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Impact of asprosin, interleukin-6, and adiponectin on exocrine pancreatic insufficiency in patients with type 2 diabetes mellitus and chronic pancreatitis

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Exocrine pancreatic insufficiency (EPI) is a clinical condition often associated with chronic pancreatitis (CP) and type 2 diabetes mellitus (T2DM) leads to malabsorption and nutritional deficiencies, severely impacting patients' quality of life. It has been identified that asprosin, a glucogenic protein, is a key player in carbohydrate metabolism and chronic inflammation. Elevated levels of asprosin, along with changes in interleukin-6 (IL-6) and adiponectin, may contribute to the pathophysiology of EPI in patients with T2DM and CP.

Objective — to investigate the levels of asprosin, IL-6, and adiponectin and their correlation with exocrine pancreatic function in patients with T2DM and CP.

Materials and methods. The study included 100 patients treated at Kharkiv Regional Clinical Hospital from 2020 to 2022. Patients were divided into two groups: Group 1 (n = 70) with comorbid T2DM and CP, and Group 2 (n = 30) with T2DM alone. The control group included 20 healthy individuals. Blood levels of asprosin, IL-6, and adiponectin were measured using immunoassays. Fecal elastase-1 (FE-1) levels were used to assess exocrine pancreatic function. Pearson's correlation coefficient was calculated to determine the relationships between these markers.

Results. Asprosin levels were significantly higher in Group 1 compared to Group 2 and the control group, with the highest levels observed in patients with comorbid T2DM and CP (10.06 ± 3.56 ng/ml). IL-6 levels were also elevated in Group 1 (64.44 ± 4.35 pg/ml), indicating heightened inflammation. Adiponectin levels were lower in Group 1 (2.80 ± 0.38 ng/ml), correlating with worse metabolic profiles. FE-1 levels were markedly reduced in Group 1 (142.2 ± 6.3 mg/g), confirming severe EPI. Significant correlations were found between asprosin levels and IL-6 (positive correlation, $r = 0.49$, $p < 0.01$), as well as between asprosin levels and FE-1 (negative correlation, $r = -0.52$, $p < 0.01$). In the group of patients with isolated T2DM, significant moderate negative correlations were found between asprosin levels and adiponectin ($r = -0.47$, $p < 0.05$) and FE-1 ($r = -0.44$, $p < 0.05$). The study also demonstrated a strong negative correlation between adiponectin and IL-6 ($r = -0.61$, $p < 0.01$) in patients with comorbid T2DM and CP.

Conclusions. Elevated asprosin levels in patients with T2DM and CP are associated with increased inflammation and reduced exocrine pancreatic function. The significant correlations between asprosin, IL-6, and FE-1 suggest that asprosin could serve as a clinically valuable biomarker for assessing metabolic and inflammatory status in these patients. The findings highlight the complex interaction between metabolic and inflammatory pathways in the development of EPI.

Keywords:
type 2 diabetes mellitus, chronic pancreatitis, exocrine pancreatic insufficiency, asprosin.

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Exocrine pancreatic insufficiency (EPI) is a clinical condition marked by the pancreas's insufficient production of digestive enzymes, resulting in malabsorption and nutritional deficiencies [10]. EPI is often associated with chronic pancreatitis (CP) but can also be exacerbated by metabolic disorders such as type 2 diabetes mellitus (T2DM) [4]. The presence of EPI significantly compromises patients' quality of life, leading to weight loss,

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diarrhea, and vitamin deficiencies due to impaired nutrient digestion and absorption.

Emerging research has focused on asprosin, a fasting-induced glucogenic protein, which plays a critical role in metabolic regulation [6]. Asprosin is secreted by adipose tissue during fasting and stimulates hepatic glucose release, thereby contributing to insulin resistance and hyperglycemia — key features of T2DM [13]. Elevated levels of asprosin have been observed in individuals with metabolic syndrome, underscoring its significant role in glucose metabolism and inflammation [3].

