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Modern Concepts of the Pathogenesis of Gastro-Oesophageal Reflux Disease

Introduction. Gastro-oesophageal reflux disease is one of the most common disorders of the gastrointestinal tract; it is characterised by considerable clinical variability and has a significant impact on patients' quality of life.

The aim of this study was to summarise current scientific evidence regarding the pathophysiological mechanisms underlying the development of GERD, the role of risk factors and psycho-emotional factors, as well as the characteristics of clinical phenotypes of GERD.

Materials and methods. A literature review was conducted to summarise current scientific data. The literature search was carried out in the international electronic databases PubMed/MEDLINE, Scopus, Web of Science Core Collection and Google Scholar. The publications included in the analysis covered the period from 2015 to 2026, with priority given to clinical guidelines, consensus documents, systematic reviews, meta-analyses and original scientific studies.

Results and discussion. The analysis showed that the development of GERD is a complex process involving mechanical, chemical, sensory and neuroregulatory factors. An important role is played by dysfunction of the anti-reflux barrier at the oesophagogastric junction, transient relaxation of the lower oesophageal sphincter, impaired oesophageal clearance and reduced resistance of the epithelial barrier. The role of modifiable risk factors, such as abdominal obesity, lifestyle factors, diet and sleep disturbances, has been established. There is a bidirectional association between GERD and psychological disorders, mediated by dysfunction of the 'brain-gut' axis, visceral sensitivity and oesophageal hypersensitivity. The current classification of GERD is based on phenotype, which is divided into erosive reflux disease,

non-erosive reflux disease, reflux hypersensitivity and functional heartburn, each of which has its own pathophysiological mechanism and treatment approach.

Conclusions. GERD is a multifactorial and heterogeneous condition involving structural, functional and psychosocial mechanisms. An understanding of current pathophysiological concepts enables a shift from a symptomatic approach to a personalised approach based on clinical, endoscopic and functional diagnostic assessment.

Key words: gastro-oesophageal reflux disease, pathogenesis, risk factors, visceral hypersensitivity, psychological disorders, clinical phenotypes.

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Сучасні уявлення про патогенез гастроезофагеальної рефлюксної хвороби

Вступ. Гастроезофагеальна рефлюксна хвороба є одним із найпоширеніших захворювань шлунково-кишкового тракту та характеризується значною клінічною варіабельністю та суттєво впливає на якість життя пацієнтів.

Метою цього дослідження було узагальнити сучасні наукові дані щодо патофізіологічних механізмів, що лежать в основі розвитку ГЕРХ, ролі факторів ризику та психоемоційних факторів, а також характеристик клінічних фенотипів ГЕРХ.

Матеріали та методи. Для узагальнення сучасних наукових даних було проведено огляд літератури. Пошук літератури здійснювався в міжнародних електронних базах даних PubMed/MEDLINE, Scopus, Web of Science Core Collection та Google Scholar. Публікації, включені до аналізу, охоплювали період з 2015 по 2026 рік, при цьому пріоритет надавався клінічним настановам, консенсусним документам, систематичним оглядам, метааналізам та оригінальним науковим дослідженням.

Результати та обговорення. Виявлено, що розвиток ГЕРХ є складним процесом, у якому беруть участь механічні, хімічні, сенсорні та нейрорегуляторні фактори. Важливу роль відіграють дисфункція антирефлюксного бар'єру на стравохідно-шлунковому з'єднанні, транзиторне розслаблення нижнього стравохідного сфінктера, порушення очищення стравоходу та зниження резистентності епітеліального бар'єру. Встановлено роль модифікованих факторів ризику, таких як абдомінальне ожиріння, фактори способу життя, раціон харчування та порушення сну. Між ГЕРХ та психологічними розладами існує двонаправлений зв'язок, опосередкований дисфункцією осі 'мозок-кишечник', вісцеральною чутливістю та гіперчутливістю стравоходу. Сучасна класифікація ГЕРХ ґрунтується на фенотипі, який поділяється на ерозивний рефлюкс, неерозивний рефлюкс, рефлюксну гіперчутливість та функціональну печію, кожна з яких має власний патофізіологічний механізм та підхід до лікування.

