DOI: https://doi.org/10.30841/2786-720X.4.2025.349491
UDC 616.329-002-008.6:616.333:616.12-009.7/.72]-06-07-053.9:614.253.1/2-051:316.36

Problems and opportunities of diagnosing reflux-induced cardialgia in the elderly at the health care primary level

N. D. Chukhrienko
Dnipro State Medical University

There are data suggesting that 20–40% of the global population exhibits symptoms representative of gastroesophageal reflux disease (GERD). GERD is more commonly diagnosed in mature, elderly, and senile individuals, accounting for approximately 23% among this group. The presence of comorbidities, the rate of which is increased with age, in combination with atypical extraesophageal GERD manifestations, particularly, cardialgia, form additional challenges for timely diagnosis of coronary heart disease (CHD) and management of patients, especially at the primary care level.
The objective: **to study the causes and manifestations of cardialgia in elderly patients which are associated with GERD and to provide general practitioners with recommendations regarding medical management for this category of patients.**
Materials and methods**. A study was conducted on 69 patients, in whom the indicators of gastric acid-forming function, indicators of basal gastric secretion, the presence of comorbid pathology and reflux score were studied. The features of electrocardiogram (ECG) changes depending on the number of refluxes were determined.**
Results**. In 20 patients (37%), no significant number (above normal) of gastroesophageal reflux (GER) episodes were detected. However, episodes of ST-segment depression on ECG were more than 2 mm in depth and lasted from 5 to 83 minutes. This allowed us to associate the pain in these patients with the presence of CHD. In 19 patients (33%), a significant number of GER episodes (> 50) was recorded, while no changes were observed on Holter ECG monitoring, which allowed us to associate this pain with extraesophageal non-cardiac manifestations of GERD. In 6 (11%) patients, both a significant number of reflux episodes (> 50) and ischemic changes on ECG were found. During the study, episodes of ST-segment depression occurred 2 to 9 times per day in these patients. In 2 of these patients, the clinical picture was similar, but the reflux episodes and ECG changes were not temporally correlated. In 3 other patients, an increased rate of GER was observed 10–15 minutes prior to ECG changes, in one of these cases, supraventricular extrasystoles occurred during the GER spike. In 10 (19%) patients, despite having complaints, no abnormalities were detected during simultaneous monitoring.**
Thus, the method of simultaneous 24-hour pH and ECG monitoring used in this study expands the possibilities for understanding the causes, conditions, and nature of cardialgia. In some patients, it allows for differential diagnosis between GERD and CHD, as well as the recognition of their combined presence. There is a reason to believe that in some cases, GER in GERD acts as a trigger mechanism for manifestations in the form of pain similar to cardialgia, which can be assessed as angina attacks. Given the diagnostic difficulties of differential diagnosis of cardialgia in the elderly, an algorithm of actions for a doctor at the primary level of medical care has been developed.
Conclusions**. GERD, defined as a chronic, recurrent condition which is characterized by spontaneous or regularly repeated reflux of gastric and duodenal contents into the esophagus, combined with cardialgia, poses specific challenges for primary care physicians in terms of differential diagnosis. In some elderly patients, the coexistence of GERD and CHD is highly probable, even in the absence of pronounced pain, and this must be taken into account when prescribing antianginal medications that contribute to the relaxation of the lower esophageal sphincter.**
Timely diagnosis and treatment, as well as adherence to lifestyle recommendations, improve the prognosis of GERD and reduce its impact on the development of cardialgia.
In addition to treating GERD with proton pump inhibitors and H_2-receptor antagonists, depending on the severity (classified as A, B, C or D), patients should be advised to follow simple lifestyle measures, such as: elevating the head of the bed (in cases of nighttime symptoms); reducing body mass index (obesity plays a key role in the development of refractory GERD). In the presence of GERD there is a risk of developing Barrett's esophagus, adenocarcinoma, strictures and dysphagia, which should be taken into account by the general practitioner – family medicine during long-term monitoring of the patient.
Keywords: *reflux-induced cardialgia, comorbidity, differential diagnosis, chest pain, advanced age, primary care level.*

Проблеми та можливості діагностики рефлюкс-індукованої кардіалгії в осіб похилого віку на первинному рівні медичної допомоги

Н. Д. Чухрієнко

Існують дані, що 20–40% населення у світі мають симптоми, характерні для гастроезофагеальної рефлюксної хвороби (ГЕРХ). ГЕРХ діагностується частіше в зрілому, похилому та старечому віці й становить серед цієї категорії населення приблизно 23%. Наявність коморбідних захворювань, кількість яких збільшується з віком, у поєднанні з нетиповими позазравохідними проявами ГЕРХ, а саме кардіалгією, створює додаткові труднощі у своєчасній діагностиці ішемічної хвороби серця (ІХС) і веденні хворих, особливо на первинному рівні медичної допомоги.

