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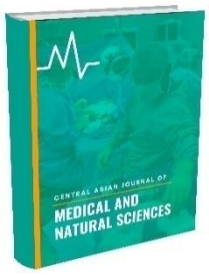

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Obesity and Pancreatitis

1. Nurova N.

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[1] Bukhara State Medical Institute, UZBEKISTAN

Annotation: Obesity is currently affecting people on a pandemic scale. Visceral abdominal obesity has the greatest impact on the course of acute pancreatitis. Although the definition of obesity varies, globally over 35% of adults are overweight (BMI > 25 kg/m2) and over 10% are obese (BMI > 30 kg/m2).

Keywords: Obesity, gastrointestinal tract.

Among the common causes of pancreatitis, one cannot ignore a number of factors that occur with a high frequency in pancreatitis and have a significant impact on the development and course of the disease. If they are still not recognized as etiological, then they can definitely be considered predisposing. Obesity, diabetes mellitus, hyperlipidemia and cholelithiasis are extremely often associated with each other, while the last two causes are included in a number of current classifications as etiological ones [3,9].

Interestingly, when defined according to the criteria adopted by national organizations, the proportion of people suffering from obesity increases. For example, while only about 4% of men in China or Japan and 34% in the US have a BMI over 30 [9]; Based on the waist circumference limits applicable to these countries, more than 35% of adult males in all three countries are obese [8]. Thus, despite different definitions of obesity, rates of abdominal obesity are equally high worldwide. This is important because both the prevalence of obesity [1] and the incidence of pancreatitis [3,5] have increased over the past 3 decades. Moreover, as we discuss below, obese patients have a higher risk of developing severe acute pancreatitis (SAP). In the next section, we will separately discuss the pathophysiology of how obesity can increase the incidence as well as worsen the severity of acute pancreatitis.

Gallstone disease: overweight and obese patients have a higher incidence of biliary tract disease [7] and pancreatitis [2]. Biliary tract disease causes acute pancreatitis with stones, sludge, or microlithiasis in the biliopancreatic ducts, either by causing bile reflux or by increasing pressure in the pancreatic duct [6]. Obesity may influence the formation of gallstones by several mechanisms. A Western high-fat diet may predispose to the formation of cholesterol-rich crystals or bile stones [1] by increasing the number of cholesterol crystals [2] or their growth [2]. This is supported by obese children with biliary pancreatitis, who are more likely to form stones than sludge [4]. Additional factors may include decreased circulating bile acids and gallbladder congestion due to increased intervals between meals in an attempt to lose weight or prevent obesity [9]. Obesity may also affect the diagnosis of gallstones, as

one study сообщается о снижении чувствительности магнитно-резонансной холангиопанкреатографии при обнаружении камней в желчном пузыре у пациентов с ожирением и избыточной массой тела [3].

Hypertriglyceridemia (HTG): HTG is associated with obesity and pancreatitis [4]. Obesity can reveal primary HTH from genetic causes [5] and is a risk factor for secondary HTH [1]. Weight loss, a treatment modality for HTH [6], is an additional risk factor for pancreatitis. Among the potential mechanisms of HTG-induced pancreatitis is the insolubility of lipid triglycerides in the aqueous medium of the blood, which leads to microthrombi in the vessels of the pancreas, causing ischemia and pancreatic infarction. Interestingly, hypertriglyceridemic pancreatitis tends to be severe [7] more often than other causes. This may be due to lipolysis of circulating triglycerides and the resulting unsaturated fatty acids (EFAs) causing FAP, as discussed below.

Type 2 diabetes and obesity are closely linked. Although diabetes may occur as a complication of acute pancreatitis due to loss of pancreatic mass or function, type 2 diabetes mellitus may increase the risk of acute pancreatitis due to HTG [7], gallstones [4] due to a diet high in fat and incretin - on the basis of treatment - possibly through hypertrophy of β-cells [7]. Although the exact mechanisms are unknown, islet cell hypertrophy, such as in nesidioblastosis, can lead to duct obstruction and pancreatitis [8]. Some studies and meta-analyses support an increased risk of acute pancreatitis [3,7] either, for example, with the use of glucagon-like peptide-1 receptor agonists [1] or dipeptidyl peptidase-4 inhibitors [8]; however, others refute this [2]. Data on the outcomes of acute pancreatitis in patients with diabetes are conflicting, with some reporting worse outcomes [3] and others reporting better outcomes [7] and need further research.

Therapeutic interventions for obesity. The morbidity associated with obesity has led to a range of interventions aimed at preventing or reversing it. Although strong evidence that medical treatment of obesity is associated with pancreatitis is lacking, the literature linking surgical or minimally invasive interventions to pancreatitis is fairly strong and is discussed below:

Gallstones and pancreatitis account for 5 and 10% of all complications in the first 3 years after surgery [5]. Although the risk of pancreatitis is higher than in the general population (0.02–0.04%), it is too low to justify cholecystectomy without associated gallstones or cholecystitis. The main mechanisms associated with an increased risk of gallstones and pancreatitis are postoperative rapid weight loss [6] and gallbladder stasis. Other mechanisms thought to cause pancreatitis include ampullary stenosis, sphincter of Oddi dysfunction, closed circuit obstruction [8], and nesidioblastosis [5,6]. Hyperamylasemia and lipasemia are observed in a significant proportion of patients with postoperative small bowel obstruction of the biliopancreatic pedicle [1] without a clinical diagnosis of pancreatitis.

In acute pancreatitis after the installation of gastric balloons for weight loss [8]. The largest series includes 301 patients followed up for 6 months, two of whom developed pancreatitis [7]. Pancreatitis can occur due to displacement or pressure on the pancreas. Interestingly, a new therapy for pancreatitis is a rapidly reversible cooling balloon placed in the stomach [6] that cools the pancreas transgastrically and can simultaneously slow down multiple mechanisms active in pancreatitis.

Conclusion Chronic pancreatitis, combined with obesity and type 2 diabetes mellitus, is accompanied by the presence of blood lipid profile disorders in the form of mixed hypertriglyceridemia. Clinically, patients with obesity were more likely to experience relapses of chronic pancreatitis and more pronounced abdominal pain, dyspeptic syndromes, indicating a relationship with the severity of CP. Patients with obesity were more likely to have comorbidities that have common pathogenetic mechanisms with DL (ischemic heart disease, chronic non-calculous cholecystitis, hypertension, type 2 diabetes mellitus).

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