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ORIGINAL ARTICLE

CONTENTS

Multivariate analysis and mathematical modeling of the informativeness of patients cases data in chronic pancreatitis associated with concomitant pathology

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ABSTRACT

Aim: To investigate and analyze homeostatic disorders in patients with a combination of Chronic Pancreatitis(CP) and Arterial Hypertension (AH) and to develop correcting ways of the detected changes.

Materials and Methods: General clinical, laboratory-instrumental examination of 121 patients, who were undergoing inpatient treatment with a diagnosis of Chronic Pancreatitis in combination with Arterial Hypertension of the II stage during 2021–2022.

Results: In the majority of cases of patients signs the increasing in IL-1,6 and Cortisol levels were found. A decrease in Ca to the lower limit of the norm was observed (2.18 ± 0.26 mmol/l to the data of control group patients (2.32 ± 0.12 mmol/l, p= 0.01), the levels of trace elements Zn and Se were determined within the reference values. The Atherogenic Index was increased 1.8 times and was significantly different from the control group date. During the FE-1 study, a decrease in the level of this indicator was revealed by 151.71±13.91 mg/g of feces, both to the values of reference values and a significant difference to the data of the control group (241.28±29.17 mg/g of feces, p<0 .05)

Conclusions: Based on the multivariate linear regression analysis of the obtained data, formulas have been developed that can be used to predict the dynamics of the dependent variable (FE-1, IL-1, Selenium level, Glutathione Peroxidase, blood pressure) according to changes in the studied influencing factors.

KEY WORDS: Chronic pancreatitis, Hypertension, associated pathology of the Digestive tract (Non-alcoholic Fatty Liver disease, Gastroesophageal Reflux disease), inflammatory response, patient

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INTRODUCTION

In the structure of diseases of the gastrointestinal tract (GIT), CP is from 5.1 to 9% per 100,000 population, and in general clinical practice - from 0.2 to 0.6% [1]. In modern studies, CP is considered not only as a combination of local lesions due to the influence of pathochemical inflammatory mechanisms, but as a systemic gastroenterological disease, in the development of which hereditary-constitutional, immunological, psychosocial and psychosomatic mechanisms are involved. At the same time, a cohort of patients in whom a clear relationship between the genesis, the onset of the disease, the phase of the disease and the features of the premorbid condition can be traced is quite common [2, 3]. The situation is exacerbated by the frequent combination of CP not only with diseases of the digestive organs, but also with CVD, among which hypertension is considered the unchanging leader, since the combination of CP and Arterial Hypertension (AH) is pathogenetically determined, in particular, as a result of generalized systemic damage to the vascular bed, which is the basis for the formation of ischemic changes, activation local inflammatory component of diseases and triggering of systemic inflammatory response, metabolic disorders [4, 5].

The etiological factors of Chronic Pancreatitis (CP) include various types of pathology of organs that are anatomically and physiologically related to Pancreas. According to the literature, in 35-56% of cases, pathology of the biliary tract is recognized as a factor that leads to the development and exacerbation of CP. Pancreatitis develops much faster in patients with gallstone disease, which confirms the role of gallstone disease in the development of CP, therefore, patients suffering from gallstone disease are a group at risk of developing CP [6, 7].

Peculiarities of the etiology and pathogenesis of CP combined with hypertension should be taken into account in clinical practice when examining and treating this cohort of patients. At the same time, management