Мета дослідження: вивчення причин і проявів кардіалгії у пацієнтів похилого віку, пов'язаних із ГЕРХ, і надання лікарям загальної практики рекомендацій щодо тактики лікування цієї категорії хворих.
Матеріали та методи. Проведено дослідження 69 пацієнтів, в яких вивчали показники кислотоутворювальної функції шлунка, базальної шлункової секреції, наявність коморбідної патології та оцінку рефлюксу в балах. Визначали особливості змін на електрокардіограмі (ЕКГ) залежно від кількості рефлюксів.
Результати. У 20 (37%) хворих не виявлено значущої (більше норми) кількості шлунково-стравохідних рефлюксів (ШСР), причому зазначені епізоди депресії сегмента ST на ЕКГ були більш ніж 2 мм, тривалістю 5–83 хв. Це дозволило асоціювати біль у цих пацієнтів із наявністю ІХС. У 19 (33%) пацієнтів виявлено як значущу кількість (> 50) ШСР, так і відсутність змін при холтерівському ЕКГ-моніторуванні, що дозволило асоціювати цей біль із позастравохідними некардіальними проявами ГЕРХ. У 6 (11%) хворих виявлено як значущу кількість ШСР (> 50), так і ішемічні зміни на ЕКГ. У цих пацієнтів під час дослідження епізоди депресії сегмента ST відзначалися від 2 до 9 разів на добу. У 2 хворих цієї групи при однотипній клінічній картині реєструвалися епізоди збільшення кількості ШСР та зміни на ЕКГ, не пов'язані один з одним у часі. ІЩе у 3 хворих відзначалися епізоди збільшення частоти ШСР, що передували на 10–15 хв змінам на ЕКГ, а в 1 із цих випадків виникали надшлуночкові екстрасистоли під час збільшення частоти ШСР. У 10 (19%) пацієнтів, попри наявність скарг, зміни при паралельному моніторуванні не виявлялися.
Отже, використаний у дослідженні метод одночасного 24-годинного pH- та ЕКГ-моніторування розширює можливості щодо розуміння причин, умов виникнення і характеру кардіалгій. У частини пацієнтів можливе проведення диференційної діагностики між ГЕРХ та ІХС, а також констатація їх поєднання. Є підстави вважати, що іноді ШСР при ГЕРХ є пусковим механізмом проявів у вигляді болю, подібних до кардіалгії, що може бути оцінено як напади стенокардії. Зважаючи на діагностичні труднощі диференційної діагностики кардіалгії в осіб похилого віку, розроблено алгоритм дій лікаря на первинному рівні медичної допомоги.
Висновки. ГЕРХ визначається як хронічний рецидивуючий процес, що характеризується спонтанними або регулярно повторюваними епізодами закидання у стравохід шлункового і дуоденального вмісту поєднано з кардіалгією, що створює певні труднощі в диференційній діагностиці для лікаря первинної ланки. У деяких пацієнтів похилого віку найбільш імовірним є поєднання ГЕРХ і ІХС, навіть за відсутності вираженого больового синдрому, що слід враховувати при призначенні антиангінальних препаратів, які призводять до розслаблення нижнього сфінктера стравоходу.
Своєчасна діагностика і лікування, а також дотримання рекомендацій щодо способу життя роблять прогноз ГЕРХ більш сприятливим, тим самим зменшуючи вплив ГЕРХ на розвиток кардіалгії.
Крім лікування ГЕРХ інгібіторами протонної помпи й антагоністами H_2-рецепторів залежно від ступеня тяжкості (класи A, B, C, D), слід рекомендувати хворим дотримуватися простих правил: підняття узголів'я ліжка (при нічних симптомах); зменшення індексу маси тіла (ожиріння відіграє особливу роль у розвитку рефрактерної ГЕРХ). При наявності ГЕРХ існує ризик розвитку стравоходу Барретта, аденокарциноми, стриктури та дисфагії – що потрібно враховувати лікарю загальної практики – сімейної медицини при довготривалому спостереженні за хворим.
Ключові слова: *рефлюкс-індукована кардіалгія, коморбідність, диференційна діагностика, біль у грудях, похилий вік, первинний рівень.*

Problems and possibilities of diagnosing reflux-induced cardialgia in elderly people at the primary care level. There is data suggesting that 20–40% of the global population exhibits symptoms representative of gastroesophageal reflux disease (GERD). GERD is more commonly seen in mature, elderly, and senile individuals, accounting for approximately 23% among this group. The presence of comorbidities, which increase with age, in combination with atypical extraesophageal GERD manifestations – particularly cardialgia – creates additional challenges for timely diagnosis of coronary heart disease (CHD) and management of patients, especially at the primary care level. A study of 69 patients was conducted, in which the indicators of gastric acid-forming function, indicators of basal gastric secretion, the presence of comorbid pathology and reflux score were studied. The features of electrocardiogram (ECG) changes depending on the number of refluxes were determined. Given the diagnostic difficulties of differential diagnosis of cardialgia in the elderly, an algorithm of actions for a doctor at the primary level was developed.

Problem Statement

GERD is a chronic condition with a prolonged course that significantly deteriorates a patient's quality of life [3, 4, 8]. Unfortunately, both patients and primary care physicians often underestimate the importance of this disease.

There is evidence that 20–40% of the global population experience symptoms typical of GERD. In industrialized countries, 10–20% of people suffer from GERD symptoms [8, 19, 22].

Analysis of Recent Research and Publications

Over the past 10 years, the incidence of GERD has tripled, with an annual increase of approximately 5%. It has been proven that heartburn and regurgitation, which are clinical signs of GERD, lead to a fivefold increase in the risk of developing esophageal cancer. This risk increases fourfold in individuals with symptoms persisting for more than 20 years [16].

Thus, GERD occurs more frequently and carries more significant negative consequences in middle-aged, elderly, and senile individuals, and is identified in 23% of surveyed patients in this category.

