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Consensus of the expert council on the management of pancreatic exocrine insufficiency in patients with diabetes mellitus

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The objective: **to provide a summary of expert recommendations for the early detection and management of pancreatic exocrine insufficiency (PEI) in patients with diabetes mellitus (DM).**
The publication is based on materials and conclusions of the International Expert Advisory Council, convened on 28 November 2025, which included gastroenterologists and endocrinologists from Ukraine and Israel. The council conducted an analysis of current guidelines and the available evidence regarding the relevance, significance, diagnosis, and treatment of PEI in patients with DM.
PEI in the context of DM is a prevalent yet frequently underdiagnosed and underestimated condition. It results in maldigestion, malabsorption, nutritional deficiencies, reduced quality of life, increased glycaemic variability, and worsened long-term prognosis, including elevated cardiovascular risk. In primary care, gastrointestinal symptoms are often incorrectly attributed to adverse effects of antihyperglycaemic therapies such as metformin, GLP-1 agonists, and DPP-4 inhibitors, which can delay accurate diagnosis. Given the combined burden and poor prognosis associated with coexisting PEI and DM, experts recommend proactive screening for PEI in at-risk patients with DM. Risk groups include those with type 1 DM, insulin dependence or low C-peptide levels, type 3c DM, long-standing DM, inadequate glycaemic control, or unexplained weight loss. Recommended screening tools include faecal elastase-1 measurement, the PEI-Q questionnaire, and systematic assessment of nutritional status. Initial pancreatic enzyme replacement therapy should utilise pancreatin in small granules less than 2 mm in diameter (optimally less than 1.8 mm) with enteric coating, at starting doses of 40,000–50,000 units of lipase per main meal and 20,000–25,000 units per snack. Therapy should be long-term, with dose titration based on clinical response and nutritional markers, and should be combined with dietary support and correction of micronutrient deficiencies.
***Conclusions*. PEI in patients with DM represents a clinically significant and frequently underdiagnosed condition in family medicine. The implementation of proactive screening in risk groups, along with a multidisciplinary management approach, is expected to improve quality of life, enhance metabolic control, and reduce long-term adverse prognostic outcomes.**
Keywords: *pancreatic exocrine insufficiency, diabetes mellitus, type 1 diabetes, type 2 diabetes, glycaemic variability, HbA1c control, faecal elastase-1, PEI-Q questionnaire, EPC guidelines, proactive screening, maldigestion and malabsorption, fat soluble vitamin deficiency (A, D, E, K), vitamin B_{12} deficiency, sarcopenia, osteoporosis and fracture risk, gastrointestinal symptoms (steatorrhea, bloating, abdominal pain), pancreatic enzyme replacement therapy, dose titration, nutritional support, cardiovascular risk, quality of life, morbidity and worsening of prognosis.*

Експертний консенсус щодо ведення екзокринної недостатності підшлункової залози у пацієнтів із цукровим діабетом

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Мета: узагальнення експертних рекомендацій щодо раннього виявлення та ведення екзокринної недостатності підшлункової залози (ЕНПЗ) у пацієнтів із цукровим діабетом (ЦД).
Публікація ґрунтується на матеріалах і висновках Міжнародної експертної консультативної ради (28.11.2025 р.) за участю гастроентерологів і ендокринологів України та Ізраїлю. Здійснено аналіз сучасних настанов і доказової бази щодо актуальності, значущості, діагностики та лікування ЕНПЗ у пацієнтів із ЦД.
ЕНПЗ на тлі ЦД є поширеним та водночас недостатньо діагностованим і недооціненим станом, що призводить до мальдигестії/мальабсорбції, нутритивної недостатності, зниження якості життя пацієнтів, підвищення глікемічної варіабельності та погіршення довгострокового прогнозу, включно із серцево-судинними ризиками. Частою проблемою на первинній ланці надання медичної допомоги є хибна атрибуція гастроінтестинальних симптомів до побічних ефектів антигіперглікемічної терапії (метформін, агоністи GLP-1, інгібітори DPP-4), що уповільнює своєчасну діагностику. Враховуючи взаємне обтяження цих двох діагнозів та прогностичну несприятливість такого поєднання, експерти рекомендують проактивний скринінг на ЕНПЗ у пацієнтів із ЦД у групах ризику (ЦД 1-го типу, інсулінозалежність або низький рівень С-пептиду, ЦД 3с типу, тривалий перебіг ЦД, незадовільний глікемічний контроль, немотивоване зменшення маси тіла) із застосуванням визначення фекальної еластази-1, опитувальника PEI-Q та