Висновки. ГЕРХ це багатофакторне та гетерогенне захворювання, у якому задіяні структурні, функціональні та психосоціальні механізми. Розуміння сучасних патофізіологічних концепцій дає змогу перейти від симптоматичного підходу до персоналізованого підходу, заснованого на клінічній, ендоскопічній та функціональній діагностичній оцінці.

Ключові слова: гастроезофагеальна рефлюксна хвороба, патогенез, фактори ризику, вісцеральна гіперчутливість, психологічні розлади, клінічні фенотипи.

Introduction. Gastro-oesophageal reflux disease (GERD) is one of the most common disorders of the digestive system and one of the most frequent reasons for patients to consult primary care doctors [1, 2, 3]. It is characterised by clinical symptoms and/or complications caused by the reflux of gastric contents into the oesophagus, which leads to a reduced quality of life for patients [4, 5]. Epidemiological studies have shown that GERD may affect up to 20 per cent of the population in Western countries, which is thought to be linked to lifestyle changes, the rising prevalence of obesity, dietary habits and an ageing population [6, 7].

The relevance of the GERD problem is further emphasised by the influence of contemporary socio-psychological factors. There is a bidirectional relationship between GERD and psycho-emotional stress, anxiety, depressive disorders and sleep disturbances, which is associated with more pronounced GERD symptoms [8]. Psycho-emotional factors may manifest as disturbances in gastrointestinal motility, hypersensitivity of internal organs, hypervigilance of the oesophagus, and disturbances in the interaction between the central nervous system and the gastrointestinal tract. At the same time, these mechanisms may exacerbate the perception of symptoms without necessarily increasing the objective reflux burden [9, 10, 11]. Certain dietary habits and changes to the diet may also influence GERD symptoms; however, evidence regarding the effectiveness of specific dietary changes is conflicting [12, 13].

The modern concept of GERD goes beyond the traditional view of this condition as an exclusively acid-dependent process and regards it as a heterogeneous, multifactorial disease. The Lyon Consensus 2.0 recommends that the diagnostic approach should take into account the type of clinical symptoms, the presence or absence of previously objectively confirmed GERD, as well as the results of endoscopy and instrumental reflux monitoring [14]. The information obtained should be used to confirm or rule out pathological reflux and to tailor the patient's treatment strategy individually.

The pathogenesis of GERD is associated with structural and functional abnormalities of the oesophagogastric junction, lower oesophageal sphincter insufficiency, transient relaxation of the lower oesophageal sphincter, impaired volumetric and chemical clearance of the oesophagus, abnormalities in oesophageal and gastric motility, reduced mucosal resistance, and increased visceral sensitivity of the oesophagus [15, 16].

Acid is one of the most significant noxious factors in the refluxate, but pepsin and bile acids can also cause damage to the oesophageal mucosa [17]. At the same time, the lack of response to adequate therapy aimed at suppressing acid secretion does not necessarily mean that non-acidic components are the cause. In such patients, an individualised approach is required to distinguish between persistent pathological low-acid or non-acid reflux, hypersensitivity to reflux, functional heartburn, oesophageal hypersensitivity and other causes of the symptoms [18, 19].

The clinical manifestations of GERD can be highly varied. The classic symptoms are heartburn and regurgitation; however, some patients may experience atypical or extra-oesophageal symptoms, including chronic cough, hoarseness, a sensation of a foreign body in the throat, and chest discomfort [20]. These manifestations can make diagnosis difficult at the primary care level, where they must be distinguished from cardiac, respiratory and otolaryngological diseases. Non-cardiac chest pain is of particular concern, as gastro-oesophageal reflux is one of the most common causes of non-cardiac chest pain once cardiovascular diseases have been ruled out [21].