With age, not only does the frequency and severity of reflux increase, but also the presence of atypical, including extraesophageal, manifestations. It is important to consider the fact that significant cases of GERD may have latent or minimally symptomatic presentations [3, 28].

GERD is defined as a chronic relapsing condition caused by impaired motor and evacuation function of the gastroesophageal zone, characterized by spontaneous or regularly recurring reflux of gastric and duodenal contents [3, 8, 22].

This is especially relevant for elderly patients, who often self-medicate and are unaware that heartburn and the associated pain are not merely unpleasant sensations, but may lead to or reflect other diseases, including CHD [12]. With age, the number of patients with comorbidities increases. The presence of comorbidity complicates both the

diagnosis and treatment of these patients [9, 14, 28].

Our study has focused on cardialgia, which may be induced by GERD, but may also represent independent CHD that presents under the mask of GERD.

The international GERD consensus group in the 2006 Montreal Consensus formulated an expert opinion that the diagnosis of GERD can be established without additional examinations, based on characteristic clinical symptoms (both esophageal and extraesophageal) [26]. This allows the primary care physician to reasonably suspect GERD and its associated complications and clinical challenges. Testing may be unnecessary in young patients with a short disease history [3].

According to the Montreal definition, GERD is a condition that develops when the reflux of stomach contents causes troublesome symptoms and/or complications. In real life, patients describe it as heartburn, acid regurgitation, or a sensation of a "lump in the throat" [4, 8, 26].

In a number of elderly patients, GERD may have a subtle course, with mild, atypical, or purely extraesophageal symptoms – a situation seen in up to 25% of cases – making diagnosis even more difficult [1, 24].

We would like to draw the attention of primary care physicians to the specific challenges in diagnosing and treating GERD that intensify with age and to highlight that in elderly individuals, the disease may manifest solely through extraesophageal symptoms [23].

The objective: to study the causes and manifestations of cardialgia in elderly patients associated with GERD and to provide general practitioners with recommendations regarding medical management for this category of patients.

Main Data

Given the conflicting data regarding the role of GERD in the development of cardialgia, we set out to detail the characteristics of the cardialgic syndrome in patients with an established diagnosis of CHD, identify possible manifestations of reflux disease, and trace the relationship through simultaneous parallel investigations using 24-hour pH monitoring and 24-hour (Holter) ECG monitoring.

For a family doctor, as the first point of contact, it is especially important to be attentive to retrosternal pain and to exercise caution when associating it solely with esophageal pathology, particularly in elderly individuals. Esophageal-related pain typically presents as a sensation of pressure behind the sternum in its middle third, which closely resembles cardiac pain. In such cases, reflux can act as a trigger, provoking changes in cardiac function.

Differential diagnostic challenges also arise because these sensations may radiate to the interscapular region, arms, or jaw. Esophageal distension or chemical stimulation by acid can be perceived as pain due to reflex mechanisms. The literature describes a wide spectrum of extraesophageal symptoms of GERD [12], but we would like to focus attention on those that may either mimic or truly be associated with heart disease [1, 13, 18, 21]. These include not only cough, which usually worsens in the evening or at night, but also episodes of dyspnea with a spasmodic component, which, in the presence of other clinical signs (e.g., elevated blood pressure, edema), may be interpreted as chronic heart failure. There may also be arrhythmias, perceived by patients as irregular heartbeats, and chest pain that worsens after eating or overeating – on which we have placed special emphasis in our study.

According to several authors, pain syndrome in GERD ranks second after heartburn [11, 14]. It is a very common symptom, referred to as odynophagia (pain during the passage of food through the esophagus due to mucosal injury). Such complaints often lead the physician to suspect a coronary origin of the symptom. Quite frequently, the pain resembles angina pectoris. It often appears behind the sternum, radiates to the neck, shoulder, or back, or is localized in the precordial area [4, 5].

To date, the etiology of esophageal pain has not been fully established. It is believed that esophageal spasm or motility disorders may be involved [4, 26]. Pain almost never occurs in the absence of esophageal contractions, which are triggered by the reflux of acidic contents, and it is this reflux that provokes the pain syndrome in the cardiac region. It cannot be excluded that inflammation of the esophageal mucosa, caused by gastric content reflux, may itself be a contributing factor to chest pain of coronary character. Or perhaps it acts as a trigger for reflux-induced angina, superimposing on pre-existing CHD in elderly patients?

It is important to consider that the function of the esophagus and stomach is largely regulated by the vagus nerve, which also plays a key role in the regulation of other organs. Irritation of vagal nerve fibers can lead to symptoms in many patients that, at first glance, seem unrelated to reflux esophagitis.

Pain, including in the heart region, arises partly because the heart and esophagus share the same neural plexus [11, 14]. Stimulation of the reflex arc, which includes both afferent and efferent gastric pathways, can lead to coronary artery spasm.

This issue has been highlighted in the works of both international and domestic researchers [1, 3–5, 8, 14]. The connection between reflux and chest pain is clearly illustrated by synchronous recordings of pH monitoring and the onset of pain symptoms in the heart area (Figure).