системної оцінки нутритивного статусу. Базова замісна ферментна терапія панкреатином у формі дрібних гранул розміром менше ніж 2 мм (оптимально – менш як 1,8 мм) з ентеросолюбільним покриттям рекомендована у стартових дозах 40 000–50 000 ОД ліпази на основний прийом їжі та 20 000–25 000 ОД на перекус; терапія має бути тривалою з подальшим титруванням дози відповідно до клінічної відповіді й нутритивних маркерів; лікування слід поєднувати з дієтологічною підтримкою та корекцією мікронутрієнтних дефіцитів.

Висновки. ЕНПЗ у хворих на ЦД є клінічно значущим і часто недіагностованим станом у практиці сімейних лікарів. Впровадження проактивного скринінгу в групах ризику й мультидисциплінарного підходу до ведення таких пацієнтів сприятиме покращенню якості життя, досягненню кращого метаболічного контролю та зменшенню довгострокових несприятливих прогностичних наслідків для пацієнтів.

Ключові слова: *екзокринна недостатність підшлункової залози, цукровий діабет, цукровий діабет 1-го типу, цукровий діабет 2-го типу, варіабельність глікемії, контроль HbA1c, фекальна еластаза-1, опитувальник PEI-Q, рекомендації EPC, проактивний скринінг, порушення травлення та мальабсорбції, дефіцит жиророзчинних вітамінів (A, D, E, K), дефіцит вітаміну B_{12}, саркопенія, остеопороз та ризик переломів, шлунково-кишкові симптоми (стеаторея, здуття живота, біль у животі), замісна терапія панкреатичними ферментами, титрування дози, нутритивна підтримка, серцево-судинний ризик, якість життя, захворюваність та погіршення прогнозу.*

On 28 November 2025, the International Expert Advisory Council (hereinafter, the Expert Council) convened to discuss the topic of "Exocrine pancreatic insufficiency in patients with diabetes mellitus". Leading specialists in the fields of gastroenterology and endocrinology exchanged views on this relevant topic via an online format. Professor Igor Skrypnyk (Poltava State Medical University), President of the Ukrainian Gastroenterological Association, served as the meeting moderator. The Expert Council comprised an international and interdisciplinary team of specialists. Members included leading Israeli gastroenterologist Dr. Yulia Ron (Tel Aviv Sourasky Medical Center – Ichilov), Professor Liubov Sokolova (SI V. P. Komisarenko "Institute of Endocrinology and Metabolism of NAMS of Ukraine"), Professor Olha Bondarenko (SNPE "Danylo Halytsky Lviv National Medical University"), Professor Ganna Maslova (Poltava State Medical University), and Professor Oleksandra Filippova (Dnipro State Medical University). The meeting addressed clinical aspects of managing patients with diabetes mellitus (DM) and confirmed or suspected pancreatic exocrine insufficiency (PEI), emphasizing practical issues in diagnosis and treatment.

The objective: to provide a summary of expert recommendations for the early detection and management of PEI in patients with DM.

Background

DM continues to represent a major medical and social challenge globally. The International Diabetes Federation (IDF) reported that in 2024, 589 million adults worldwide were living with DM, equating to 1 in 9 individuals [4]. Projections suggest that by 2050, this number will rise to 853 million, with over 40% of affected individuals potentially remaining undiagnosed [4]. In Ukraine, the disease burden is similarly substantial. As of November 2025, the National Health Service of Ukraine recorded more than 1.3 million individuals with DM in the electronic health system [6], while IDF epidemiological estimates indicate an even higher prevalence among adults [4]. Most cases (nearly 90%) are attributed to type 2 DM [5], which is commonly associated with obesity [7], arterial hypertension [7], dyslipidaemia [7], metabolically associated steatosis liver disease [8], and pancreatic steatosis [11]. These comorbidities contribute to a high cumulative risk of complications [7–10] and impede achieving stable glycaemic control [12, 13]. In this context, PEI is increasingly recognised as an underappreciated but clinically significant factor. PEI may influence nutritional status, gastrointestinal symptoms, and quality of life [1–3], is associated with glycaemic variability and therapeutic challenges in certain patients with DM [13, 14], and may also exacerbate cardiovascular outcomes [1–3].