An important feature of the modern management of patients with GERD is the need to move away from symptomatic treatment in favour of an individualised strategy that takes into account the clinical phenotype of the disease, the presence of risk factors, possible complications and the impact of the disease on quality of life [22]. Despite the effectiveness of proton pump inhibitors (PPIs), which remain the mainstay of medical treatment for GERD, the issue of their rational use remains relevant. In some patients, long-term PPI therapy is used without clear indications, whilst others do not respond to PPIs, necessitating a review of the diagnosis, an assessment of adherence to the treatment regimen and further diagnostic investigations [23, 24].

The family doctor plays an important role in the early diagnosis and management of patients with GERD, as they are the first point of contact with the healthcare system [25]. The role of the GP is to assess the nature of the symptoms, decide whether an endoscopy is necessary, identify 'red flags', prescribe initial treatment and refer the patient to a gastroenterologist in a timely manner, if necessary [26]. One of the most important challenges is to establish a clearly defined algorithm for the management of patients with GERD in clinical practice, in order to prevent inadequate treatment and the prescription of unnecessary medicines.

GERD is a pressing clinical problem that requires a comprehensive approach to diagnosis and treatment based on the latest evidence-based clinical guidelines. A review of current knowledge regarding its pathogenesis, clinical manifestations and treatment strategies enables the optimisation of patient management and an improvement in the quality of clinical care.

The aim of this study was to summarise the current scientific evidence regarding the pathophysiological mechanisms underlying the development of GERD, the role of risk factors and psycho-emotional factors, and the characteristics of GERD clinical phenotypes.

Materials and methods. The analysis and synthesis of existing scientific information on GERD have been conducted, including the most recent information regarding the pathogenetic mechanisms of the development of the disease, risk factors, the role of psycho-emotional factors and the features of clinical phenotypes.

The literature search was conducted in the international electronic databases PubMed/MEDLINE, Scopus, Web of Science Core Collection and Google Scholar. The analysis covered scientific publications, predominantly from the last decade (2015-2026), with priority given to current clinical guidelines, consensus documents, systematic reviews, meta-analyses and original research. In addition, selected fundamental works of significant importance for shaping the current understanding of the pathogenesis and treatment of GERD were included.

The search was conducted using the following keywords in English: 'gastro-oesophageal reflux disease', 'GERD', 'pathophysiology', 'risk factors', 'clinical phenotypes', 'erosive reflux disease', 'non-erosive reflux disease', 'reflux hypersensitivity', 'functional heartburn', 'brain-gut axis', 'anxiety', 'depression', 'stress', 'sleep disturbance', 'oesophageal hypersensitivity', 'oesophageal hypervigilance'.

Publications containing data on the epidemiology, risk factors, mechanism of development, clinical manifestations and phenotypic characteristics of GERD were included, as well as articles focusing on the relationship between gastro-oesophageal reflux disease, psychoemotional factors and disorders of the "brain-gut" axis. The selected sources were analyzed using descriptive and comparative methods, after which the obtained data were synthesized regarding the current understanding of gastro-oesophageal reflux disease as a multifactorial condition in the development of which organic, sensory, behavioral and psychoemotional factors play a role.

Results and Discussion. Gastro-oesophageal reflux disease is one of the most frequent chronic digestive diseases (incidence very different by geographic area). The prevalence of GERD has increased from nearly 450 million patients in 1990 to more than 825 million in 2021, according to a study by L. Chen et al. [27]. A study published by J. Nirwam et al. (2020) shows that there are

regional variations in the prevalence of GERD with highest prevalence in North America and Europe and lowest prevalence in East Asian countries [3]. These disparities could be associated with obesity rates, nutrition, and urbanization and lifestyle. The burden of GERD is a major health and social problem worldwide because of its adverse effects on patient's quality of life and the high demands for healthcare resources.

