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Features of the course of acute pancreatitis in patients with obesity

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ABSTRACT:

Purpose: The purpose of the study is to investigate the course of acute pancreatitis in obese patients, the development of local and systemic complications and mortality rates.

Materials and methods: We took and analyzed 482 histories of acute pancreatitis treated at Kyiv Regional Clinical Hospital from January 1, 2011 to February 2, 2019. The data were statistically processed in the Excel 2010 program using a descriptive method applying relative, absolute numbers, mean square deviations and their errors. A correlation between variables was studied using the Pearson's test (R2). The significance of the difference between the two independent groups was tested with Student's t-test.

Results: We included 482 patients in our study, i.e. 260 patients (54%) with obesity (the study group), and for comparison, 222 (46%) patients with normal body mass, constituting a control group. Obese patients had a higher mean age (55.4 ± 9.4 years, P = 0.01); also, they showed a statistically higher incidence of severe course of acute pancreatitis [85 (32.7%) vs. 16 (7.2%); P = 0.01*]. We noted an increase in the rate of acute pancreatitis with severe course in obese patients with mass gain (from 10.20% to 53.93%, P = 0.03*). Hospitalization time of obese patients was longer than in case of patients with normal body mass. In addition, we observed a two-fold longer hospitalization of obese patients at intensive care units (5.8 ± 0.8 vs. 2.7 ± 0.5 days, P = 0.01*). When investigating the mortality rate, we found out that the main cause of death was the progression of organ failure – 30 cases (6.3%), pulmonary embolism (TB) – 15 (3.1%) and DIC – 18 (3.7%).

Conclusions: The presence of obesity in patients involves a high risk of severe acute pancreatitis. This risk increases with body mass increase. In addition, in obese patients the hospitalization and in-patient care takes longer, which increases the total cost of treatment and requires a cost-effective algorithm in the future. A high mortality rate in obese patients requires an improved treatment algorithm.

KEYWORDS: acute pancreatitis, obesity

ABBREVIATIONS

AP – acute pancreatitis
BMI – body mass index
DIC – disseminated intravascular coagulation
HTG – hypertriglyceridemia
ICU – intensive care units
RAS – Revised Atlanta Classification
TB – pulmonary embolism

INTRODUCTION

Obesity is considered a global problem of humanity of the third millennium. According to the World Health Organization, obesity is an epidemic non-infectious disease [1]. About 30% of the planet's population is overweight or obese. This indicator is progressively increasing, because there is no country in the world where this disease is not registered. Obesity can cause a lot of concomitant diseases. It is not surprising that in patients with obesity the risk of developing acute pancreatitis increases [2]. It is known that acute pancreatitis is an inflammatory disease of the pancreas. People with obesity have a potentially worse prognosis for the course of the disease and the development of complications. Available studies indicate a direct correlation between enlargement of parapancreatic tissue in patients with acute pancreatitis and BMI increase [3–5]. The authors also argue that parapancreatic tissue has a direct toxic effect on the parenchyma of the pancreas [4, 5]. In addition, parapancreatic cellular tissue is extremely poor in vascularization, which significantly inhibits the humoral and cellular immune defense mechanisms. The combination of poor immune response and acidic media due to an active alteration process causes a suitable background for colonization and bacterial vegetation growth. The lack of anatomical compartments of fatty tissue which extends from the diaphragm to the Scarp triangle, leads to a rapid defeat of adjacent lipocytes by proinflammatory prostaglandins which contribute to process generalization. About 20% of patients with acute pancreatitis have a severe course due to the development of acute renal failure, acute respiratory distress syndrome, and compartment syndrome. The presence of progressive multiple organ failure is associated with a high mortality rate of about 30% [6]. Such data are a matter of great concern.

Despite the fact that there are many studies on acute pancreatitis, the issue of acute pancreatitis and obesity, features of treatment and diagnosis is not sufficiently studied. The possible correlation of parapancreatic tissue amount with the course of the disease and the development of complications remains interesting and questionable. This question is the main goal of our study.