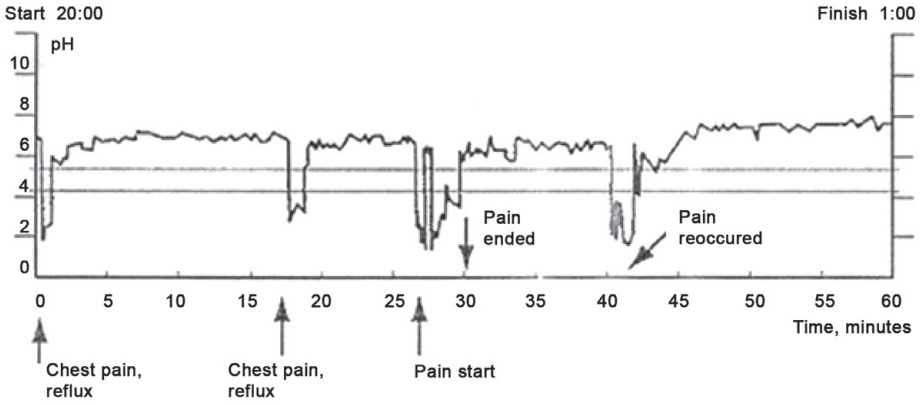
This raises important questions: Could this pain indeed be of coronary origin as a result of reflex influences? How often does it arise from such mechanisms?

The problem becomes even more complex in elderly patients, whose pain threshold is higher compared to younger individuals. This may be due to an age-related reduction in the number of myocentric neurons. As a result, older patients often seek medical attention at later stages, when the disease has already progressed significantly. Could it be that GERD acts as a trigger for a cascade of pathological processes that initiate destabilization of coronary blood flow?

The issue becomes particularly relevant given that many patients with verified CHD and clinical signs of angina, along with other comorbid conditions, have been taking nitrates or their analogs, NSAIDs (Non-Steroidal Anti-Inflammatory Drugs), antihypertensives, and calcium channel blockers over a long period of time. These drugs reduce the tone of the lower esophageal sphincter, thereby creating favorable conditions for the development and progression of GERD and its complications [4, 5, 14].

On the other hand, reflux disease is often masked as angina pectoris, presenting as burning retrosternal pain [12], and the prescription of nitrates further reinforces the vicious cycle of cardialgia progression.

FAMILY MEDICINE. EUROPEAN PRACTICES / СІМЕЙНА МЕДИЦИНА. ЄВРОПЕЙСЬКІ ПРАКТИКИ •
ISSN 2786-7218 (Online) | ISSN 2786-720X (Print)
№4 (114)/2025



Episodes of recurrent retrosternal pain correlate with episodes of reflux with pH < 4 (V. D. Pasechnikov, 2000)

One of the GERD reasons difficult to detect is impaired neural regulation of the lower esophageal sphincter. Studies have shown that in some patients, there is an insufficient number of nerve endings in this part of the esophagus responsible for triggering contraction of the muscular sphincter. In GERD, its tone is reduced, creating conditions for repeated mucosal damage due to gastroesophageal reflux [3, 6, 8].

It can be assumed that the prevalence of GERD in the elderly increases due to these pathophysiological and anatomical changes characteristic of aging, including homeostatic dysfunctions in various organs and tissues. With age, there is a reduction in saliva production. Saliva plays a physiological role in neutralizing refluxate, but with aging, the bicarbonate content decreases and other biochemical changes occur. Considering that saliva neutralizes the refluxate, this increases the risk of GERD [3, 6, 8]. Interesting studies have shown that in patients with comorbid GERD and thyroid pathology [17], particularly with involutional changes in the thyroid gland, there is a reduction in antioxidant activity, impairment of mitochondrial function, and reduced control over toxic radicals [16, 28].

Recently, scientists have been paying attention to the peculiarities of the course of GERD in hypothyroidism. The number of cases increases with age, which contributes to the development and progression of metabolic disorders [18] and presumably affects the development of cardiovascular changes. Against the background of reduced thyroid function, the level of atherogenic lipids increases with the progression of the severity of hypothyroidism.

In addition, age-related factors arise that promote the formation of GERD, such as increased body weight, which leads to elevated intra-abdominal pressure.

Moreover, evidence suggests that in obesity [11], there is an increased concentration of glycocholic acid (GCA) in gastric aspirate. This is associated with a more severe course of esophagitis, especially when GCA is in its lipophilic form under acidic pH conditions.

With age, there is a decline in esophageal motility and reduction in the tone of the lower esophageal sphincter. The incidence of hiatal hernia increases to up to 50%, resulting in a weakened epithelial defense of the esophagus. The risk of microcirculatory disturbances also increases, leading to shifts in tissue acid-base balance and compromised epithelial integrity – which is a critical protective factor of the esophagus against the aggressive effects of gastric reflux [4, 5, 27].

Thus, there is evidence that GERD can be both a consequence of pathological changes in the human body and a triggering mechanism for a cascade of pathological processes that initiate destabilization of coronary blood flow, leading to myocardial ischemia that clinically resembles an angina attack. In such cases, ECG changes may occur, such as ST segment depression, T-wave inversion, and manifestations of arrhythmias. On the other hand, GERD is considered to be the main cause of non-cardiac chest pain [8, 14].

Given the conflicting data regarding the role of GERD in the development of cardialgia and its interpretation, we conducted a study in accordance with the stated objective to identify the relationship between GERD manifestations and cardialgia.