There were several modifiable risk factors for developing reflux oesophagitis among which abdominal obesity was noted by J. Zhan et al. (2022) [28] as one such factor. There is an association between accumulation of visceral adipose tissue and a higher risk of reflux oesophagitis. The major pathophysiological mechanisms involved in this correlation are: increased intra-abdominal pressure, decreased function of the oesophagogastric junction, increased prevalence of hiatal hernias, and motility disorders of the upper gastrointestinal tract. Data from a Mendelian randomisation study by Shuai Yuan and Susanna C. Larsson (2022) confirm a possible causal link between obesity and the development of GERD. A higher genetically determined BMI (per 1 standard deviation) was linked to an increased risk for GERD (OR 1.49; 95% CI 1.40-1.60) [29]. The cause of this association could be through a number of mechanisms such as: increased intra-abdominal pressure, decreased oesophageal motility, lower oesophageal sphincter dysfunction and development of inflammatory changes in the mucosa. There are several lifestyle factors that are important in the occurrence of GERD symptoms. Factors that may be harmful are smoking, lack of physical activity, irregular eating habits, eating too many energy dense foods and sleep problems. At the same time, recent research has shown that the impact of individual foods on GERD is unique to the individual, and dietary recommendations should not be made to exclude specific foods, but rather to identify foods that trigger symptoms.

The age has been shown as an important factor in the clinical course of gastro-oesophageal reflux disease (GERD) through the study of Shi et al. (2021) [30]. Ineffective oesophageal motility, reduced anti-reflux function, increased acid exposure were more common in older patients (age ≥60 years) than in younger patients. Severe forms of reflux oesophagitis and dysfunction of the oesophageal hiatus were more frequently detected in elderly patients, which justifies the need for more careful clinical monitoring of this patient group.

Recent epidemiological data indicate that many factors contribute to the development of GERD, with obesity, lifestyle, dietary factors and age-related changes being the most important. Taking these factors into account is a key component of prevention and the development of a personalised management strategy for patients.

The prevailing view of the pathogenesis of gastro-oesophageal reflux disease (GERD) considers it to be a multifactorial disorder, with a breakdown in the function of the oesophagogastric junction (EGJ), refluxate characteristics, the mucosal barrier and sensory perception. The current paradigm of GERD is different from the old acid-based model, which sees GERD as a failure of the oesophagus's defence mechanisms to cope with aggressive reflux factors. The key mechanism underlying the development of pathological gastro-oesophageal reflux is the insufficiency of the EGJ's anti-reflux barrier, which comprises the lower oesophageal sphincter, the diaphragmatic crura and the anatomical configuration of the cardia. Disruption of these interactions, especially when a hiatal hernia is present, is a factor in the frequency and length of reflux episodes. An important role is played by transient lower oesophageal sphincter relaxations (TLESR); these are a physiological mechanism for belching, but they can be a part of the reflux events in patients with a weak EGJ [31, 32, 33].

The length of time that the oesophageal mucosa is in contact with the refluxate is an important part of the pathogenesis of gastro-oesophageal reflux disease, and is heavily influenced by the efficiency of oesophageal clearance. The impaired oesophageal clearance mechanisms cause the mucosa to be exposed to refluxate for a longer period of time, which can worsen damage to the epithelial barrier. Volumetric clearance of the oesophagus is accomplished mainly by the peristaltic contractions (secondary peristalsis) stimulated by distension of the oesophageal wall by refluxate. Impaired oesophageal motility and decreased clearance are linked to the onset of GERD and are responsible for increased contact time between the acidic refluxate and the mucosa [34, 35].