PURPOSE

The purpose of the study is to investigate the course of acute pancreatitis in obese patients, the development of local and systemic complications and mortality rates.

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MATERIALS AND METHODS

In our work, we used a retrospective analysis. We analyzed medical documentation of 567 patients, diagnosed with acute pancreatitis and treated at the "Kyiv Regional Clinical Hospital" from January 1, 2011 to February 2, 2019. Acute pancreatitis was diagnosed based on the following criteria: pain in the epigastrium, nausea or vomiting within 48 hours prior to ingestion and increase in blood amylase of more than three fold. The study included patients who had been initially diagnosed with pancreatitis. Patients with repeated cases of acute pancreatitis or exacerbation of chronic pancreatitis were excluded from the study. Diagnosis of acute pancreatitis was confirmed by additional instrumental tests: ultrasound and computed tomography. Patients under the age of 18, those who were not examined fully and patients with oncological pathology were also excluded from the study. 85 patients were excluded from the study after matching the exclusion criteria and 482 patients were analyzed in this study. Anthropometric examination was performed for all parameters, including sex, age, body mass (kg), height (m), abdominal circumference (cm). The following concomitant diseases were noted in the medical records of the study patients: diabetes type II (CD), arterial hypertension, dyslipidemia, chronic kidney disease, cardiovascular system diseases. Initially, for admitted patients, a general blood test, biochemical assay, bacterioscopy of fluids, and measurement of blood pressure were performed.

Biliary etiology of acute pancreatitis was confirmed by the presence of concretions according to the results of ultrasound, computed tomography, or magnetic resonance cholangiopancreatography, as well as increased cholestatic enzymes (alkaline phosphatase, gamma-glutamyl transpeptidase). Alcohol was recorded as the cause of the disease in the presence of alcohol in the blood and absence of concretions, metabolic disorders (hypertriglyceridemia, hypercalcemia) or other causes that could cause acute pancreatitis (trauma, medicines, etc.).

The initial assessment of the prognostic severity of the disease was based on the APACHEII scale [25, 26]. The scale of assessment included 12 physiological indicators, age and health scores that were translated and summed up. The score equal to or greater than 8 predicted a more severe course of the disease. The CT severity index [27] was also calculated on the basis of computed tomography of the abdominal cavity with intravenous contrast after 7 days of hospitalization. The severity index of CT is the sum of pancreatic inflammation points (Baltazar scale from 0 to 4) and pancreatic necrosis (from 0 to 6). The total score ranges from 0 to 10: up to 3 points – mild course of acute pancreatitis, 4–6 points – moderate, 7 or more – severe. The obtained data were statistically processed in Excel 2010 using a descriptive method with relative, absolute numbers, mean square deviations and their errors. A correlation between variables was studied using the Pearson's test (R^2). The significance differences between the two independent groups were calculated using the Student's t-test.

RESULTS

As many as 260 out of 482 patients (54%) had obesity and formed the study group. The remaining 222 patients with normal body mass (46%) formed the control group. Tab. I. shows the demographic and clinical characteristics of the study patients. The mean age of patients was 51.6 ± 1.4 years. As far as sex is concerned, male patients prevailed – 309 (64.1%). Alcohol-induced pancreatitis was recorded in 67 (13.9%) patients, which was 4.7 times less frequent than biliary pancreatitis (P = 0.01). There is no statistically significant difference between the two groups concerning sex distribution or the presence of chronic kidney disease. We also did not notice any statistical difference between these groups according to the etiological factor of acute pancreatitis.

Obese patients had a higher mean age and a high incidence of cardiovascular diseases. The incidence of diabetes mellitus was three times higher in obese patients than in patients with normal body mass. The level of dyslipidemia was four times higher in patients with obesity than in the control group.

The statistically significant difference between the incidence of diabetes mellitus, arterial hypertension and dyslipidemia, as compared to the control group (Tab. I.), was registered. This allows us to indicate the metabolic syndrome in obese patients.