MATERIALS AND METHODS

We have examined 69 patients with clinical and subjective manifestations of GERD, who were selected using questionnaires and the GerdQ survey tool [10, 19, 22]. The study included 41 men and 28 women, aged from 25 to 85 years. All patients were divided into three groups:

- Group A – young adults (25–40 years): 20 patients;
- Group B – elderly patients (60–74 years): 32 patients;
- Group C – senile patients (75 years and older): 17 patients.

A comparative analysis was conducted with a group of practically healthy individuals. These healthy individuals were selected based on preliminary general clinical and endoscopic examinations, which excluded diseases of the digestive organs and other systems.

The characteristics of gastric acid-producing function (GAPF) in basal conditions were assessed in accordance with the functional pH intervals (F1-pH) proposed by V. M. Chornobrovyi [2], as follows:

- pH 7.0–7.5 – achlorhydria;
- pH 3.6–6.9 – pronounced hypochlorhydria;
- pH 2.3–3.5 – moderate hypochlorhydria;
- pH 1.6–2.2 – normochlorhydria;
- pH 1.3–1.5 – moderate hyperchlorhydria;
- pH 0.9–1.2 – pronounced hyperchlorhydria.

RESULTS AND DISCUSSION

The results of GAPF assessment in GERD patients are presented in Table 1, and in practically healthy individuals – in Table 2.

During the analysis, it was found that in patients of the Group A with GERD during exacerbation, there was an increase in GAPF – hyperchlorhydria was observed in 14 patients (70%). In the Group B, enhanced GAPF and hyperchlorhydria were found in 19 patients (59.4%), while hypochlorhydria was observed in 6 patients (28.7%). In the Group C, a clear decline in GAPF was recorded – hypochlorhydria was present in 11 patients (58.8%), and hyperchlorhydria in only 3 patients (17.7%), which can be explained by the age-related atrophic changes in the gastric mucosa. Normochlorhydria, with minor variations, was noted in all three groups and accounted for 17 patients (24.6%) overall.

Among practically healthy individuals across all age groups (Table 2), baseline gastric secretion parameters were mainly characterized by predominance of normochlorhydria GAPF – 7 individuals (70%) in the Group A, 13 (65%) in the Group B, and 6 (60%) in the Group C.

Hyperchlorhydria GAPF was observed in 3 individuals (30%) in the Group A and 3 individuals (15%) in the Group B. In individuals aged 75 and older, there was a tendency toward hypochlorhydria – 4 individuals (40%), which confirms the progression of atrophic changes in the gastric mucosa with age, and as a result, a decline in hydrochloric acid production.

Thus, during GERD exacerbation, patients in the Groups A and B demonstrated increased GAPF in basal conditions, with predominant F1-pH values of 4.5 – 70% and 59.4%, respectively. In patients of the Group C, decreased GAPF was noted, with predominant F1-pH values of 1.2 – 58.8%, due to age-related atrophic changes in the mucosa. These findings should be taken into account when prescribing differentiated pharmacotherapy, especially acid-suppressive medications, in order to prevent potential complications and recurrences of duodenal ulcer disease.

It is known that with age, pathological conditions and diseases increase affecting the development and prognosis of the cardiovascular continuum. In 50 examined individuals, comorbid conditions were identified (Table 3).

An increase in body weight with age in patients with dyslipidemia is noteworthy.

To confirm GERD, the GAPF was assessed using an express method of computer-based intraesophageal pH-metry along the gastric canal using the acidogastrometer AGID-01, according to the method of V. M. Chornobrovyi [2].

After excluding 19 patients, 50 patients remained in the analysis. The severity of reflux was evaluated in points, based on which all patients were divided into three groups: Group 1 – patients with a minimal degree of reflux (less than 20 points): 25 patients with CHD (50% of all examined); Group 2 – patients with a moderate degree of reflux (20–30 points): 19 patients (38%); Group 3 – patients with severe reflux symptoms (more than 30 points): 6 patients (12%).

Table 1

Distribution of Examined GERD Patients by Baseline Gastric Secretion Parameters According to Functional pH Intervals

Study Group	Functional Intervals				
	Pronounced hypochlorhydria, n (%)	Moderate hypochlorhydria, n (%)	Moderate hypochlorhydria, n (%)	Moderate hyperchlorhydria, n (%)	Pronounced hyperchlorhydria, n (%)
A (n = 20)	–	–	6 (30)	8 (40)	6 (30)
B (n = 32)	2 (6.2)	4 (12.5)	7 (21.9)	18 (56.3)	1 (3.1)
C (n = 17)	4 (23.5)	7 (41.2)	4 (23.5)	2 (11.8)	–

Table 2

Distribution of Examined Practically Healthy Individuals by Baseline Gastric Secretion Parameters According to Functional pH Intervals

Study Group	Functional Intervals				
	Pronounced hypochlorhydria, n (%)	Moderate hypochlorhydria, n (%)	Moderate hypochlorhydria, n (%)	Moderate hyperchlorhydria, n (%)	Pronounced hyperchlorhydria, n (%)
A (n = 10)	–	–	7 (70)	3 (30)	–
B (n = 20)	–	4 (20)	13 (65)	3 (15)	–
C (n = 10)	1 (10)	3 (30)	6 (60)	–	–

