBV, CMV), autoimmune hepatitis (AIH), and toxic and metabolic (Wilson's disease, α-1-antitrypsin deficiency) liver diseases as a reason for elevated activity of liver enzymes were excluded. C-reactive protein remained normal, and her stool was negative for occult blood, bacteria, parasites, and viruses (rotavirus, norovirus, adenovirus). Due to the typical signs of insulin resistance on physical examination, an oral glucose tolerance test together with insulin levels was performed. The test revealed flat glucose curve and hyperinsulinaemia. Homeostatic model assessment – insulin resistance (HOMA-IR) was calculated, and insulin resistance was confirmed (HOMA IR > 2.5). The patient underwent lactose hydrogen breath test. The result was negative, but typical symptoms of lactose intolerance were observed. Abdominal ultrasonography revealed enlarged (right lobe AP 120 mm), hyperechogenic liver with irregular hypoechogenic areas (steatosis) and hyperechogenic, steatotic pancreas (head – 27 mm, body – 17 mm, tail – 29 mm). Other organs of the abdominal cavity were described as normal. The patient was diagnosed with NAFPD, NAFLD (elevated alanine transaminase activity with concomitant liver steatosis in USG), and lactose intolerance.

## PATIENT 2

An 18-year-old boy was admitted to the hospital due to obesity and suspected NAFLD. Additionally, the patient had complained of dry cough during the previous year. Physical examination revealed excessive subcutaneous adipose tissue, steatomastia, and acne on the boy's face and back. On admission, his BMI was 31.5 kg/m$^2$. Anthropometric measurements and significant laboratory tests are included in Table 1. Similarly to the previous patient, other causes of hypertransaminasaemia were excluded – viral hepatitis, autoimmune hepatitis, and toxic and selected metabolic liver diseases.

Abdominal ultrasonography showed enlarged (right lobe AP 151 mm), hyperechogenic, fatty liver with hypoechogenic areas and hyperechogenic pancreas (steatosis).

There were no abnormalities detected in other organs of the abdomen. The patient was diagnosed with NAFPD and NAFLD.

### PATIENT 3

A 15-year-old female patient was admitted to the department due to obesity and suspected NAFLD. Additional problems included abnormalities of menstrual cycle treated with progesterone by a gynaecologist. On admission, her BMI was 38.72 kg/m$^2$. Physical examination revealed typical features of obesity – excessive subcutaneous adipose tissue and stretch marks on the skin of the abdomen. Her anthropometric measurements together with significant laboratory test results are presented in Table 1. Moreover, insulin resistance was confirmed after calculating HOMA-IR (HOMA IR > 2.5). Laboratory tests needed to exclude other causes of hypertransaminasaemia (viral hepatitis, autoimmune hepatitis, and toxic and selected metabolic liver diseases) were all negative. Abdominal ultrasonography revealed liver steatosis (normoechogenic liver with hyperechogenic areas of steatosis) and hyperechogenic, fatty pancreas. Other organs of the abdominal cavity did not show any abnormalities. The girl received a diagnosis of NAFPD and liver steatosis.

All children were recommended with weight loss, a healthy, balanced diet low in carbohydrates and saturated fats, and regular physical activity.

## DISCUSSION

The first investigation into lipid accumulation in the pancreas of obese people was conducted in 1933 by Ogilvie [6]. The subject of pancreatic steatosis was continued by Olsen, who described its relation to obesity based on 394 autopsies [7]. According to the current nomenclature proposed by Smits and van Geenen, NAFPD can be described as excessive lipid accumulation in the pancreas, related to metabolic syndrome and obesity [8]. The prevalence of NAFPD in Polish children remains unknown. The only study conducted on a paediatric population (in the USA) reported that 10% of patients had pancreatic steatosis [9]. The exact pathophysiology of this disease has not been established yet; however, its relation to obesity and NAFLD has been described [10–12]. A growing body of literature suggests that this relation is bi-directional – obesity may promote fat accumulation in the pancreas, which leads to B-cell dysfunction, insulin resistance, and impaired glucose metabolism [13]. In a study by Della Corte *et al.* the presence of NAFPD was related to more advanced liver disease. Nevertheless, more studies are needed to describe the exact relationship between obesity, pancreatic steatosis, insulin resistance, and NAFLD. NAFPD was also linked to significantly increased risk of MetS and its components, but the exact cause and effect relationship has not been established yet [14, 15]. All of our patients fulfilled the first criterion of MetS (central obesity), but only Patient 2 fulfilled an additional two criteria (fasting glucose level > 100 mg/dl and hypertension). Moreover, not all of the patients demonstrated lipid profile disturbances – hypertriglyceridaemia was observed only in Patient 1. In a study by Della Corte *et al.* the mean triglyceride concentration in children with NAFPD was within the normal range, which means that some children with this disease do not necessarily have lipid profile disturbances [12]. NAFPD can progress to nonalcoholic steatopancreatitis (NASP), which can be reversible after body weight reduction [8]. So far, there are no official criteria for diagnosing NAFPD. The diagnosis is based on pancreatic steatosis in the imaging studies (ultrasonography, computed tomography, magnetic resonance, or proton magnetic resonance spectroscopy) accompanying obesity or MetS [16, 17]. There are no recommendations on NAFPD treatment – it seems that the only way to stop or even reverse the progression of NAFPD is to reduce weight [18].

## CONCLUSIONS

To conclude, we present three cases of NAFPD in children to point out that it may be another disease coexisting with obesity and NAFLD. Early detection together with lifestyle changes leading to weight reduction can possibly prevent serious complications of this disease.

## DISCLOSURE

The authors declare no conflict of interest.

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