In addition to asprosin, other biomarkers such as interleukin-6 (IL-6) and adiponectin have been implicated in the pathophysiology of EPI. IL-6, a pro-inflammatory cytokine, is elevated in chronic inflammatory states including CP and T2DM [9]. High levels of IL-6 are associated with systemic inflammation and have been linked to the progression of pancreatic damage and insufficiency [12]. Conversely, adiponectin, an anti-inflammatory adipokine, is typically reduced in metabolic disorders. It has insulin-sensitizing properties and its lower levels are associated with increased inflammation and metabolic dysfunction [2].

The interplay between T2DM and CP presents a unique clinical challenge, as both conditions independently and synergistically impair pancreatic function [7]. Understanding the roles of asprosin, IL-6, and adiponectin in this context is crucial for elucidating the mechanisms underlying EPI. Elevated asprosin and IL-6 levels, coupled with reduced adiponectin levels, may exacerbate pancreatic inflammation and fibrosis, contributing to the severity of EPI [5].

Objective — to investigate the levels of asprosin, IL-6, and adiponectin and their relationships with other metabolic and inflammatory markers, with a specific focus on their impact on exocrine pancreatic function in patients with T2DM and CP.

By examining these correlations, we seek to provide insights into the potential mechanisms through which these biomarkers influence pancreatic health and to identify new therapeutic targets for managing EPI in this patient population.

Materials and methods

This study examined 100 patients treated at the Kharkiv Regional Clinical Hospital from 2020 to 2022. The patients were divided into two groups. Group 1 (n = 70) included patients with comorbid T2DM and chronic pancreatitis (CP), consisting of 64 % men and 36 % women, with an average age of 62.3 ± 6.7 years. The mean BMI in this group was 29.4 ± 4.69 kg/m^2. Group 2 (n = 30) comprised individuals with type 2 diabetes mellitus (T2DM), with a gender distribution of 63 % men and 37 % women, and an average age of 64.4 ± 6.6 years. The mean body mass index (BMI) in this group was 29.6 ± 3.92 kg/m^2. The mean duration of T2DM in the first group was 12.6 ± 5.4 years, whereas in the second group, it was 11.2 ± 6.8 years. The control group (CG) included 20 relatively healthy individuals of corresponding age.

Informed consent was obtained from all participants under the Code of Ethics of the World Medical Association (Declaration of Helsinki). The study received ethical approval from the Ethics and Bioethics Committee of Kharkiv National Medical University (decision dated October 7, 2020).

The diagnoses of chronic pancreatitis and T2DM were established based on the regulations set by the Ministry of Health of Ukraine.

Inclusion criteria were: the presence of voluntary consent to participate in the study, the presence of chronic pancreatitis, and type 2 diabetes mellitus.

Exclusion criteria included: patients aged under 18 years, with HbA1c level > 9 %, patients with type 1 diabetes mellitus, patients with acute pancreatitis, patients with chronic kidney disease, patients with stage III hypertension, patients with stage IIB-III heart failure, acute inflammatory processes, oncological diseases, history of alcohol and substance abuse, and presence of HIV/AIDS.

Blood asprosin, adiponectin and IL-6 levels were measured using an indirect non-competitive heterogeneous immunoassay on the «Labline-90» analyzer (Austria) with a commercial test system from «Elabscience» (China). To evaluate exocrine pancreatic insufficiency, fecal elastase-1 (FE-1) levels were determined.

Statistical analyses were performed using Prism 9.0 software (GraphPad Software, USA). Pearson's linear correlation coefficient (r) was used to determine the relationships between the studied characteristics. Correlation was considered inverse for r values between 0 and -1.0 and direct for values between 0 and 1.0. An r coefficient from 0 to 0.3 (or from 0 to -0.3) indicated a weak relationship; from 0.4 to 0.7 (or from -0.4 to -0.7), a moderate relationship; and from 0.7 to 1.0 (or from -0.7 to -1.0), a strong relationship. The results were presented as r coefficients with the corresponding significance level (p). Differences between independent groups were assessed using analysis of variance (ANOVA).

Results and discussion

The following markers were studied: adiponectin, IL-6, FE-1. The results show significant differences in the levels of these parameters, as well as asprosin levels, in the patient groups compared to the control group and between group 1 and 2. The levels all markers of the studied groups are presented in Table 1.