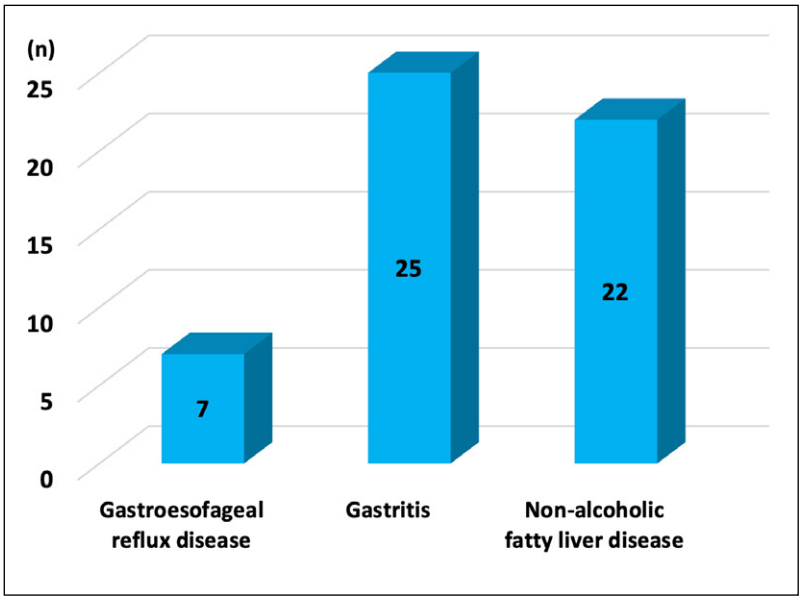

Fig. 1. Associated pathology of the digestive tract in patients with Chronic Pancreatits and Arterial Hypertension.

of patients with comorbidities still remains a methodologically complex task. The variety of reasons for the formation of CP combined with hypertension motivates researchers to study in-depth the causes and features of the development of this combined pathology.

AIM

The aim is to investigate and analyze homeostatic disorders in patients with a combination of chronic pancreatitis and hypertension and to develop ways of correcting the detected changes.

MATERIALS AND METHODS

A general clinical, laboratory-instrumental examination of 121 patients (average age of patients 48.6 ± 8.9 years) who were undergoing inpatient treatment in the therapeutic department of the Khust Central Regional Hospital with a diagnosis of chronic pancreatitis (CP) in combination with hypertension of the II stage during 2021–2022. Control group include 25 healthy person identified the studied parameters. The diagnostic algorithm for CP and associated pathology was carried out in accordance with the clinical protocols of the Ministry of Health of Ukraine and guidelines.

RESULTS

To assess the features of the clinical course of the combined pathology, we performed a comprehensive analysis of the results of objective and subjective examinations of the studied selection of patients. The diagnosis of hypertension was also confirmed in all patients with chronic pancreatitis (121/100%). At the level of the esophago-gastro-duodenal segment of the digestive system, pathological changes were established, which were finally confirmed thanks to endoscopic esophagogastroduodenoscopy. Gastritis (n=25; 24.5%) was most often registered, and Gastroesophageal Reflux disease was registered in 7 (6.9%) of the examined. Non-alcoholic Fatty Liver disease was established in 22 (21.6%) patients examined using steatometry (Fig. 1.).

From the group of patients with fixed signs of fatty inclusions in the Liver parenchyma, grade S1 was determined in 11 (50 %), S2 – 9 (41 %), and S3 – in 2 patients (9 %) (Fig. 2)

The complex of laboratory examinations included the study of the links of patients' homeostasis and the identification of imbalances. The first stage was a study of biochemical blood analysis (Table 1).

The following changes were noted in the results of the biochemical study, in particular, the level of total protein had a direction towards the lower limit of reference values and was significantly different from the indicators of the control group (68.51 ± 3.66 to 75.01 ± 2.77 g/l, $p<0.05$) due to the low value of albumin (39.56 ± 2.91 g/l against the data of the control group 45.12 ± 2.04 $p< 0.05$). A slight increase in the levels of transaminases ALT (0.97 ± 0.49 mmol/l) and AST (0.97 ± 0.49 mmol/l) was also observed and had significant differences from the data of the control group (0.65 ± 0.12 mmol /l, $p< 0.002$ and 0.42 ± 0.11 mmol/l, $p< 0.05$, respectively). Increasing levels of the last two indicators show the influence of pathological changes at the level of the pancreas on the functioning of the liver, which confirms the presence of associated pathology. Biochemical blood analysis indicated the presence of cytolytic syndrome (increased levels of transaminases), a decrease in the concentration of Total Protein, due to a low level of Albumin.

