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Algorithm for Early Detection of Pancreatic Steatosis in Primary Healthcare

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Abstract

Aim: to develop and evaluate an algorithm for the early detection of pancreatic steatosis in a primary healthcare setting, and to substantiate measures for primary and secondary prevention.

Materials and Methods: the study was conducted in three stages. In the first stage, a questionnaire survey was performed among 3,135 individuals aged 34–75 years attached to family polyclinics of the Mirzo-Ulugbek and Shaykhantakhur districts. In the second stage, 1,530 patients with moderate and high risk underwent comprehensive clinical, laboratory, and instrumental examination, including anthropometry, assessment of carbohydrate and lipid metabolism and liver enzymes, and ultrasound examination of the liver and pancreas. In the third stage, risk factors were analyzed and the algorithm was validated in 300 patients from the risk group. Statistical analysis included descriptive statistics, the Pearson χ^2 test, and logistic regression analysis.

Results: moderate and high risk of pancreatic steatosis was identified in 1,530 of 3,135 examined individuals (48.8%). According to ultrasound findings, pancreatic steatosis was diagnosed in 1,102 of 1,530 risk-group patients (72.0%). Independent predictors of the disease were hepatic steatosis (OR=4.57), abdominal obesity (OR=4.10), obesity (OR=3.46), hypertriglyceridemia (OR=2.94), age ≥50 years (OR=2.41), and impaired carbohydrate metabolism (OR=2.27), $p<0.001$. Upon validation, the algorithm demonstrated a sensitivity of 92.5%, specificity of 72.1%, and overall diagnostic accuracy of 86.7%.

Application of the algorithm reduced conditional costs for comprehensive examination by 49.5%.

Conclusion: the developed algorithm for early detection of pancreatic steatosis is an accessible and practically applicable tool for primary healthcare. Its implementation allows timely identification of risk-group patients, optimizes referral pathways, improves the efficiency of preventive examinations, and enables rational use of healthcare system resources.

Keywords: Pancreatic steatosis; fatty pancreatic infiltration; primary healthcare; screening; abdominal obesity; hepatic steatosis; prevention.

Introduction

In recent decades, there has been a rise in the prevalence of obesity, type 2 diabetes mellitus, metabolic syndrome, and other conditions associated with impaired energy metabolism. Among metabolically associated diseases, increasing attention is being paid to pancreatic steatosis — fatty infiltration of the pancreas, which is regarded not only as a morphological change of the organ but also as a possible marker of systemic metabolic dysfunction.

Pancreatic steatosis develops against a background of lipid metabolism disorders, insulin resistance, obesity, and chronic low-grade inflammation. Accumulation of adipose tissue in the pancreas may be accompanied by impairment of the organ's endocrine and exocrine function and an increased risk of type 2 diabetes mellitus, chronic pancreatitis, and other metabolically associated complications.

The relevance of the problem is due to the prolonged asymptomatic course of the disease, the absence of unified approaches to screening, and insufficient awareness among primary care physicians. In most cases, fatty infiltration of the pancreas is detected incidentally during abdominal ultrasound examination. In this regard, the development of a simple algorithm for early detection and risk stratification is of considerable practical importance for family physicians, internists, gastroenterologists, and endocrinologists.

Aim of the Study

To improve early detection of pancreatic steatosis at the primary healthcare level through the development and implementation of a screening algorithm for risk-group patients, and to substantiate a set of primary and secondary prevention measures.

Methods

The study was conducted in three consecutive stages. In the first stage, an analysis was performed of the results of preventive medical examinations of the population attached to family polyclinics of the Mirzo-Ulugbek and Shaykhantakhur districts. A total of 3,135 individuals aged 34–75 years participated in the survey.

For initial patient selection, a Pancreatic Steatosis Risk Assessment Scale (PSRAS) was developed, comprising age, body mass index, waist circumference, physical activity, dietary characteristics, smoking status, presence of arterial hypertension, carbohydrate metabolism disorders, and ultrasound data on pancreatic steatosis. A total score of 0–7 points corresponded to low risk, 8–14 points to moderate risk, and more than 15 points to high risk.

In the second stage, 1,530 patients with moderate and high risk were enrolled in a comprehensive examination program. All patients underwent anthropometry and assessment of fasting glucose, HbA1c, lipid profile, ALT, AST, and GGT. Abdominal ultrasound examination was performed after fasting, with evaluation of the liver and pancreas.

The principal ultrasound criterion for pancreatic steatosis was diffuse hyperechogenicity of the pancreatic parenchyma relative to the echogenicity of the left renal cortex and/or liver, in the absence of signs of acute or chronic inflammatory processes. According to the severity of changes, grade I (mild), grade II (moderate), and grade III (severe) were distinguished.

In the third stage, clinical-anamnestic, anthropometric, metabolic, and behavioral risk factors were analyzed. The data obtained were used to construct the algorithm for early detection of pancreatic steatosis and to develop primary and secondary prevention measures. Additionally, the algorithm was validated in 300 risk-group patients.