Asprosin, adiponectin, IL-6, and FE-1 levels in the studied groups were analyzed. Asprosin levels were significantly higher in Group 1 (T2DM and CP) and Group 2 (T2DM) compared to the control group. The highest asprosin levels were observed in Group 1 (10.06 ± 3.56 ng/ml), which were significantly higher compared to Group 2 (7.34 ± 2.08 ng/ml, $p < 0.001$) and the control group (2.81 ± 1.34 ng/ml, $p < 0.001$). Adiponectin levels were lower in Group 1 (2.80 ± 0.38 ng/ml) compared to Group 2 (3.29 ± 0.31 ng/ml, $p < 0.05$) and the control group (4.92 ± 0.37 ng/ml, $p < 0.001$). The differences were statistically significant. IL-6 levels were also highest in Group 1 (64.44 ± 4.35 pg/ml), significantly higher than in Group 2 (28.67 ± 3.05 pg/ml, $p < 0.001$) and the control group (3.10 ± 0.34 pg/ml, $p < 0.001$). FE-1 levels were lower in Group 1 (142.2 ± 6.3 μg/g) compared to Group 2 (192.7 ± 4.3 μg/g, $p < 0.001$) and the control group (210.2 ± 1.2 μg/g, $p < 0.001$). The differences were statistically significant.

The obtained results of asprosin, adiponectin, IL-6, and FE-1 levels in patients with T2DM and comorbid T2DM and CP indicate correlational relationships (mostly of moderate strength) between these indicators. The results are presented in Tables 2 and 3.

In the group of patients with comorbid T2DM and CP, significant correlation relationships were observed between asprosin and other studied indicators. Asprosin levels demonstrated a moderate negative correlation with adiponectin levels ($r = –0.42$; $p < 0.05$) and a moderate negative correlation with FE-1 levels ($r = –0.52$; $p < 0.01$). There was a moderate positive correlation between asprosin and IL-6 levels ($r = 0.49$; $p < 0.01$). Additionally, adiponectin levels exhibited a strong negative correlation with IL-6 levels ($r = –0.61$; $p < 0.01$) and a moderate positive correlation with FE-1 levels ($r = 0.41$; $p < 0.01$). IL-6 levels had a weak negative correlation with FE-1 levels ($r = –0.34$; $p < 0.05$).

In the group of patients with T2DM (n = 30), significant correlation relationships were observed between asprosin and other studied indicators. Asprosin levels demonstrated a moderate negative correlation with adiponectin levels ($r = –0.47$; $p < 0.05$) and a moderate negative correlation with FE-1 levels ($r = –0.44$; $p < 0.05$). There was a moderate positive correlation between asprosin and IL-6

Table 1. Asprosin, adiponectin, IL-6 and FE-1 levels in the studied groups

Indicator	Group 1 (T2DM and CP) (n = 70)	Group 2 (T2DM) (n = 30)	Control group (n = 20)
Asprosin, ng/ml	10.06 ± 3.56	7.34 ± 2.08*	2.81 ± 1.34*#
Adiponectin, ng/ml	2.80 ± 0.38	3.29 ± 0.31**	4.92 ± 0.37*#
IL-6, pg/ml	64.44 ± 4.35	28.67 ± 3.05*	3.10 ± 0.34*#
FE-1, μg/g	142.2 ± 6.3	192.7 ± 4.3*	210.2 ± 1.2*#

Note. The difference from the control group is statistically significant: * $p < 0.001$; ** $p < 0.05$.
The difference from the control group is statistically significant: # $p < 0.001$.

Table 2. Correlation between asprosin and other studied indicators in patients with comorbid T2DM and CP (n = 70)

Indicator	Asprosin, ng/ml	Adiponectin, ng/ml	IL-6, pg/ml	FE-1, μg/g
Asprosin, ng/ml	—	–0.42*	+0.49**	–0.52**
Adiponectin, ng/ml	–0.42*	—	–0.61**	0.41**
IL-6, pg/ml	+0.49**	–0.61**	—	–0.34*
FE-1, μg/g	–0.52**	0.41**	–0.34*	—

Note. * $p < 0,05$, ** $p < 0,01$.