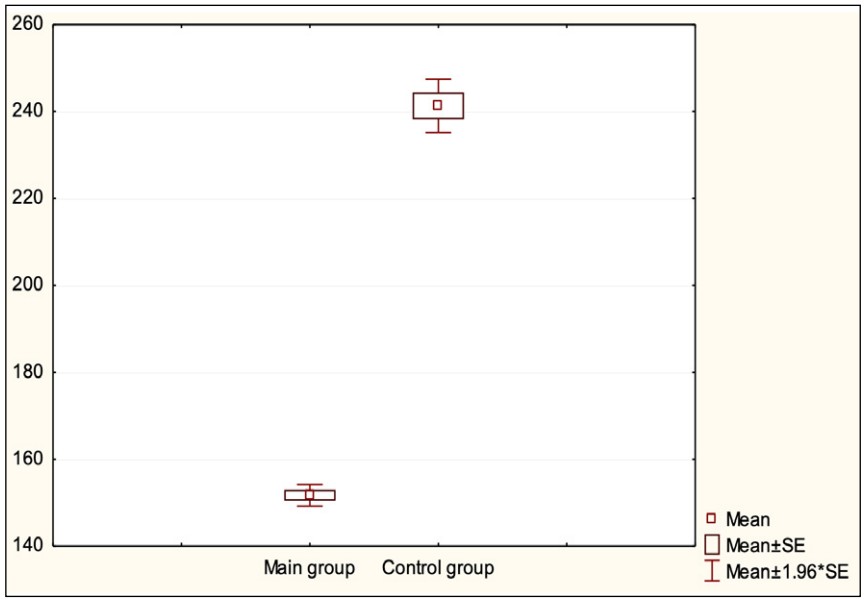

Fig. 2. Percentage value of steatometry results in patients.

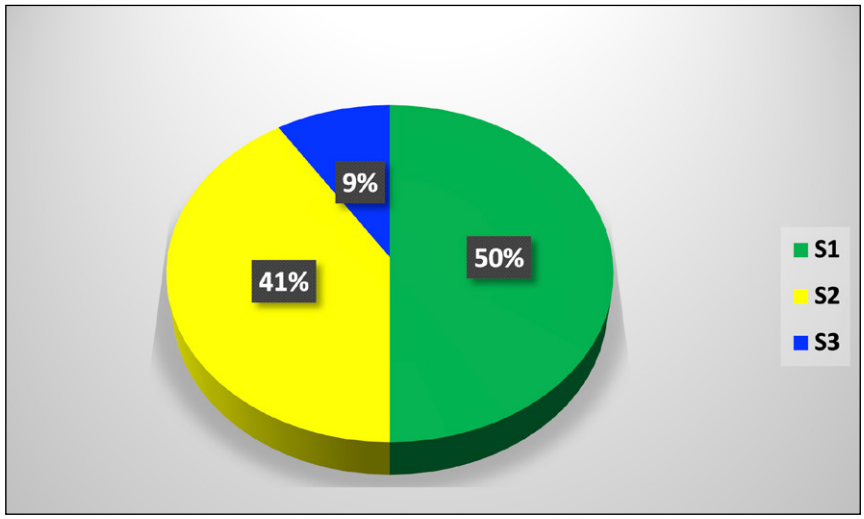

Fig. 3. Comparing levels diagram of Fecal Elastase-1 between the main group and the control group.

The state of inflammatory markers in CP patients with comorbid pathology (Table 2) is bringing to your attention.

Analyzing the obtained data, all the studied indicators are significantly different from the corresponding parameters of patients from the control observation group ($p<0.05$). Specifying individual indicators, it was found that the level of leukocytes (8.95 ± 2.95 G/l is significantly different from the data of the control group, 6.12 ± 1.77 G/l), but was near the upper limit of the physiological norm. The levels of ESR and fibrinogen are also within the reference values and may be a sign of insufficient immunological reactivity of the studied contingent. There was a slight increase in the level of pro-inflammatory IL-6 (11.99 ± 1.64 pg/ml and 6.94 ± 1.79 pg/ml in comparison with the control group), and a reliable variation of IL-4 within the reference values with the data of the control group. One can think about the presence of an inflammatory reaction of the body in conditions of persistence of the inflammatory process.