The European guidelines for the diagnosis and treatment of PEI, prepared under the auspices of the European Pancreatic Club (EPC, 2024), which consider the management of PEI in patients with type 1 and type 2 DM, emphasizes the relevance of the problem [1]. This justifies the need to implement systematic approaches to the timely detection and treatment of PEI in patients with DM, adapted to the Ukrainian clinical context.

Pathogenesis

The key pathogenetic mechanisms of PEI in the context of DM are recognised as multifactorial, as outlined in the EPC (2024) guideline [1] (Fig. 1). PEI arises from overlapping hormonal, metabolic, neurohumoral, and structural changes within the pancreas, which can mutually potentiate one another [16–19]. Central to these processes is the endocrine-exocrine interaction: disruption of islet cell function alters trophic support and regulation of acinar cell viability and secretion, while exocrine dysfunction may further complicate metabolic control [15, 18, 19].

The principal pathogenetic mechanisms contributing to the development of PEI in patients with DM include the following [1, 16]:

- loss of the trophic and stimulatory effects of insulin on acinar cells and exocrine pancreatic secretion;
- atrophy and fibrosis of the pancreas, often resulting from microangiopathy and chronic tissue injury;
- hyperglycaemia, which promotes fibrogenesis and exacerbates gland dysfunction;
- diabetic autonomic neuropathy and impaired enteropancreatic reflexes, leading to altered regulation of secretion;
- steatosis as a manifestation of metabolic pancreatic injury;
- dysregulation of other islet hormones, particularly glucagon and somatostatin, which influence exocrine pancreatic function;
- concurrent involvement of endocrine and exocrine tissue in autoimmune, infectious, or genetic diseases.