Hydrochloric acid is one of the most important factors that is aggressive to the pathogenesis of gastro-oesophageal reflux disease. In parallel, Samuels T. and Johnston N. (2020) showed that pepsin, a non-acidic component of refluxate, may have a role to play [36]. Pepsin is one of the most active components of refluxate, which is capable of damaging the epithelium and triggering an inflammatory response, and can have a pathological effect even at neutral pH. But, the pathogenetic role of pepsin and its role in the pathogenesis of different GERD phenotypes should be further investigated.

Non-acid reflux, or pathological low-acid reflux, is not always present when symptoms persist after proton pump inhibitor (PPI) treatment in patients with gastro-oesophageal reflux disease (GERD). In some patients, symptoms may be related to reflux hypersensitivity, in which the reflux is not associated with acid levels that are above normal, but there is a strong temporal association between reflux and the appearance of symptoms. Another phenotype is functional heartburn, which is defined as no pathological acid exposure, and is not associated with reflux events. Therefore, if the response to PPIs is inadequate, the mechanism responsible for the symptoms needs to be clarified, using functional testing, such as pH-impedance monitoring [37].

The modern concept of GERD involves one of its major elements as a weakened resistance of the oesophageal mucosa [38]. The increase in epithelial permeability, widening intercellular spaces, changes in expression of tight junction proteins and decrease in basal impedance may indicate a disruption in the integrity of the epithelial barrier [39]. In these circumstances the chemical components of refluxate are more likely to gain access to subepithelial structures and to sensory nerve endings, thereby potentially contributing to peripheral sensitisation.

Damage to the epithelial barrier has been associated with the development of oesophageal hypersensitivity, and activation of neuroimmune and neuroinflammatory signalling pathways is one of the potential mechanisms that could link these two events together [40]. Pro-inflammatory mediators, sensory receptors, neuropeptides and immunocompetent cells of the mucosa may be involved in this process. However, the severity of clinical symptoms is not always related to the severity of the endoscopic findings: severe clinical symptoms can occur without erosions, and severe endoscopic changes can occur with relatively mild symptoms [41, 42].

The pathogenesis of GERD is multifactorial and arises from the interaction of mechanical insufficiency of the oesophagogastric junction, characteristics of the refluxate, impaired oesophageal clearance, reduced mucosal resistance and neurosensory dysregulation. This integrative model helps to explain the clinical heterogeneity of the disease and the discrepancy between symptoms and morphological changes, whilst also justifying a phenotype-oriented and personalised approach to the diagnosis and treatment of patients with GERD.

The current understanding of GERD combines the traditional somatic model with a biopsychosocial perspective, according to which the clinical severity of symptoms depends not only on acid load but also on psychological and neuroregulatory mechanisms of sensory processing within the 'brain-gut' axis. In a systematic review and meta-analysis of nine observational studies involving 1,485,268 participants, He et al. (2022) investigated the association between psychosocial disorders (anxiety, depression and other psychological disorders) and GERD [43]. The authors found that the presence of psychosocial disorders was associated with an increased risk of GERD (OR 2.23; 95% CI 1.42-3.51), whilst in patients with GERD, the risk of psychosocial disorders was also higher (OR 2.57; 95% CI 1.87-3.54). Subgroup analysis showed that patients with non-erosive reflux disease (NERD) had a stronger association with anxiety disorders compared with patients with the erosive form (ERD), which may indicate a greater role for disturbances in sensory regulation and visceral hypersensitivity in NERD. The authors concluded that there is a bidirectional relationship between psychosocial factors and GERD, whereby psychological disorders may influence the perception of symptoms via the 'brain-gut' axis, whilst GERD symptoms themselves may contribute to the development of psychological distress. In a comprehensive review of current mechanisms underlying GERD, Zheng J. and Tao L. emphasise the key role of the microbiota-gut-brain axis and its involvement in the bidirectional interaction between psychological stress, immune activation, and motor and sensory dysregulation [44]. The authors describe how psychological factors and stress can influence oesophageal motor function and lower oesophageal sphincter pressure, increasing sensory sensitivity and sustaining inflammatory processes, which directly determine the clinical severity of symptoms. This study reinforces the argument regarding the neurosensory and immune mechanisms underpinning GERD symptoms and demonstrates that the biopsychosocial model is key to understanding both NERD and ERD.