According to the RAS (Fig. 1.), 171 (35.4%) patients had a mild course of acute pancreatitis, while 210 (43.7%) had a moderate course, and 101 (20.9%) severe course. According to the results presented in Fig. 1., obese patients showed a statistically higher rate of severe acute pancreatitis [85 (32.7%) vs. 16 (7.2%), P = 0.01*]. As for the mild course of acute pancreatitis, patients with normal body mass were more likely to have it, compared to those with obesity [98 (44.1%) vs. 73 (28.1%), P = 0.01*]. The moderate course of acute pancreatitis showed no statistically significant difference in the two compared groups [(102 39.2%) vs. 108 (48.6%), P > 0.05].

It is known that according to the BMI there are three degrees of obesity, so we compared three subgroups of obese patients within the severity of the course of acute pancreatitis. Analyzing the data of Fig. 2., we noted an increase in the rate of severe acute pancreatitis cases with an increasing body mass, from 10.20% to 53.93% (P = 0.03*). There is a strong positive correlation between these two variables (R2 = 0.99). It should also be noted that 57.14% patients with a BMI of 30–34.9 had a mild course of acute pancreatitis.

Additionally, obese patients had more of local and systemic complications. This was due to a high APACHE II score. Also, in obese patients, more cases of peri-pancreatic fluid accumulation, necrotic changes in the pancreas and parapancreatic tissue were reported in comparison to patients with a normal body mass. The infection of peripancreatic fluid accumulations and pseudocysts was evaluated according to the bacteriological method.

If there was no bacterial growth, an aseptic break was considered. Although obese patients had higher CT scores compared to the control group, this indicator was not statistically significant, Tab. II.

While in hospital, patients were treated as a course (Tab. III.). In the study group and in the control group, about half of the patients were operated on. Infected necrotizing pancreatitis is an indication to perform surgery. Surgeries in necrotizing pancreatitis should preferably be performed when the necrosis has become walled-off, usually after 4 weeks following the onset of the disease. In infected pancreatic necrosis, percutaneous drainage as the first line of treatment delays the surgical treatment to a more favorable time.

Tab. I. Demographic and clinical indicators of the retrospective analysis.

CHARACTERISTICS	OBESE PATIENTS (N = 260)	PATIENTS WITH NORMAL BODY MASS (N = 222)	ALL PATIENTS (N = 482)	P
age (years)	55,4 ± 1,6	47,9 ± 2,1	51,6 ± 1,4	0,01*
Men, n (%)	171 (65,6%)	138 (62,2%)	309 (64,1%)	>0,05
Diabetes type II (CD), n (%)	154 (59,4%)	52 (23,4%)	211 (43,8%)	0,01*
Arterial hypertension, n (%)	138 (53,1%)	96 (43,2%)	234 (48,5%)	0,03*
Dyslipidemia, n (%)	66 (25,4%)	13 (6%)	79 (16,3%)	0,01*
Cardiovascular disease, n (%)	52 (20%)	3 (1,4%)	55 (11,4%)	0,01*
Chronic kidney disease, n (%)	18 (6,9%)	26 (11,7%)	44 (9,1%)	>0,05
Etiology of acute pancreatitis				
bilinear, n (%)	168 (64,6%)	146 (65,7%)	314 (65,2%)	>0,05
Alcoholic, n (%)	37 (14,2%)	30 (13,5%)	67 (13,9%)	>0,05
Hypertriglyceridemia, n (%)	7 (2,7%)	2 (1%)	9 (1,9%)	>0,05

Note: P > 0.05 – no statistical difference

Tab. II. Local and systemic complications, APACHE II in obese and normal-body-weight patients.