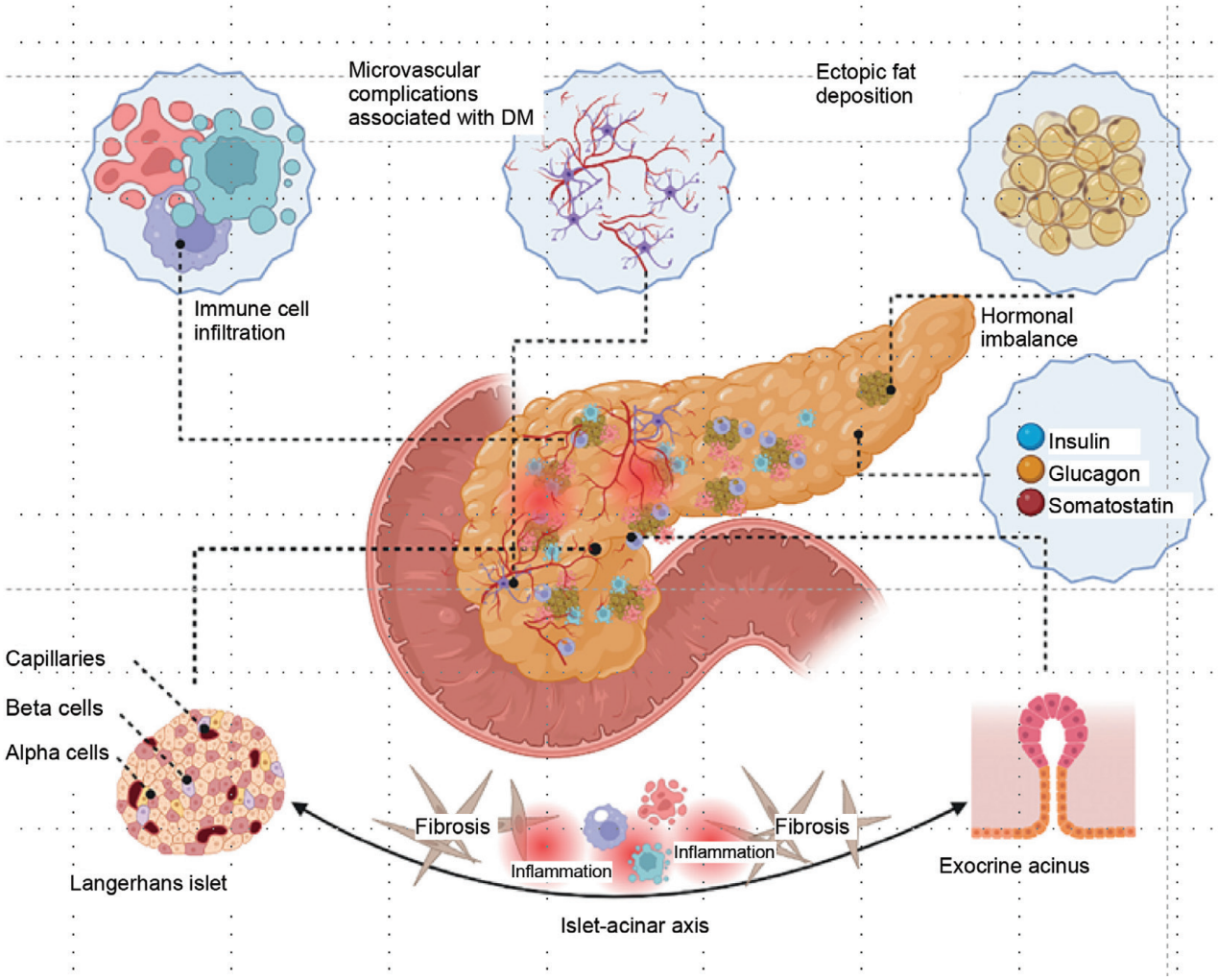

Fig. 1. Pathogenesis of PEI associated with DM: multifactorial process [16]

Notes: PEI – pancreatic exocrine insufficiency; DM – diabetes mellitus.

Clinical significance of PEI in DM

PEI in patients with DM has not only a gastroenterological, but also a systemic clinical dimension, manifested by the phenomenon of "mutual burden": DM promotes the development and progression of exocrine pancreatic dysfunction, while PEI, in turn, worsens the course of DM [15, 17, 19, 20]. Prognostically, the most significant, though not the only, adverse consequences of this are the bidirectional effects on increased risk of osteoporosis [21, 22], cardiovascular diseases [23, 24], and glycaemic variability [15, 17, 19, 20]. The key mechanism underlying glycaemic variability is unpredictable digestion and absorption of nutrients, with altered carbohydrate availability due to impaired incretin response, which may clinically manifest as increased glycaemic variability (episodes of hyper- and/or hypoglycaemia), complicating the achievement of target glycated haemoglobin (HbA1c) levels and stable metabolic control [15, 17, 19, 20].

A key clinical consequence of PEI in patients with DM is progressive nutritional deficiency. Maldigestion and malabsorption result in weight loss, sarcopenia, protein-calorie deficiency, and micronutrient deficiencies, primarily of fat-soluble vitamins (A, D, E, K), which have direct clinical consequences [1, 3]. Vitamin D deficiency and disturbances of calcium-phosphorus homeostasis in PEI are associated with decreased bone mineral density, osteoporosis, and increased fracture risk. Clinical guidelines recommend monitoring for metabolic bone disease and correcting deficiencies as part of comprehensive nutritional support and pancreatic enzyme replacement therapy (PERT) [1, 33].

Vitamin A deficiency may present as night blindness and impaired trophism of mucous membranes, which has additional clinical significance in DM; vitamin E deficiency is mainly associated with neurological and muscular symptoms (peripheral neuropathy, weakness, rapid fatigability), which further worsen the functional status of patients with DM [1, 3].

Vitamin K deficiency may lead to coagulopathy with a tendency to bleed (prolonged coagulation parameters, easy bruising), which is especially significant in patients receiving antiplatelet or anticoagulant therapy [1, 3].

Vitamin B_{12} deficiency requires particular attention: in PEI it may manifest as macrocytic anaemia, fatigue,

and neurological symptoms (paraesthesia, gait disturbances, cognitive complaints), which justifies screening and correction as part of comprehensive management of patients with DM [3].

Impaired hydrolysis and absorption of lipids in PEI, particularly essential fatty acids, are associated with dermatological manifestations (dry skin, dermatitis) and delayed tissue repair, which in patients with DM may correlate with increased risks of infection and impaired wound healing [1, 2].

At the same time, existing nutritional deficiencies and inadequate absorption of vitamins and trace elements in PEI can reduce immune response, increase susceptibility to infections, and worsen the course of diabetic complications due to impaired reparative processes and overall metabolic status. Clinical guidelines emphasize the need for systematic assessment of nutritional status, correction of deficiencies, and adequate PERT in these patients [1, 3].