The level of cortisol in the blood of patients showed an increase in values (972.61 ± 220.08nmol/l) by 1.5 times, which indicates the initiation of activation of anti-inflammatory mediators and inhibition of the synthesis of pro-inflammatory ones. Values of Glutathione Peroxidase(GPO), as a representative of the enzymatic link of Antioxidant protection, show an approach to the lower limit of normal, which indicates insufficient activity of GPO in the conditions of oxidative stress (OS) launch in patients with associated pathology. So, the results indicate that in most patients, during the initial examination, signs of persistence of the inflammatory reaction due to the increase in the levels of IL-1,6 and Cortisol were found. Other indicators of biochemical blood analysis in the examined patients were less indicative. The levels of the Leukocyte pool and ESR varied within the reference values, but with statistical probability with the indicators of the control group.

Violation of mineral metabolism in diseases of the digestive tract is important (Table 3).

Table 1. Biochemical indicators of blood serum in the studied contingent

Parameters	Main group (n = 121)	Control group (n = 25)	Statistical significance of differences (p)
Total protein (65-85, g/l)	68,51 ± 3,66	75,01 ± 2,77	< 0,05
Albumin (38-51, g/l)	39,56 ± 2,91	45,12 ± 2,04	< 0,05
Total bilirubin (3,4-20,05, μmol/l)	14,36 ± 11,89	10,31 ± 3,22	0,09
Alanine aminotransferase (0,1-0,45, mmol/l)	0,97 ± 0,49	0,65 ± 0,12	0,002
Aspartate aminotransferase 0,1-0,68, mmol/l)	0,52 ± 0,25	0,42 ± 0,11	0,05

Note: p- statistical significance between the indicators of the main and control groups.

Table 2. Analysis of inflammatory markers in CP patients with comorbid pathology

Indexes	Main group (n = 121)	Control group (n = 25)	Statistical significance of differences (p)
Leukocytes (4.0-9.0, G/l)	8,95 ± 2,95	6,12 ± 1,77	< 0,05
ESR (2-15, mm/h)	9,93 ± 5,72	5,42 ± 2,93	< 0,05
Fibrinogen (1.8-3.5, g/l)	2,94 ± 0,54	2,44 ± 0,36	< 0,05
Cortisol (in the morning, 190-690, nmol/l)	972,61 ± 220,08	495,62 ± 159,54	< 0,05
IL-1 (0-11, pg/ml)	23,77 ± 8,61	2,57 ± 1,02	< 0,05
IL-4 (0-4, pg/ml)	1,94 ± 0,21	2,39 ± 0,62	< 0,05
IL-6 (0-10, pg/ml)	11,99 ± 1,64	6,94 ± 1,79	< 0,05
Glutathione peroxidase (12.5-200, ng/ml)	45,55 ± 10,98	65,28 ± 9,47	< 0,05

Note: p- statistical significance between the indicators of the main and control groups.

According to the data in Table III, the concentrations of Na, K, Cl in both studied groups were not significantly different from the results of the control group, which indicates the absence of changes in the metabolism of these elements in the combined pathology of CP. However, our results indicate a tendency to decrease Ca content to the lower limit of normal (2.18 ± 0.26 mmol/l in comparison with the data of control group patients (2.32 ± 0.12 mmol/l, p=0.01) The balance of Ca plays an important role in the synthesis and excretion of Pancreas enzymes, as well as in the regulation of vascular tone [9].