Table 3

Comorbid Conditions

Parameters		Group 1 (n = 25)	Group 2 (n = 19)	Group 3 (n = 6)
Gender, n (%)	Men Women	18 (71.18) 7 (28.82)	10 (52.6) 9 (47.4)	4 (73.7) 2 (26.3)
Comorbidity, n (%)	Type 2 Diabetes Mellitus Peptic Ulcer Disease	– 4 (15)	– –	4 (69) 2 (30)
Dietary Characteristics, n (%)	Habit of Late-Night Overeating	3 (10)	18 (68)	5 (87)
	Weight Gain	3 (10)	18 (68)	5 (87)
	Dyslipidemia	12 (42)	9 (48)	4 (70)

FAMILY MEDICINE. EUROPEAN PRACTICES / СІМЕЙНА МЕДИЦИНА. ЄВРОПЕЙСЬКІ ПРАКТИКИ • ISSN 2786-7218 (Online) | ISSN 2786-720X (Print) №4 (114)/2025

To analyze the ECG manifestations of CHD and its complications, ECG data of patients from different groups were reviewed.

In 66.6% of patients in Group 3, scarring changes were observed on ECG, whereas in Group 1, similar changes were present in only 32% of patients ($p < 0.05$). These identified ECG features logically suggest a more intensive antianginal treatment regimen in Group 3, due to pronounced coronary changes and a history of myocardial infarction.

Among patients in this group, 25% continuously received calcium channel blockers as baseline therapy, 25% were treated with nitrates, 35% received a combination of both, and 15% were on beta-blockers.

In Group 1 patients, the intensity and duration of antianginal therapy with nitrates and calcium channel blockers were significantly lower. The duration and frequency of beta-blocker use did not differ significantly. Thus, it was concluded that long-term use of nitrates and calcium channel blockers in patients with CHD combined with GERD – as factors that reduce closure of the lower esophageal sphincter – may aggravate clinical manifestations of gastroesophageal reflux. Given the shared innervation and functional unity of the body, this may be perceived as cardialgia, or may actually lead to coronary artery spasm, myocardial ischemia, arrhythmias, and ultimately a decrease in quality of life in this patient group.

We also have noted that in some cases, pain disappeared after taking nitroglycerin, while in others, it intensified. Some patients reported increased discomfort and retrosternal pain after meals, while others noted symptom relief. All patients kept a detailed diary documenting all unpleasant sensations, and the results were analyzed synchronously.

It should be noted that functional esophageal spasm may also present with retrosternal pain. However, this pain is not always associated with food intake and is often relieved by nitroglycerin, which further complicates differential diagnosis [7, 8, 14, 21].

A characteristic feature may be the combination of pain with dysphagia, which may also be functional in nature. Dysphagia can occur when ingesting liquid food and may be worsened by cold meals.

The standard technique was used to perform pH monitoring [2]. Electrodes of the acidogastrometer were placed as follows: the first in the esophagus, 2.5–3 cm above the lower esophageal sphincter; the second in the cardia; and the third in the body of the stomach.

The Holter monitoring system was installed using a standard technique with V4, V6, and aVF leads. A total of 54 patients were examined – 21 men (39%) and 33 women (61%), aged from 21 to 73 years. These patients periodically experienced pain both at rest and during physical exertion, and retrosternal heartburn was reported by the majority. In some patients, there was no consistent response to nitroglycerin: in some, the pain resolved, while in others, it worsened.

In addition to 24-hour pH monitoring and Holter ECG monitoring, 47 patients underwent esophagogastroduodenoscopy (EGD). In 41 of them (87%), cardiac insufficiency and hiatal hernia were detected.

Some patients reported increased discomfort and retrosternal pain after eating, while others noted a reduction of symptoms. All patients maintained a detailed diary documenting all unpleasant sensations, and the results were analyzed synchronously using a personal computer.

The following results were obtained from the conducted studies.

In 20 patients (37%), no significant number (above normal) of gastroesophageal reflux episodes were detected. However, episodes of ST-segment depression on ECG were observed to exceed 2 mm in depth and lasted from 5 to 83 minutes. This allowed the pain in these patients to be attributed to CHD.

In 19 patients (33%), a significant number of gastroesophageal reflux episodes (> 50) was recorded, while no changes were observed on Holter ECG monitoring. These pain episodes were therefore considered non-cardiac, extraesophageal manifestations of GERD.

In 6 patients (11%), both a significant number of reflux episodes (> 50) and ischemic changes on ECG were found. During the study, episodes of ST-segment depression occurred 2 to 9 times per day. In 2 of these patients, the clinical picture was similar, but the reflux episodes and ECG changes were not temporally correlated. In 3 other patients, an increase in reflux frequency was observed 10–15 minutes prior to ECG changes; in one of these cases, supraventricular extrasystoles occurred during the reflux spike.

In 10 patients (19%), despite having complaints, no abnormalities were detected during simultaneous monitoring.

Thus, the method of simultaneous 24-hour pH and ECG monitoring used in this study expands the possibilities for understanding the causes, conditions, and nature of cardialgia. In some patients, it allows for differential diagnosis between GERD and CHD, as well as the recognition of their combined presence.

There is reason to believe that in some cases, GERD-related reflux acts as a trigger for pain symptoms resembling cardialgia, which may be clinically misinterpreted as angina pectoris attacks.