Gastrointestinal manifestations of PEI (diarrhoea, steatorrhea, bloating, abdominal pain, flatulence, postprandial discomfort, and a feeling of heaviness after eating) are frequent and clinically significant, as they reduce quality of life, affect eating behaviour and adherence to therapy, and may be accompanied by psychological distress [1, 3].

In patients with DM, these nonspecific symptoms are often mistakenly attributed as side effects of antihyperglycaemic agents (notably metformin, GLP-1 (Glucagon-Like Peptide-1) receptor agonists, and/or DPP-4 (Dipeptidyl Peptidase-4) inhibitors). This misattribution is explicitly highlighted in section 10 of the European Guidelines (2024) on PEI in DM and is considered one of the reasons for underestimation of PEI and delayed diagnosis [1].

As a result of this diagnostic trap, the clinical focus often shifts to "treatment intolerance": patients independently discontinue medications, reduce dosages, or insist on unwarranted changes in therapy. According to the guidelines [1], such actions may worsen glycaemic control but do not address the root cause – maldigestion and malabsorption due to PEI [1, 3]. This can lead to progression of nutritional deficiencies and further decline in quality of life [1, 3].

For the appropriate interpretation of symptoms in patients with DM, the guidelines recommend:

– suspecting PEI when symptoms of malabsorption (steatorrhea, bloating, flatulence, diarrhoea, abdominal pain, heaviness) are present in combination with signs of nutritional deficiency (weight loss, deficiencies of fat-soluble vitamins and/or trace elements) [1, 3];
– performing non-invasive testing, primarily determination of faecal elastase-1 (FE-1), combined with assessment of nutritional status (vitamins A, D, E, K, B_{12}; albumin/prealbumin; anthropometry) [1, 3];
– if PEI is confirmed, initiating PERT with adequate doses of pancreatic enzymes and correction of nutritional deficiencies, which not only reduces symptom severity but also contributes to stabilization of glycaemic control, avoiding unjustified discontinuation of antihyperglycaemic therapy [1, 3]. According to cohort studies, PEI is associated with increased morbidity, mortality, and worse cardiovascular prognosis [1]. PEI is considered a marker of a more unfavourable course of chronic pancreatic diseases, which should be taken into account during risk stratification, prevention, and management of complications [1, 3].

Given the high likelihood of underdiagnosis of PEI in patients with DM and the nonspecific nature of symptoms (abdominal pain, heaviness, flatulence, bloating, diarrhoea, steatorrhea), experts have identified clinical situations in which the risk of PEI increases (Table 1). This justifies a proactive approach – targeted assessment of PEI in risk groups even before the onset of pronounced gastrointestinal symptoms or nutritional deficiencies.

An additional marker of risk for PEI, as recommended by experts, is pronounced glycaemic variability that cannot be explained solely by the antihyperglycaemic therapy regimen [20].

Important modifiers of risk and/or consequences of PEI are conditions of the so-called "metabolic continuum": pancreatic steatosis (referred to in English literature as fatty pancreas disease) [1], sarcopenia and sarcopenic obesity [34], as well as age-related structural and functional changes in the pancreas (so-called "aging" pancreas) [1, 36].

It is also necessary to consider concomitant pathological conditions associated with an increased risk of PEI and digestive disorders: gut microbiome disturbances [37], chronic heart [35] and renal insufficiency [1], inflammatory bowel diseases, pancreatic duct obstruction, previous pancreatic and/or gastrointestinal surgeries, and iatrogenic pancreatic injuries [1] (Fig. 2).

Table 1

Risk factors for the development of PEI in patients with DM

Aggravating factors	Clinical significance
Type 1 DM	The likelihood of PEI is higher compared to type 2 DM [1, 25]
Insulin dependence, low C-peptide	Markers of insulin deficiency, associated with more pronounced reduction of exocrine pancreatic function [26, 27]
Type 3c DM (pancreatogenic)	Frequently coexists with PEI [1, 28]
Poor glycaemic control (high HbA1c)	Associated with higher frequency of PEI [20, 29, 30, 32]
Long-standing DM	Risk of PEI increases with the duration of disease; increased vigilance is warranted in patients with a long history of DM [1, 31, 32]
Unintentional weight loss	A marker of possible maldigestion/malabsorption; basis for proactive assessment of PEI

Notes: DM – diabetes mellitus; PEI – exocrine pancreatic insufficiency.