According to our data, the levels of trace elements Zn and Se are determined within the reference values, but with statistically significant differences from the data of patients of the control group: Se (63.74 ± 18.03μg/l vs. 87.58 ± 14.03μg/l, p<0 ,01); Zn (743.33 ± 206.01 μg/l vs. 901.23 ± 168.57 μg/l (p=0.05)). In our opinion, the lower content of Se and Zn in patients with associated pathology is caused by functional disorders of the liver and, as a result, disorders of mineral homeostasis and disruption of microelement assimilation processes due to enzymatic dysfunction of the liver.

The components of lipid metabolism in the pathology under study in our patients (Table 4) was considered.

Evaluating the studied indicators, it can be concluded that in patients with associated pathology, a statistically significant difference was established in all parameters of the Lipidogram compared to the data of the indicators of patients in the control group (p<0.05). In particular, slight Hypercholesterolemia (5.13 ± 1.07 mmol/l against the data of the control group 4.21 ± 0.45 mmol/l, p < 0.05) and Hypertriglyceridemia (1.79 ± 0.45 mmol/l against 1.29 ± 0.22 mmol/l, p < 0.05), a reduced level of HDL Cholesterol in the blood serum of patients (0.85 ± 0.17 mmol/l, against 1.45 ± 0.32 mmol/l, p < 0, 05),

Table 3. Indicators of mineral metabolism in patients

Parameters	Main group (n = 121)	Control group (n = 25)	Statistical significance of differences (p)
K (3,6-5,5, mmol/l)	4,39 ± 0,37	4,48 ± 0,36	0,27
Na (135-155, mmol/l)	143,19 ± 3,20	142,71 ± 3,22	0,49
Ca (2,1-2,6, mmol/l)	2,18 ± 0,26	2,32 ± 0,12	0,01
Cl (95-108, mmol/l)	101,88 ± 3,14	103,17 ± 2,48	0,06
Se (23-190, µg/l)	63,74 ± 18,03	87,58 ± 14,03	< 0,05
Zn (543-1130, µg/l)	743,33 ± 206,01	901,23 ±168,57	< 0,05

Note: p- statistical significance between the indicators of the main and control groups.

Table 4. Lipidogram indicators of the studied contingent

Parameters	Main group (n = 121)	Control group (n = 25)	Statistical significance of differences (p)
Total cholesterol (< 5.0 mmol/l)	5,13 ± 1,07	4,21 ± 0,45	< 0,05
Triglycerides (< 1.7, mmol/l)	1,79 ± 0,45	1,29 ± 0,22	< 0,05
HDL (> 1.0, mmol/l)	0,85 ± 0,17	1,45 ± 0,32	< 0,05
LDL (< 3.0, mmol/l)	3,46 ± 1,04	2,37 ± 0,55	< 0,05
VLDL (0.26-1.04, mmol/l)	0,82 ± 0,20	0,65 ± 0,09	< 0,05
IA (≤3)	5,43 ± 2,24	2,38 ± 1,01	< 0,05

Note: p- statistical significance between the indicators of the main and control groups.

increased levels of LDL Cholesterol (3.46 ± 1.04 vs. 2.37 ± 0.55 mmol/l, $p < 0.05$). Levels of VLDL Cholesterol (0.82 ± 0.20 mmol/l and 0.8 ± 0.22 mmol/l vs. 0.65 ± 0.09 mmol/l, $p < 0.05$) varied within the reference limits. The Atherogenic Index was increased in 1.8 times and significantly differed from the values of the control group (5.43 ± 2.24 vs. 2.38 ± 1.01, $p < 0.05$).

Identified Dyslipidemic disorders have pathological effects on the state of functioning of the Pancreas and, along with this, the vascular component of the body, which can be considered as a risk factor for the development of pathology of the Cardiovascular system and Digestive tract.

Monitoring of pressure values showed the following changes (Table 5).

Average Blood pressure(BP) levels in patients corresponded to the parameters of Arterial Hypertension of the 1st-2nd degree. Statistically significant differences were observed between the groups of patients with associated pathology in the levels of SBP (153.47 ± 9.42 mmHg) and DBP (94.75 ± 8.61 mmHg)) with indicators in the control group ($p<0.05$).