Statistical analysis was performed using Statistica 13.0 software. Quantitative variables are presented as mean and standard deviation (M±SD). The Pearson χ^2 test was used to compare categorical variables. Logistic regression analysis was used to identify independent predictors of the disease. Differences were considered statistically significant at $p<0.05$.

Results

According to the initial survey, low risk of pancreatic steatosis was established in 1,605 examined individuals (51.2%),

moderate risk in 890 individuals (28.4%), and high risk in 640 individuals (20.4%). Thus, 1,530 patients (48.8%) were classified into the moderate- and high-risk groups and referred for comprehensive examination.

Table 1. Distribution of examined individuals by degree of risk of pancreatic steatosis (n=3,135)

Degree of risk	Number, n	Proportion, %
Low risk	1,605	51.2
Moderate risk	890	28.4
High risk	640	20.4
Total	**3,135**	**100.0**

As the degree of risk increased, a significant increase in age, body mass index, and waist circumference was observed. In the high-risk group, mean age was 58.9±9.1 years, BMI was 34.2±4.5 kg/m², and waist circumference was 104.5±11.2 cm, significantly exceeding the corresponding values in the low-risk group ($p<0.001$).

Table 2. Age and anthropometric characteristics of examined individuals

Parameter	Low risk	Moderate risk	High risk	p
Age, years	43.8±7.2	51.6±8.4	58.9±9.1	<0.001
BMI, kg/m²	24.8±2.6	29.7±3.1	34.2±4.5	<0.001
Waist circumference, cm	83.6±8.4	94.8±9.7	104.5±11.2	<0.001

Analysis of risk factors showed that abdominal obesity, low physical activity, poor dietary habits, smoking, and arterial hypertension were significantly more common in the high-risk group. The frequency of abdominal obesity increased from 17.8% in the low-risk group to 83.8% in the high-risk group ($p<0.001$).

Table 3. Distribution of risk factors according to degree of risk

Parameter	Low risk, n (%)	Moderate risk, n (%)	High risk, n (%)	p
Abdominal obesity	285 (17.8)	548 (61.6)	536 (83.8)	<0.001
Low physical activity	342 (21.3)	536 (60.2)	492 (76.9)	<0.001
Poor diet	418 (26.0)	612 (68.8)	514 (80.3)	<0.001
Smoking	224 (14.0)	196 (22.0)	186 (29.1)	<0.001

Arterial hypertension	198 (12.3)	356 (40.0)	382 (59.7)	<0.001

Patients in the high-risk group demonstrated significantly poorer carbohydrate and lipid metabolism parameters compared with the moderate-risk group. Mean fasting glucose was 6.7±1.2 mmol/L versus 5.8±0.7 mmol/L, HbA1c was 6.8±0.9% versus 5.9±0.5%, and triglycerides were 2.8±0.9 mmol/L versus 1.9±0.6 mmol/L, respectively (p<0.001).

Table 4. Metabolic parameters in moderate- and high-risk patients (n=1,530)

Parameter	Moderate risk	High risk	p
Fasting glucose, mmol/L	5.8±0.7	6.7±1.2	<0.001
HbA1c, %	5.9±0.5	6.8±0.9	<0.001
Total cholesterol, mmol/L	5.5±0.8	6.3±1.1	<0.001
Triglycerides, mmol/L	1.9±0.6	2.8±0.9	<0.001
LDL, mmol/L	3.4±0.7	4.1±0.8	<0.001
HDL, mmol/L	1.18±0.24	0.96±0.18	<0.001
GGT, U/L	34.6±14.8	56.2±23.4	<0.001

According to ultrasound findings, pancreatic steatosis was identified in 1,102 of 1,530 risk-group patients, corresponding to 72.0%. In the moderate-risk group, the disease was detected in 536 patients (60.2%), whereas in the high-risk group it was detected in 566 patients (88.4%; p<0.001). Grade II and III fatty infiltration of the pancreas were more frequently recorded in high-risk patients.

Table 5. Ultrasound characteristics of the pancreas

Parameter	Moderate risk, n (%)	High risk, n (%)	p
Increased parenchymal echogenicity	536 (60.2)	566 (88.4)	<0.001
Structural heterogeneity	268 (30.1)	348 (54.4)	<0.001
Pancreatic steatosis, grade I	286 (32.1)	134 (20.9)	<0.001
Pancreatic steatosis, grade II	198 (22.2)	270 (42.2)	<0.001
Pancreatic steatosis, grade III	52 (5.8)	162 (25.3)	<0.001
Total pancreatic steatosis	**536 (60.2)**	**566 (88.4)**	**<0.001**

Univariate analysis revealed a statistically significant association of pancreatic steatosis with age ≥50 years, BMI ≥30 kg/m², abdominal obesity, low physical activity, poor diet, arterial hypertension, hyperglycemia, hypertriglyceridemia, and hepatic steatosis. The strongest associations were observed for abdominal obesity (OR=6.39), obesity (OR=5.44), hepatic steatosis (OR=4.96), and hypertriglyceridemia (OR=4.41), p<0.001.