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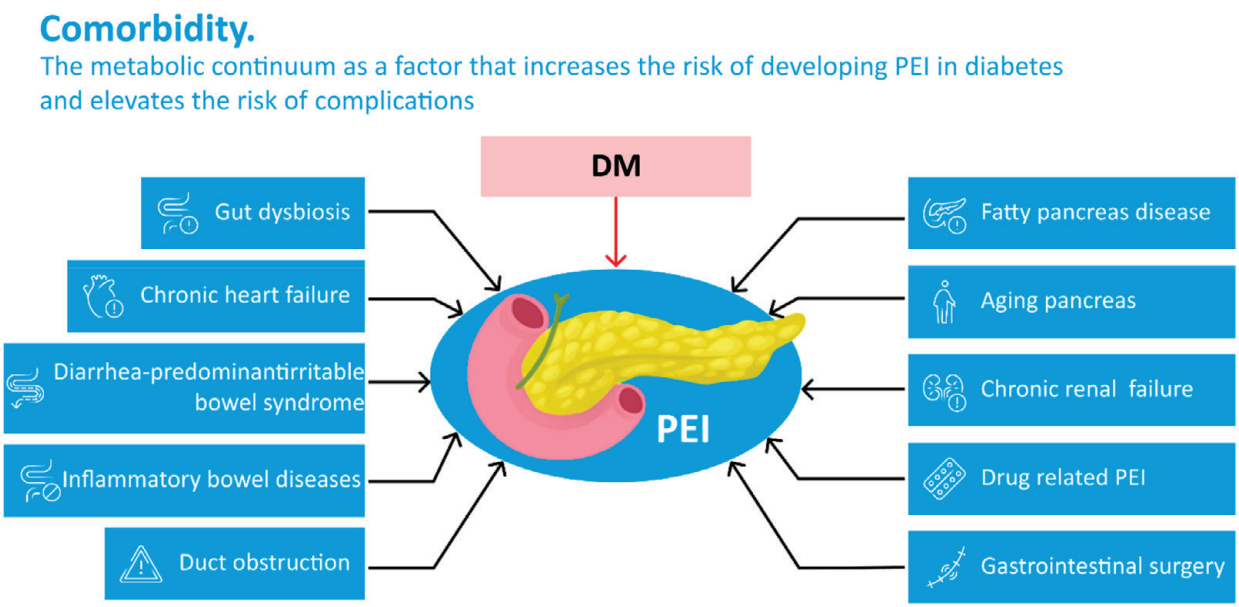

Fig. 2. Metabolic continuum: diseases increasing the risk of PEI in DM [1–3, 33–37]

Notes: DM – diabetes mellitus; PEI – exocrine pancreatic insufficiency.

Diagnosis

Experts have proposed an active approach to the management of patients with DM, which involves a shift from the strategy of "assessing PEI only in cases of pronounced steatorrhea and significant weight loss" to a systematic strategy of early detection. The practical aim of this approach is to reduce the proportion of unrecognised cases of PEI and prevent the consequences of malabsorption, which, in addition to the general effects of PEI, may influence glycaemic variability, bone metabolism, cardiovascular, and overall prognosis. Implementation of an active strategy is based on identification of patients at increased risk (Table 1, Fig. 2), active detection of "minor" gastrointestinal symptoms using validated questionnaires, early use of available methods for assessing exocrine pancreatic function, and parallel evaluation of nutritional status. Systematic screening in risk groups is viewed as a tool capable of improving early detection of PEI and optimizing treatment outcomes.

Experts noted that the diagnosis of PEI in patients with DM should be based on general principles, combining clinical assessment of symptoms, analysis of nutritional status, and available tests for assessment of exocrine pancreatic function [1–3]. At the same time, specialists emphasized the high risk of diagnostic errors due to the nonspecificity of gastrointestinal manifestations: bloating, abdominal discomfort, diarrhoea, and steatorrhea may be mistakenly interpreted as functional bowel disorders or gastroparesis, side effects of hypoglycaemic agents (notably metformin, GLP-1 receptor agonists), or manifestations of diabetic autonomic neuropathy [1, 38–40]. Such misinterpretations can delay timely diagnosis of PEI, which may be accompanied by progression of nutritional deficiencies and potential worsening of glycaemic control.

According to the recommendations of the EPC guideline (2025), determination of FE-1 is recommended as a method of primary assessment of PEI. Although it is not the gold standard and has certain limitations, it remains the most accessible test in routine clinical practice [1].