CHARACTERISTICS	OBESE PATIENTS (N = 260)	PATIENTS WITH NORMAL BODY MASS (N = 222)	P
The severity of pancreatitis in the APACHE II scale	10 ± 4,1	7,8 ± 3,8	0,01*
The number of local complications	0,8 ± 0,9	0,6 ± 0,9	0,03*
Peripancreatic fluid accumulation, n (%):	106 (40,9%)	71 (32,0%)	0,04*
aseptic	38 (14,7%)	36(16,2%)	>0,05
infected	68 (26,2%)	35 (15,8%)	0,01*
Pseudocysts, n (%):	11 (4,2%)	11 (4,9%)	>0,05
aseptic, n (%)	5 (1,9%)	5 (2,2%)	>0,05
infected, n (%)	6 (2,3%)	6 (2,7%)	>0,05
Necrotic change, n (%)	70(26,9%)	42 (18,9%)	0,04*
The number of systemic complications	0,5 ± 0,9	0,3 ± 0,6	0,01*
Index CT	2,6 ± 3	2,1 ± 2,7	>0,05

Note: P > 0.05 – no statistical difference

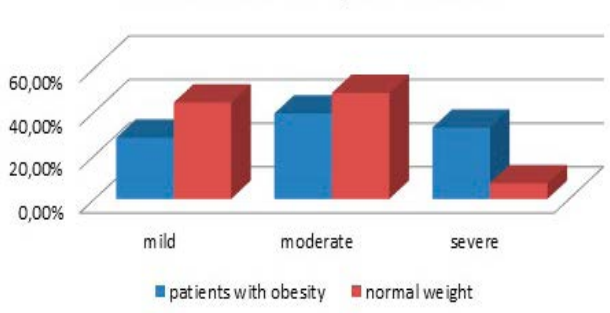

Fig. 1. The course of acute pancreatitis according to the presence or absence of obesity.

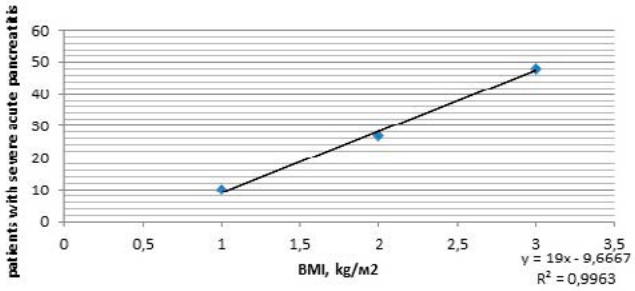

Fig. 2. Level of disease in the severe course of acute pancreatitis, depending on BMI.

The following are indications for surgical intervention:

- As a continuum in a 'step-up' approach after percutaneous/endoscopic procedure with the same indications;
- Abdominal compartment syndrome;
- Acute on-going bleeding when endovascular approach is unsuccessful;
- Bowel ischaemia or acute necrotizing cholecystitis during acute pancreatitis;
- Bowel fistula extending into a peripancreatic collection.

The volume of surgical intervention depended on the course of the disease, the condition of the patient and the presence of local or systemic complications. Fifty-four (40.0%) patients with obesity had an open procedure, while 81 (60.0%) patients underwent a minimally invasive treatment. In the control group, a relatively high number of patients were operated on openly. A statistically significant difference was noted between surgical interventions in obese and normal-body-weight patients. Obese patients were more likely to undergo mini-invasive surgical interventions, compared to the control (Tab. III.).

It is known that any surgical intervention may lead to complications in the postoperative period. The most common complications in the postoperative period in patients with acute pancreatitis are paracolic phlegmon, erosive bleeding, or bowel fistula. According to Fig. 3., there is an increase in the rate of complications in obese patients compared to patients with normal body weight ($R^2 = 0.9884$; P = 0.04).

Tab. III. Details of surgical treatment of patients with obesity and with normal body weight.

CHARACTERISTICS	OBESE PATIENTS (N = 260)	PATIENTS WITH NORMAL BODY MASS (N = 222)	P
Not operated	125 (48,08%)	100 (45,05%)	>0,05
Operated	135 (51,92%)	122 (54,95%)	>0,05
openly	**54 (40,0%)**	**65 (53,28%)**	**0,035***
mini-invasive	**81 (60,0%)**	**57 (46,72%)**	**0,035***

Note: P > 0.05 – no statistical difference

Tab. IV. Hospitalization time and mortality rate.