Table 3. Correlation between asprosin and other studied indicators in patients with T2DM (n = 30)

Indicator	Asprosin, ng/ml	Adiponectin, ng/ml	IL-6, pg/ml	FE-1, μg/g
Asprosin, ng/ml	—	–0.47*	+0.51**	–0.44*
Adiponectin, ng/ml	–0.47*	—	–0.53**	+0.31*
IL-6, pg/ml	+0.51**	–0.53**	—	–0.32*
FE-1, μg/g	–0.44**	+0.31**	–0.32*	—

Note. * $p < 0,05$, ** $p < 0,01$.

levels ($r = 0.51$; $p < 0.01$). Additionally, adiponectin levels exhibited a strong negative correlation with IL-6 levels ($r = –0.53$; $p < 0.01$) and a moderate positive correlation with FE-1 levels ($r = 0.31$; $p < 0.05$). IL-6 levels had a weak negative correlation with FE-1 levels ($r = –0.32$; $p < 0.05$).

These findings indicate that asprosin is significantly correlated with several key metabolic and inflammatory markers in patients with comorbid T2DM and CP. Elevated asprosin levels are associated with decreased adiponectin and increased IL-6, which are both implicated in the pathogenesis of exocrine pancreatic insufficiency. These findings suggest a potential role for asprosin in the development of pancreatic dysfunction in T2DM patients with chronic pancreatitis [3, 8, 13].

Adipose tissue has been established as an endocrine organ that plays a regulatory role in metabolism and energy balance. Research has demonstrated that adipocyte-derived factors can modulate insulin action and inflammation [1]. Excess adipose accumulation is linked to insulin resistance, a key driver of T2DM. Consequently, obesity is associated with a range of metabolic disorders, including T2DM and metabolic syndrome.

The present study indicates that asprosin, an adipokine secreted by white adipose tissue, may contribute to the development of exocrine pancreatic insufficiency in patients with T2DM and chronic pancreatitis. Elevated asprosin has been associated with insulin resistance and impairment of pancreatic beta-cell function [13]. Furthermore, asprosin appears to have inflammatory effects, as evidenced by its positive correlation with interleukin-6 (IL-6) levels [11].

The observed negative correlation between asprosin and the FE-1 level, a marker of exocrine pancreatic function, suggests that asprosin may adversely impact pancreatic exocrine secretion. This is consistent with previous research demonstrating the deleterious effects of asprosin on pancreatic beta-cell function and the development of diabetic complications [3]. Notably, the inverse relationship between asprosin and adiponectin, an adipokine with anti-inflammatory and insulin-sensitizing properties, further supports the notion that asprosin contributes to the impairment of pancreatic function in this patient population [6].

In summary, the present study provides evidence that elevated asprosin, in conjunction with altered levels of adiponectin and interleukin-6, may be associated with the development of exocrine pancreatic insufficiency in patients with T2DM and chronic pancreatitis. These findings highlight the potential role of asprosin as a pathogenic factor and a potential therapeutic target in the management of pancreatic dysfunction in this patient population.

Conclusions

Thus, the analysis of the correlation relationships between asprosin, IL-6, adiponectin, and FE-1 levels in patients with T2DM and CP revealed:

1. The highest asprosin levels were noted in the group with comorbid T2DM and CP, significantly higher than in the group with isolated T2DM and the control group.

2. Significant moderate positive correlations were established between asprosin levels and IL-6 in both the group of patients with isolated T2DM and the group with combined T2DM and CP.

3. In the group of patients with isolated T2DM, significant moderate negative correlations were found between asprosin levels and adiponectin and FE-1.

4. In the group of patients with combined T2DM and CP, significant moderate negative correlations were observed between asprosin levels and adiponectin and FE-1.

5. The findings suggest that elevated asprosin levels are associated with increased inflammatory markers (IL-6) and decreased anti-inflammatory markers (adiponectin), as well as impaired exocrine pancreatic function (FE-1), indicating a complex interplay between metabolic and inflammatory pathways in these patients.

This study identifies substantial changes in the levels of the asprosin, the inflammatory cytokine interleukin-6, and adiponectin in patients with type 2 diabetes mellitus and chronic pancreatitis. The observed associations between these biomarkers and exocrine pancreatic function underscore their potential involvement in the pathogenesis of these comorbid conditions, thereby warranting further research into their clinical applications as diagnostic and therapeutic targets.