To determine the exocrine capacity of the parenchymal organ, we measured the concentration of elastase-1 in feces (Table 6).

The level of fecal elastase-1 plays an important role in the diagnosis of Pancreatic Exocrine insufficiency (PEI), as a specific indicator of the exocrine function of the pancreas. During the FE-1 study, a decrease in the level of this indicator was revealed by 151.71±13.91 mg/g, both to the values of reference values and a significant difference to the data of the control group (241.28±29.17 mg/g of feces, $p<0.05$), which can be regarded as a mild degree of PEI [10].

To confirm and optimally interpret the indicators of the studied contingent with a diagnosis of CP in association with concomitant pathology, a multivariate linear regression analysis of the obtained data was conducted. According to the calculated regression formula, it is possible to predict the dynamics of the dependent variable

Table 5. The value of blood pressure monitoring in patients

Parameters	Main group (n = 121)	Control group (n = 25)	Statistical significance of differences (p)
Systolic blood pressure (mm Hg)	153,47 ± 9,42	125,73 ± 6,38	< 0,05
Diastolic blood pressure (mm Hg)	94,75 ± 8,61	72,41 ± 5,92	< 0,05

Note: p- statistical significance between the indicators of the main and control groups.

Table 6. Value of fecal elastase level in patients

Indexes	Main group (n = 121)	Control group (n = 25)	Statistical significance of differences (p)
Fecal elastase-1 (> 200, μg/g stool)	151,71 ± 13,91	241,28 ± 29,17	< 0,05

Note: p- statistical significance between the indicators of the main and control groups.

according to the changes in the studied influencing factors [11]. The created mathematical models of the obtained data are presented in formulas. Credible levels of indicators were used to create mathematical models.

1. Fecal elastase-1 = 167.29 – 0.22 * Total Bilirubin + 2.30 * Total Cholesterol – 5.91 * Fibrinogen. (formula 1)
EXPLANATION:
- with an increase in the level of Total Bilirubin by 1 μmol/l, the value of FE-1 will decrease by 0.22 μg/g of feces;
- with an increase in the level of Total Cholesterol by 1 mmol/l, the concentration of FE-1 will increase by 2.30 μg/g of feces;
- with an increase in the level of Fibrinogen by 1 g/l, the FE-1 value will decrease by 5.91 μg/g of feces.

According to the data of formula 1, the highest level of influence of bilirubin values is observed, which indicates the biliary component of the development of pancreatitis. There is also a significant influence of fibrinogen values on the reduction of FE-1 level and increase of PEI. Since Fibrinogen belongs to acute phase Proteins and immunoinflammatory modulators, it can be assumed that the leading role of the inflammatory factor in the development of diseases of the studied contingent. The lowest influence in the development of CP is the level of Total Cholesterol.

2. IL-1 = 22.55 – 9.27 * HDL + 0.20 * Glutathione Peroxidase. (formula 2)
EXPLANATION:
- an increase in the concentration of HDL by 1 mmol/l will cause a decrease in the level of IL-1 by 9.27 pg/ml;
- with an increase in the level of Glutathione Peroxidase by 1 ng/ml, the level of IL-1 will increase by 0.20 pg/ml.

According to formula 2, there is a high value of the level of the HDL component on the reduction of the pro-inflammatory cytokine IL-1 and a slight Antioxidant protection due to glutathione peroxidase.

3. Se = 62, 94 + 1.08 * Atherogenicity Index – 0.69 * Na + 12.32 * Ca + 0.03 * Zn + 0.86 * Glutathione Peroxidase + 0.009 * cortisol. (formula 3)
EXPLANATION:
- if the value of Atherogenicity Index increases by 1 Unit, the concentration of Se will increase by 1.08 μg/l;
- when the level of Na increases by 1 mmol/l, the concentration of Se will decrease by 0.69 μg/l;
- when the concentration of Ca increases by 1 mmol/l, the concentration of Se will increase by 12.32 μg/l;
- if the concentration of Zn increases by 1 μg/l, the level of Se will increase by 0.03 μg/l;
- when the Glutathione Peroxidase concentration increases by 1 ng/ml, the Se concentration will increase by 0.86 μg/l;
- with an increase in the level of Cortisol by 1 nmol/l, the level of Se will increase by 0.009 μg/l.