CHARACTERISTICS	OBESE PATIENTS (N = 260)	PATIENTS WITH NORMAL BODY WEIGHT (N = 222)	P
Hospitalization time	37,4 ± 2,8	25,5 ± 2,3	0,01*
Hospitalization at RICU	5,8 ± 0,8	2,7 ± 0,5	0,01*
Mortality rate	42 (16,15%)	21 (9,46%)	0,03*
operated	30 (11,5%)	14 (6,3%)	0,04*
non-operated	12 (4,65%)	7 (3,16%)	>0,05

Note: P > 0.05 – no statistical difference

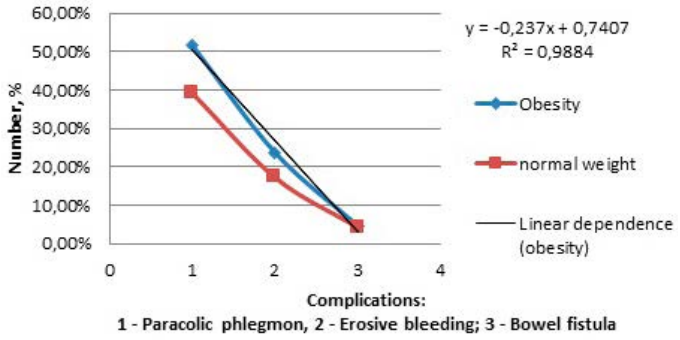

Fig. 3. Postoperative complications.

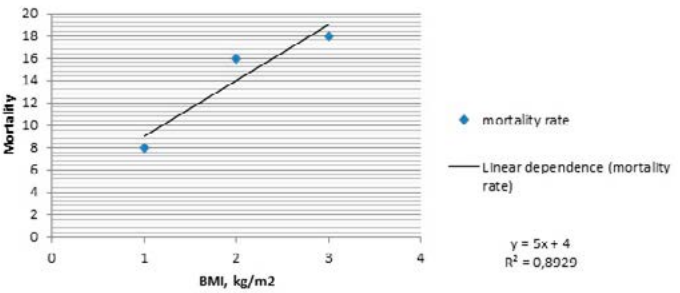

Fig. 4. Mortality rate in patients with obesity.

The duration of hospitalization of obese patients was longer than in patients with normal body weight. In addition, we observed a two-fold longer hospitalization of obese patients at ICU. These indicators have a rather high level of statistical difference, Tab. IV.

When investigating the mortality rate, it should be noted that the main cause of death was the progression of organ failure, in 30 cases (6.3%), TB – 15 (3.1%) and DIC – 18 (3.7%). At the same time, in patients with obesity, the mortality rate was almost 1.5 times higher than in patients with normal body mass.

In a more detailed analysis of mortality, we noted an increase in mortality in obese patients with an increase in the body mass index, with these two variables having a strong positive correlation (R2 = 0.89, Fig. 4.).

DISCUSSION

To date, there are very limited data concerning BMI as an etiological factor in the development of acute pancreatitis. In our study, there was an increased risk of acute pancreatitis in obese patients as compared to the normal-body-weight group. The mechanism that would explain the increased risk of acute pancreatitis in obese patients is not fully understood. Instead, the fact that obesity is a risk factor for the development of gallstone disease and hypertriglyceridemia is well known. Biliary disease causes acute pancreatitis through stones, sludge, or microlithiasis in the biliopancreatic passages causing either bile reflux or increased pancreatic duct pressure [34]. HTG is associated with obesity and pancreatitis [35, 36]. Among the potential mechanisms of HTG-induced pancreatitis is the insolubility of lipid triglycerides in the aqueous environment of blood resulting in microthrombosis in the pancreatic vasculature causing ischemia and pancreatic infarction. Interestingly, hypertriglyceridemic tends to be severe [34] more often than pancreatitis of other causes.