The pathogenesis of GERD is a multifactorial process and is characterized not only by heartburn, pain, and arrhythmias, but also by various manifestations and changes depending on the clinical form of the reflux. At the same time, functional esophageal spasm can also present with pain. In such cases, pain is relieved after taking antacids, undergoing a course of proton pump inhibitors (PPIs), and adhering to specific behavioral and dietary restrictions. Pain syndrome in GERD is of particular importance in terms of diagnosis and treatment, as it can trigger a chain reaction leading to coronary artery spasm and ischemia. In other words, in certain cases, it becomes a trigger for the development of CHD in elderly patients who already have existing coronary vessel changes, diabetes mellitus, or dyslipidemia-requiring personalized and precise actions from the physician.

In some instances, pain is not always related to food intake and may be relieved by nitrates or their analogs, which further complicates differential diagnosis [7, 8, 14, 15] and the selection of clinical management strategies, especially in elderly patients with comorbidities at the primary care level [20, 25].

To help solve these complex challenges in managing GERD patients, we have developed a diagnostic algorithm for use by primary care physicians (Addition).

CONCLUSIONS

1. GERD, defined as a chronic, recurrent condition characterized by spontaneous or regularly repeated reflux of gastric and duodenal contents into the esophagus, combined with cardialgia, poses specific challenges for primary care physicians in terms of differential diagnosis.

2. In some elderly patients, the coexistence of GERD and CHD is highly probable, even in the absence of pronounced pain, and this must be taken into account when prescribing antianginal medications that contribute to the relaxation of the lower esophageal sphincter.

3. Timely diagnosis and treatment, as well as adherence to lifestyle recommendations, improve the prognosis of GERD and reduce its impact on the development of cardialgia.

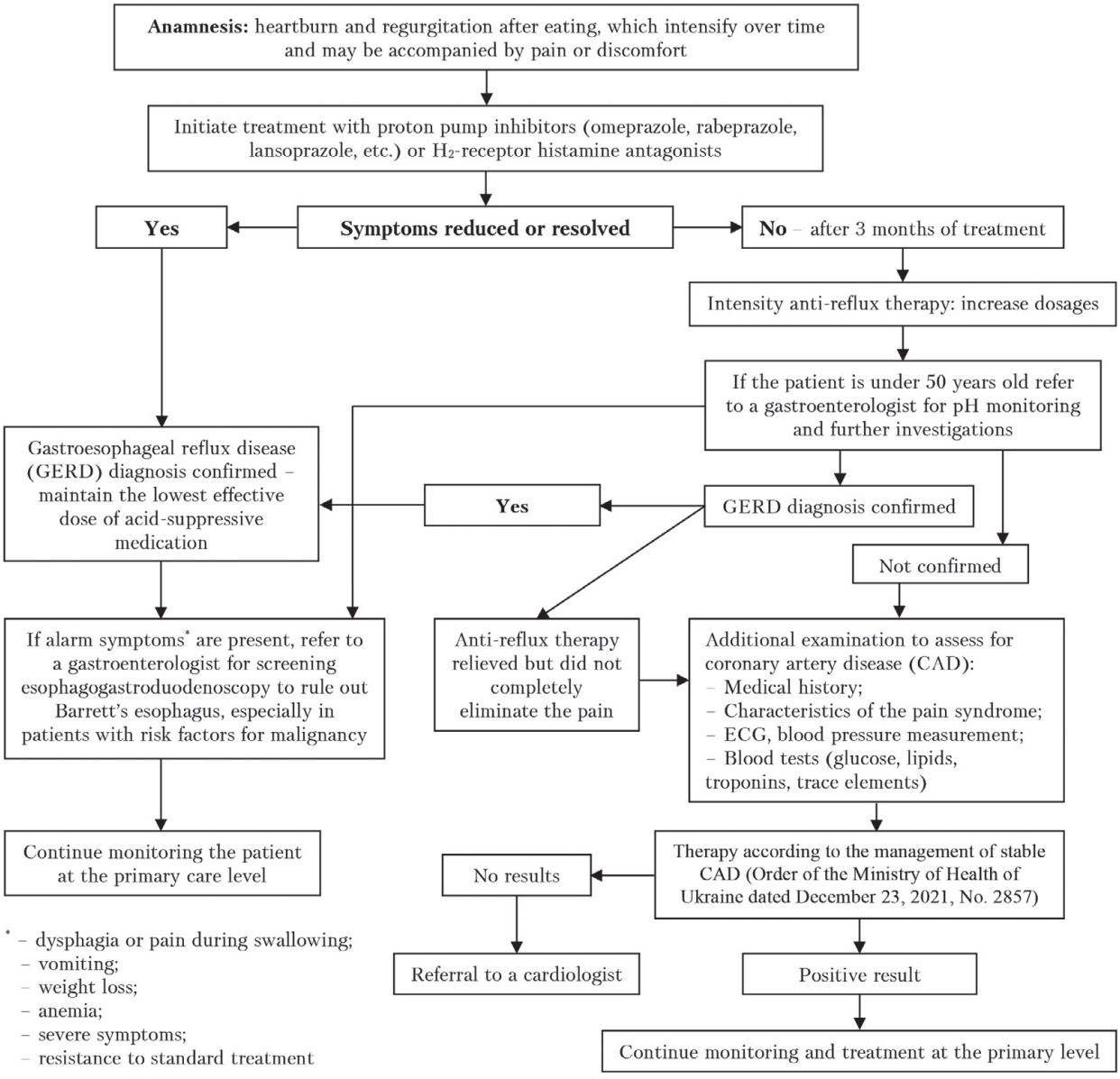
4. It is important to consider that long-term use of PPIs may lead to malabsorption of calcium, magnesium, vitamin B_{12}, and iron, especially in elderly patients. It should also be noted that PPIs can reduce the effectiveness of antithrombotic drugs.