Future studies should focus on elucidating the mechanisms by which asprosin, IL-6, and adiponectin influence pancreatic function and exploring potential therapeutic interventions that target these biomarkers to mitigate their adverse effects on both endocrine and exocrine pancreatic functions. Such interventions could significantly improve the management of patients with T2DM and comorbid CP, addressing both metabolic control and pancreatic health.

Conflicts of interest: none.

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Вплив аспросину, інтерлейкіну-6 і адипонектину на екзокринну недостатність підшлункової залози в пацієнтів із цукровим діабетом 2 типу та хронічним панкреатитом

Екзокринна недостатність підшлункової залози (ЕНПЗ) — клінічний стан, який часто асоціюється з хронічним панкреатитом (ХП) і цукровим діабетом (ЦД) 2 типу, призводить до мальабсорбції та дефіциту харчування, що значною мірою впливає на якість життя пацієнтів. Установлено, що аспросин, глюкогенний білок, є ключовим учасником глікемічного обміну та хронічного запалення. Підвищений рівень аспросину разом зі змінами інтерлейкіну-6 (ІЛ-6) і адипонектину можуть спричинити патофізіологію ЕНПЗ у пацієнтів із ЦД 2 типу та ХП.

Мета — дослідити рівень аспросину, ІЛ-6 і адипонектину та їхню кореляцію з екзокринною функцією підшлункової залози в пацієнтів із ЦД 2 типу та ХП.

Матеріали та методи. У дослідження було залучено 100 пацієнтів, які перебували на лікуванні в Харківській обласній клінічній лікарні в період із 2020 до 2022 р. Пацієнтів розподілили на дві групи: група 1—70 пацієнтів із поєднанням ЦД 2 типу і ХП, група 2—30 пацієнтів із ЦД 2 типу без ХП. Контрольну групу створено із 20 здорових осіб. Рівень аспросину, ІЛ-6 і адипонектину в крові вимірювали за допомогою імуноаналізів. Вміст фекальної еластази-1 (ФЕ-1) використовували для оцінки екзокринної функції підшлункової залози. Коефіцієнт кореляції Пірсона розраховували для визначення взаємозв'язків між зазначеними маркерами.

Результати. Рівень аспросину був значно вищим у групі 1 порівняно з групою 2 та контрольною групою, з найвищими показниками в пацієнтів із поєднанням ЦД 2 типу та ХП ((10,06 ± 3,56) нг/мл). Концентрація ІЛ-6 також була високою в групі 1 ((64,44 ± 4,35) пг/мл), що свідчило про значне запалення. Рівень адипонектину був нижчим у групі 1 ((2,80 ± 0,38) нг/мл), що корелювало з гіршими метаболічними профілями. Вміст ФЕ-1 був значно зниженим у групі 1 ((142,2 ± 6,3) мкг/г), що підтверджувало тяжку ЕНПЗ. Виявлено сильні кореляції між рівнями аспросину й ІЛ-6 ($r = 0,49$; $p < 0,01$), а також між рівнями аспросину та ФЕ-1 ($r = -0,52$; $p < 0,01$). У групі пацієнтів з ізольованим ЦД 2 типу зафіксовано помірні обернено пропорційні зв'язки між рівнями аспросину й адипонектину ($r = -0,47$; $p < 0,05$) та ФЕ-1 ($r = -0,44$; $p < 0,05$), у групі пацієнтів з поєднанням ЦД 2 типу і ХП — сильний обернено пропорційний зв'язок між адипонектином та ІЛ-6 ($r = -0,61$; $p < 0,01$).

Висновки. Підвищений рівнень аспросину в пацієнтів із ЦД 2 типу та ХП асоціюється з підвищеним запаленням та зниженою екзокринною функцією підшлункової залози. Значні кореляції між аспросином, ІЛ-6 та ФЕ-1 свідчать, що аспросин може бути клінічно значущим біомаркером для оцінки метаболічного та запального статусу в цих пацієнтів. Отримані дані свідчать про складну взаємодію між метаболічними та запальними шляхами в розвитку ЕНПЗ.

Ключові слова: цукровий діабет 2 типу, хронічний панкреатит, екзокринна недостатність підшлункової залози, аспросин.

ДЛЯ ЦИТУВАННЯ

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