According to the obtained data of formula 3, the level of Selenium will increase due to the concentration of Ca, the value of Atherogenicity Index, and with the minimum values of the concentration of GPO, Zn, and Cortisol. The level of Na helps to reduce the content of Selenium

4. Glutathione peroxidase = -10.66 +10.64 * HDL Cholesterol + 0.48 * Se + 0.28 * ESR – 0.82 * IL-6 + 0.16 * systolic blood pressure. (formula 4)
EXPLANATION:
- an increase in the concentration of HDL Cholesterol by 1 mmol/l will cause an increase in the level of GPO by 10.64 ng/ml;

- when the concentration of Se increases by 1 μg/l, the concentration of GPO will increase by 0.48 ng/ml;
- when the ESR concentration increases by 1 mm/h, the GPO concentration will increase by 0.28 ng/ml;
- when the level of IL-6 increases by 1 pg/ml, the concentration of GPO will decrease by 0.82 ng/ml;
- with an increase in SBP value by 1 mm. Hg the GPO concentration will increase by 0.16 ng/ml.

Based on the components of formula 4, it can be noted that with an increase in the concentration of HDL Cholesterol, an increase in the level of GPO is observed and, as a result, an increase in the body's antioxidant capacity in CP with Arterial Hypertension. Concentrations of Se, ESR, IL-6, and SBP are characterized by minimal contributions to the maintenance of the body's Antioxidant protection.

5. Systolic blood pressure = 225.93 – 0.57 * Total Protein - 0.12 * Se – 1.09 * Leukocytes – 1.36 * IL-6. (formula 5)

- when the Total Protein concentration increases by 1 g/l, the SBP level will decrease by 0.57 mm Hg;
- with an increase in the concentration of Se by 1 μg/l, the SBP level will decrease by 0.12 mm Hg;
- with an increase in the concentration of leukocytes by 1 G/l, the SBP level will decrease by 1.09 mm Hg;
- with an increase in the concentration of IL-6 by 1 pg/ml, the SBP level will decrease by 1.36 mm Hg.

According to formula 5, the category of systolic pressure increases due to the increase in the concentration of IL-6, Leukocytes and with minor effects of the levels of the concentration of Total Protein, the concentration of Se. That is, inflammatory factors of influence are decisive.

DISCUSSION

Usually, the patient has a comorbid pathology, which is based on common risk factors, pathogenetic mechanisms and requires a systematic approach to solving patient management tactics, which is significant in the choice of treatment and monitoring

CP is often determined by its combination with other diseases in the conditions of polymorbidity of the modern patient, which is an indication for expanding therapy [5,12]. Since CP is known to be associated with an increased risk of cardiovascular disease (CVD), D. De la Iglesia et al. (2019) interpret the risk of cardiovascular events in CP patients with pancreatic exocrine insufficiency [9]. Dysfunctional motor disorders of the upper parts of the alimentary canal, processes of stomach accommodation form the pathophysiological basis for associated pathologies in CP and non-erosive GERD. Numerous data on the most frequent risk factors that prevent the development and progression of chronic pancreatitis and the high probability of combining chronic pancreatitis with diseases of the gastroduodenal and biliary zones and as a result - a violation of the regular course of the disease and changes in clinical presentations have been presented in scientific research [13]. According to our data, hypercholesterolemia and hypertriglyceridemia, significant hyperproduction of pro-inflammatory cytokines (IL-1.6), increased cortisol levels, imbalance of macro- and microelements were identified as common components. Chronic oxidative stress, hyperproduction of proinflammatory cytokines, and hyperlipidemia are essential factors in the pathogenesis of both CVD and CP. Dyslipidemia acts as a common mechanism for the formation of fatty infiltration of the liver, which contributes to the formation of steatopancreatitis, non-alcoholic fatty liver disease and the development of vascular atherosclerosis in the comorbid course of CP with hypertension. At the same time, the nature and depth of lipid metabolism disorders in patients with a combined course of CP and hypertension have not yet been definitively investigated.