According to multivariate logistic regression analysis, independent predictors of pancreatic steatosis were hepatic steatosis, abdominal obesity, obesity, hypertriglyceridemia, age ≥50 years, and impaired carbohydrate metabolism.

Table 6. Independent risk factors for pancreatic steatosis according to logistic regression analysis

Factor	OR	95% CI	p
Age ≥50 years	2.41	1.72–3.39	<0.001
BMI ≥30 kg/m²	3.46	2.41–4.97	<0.001
Abdominal obesity	4.10	2.89–5.81	<0.001
Glucose ≥5.6 mmol/L or HbA1c ≥5.7%	2.27	1.61–3.19	<0.001
Triglycerides ≥1.7 mmol/L	2.94	2.09–4.13	<0.001
Hepatic steatosis	4.57	3.22–6.48	<0.001

The developed algorithm for early detection of pancreatic steatosis comprises the following sequential steps: screening questionnaire administration, calculation of the total risk score using the PSRAS scale, stratification of patients into low-, moderate-, and high-risk groups, comprehensive laboratory and ultrasound examination of moderate- and high-risk individuals, determination of the grade of pancreatic steatosis, and prescription of preventive measures.

Upon validation of the algorithm in 300 risk-group patients, pancreatic steatosis was identified in 214 individuals (71.3%). The algorithm demonstrated a sensitivity of 92.5%, specificity of 72.1%, positive predictive value of 89.2%, negative predictive value of 79.5%, and overall diagnostic accuracy of 86.7%.

Table 7. Diagnostic performance of the algorithm for early detection of pancreatic steatosis

Parameter	Value, %
Sensitivity	92.5
Specificity	72.1
Positive predictive value	89.2
Negative predictive value	79.5

Overall diagnostic accuracy	86.7

Economic calculation showed that under the standard approach, the conditional cost of comprehensive examination of all 3,135 patients would amount to 940,500,000 UZS, whereas using the algorithm and performing comprehensive examination only in moderate- and high-risk individuals amounted to 474,675,000 UZS. The savings amounted to 465,825,000 UZS, or 49.5%.

Discussion

The findings confirm the high prevalence of pancreatic steatosis risk factors among the adult population undergoing preventive medical examinations. Nearly half of the examined individuals were classified into the moderate- and high-risk groups, underscoring the need for active identification of metabolic disorders already at the primary healthcare level.

The most important independent predictors of pancreatic steatosis were hepatic steatosis and abdominal obesity. This confirms the shared pathogenetic mechanisms underlying fatty infiltration of the liver and pancreas, related to visceral obesity, insulin resistance, dyslipidemia, and chronic low-grade inflammation.

The practical value of the proposed algorithm lies in its simplicity and accessibility. In the first stage, a questionnaire and anthropometry are used, requiring no expensive equipment. Laboratory and ultrasound examinations are performed predominantly in moderate- and high-risk patients, which increases diagnostic efficiency while simultaneously reducing the cost of mass screening.

An important direction for further research is the refinement of diagnostic criteria for pancreatic steatosis, standardization of ultrasound grading of pancreatic fatty infiltration, and prospective studies to assess the impact of preventive measures on disease progression and the risk of developing type 2 diabetes mellitus.

Conclusion

The developed algorithm for early detection of pancreatic steatosis in primary healthcare settings enables effective identification of risk-group patients and timely referral for clinical, laboratory, and ultrasound examination. Pancreatic steatosis was found to be closely associated with age, obesity, abdominal obesity, impaired carbohydrate and lipid metabolism, and hepatic steatosis. Validation of the algorithm demonstrated high sensitivity and diagnostic accuracy, and economic calculation confirmed the possibility of rational use of healthcare system resources.

Practical Recommendations

• All patients aged 34–75 years undergoing preventive examination should be screened using the PSRAS questionnaire for pancreatic steatosis risk calculation.

• Patients with moderate and high risk are recommended to undergo comprehensive examination: anthropometry, fasting glucose, HbA1c, lipid profile, ALT, AST, GGT, and ultrasound of the liver and pancreas.

• When pancreatic steatosis is detected, correction of modifiable risk factors is necessary: weight reduction, increased physical activity, dietary optimization, smoking cessation, and control of blood pressure and carbohydrate and lipid metabolism.

• Patients with pancreatic steatosis require dispensary follow-up with regular clinical, laboratory, and ultrasound monitoring.

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Conflict of Interest Statement

The authors declare no conflict of interest.

Author Contributions

Pulatova S.Sh.: conceptualization, methodology, data collection, formal analysis, writing – original draft. Babadzhanov A.S.: supervision, methodology, writing – review and editing.

Data Availability Statement

The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

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