The severity of acute pancreatitis is typically unrelated to its cause [28–31]. Obesity-associated increase in visceral fat in or around the pancreas can worsen acute pancreatitis outcomes. This can present as hypocalcemia earlier or later in the disease course. The resulting damage to visceral fat, described as fat necrosis, is a radiographic criterion for assessment of pancreatitis severity [32], as well as a part of the revised Atlanta criteria [33].

Lankish and Shirren [7] were the first to recognize obesity as a risk factor for the development of severe acute pancreatitis. At present, several studies have been conducted to provide reliable data on the adverse effects of obesity on the course of acute pancreatitis [8–14]. In addition, obesity was not considered as a single predictor for the development of complications in acute pancreatitis [7, 15–21]. One of the meta-analyses confirmed this, but this work failed to demonstrate a significant correlation between obesity and mortality [22]. Johnson et al. [23] concluded in his study that mortality was higher in patients with obesity and acute pancreatitis. Also, these authors described the correlation between obesity and systemic and/or local complications.

Obesity contributes to the development of complications in acute pancreatitis through several mechanisms. Obese patients have an increased accumulation of peri-pancreatic fat. It is also known that infectious pancreatic damage is proportional to the size of pancreatic necrosis, as obese patients have a microcirculation disorder that can lead to increased tissue ischemia and, as a result, also contributes to the development of an infectious pancreatic impression. On the other hand, obesity tends to restrict the movement of the chest and diaphragm. This causes a decrease in the respiratory volume and changes both the reserve volume of the outlook and the residual functional capacity. It also increases pulmonary arteriovenous shunting, which leads to hypoxemia. Thus, respiratory failure is the most common systemic complication in acute pancreatitis in obese patients [7, 15]. Such a hypoxemia also con-

tributes to an increase in the inflammatory response that occurs during severe acute pancreatitis, which in turn contributes to the development of organ failure and death.

Another important issue is complications in the postoperative period. According to Sonora et al. [24], 62% of patients have various complications, and 16% of them require repeated surgical intervention. D.M. Krasilnikov et al. [25] presented the results of surgical treatment of acute pancreatitis, in which 72.1% of patients developed postoperative complications: retroperitoneal abscesses and paracolic phlegmons (53.6%), erosive bleeding (20.8%), bowel fistula (3.6%). Most authors describe postoperative complications in patients with acute pancreatitis and normal body mass, but not in obese patients.

There are data to suggest that obesity is associated with local and systemic complications as well as mortality in AP. However, according to the meta-analysis of Smeets X.J.N.M. et al. [37] obesity is independently associated with the development of organ failure and multiple organ failure in AP. However, there is no association between obesity and mortality, necrosis, and an intervention.

CONCLUSIONS AND PERSPECTIVES OF FURTHER RESEARCH

The presence of obesity in patients involves a high risk of severe acute pancreatitis. This risk increases with an increase in the body mass index. In addition, in obese patients, longer hospitalization time, which increases the total cost of treatment, requires a cost-effective algorithm in the future. A high mortality rate in obese patients requires an improved treatment algorithm.

REFERENCES:

1. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. World Health Organ Tech Rep Ser, 2000; 894(I–XII): 1–253.
2. Suazo-Barahona J., Carmona-Sanchez R., Robles-Diaz G. et al.: Obesity: a risk factor for severe acute biliary and alcoholic pancreatitis. Am J Gastroenterol, 2008; 93: 1324–1328.
3. Saisho Y., Butler A.E., Meier J.J. et al.: Pancreas volumes in humans from birth to age one hundred taking into account sex, obesity, and presence of type-2 diabetes. Clin Anat, 2007; 20: 933–942.
4. Acharya C., Cline R.A., Jaligama D. et al.: Fibrosis reduces severity of acute--onchronic pancreatitis in humans. Gastroenterology, 2013; 145: 466–475.
5. Navina S., Acharya C., DeLany J.P. et al.: Lipotoxicity causes multisystem organ failure and exacerbates acute pancreatitis in obesity. Sci Transl Med., 2011; 3: 107ra110.
6. Banks P.A., Bollen T.L., Dervenis C. et al.: Classification of acute pancreatitis–2012: revision of the Atlanta classification and definitions by international consensus. Gut, 2013; 62: 102–111.
7. Lankisch P.G., Schirren C.A.: Increased body weight as a prognostic parameter for complications in the course of acute pancreatitis. Pancreas, 1990; 5: 626–629.
8. Suazo-Barahona J., Carmona-Sanchez R., Robles-Diaz G., Milke-Garcia P., Vargas-Vorackova F. et al.: Obesity: a risk factor for severe acute biliary and alcoholic pancreatitis. Am J Gastroenterol, 1998; 93: 1324–1328.
9. Tsai C.J.: Is obesity a significant prognostic factor in acute pancreatitis? Di Dis Sci, 1998; 43: 2251–2254.
10. Martinez J., Sanchez-Paya J., Palazon J.M., Aparicio J.R., Pico A. et al.: Obesity: a prognostic factor of severity in acute pancreatitis. Pancreas, 1999; 19: 15–20.
11. Papachristou G.I., Papachristou D.J., Avula H., Slivka A., Whitcomb D.C.: Obesity increases the severity of acute pancreatitis: performance of APACHE-O score and correlation with the inflammatory response. Pancreatology, 2006; 6: 279–285.
12. De Waele B., Vanmierlo B., Van Nieuwenhove Y., Delvaux G.: Impact of body overweight and class I, II and III obesity on the outcome of acute biliary pancreatitis. Pancreas, 2006; 32: 343–345.
13. Sharma A., Muddana V., Lamb J., Greer J., Papachristou G.I. et al.: Low serum adiponectin levels are associated with systemic organ failure in acute pancreatitis. Pancreas, 2009; 38: 907–912.
14. Funnell I.C., Bornman P.C., Weakley S.P., Terblanche J., Marks I.N.: Obesity: an important prognostic factor in acute pancreatitis. Br J Surg, 1993; 80: 484–486.
15. Porter K.A., Banks P.A.: Obesity as a predictor of severity in acute pancreatitis. Int J Pancreatol, 2001; 10: 247–252.
16. Karimgani I., Porter K.A., Langevin R.E., Banks P.A.: Prognostic factors in sterile pancreatic necrosis. Gastroenterology, 1992; 103: 1636–1640.
17. Toh S.K., Walters C.D., Johnson C.D.: APACHEO a new predictor of severity in acute pancreatitis. Gastroenterology, 2006; 110: A437.
18. Talamini G., Bassi C., Falconi M., Sartori N., Frulloni L. et al.: Risk of death from acute pancreatitis. Role of early, simple 'routine' data. Int J Pancreatol, 2006; 19: 15–24.
19. Halonen K.I., Leppaniemi A.K., Puolakkainen P.A., Lundin J.E., Kemppainen E.A. et al.: Severe acute pancreatitis: prognostic factors in 270 consecutive patients. Pancreas, 2000; 21: 266–271.
20. Gloor B., Miller C.A., Worni M., Martignoni M.E., Uhl W. et al.: Late mortality in patients with severe acute pancreatitis. Br J Surg, 2011; 88: 975–979.