Associated diseases should be considered as an interaction of the patient, risk factors, triggers and basic protective compensatory mechanisms that underlie the development of a disease state with certain characteristic clinical manifestations. It is very important for the doctor to recognize them early in a single disease process, to prevent the generalization of homeostasis and stimulation disorders, or to model compensatory and adaptive mechanisms [14].

CONCLUSIONS

1. Biochemical blood analysis indicated the presence of cytolytic syndrome (increased levels of transaminases), a decrease in the concentration of Total Protein, due to a low level of Albumin.
2. Therefore, the results indicate that in most patients during the initial examination signs of persistence of the inflammatory reaction due to the increase in the levels of IL-1,6 and Cortisol were found. Other indicators of biochemical blood analysis in the examined patients were less indicative. The levels of the Leukocyte pool and ESR varied within the reference values, but with statistical probability with the indicators of the control group.
3. Our results indicate a tendency to decrease the content of Ca to the lower limit of normal (2.18 ± 0.26 mmol/l) in comparison with the data of patients of the control group (2.32 ± 0.12 mmol/l, p=0.01). According to our data, the levels of trace elements Zn and Se are determined within the reference values, but with statistically significant differences from the data of patients of

the control group. In our opinion, the lower content of Se and Zn in patients with associated pathology is caused by functional disorders of the liver and as a result of mineral disorders homeostasis and disruption of processes of assimilation of microelements due to Pancreatic enzyme dysfunction.

4. Evaluating the studied indicators, slight Hypercholesterolemia and Hypertriglyceridemia, a reduced level of HDL Cholesterol, in the blood serum of patients, and an increase in the level of LDL Cholesterol were found. Levels of HDL cholesterol (0.82±0.20mmol/l and 0.8±0.22mmol/l vs. 0.65±0.09mmol/l, $p<0.05$) varied within the reference limits. The Atherogenicity Index was increased 1.8 times and significantly differed from the values of the control group.
5. During the FE-1 study, a decrease in the level of this indicator was revealed by 151.71±13.91 mg/g of feces, both to the values of the reference values and a significant difference to the data of the control group (241.28±29.17 mg/g of feces, $p<0, 05$), which can be considered as a mild degree of PEI.
6. We have the next result, according to mathematical formulas,:

- formulas 1 - the highest level of influence of bilirubin values on the FE-1 level is observed, which indicates the biliary component of pancreatitis development. There is also a significant influence of Fibrinogen values on the reduction of the FE-1 level and the increase of PEI;
- formula 2 - a high value of the HDL Cholesterol level is observed for the reduction of the pro-inflammatory cytokine IL-1 and a slight Antioxidant protection due to Glutathione Peroxidase.
- formula 3 - the level of Selenium will increase due to the concentration of Ca, the value of AI, and with the minimum values of the concentration of GPO, Zn, Cortisol. The level of Na helps to reduce the content of Selenium
- formula 4 - it can be noted that with an increase in the concentration of HDL, an increase in the level of GPO is observed and, as a result, an increase in the body's Antioxidant capacity in CP with Arterial Hypertension. Concentrations of Se, ESR, IL-6, and SBP are characterized by minimal contributions to the maintenance of the organism's Antioxidant protection.
- formula 5 - the category of systolic blood pressure increases due to the increase in the concentration of IL-6, Leukocytes and with minor effects of the concentration levels of the Total Protein, Se. That is, inflammatory factors of influence are decisive.

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CONFLICT OF INTEREST

The Authors declare no conflict of interest

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