To improve the diagnosis of PEI, it is advisable to use the standardized PEI-Q (Pancreatic Exocrine Insufficiency Questionnaire), which allows for the structuring of complaints, quantitative assessment of their severity, and subsequent use for monitoring response to PERT. The questionnaire contains 18 questions, is available in Ukrainian, and can be completed online. From a practical standpoint, the optimal tactic is to fill out the PEI-Q before the consultation, which increases the structure of the visit and facilitates the identification of patients with a higher probability of PEI [41]. Use of the PEI-Q is recommended in the National Clinical Guideline "Chronic Pancreatitis" (2023) [42], based on the principles of evidence-based medicine.

If, after a comprehensive assessment of symptoms, history, nutritional status, and exocrine pancreatic function, the diagnosis of PEI cannot be established unequivocally, an additional tool may be used – a trial PERT at therapeutic doses for 4–6 weeks. Experts emphasized that a positive response to such therapy may indicate probable PEI and is both a diagnostic and therapeutic approach. Even partial clinical improvement with PERT should be considered a positive result and grounds for continuation of therapy [1, 2].

Treatment

In reviewing modern approaches to the treatment of PEI, the Expert Council relied on the provisions of the EPC guideline (2024), which recommends, among agents for PERT, a preference for enzyme preparations (EPs) in the form of small-sized granules with an acid-resistant (enterosoluble) coating. To date, only porcine-derived pancreatin preparations have demonstrated clinical efficacy [1].

The current key requirements for choosing EPs, which determine the effectiveness of PERT, are: good mixing with chyme, protection from inactivation by gastric juice, evacuation from the stomach synchronously with the food bolus, and rapid activation of enzymes in the proximal duodenum. It is specifically emphasized that modern EPs should contain small particles (less than 2 mm, optimally less than 1.8 mm), which better mix with food and, synchronously with chyme, transit from the stomach to the duodenum. These characteristics of EPs promote more effective digestion as well as higher therapeutic efficacy [1–3, 43]. Members of the Expert Council emphasized that, for the correction of PEI, it is appropriate to use medicinal products with proven efficacy rather than dietary supplements [3].

Initial (minimally effective) doses of EPs depend on age, severity of PEI, and fat content in the diet. For adults, a basic initial regimen should consider 40,000–50,000 units of lipase per main meal and 20,000–25,000 units of lipase per snack [1–3, 42]. These doses are aimed at reducing gastrointestinal symptoms, improving and maintaining nutritional status; with lower doses, even if symptomatic improvement is observed, 70% of patients continue to have pathologically reduced nutritional indicators [43].

EPs should be taken so that they enter the intestine simultaneously with food; the optimal time for administration is during meals, not before or after. Taking EPs with snacks is mandatory if the food contains fat or protein [1–3, 42].

Subsequently, the initial doses should be titrated based on clinical response, changes in body weight, and nutritional status parameters. An approximate control assessment should be performed after 4–6 weeks, taking into account symptoms, body weight, and dynamic changes in PEI-Q scores. In case of insufficient effect, adherence to therapy, the correctness of EP intake (with every meal, without skipping snacks, and avoiding "after meal" administration), and the exclusion of concomitant pathology that may impair digestion or the compliance of the prescribed preparation with modern PERT requirements should be verified. Further dose increases and individualized consideration of adding a proton pump inhibitor to the treatment regimen may be possible [1–3, 42, 43].

An insufficient response to PERT is defined by persistent diarrhoea and/or steatorrhea, bloating, flatulence, abdominal discomfort, lack of weight gain or stabilization, and persistent nutritional deficiencies. If the expected effect is not achieved after correcting the duration and dose of EPs, it is necessary to simultaneously exclude other causes of malabsorption or diarrhoea [1–3, 42, 43].

The first step in evaluating the effectiveness of PERT should be conducted after 4–6 weeks, assessing gastrointestinal symptoms (preferably using a standardized validated questionnaire). Subsequently, according to European recommendations (2024), for long-term follow-up of patients with PEI in the context of DM, monitoring of the parameters listed in Table 2 is recommended [1].

The effectiveness of PERT is assessed comprehensively – by reduction of gastrointestinal manifestations in combination with correction of nutrient deficiencies, improvement or maintenance of adequate nutritional status, and quality of life. Long-term observational data indicate that clinically significant improvement in trophological status parameters, including stabilization or increase in body weight along with symptom reduction, is possible within 6–12 months after initiation of therapy [43]. In this context, partial effect should be regarded as grounds for continuation of therapy: even incomplete but clinically noticeable reduction of some symptoms or deficiencies indicates that the chosen strategy is working and requires further optimisation (primarily of dosage and adherence) rather than discontinuation [1].