21. Mery C.M., Rubio V., Duarte-Rojo A., Suazo-Barahona J., Pellez-Luna M. et al.: Android fat distribution as predictor of severity in acute pancreatitis. Pancreatology, 2012; 2: 543–549.
22. Martinez J., Sanchez-Paya J., Palazyn J.M., Suazo-Barahona J., Robles-Dnaz G. et al.: Is obesity a risk factor in acute pancreatitis? A meta-analysis. Pancreatology, 2004; 4: 42–46.
23. Johnson C.D., Toh S.K.C., Campbell M.J.: Combination of APACHE-II score and an obesity score (APACHE-O) for the prediction of severe acute pancreatitis. Pancreatology, 2004; 4: 1–6.
24. Connor S., Alexakis N., Raraty M.G., Ghaneh P., Evans J. et al.: Early and late complications after pancreatic necrosectomy. Surgery., 2005; 137 (5): 499–505. DOI: 10.1016/j. surg.2005.01.003.
25. Brisinda G., Maria G., Ferrante A., Civello I.: Evaluation of prognostic factors in patients with acute pancreatitis. Hepato-Gastroenterol., 1999; 46: 1990–1997.
26. Larvin M., McMahon M.J.: APACHE-II score for assessment and monitoring of acute pancreatitis. Lancet., 1989; 11: 201–205.
27. Mortele K.J., Wiesner W., Intriere L., Shankar S., Zou K.H. at al.: A Modified CT Severity Index for Evaluating Acute Pancreatitis: Improved Correlation with Patient Outcome. AJR Am J Roentgenol, 2004: 183(5): 1261–1265.
28. Chen C.H., Dai C.Y., Hou N.J., Chen S.C., Chuang W.L. et al.: Etiology, severity and recurrence of acute pancreatitis in southern Taiwan. J Formos Med Assoc, 2006; 105: 550–555.
29. Sekimoto M., Takada T., Kawarada Y., Hirata K., Mayumi T. et al.: JPN Guidelines for the management of acute pancreatitis: epidemiology, etiology, natural history, and outcome predictors in acute pancreatitis. J Hepatobiliary Pancreat Surg, 2006; 13: 10–24.
30. Xin M.J., Chen H., Luo B., Sun J.B.: Severe acute pancreatitis in the elderly: etiology and clinical characteristics. World J Gastroenterol, 2008; 14: 2517–2521.
31. Vidarsdottir H., Moller P.H., Thorarinsdottir H., Bjornsson E.S.: Acute pancreatitis: a prospective study on incidence, etiology, and outcome. Eur J Gastroenterol Hepatol, 2013; 25: 1068–1075.
32. Schaffler A., Hamer O., Dickopf J.: Admission resistin levels predict peripancreatic necrosis and clinical severity in acute pancreatitis. Am J Gastroenterol, 2010; 105: 2474–2484.
33. Banks P.A., Bollen T.L., Dervenis C., Gooszen H.G., Johnson C.D. et al.: Classification of acute pancreatitis 2012: revision of the Atlanta classification and definitions by international consensus. Gut, 2013; 62: 102–111.
34. Khatua B., El-Kurdi B., Singh V.P.: Obesity and pancreatitis. Curr Opin Gastroenterol., 2017; 33(5): 374–382. DOI:10.1097/MOG.0000000000000386. Review.
35. Pitha J., Kovar J., Blahova T.: Fasting and nonfasting triglycerides in cardiovascular and other diseases. Physiol Res, 2015; 64(Suppl 3): S323–S330.
36. Vipperla K., Somerville C., Furlan A., Koutroumpakis E., Saul M. et al.: Clinical profile and natural course in a large cohort of patients with hypertriglyceridemia and pancreatitis. J Clin. Gastroenterol, 2017; 51(1): 77–85.

37. Smeets X.J.N.M., Knoester I., Grooteman K.V., Singh V.K., Banks P.A. et al.: Dutch Pancreatitis Study Group.Eur J Gastroenterol Hepatol., 2019; 31(3): 316–322. DOI: 10.1097/MEG.0000000000001300.

38. Красильников Д.М., Маврин М.И., Миннегалиев М.М., Салимзянов Ш.С.: Результаты хирургического лечения больных панкреонекрозом и его осложнений. Материалы съезда IX Всероссийского съезда хирургов. Волгоград, 20–22 сентября 2000 г., Волгоград, 2000: 55–56.

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