In an editorial discussing experimental evidence, Cho YK (2017) analysed the role of acute stress and the activation of stress-related neuroendocrine mechanisms in the development of oesophageal hypersensitivity [45]. The authors reported that corticotropin-releasing hormone (CRH), which plays an important role in the stress response and is an activator of the hypothalamic-pituitary-adrenal axis, has the ability to modulate oesophageal function. Experimental research has demonstrated that CRH administration to healthy volunteers resulted in heightened sensitivity of the oesophagus to mechanical and electrical stimuli, modifications of oesophageal contractile activity and changes in the relaxation of the lower oesophageal sphincter. The authors noted that stress-induced changes in sensory processing may play a role in the development of oesophageal hypersensitivity, even if the acid exposure in normal was not high, in patients with PPI-refractory symptoms, reflux hypersensitivity and functional heartburn.

Hypervigilance relates to oesophageal sensations and is thought to be one of the factors leading to symptom development among patients with a wide variety of GERD manifestations, especially in GERD syndromes associated with increased oesophageal sensitivity. Guadagnoli et al. examined the prevalence of oesophageal hypervigilance in reflux symptom patients and the relationship between the prevalence and severity of the symptoms [46]. AET and SI were compared with the level of acid exposure hypervigilance assessed via the Esophageal Hypervigilance and Anxiety Scale (EHAS) in a retrospective cohort study of patients who had 96-hour pH monitoring (n = 123 for AET and n = 116 for SI). The authors observed that severity of acid exposure and/or symptom association with reflux events did not affect the level of oesophageal hypervigilance. Hypervigilance also independently predicted symptom severity, even when controlling for symptom-specific anxiety, suggesting that hypervigilance is a unique factor in symptom perception in the oesophagus.

There are bidirectional associations between sleep issues and GERD symptoms.Certain sleep issues can be related to GERD symptoms and vice versa. Tan et al. (2024), in a systematic review and meta-analysis of 22 observational studies, assessed the association between GERD and sleep disturbances [47]. The authors found that insomnia, sleep disturbances and insufficient sleep duration were associated with an increased risk of GERD (OR 2.02; 95% CI 1.64-2.49; OR 1.98; 95% CI 1.58-2.50; OR 2.66; 95% CI 2.02-3.15, respectively). Concurrently, GERD, as an indication of a bidirectional relationship, was found to have a positive relationship with sleep quality disturbances. Kurin et al. (2021) found that sleep disturbances are correlated with GERD, with about half of patients with GERD claiming to have symptoms of reflux during sleep, which could lead to poor sleep quality and sleep fragmentation [48]. Meanwhile, sleep loss could worsen GERD symptoms by altering sensory processing, oesophageal sensitivity and possibly also oesophageal reflux parameters. The authors highlight the bidirectional relationship between these two and highlight the importance of considering sleep disturbances in the complete management of patients with GERD.

Current knowledge of gastro-oesophageal reflux disease (GERD) has emerged as an understanding that it is not a single disease, but rather a spectrum of clinically and pathophysiologically different phenotypes. This has become possible in view of the increasing evidence suggesting that patients with comparable symptoms might have different mechanisms to explain their clinical symptoms, objective evidence of reflux and the responses to therapeutic interventions. Current consensus documents recommend using a thorough examination of symptoms, along with an evaluation of the long-term pH monitoring, the pH-impedance testing, and other functional diagnostic test results to assess the mechanism that produces symptoms in a particular patient, and for this reason GERD should be diagnosed using a comprehensive approach [14, 49, 50].