Table 2

Parameters recommended for long-term follow-up of patients with PEI associated with DM

Nutritional/functional			
Body weight BMI Weight loss Grasping power	Anthropometry: mid-upper arm muscle circumference; quantitative analysis of muscle mass (bioelectrical impedance measurement and/or computed tomography)	Body composition and bone tissue assessment using dual-photon X-ray absorptiometry (every 2 years)	6-minute walk test
Biochemical			
Complete blood count and iron stores	Plasma proteins: albumin, retinol-binding protein, and transferrin	Micronutrient status: magnesium, fat-soluble vitamins, zinc, selenium, vitamin B_{12}, and folate	C-reactive protein Glucose and HbA1c Parathormone
Clinical			
Assessment of bowel symptoms: frequency and colour of stools; bloating/flatulence; abdominal pain after meals	Factors affecting quality of life: changing medications (especially opioids and antidiarrhoeals)	Following lifestyle considerations (smoking cessation and abstinence, physical activity, and adequate sun exposure)	
Nutritional			
Assessment of aversion to food due to abdominal symptoms	Analysis of the daily ration (dietary history / food record) compared to the appropriate PERT dose to assess adherence to therapy and consistency of PERT with nutrition	Assessment of avoidance of fatty foods	Assessment of adequate diet

Notes: DM – diabetes mellitus; PEI – exocrine pancreatic insufficiency; BMI – body mass index; PERT – pancreatic enzyme replacement therapy.

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Given the pathogenesis of PEI in the context of DM and its prognostic consequences, treatment should be long-term and primarily aimed at restoring nutritional status and correcting deficiencies, not merely at alleviating gastrointestinal symptoms [1, 3, 43].

To achieve maximum effectiveness of PERT, treatment should be combined with nutritional support and correction of micronutrient deficiencies; if indicated, involvement of a clinical dietitian is advisable [1, 3]. Experts recommended including a dietitian consultation as part of the standard of care for patients with suspected or confirmed PEI in the context of DM. It is advisable to design the diet without strict fat restrictions, with sufficient protein content and a fractional meal regimen of 5–6 times per day, individualized according to glycaemic goals. Correction of fat-soluble vitamin deficiencies should be performed with subsequent laboratory monitoring; the frequency of monitoring should be determined individually, approximately every 6–12 months. Recommended doses: vitamin D – 2,000–4,000 IU/day, vitamin A – 10,000–25,000 IU/day, vitamin E – 400–800 IU/day, vitamin K – 5–10 mg/day as needed [1].

CONCLUSIONS

Following the deliberations of the Expert Council, the subsequent resolutions were adopted:

1. PEI in the context of DM should be recognised as a clinically significant and underdiagnosed condition that adversely affects nutritional status, quality of life, glycaemic control, and long-term patient prognosis.

2. The nonspecific presentation of gastrointestinal symptoms and their frequent misinterpretation often result in delayed diagnosis and the progression of nutritional deficiencies.

3. An active approach is recommended, involving systematic assessment of PEI in high-risk groups using established diagnostic criteria, including FE-1, the PEI-Q questionnaire, and comprehensive evaluation of nutritional status.

4. It is considered appropriate to initiate PERT at recommended starting doses with subsequent titration, guided by both clinical symptoms and nutritional markers. Partial clinical response to PERT should serve as a basis for continuation and optimisation of therapy.

5. Consultation with a clinical dietitian should be incorporated into the standard of care for patients with suspected or confirmed PEI in the context of DM. Systematic correction of micronutrient deficiencies and ongoing monitoring of treatment outcomes are also recommended.

6. To enhance the quality of diagnosis and patient management, a series of scientific and educational initiatives should be launched for endocrinologists, gastroenterologists, and general practitioners or family physicians. Broad dissemination of information to the professional community is recommended through publication of this resolution and development of a Consensus document.

7. Given that PEI in the context of DM is a prevalent yet consistently underdiagnosed and underestimated condition with significant prognostic implications, it is both necessary and advisable to develop national clinical guidelines for the management of PEI in patients with diabetes, which would provide a structured, evidencebased decisionsupport framework for physicians across multiple specialties involved in the care of these complex cases.

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