One of the main phenotypes is erosive reflux disease (ERD), which is characterised by the presence of endoscopically confirmed damage to the oesophageal mucosa associated with exposure to refluxate [17]. The main pathophysiological mechanism in ERD is prolonged contact of the mucosa with acidic and other aggressive components of reflux, leading to disruption of the epithelial barrier, the development of an inflammatory response and the formation of erosive lesions. Objective reflux-like findings are more common in these patients and the response to acid-suppression medications is more predictable. Concurrently, in patients with ERD, the extent of endoscopy changes does not always reflect the degree of clinical symptoms, thus supporting the importance of other neurosensory mechanisms in the clinical picture.

NERD is one of the most common phenotypes of GERD and is characterised by the presence of typical reflux symptoms in the absence of visible erosive changes to the oesophageal mucosa on endoscopic examination. The pathogenesis of NERD is more variable than ERD and can include pathological acid or non-acid reflux, increased mucosal permeability, local inflammatory changes and impaired sensory regulation. In patients with NERD, inadequate response to PPI drugs is more common, and is associated not only with the presence of persistent reflux, but also with the potential for visceral hypersensitivity mechanisms.

The patient's symptoms are definitely associated with reflux and acid exposure is within normal ranges, even though there is reflux hypersensitivity (a distinct pathophysiological phenotype) [38]. The mechanisms that are commonly involved when this occurs are increased sensitivity of the oesophageal receptors, peripheral and central sensitisation, and the processing of the signals of the viscera in the central nervous system. As for the perception of reflux, current understanding considers reflux hypersensitivity as a condition bordering GERD and functional oesophageal disorders, clinical manifestations of which are not only due to reflux, but also to the perception of it.

Functional heartburn is defined as a functional oesophageal disorder, and is defined by symptoms of heartburn in the absence of pathological reflux, no relationship between reflux episodes and symptoms, and no structural changes of the oesophagus [51]. These patients have two main mechanisms: the first is related to sensory processing and the second is an over-sensitisation and dysregulation of the ‘brain-gut' interaction.

Functional heartburn is often associated with other ‘gut-brain’ interaction disorders and psycho-emotional disturbances, which necessitates a comprehensive approach to treatment.

The classification of gastro-oesophageal reflux disease into ERD, NERD, reflux hypersensitivity and functional heartburn reflects the current shift from a simplified acid-dependent model to the concept of a heterogeneous disease with different mechanisms underlying symptom development. A phenotype-oriented approach allows the integration of endoscopic findings, functional diagnostic results and the assessment of neurosensory factors, thereby laying the foundation for personalised medicine in GERD.

Conclusions. GERD is a heterogeneous, multifactorial condition, the pathogenesis of which involves abnormalities in the anti-reflux barrier, oesophageal motility, mucosal resistance, the properties of the refluxate, and sensory signal processing. The current concept of GERD goes beyond the acid-dependent model and takes into account the role of visceral hypersensitivity, ‘brain-gut’ axis dysfunction, psychosocial factors and modified risk factors. The clinical heterogeneity of the condition, characterised by erosive and non-erosive forms, reflux hypersensitivity and functional heartburn, necessitates a differentiated approach to diagnosis and treatment. A personalised management strategy for patients with GERD, based on a comprehensive assessment of clinical, endoscopic and functional data, enables the optimisation of treatment and an improvement in patients’ quality of life.

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Chernets Tetiana Yuriivna – conception and design of the study, formulation of the aim, analysis of scientific literature, final revision and approval of the manuscript;

Kucher Svitlana Viktorivna – literature search and collection of scientific materials, analysis and interpretation of the obtained data, preparation and editing of the manuscript, review and approval of the final version of the manuscript;

Ruda Mariia Myronivna – collection and systematisation of scientific sources, analysis of literature data, participation in preparation of the manuscript;

Kavetska Nataliia Anatoliivna – analysis of current scientific evidence, critical revision of the manuscript, contribution to the discussion and conclusions;

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