5. In addition to treating GERD with PPIs and H_2-receptor antagonists, depending on the severity (classified as A, B, C or D), patients should be advised to follow simple lifestyle measures, such as: elevating the head of the bed (in cases of nighttime symptoms); reducing body mass index, as obesity plays a key role in the development of refractory GERD; avoiding meals before bedtime and foods that trigger reflux; re-evaluating the need for certain medications (such as nitrates, calcium channel blockers, and anticholinergic agents).

6. It is also important for family doctors to be aware that long-standing GERD may lead to complications such as Barrett's esophagus, adenocarcinoma, strictures, and dysphagia, which should be considered during long-term patient follow-up.

Addition

General Algorithm for the Diagnosis and Treatment of Patients with GERD Manifestations at the Primary Care Level



FAMILY MEDICINE. EUROPEAN PRACTICES / СІМЕЙНА МЕДИЦИНА. ЄВРОПЕЙСЬКІ ПРАКТИКИ • ISSN 2786-7218 (Online) | ISSN 2786-720X (Print) №4 (114)/2025

Information about the author

Chukhrienko Neonilla D. – Dnipro State Medical University, tel.: (050) 652-03-77. *E-mail: neonilla.ch15@gmail.com*
ORCID: 0000-0003-2101-3882

Відомості про автора

Чухрієнко Неонілла Дмитрівна – Дніпровський державний медичний університет; тел.: (050) 652-03-77. *E-mail: neonilla.ch15@gmail.com*
ORCID: 0000-0003-2101-3882

REFERENCES

1. Derbak MA, Tovtyn R-MI, Hanych OT, Kurakh AV, Hodanych LI, Keshten W. Gastroesophageal reflux disease in patients with ischemic heart disease of different body weights. In: Material Int Interdisciplinary Sci Pract Conf "Modern aspects of preserving human health". Uzhhorod; 12–13 April 2024. Uzhgorod; 2024, p. 161-3.
2. Budzak IYa, Hrytsenko VI, Hrytsenko II, et al. Intraluminal pH-metry of the gastrointestinal tract: Practical manual. In: Chornobrovyi VM, editor. Vinnytsia: Logos; 1999. 80 p.
3. Ministry of Health of Ukraine. Gastroesophageal reflux disease: Unified clinical protocol for primary and secondary (specialized) medical care [Internet]. 2013. Order No. 943; 2013 Oct 31. Available from: https://www.dec.gov.ua/mtd/gastroezofagealna-reflyuksna-hvoroba/.
4. Gastroesophageal reflux disease. Clinical guideline. 2017.
5. Babinets LS, editors. Geriatrics in family medicine: Part 1. In: Educational manual. Lviv: Magnolia; 2023. 510 p.
6. Babinets LS. Geriatrics in Family Medicine: Part 2. In: Educational manual. Lviv: Magnolia; 2024. 478 p.
7. Ministry of Health of Ukraine. Unified clinical protocol for primary, secondary (specialized), and tertiary (highly specialized) medical care for stable coronary artery disease [Internet]. 2021. Order No. 2857, 2021 Dec 23. Available from: https://zakon.rada.gov.ua/rada/show/v2857282-21#Text.
8. DUODECIM Medical Publications. Gastroesophageal reflux disease (evidence-based clinical guideline) [Internet]. Guideline 00170. In: Pelttari H, editor. DUODECIM; 2017. Available from: http://guidelines.moz.gov.ua/documents/2918?id=ebm00170&format=pdf.
9. Makarova HV, Rekalova OM. Features of chronic bronchitis in patients with concomitant gastroesophageal reflux disease. Asthma Allergy. 2019;(4):37-42.
10. Palii IH, Zaika SV, Ksentshyn OO, Skichko NS. Diagnostic properties of the GerdQ questionnaire based on the results of 24-hour esophageal impedance-pH monitoring. Therapeutics. 2021;2(2):21-9.
11. Perederii VH, Tkach SM. Practical Gastroenterology: A Guide for Physicians. Vinnytsia: SPD Kashtelianov AI; 2011. 776 p.
12. Fadieienko HD, Nesen AO, Krakhmalova OO, Izmailova OV. Combination of gastroesophageal reflux disease and coronary heart disease: A non-invasive diagnostic method. Ukr Therapeutic J. 2017;(4):59-66.
13. Stan MP. Peculiarities of gastroesophageal reflux disease in patients with disorders of carbohydrate and lipid metabolism and its treatment [abstract]. Dnipro: Dnipropetrovsk Medical Academy of the Ministry of Health of Ukraine; 2020. 20 p.
14. Fadieienko GD, Nesen AO, Krakhmalova OO, Izmailova OV. Mechanisms of formation of the gastroesophageal reflux disease and coronary heart disease comorbidity. Modern Gastroenterol. 2018;(3):7-13. doi: 10.30978/MG-2018-3-7.
15. Chornobrovyi VM, Melashchenko SH, Ksentshyn OO. Gastroesophageal reflux disease: Approaches to resolving clinical problems in general and family medicine. Fam Med. 2016;(6):125-8. doi: 10.30841/2307-5112.6.2016.249566.
16. Yakhnytska MM. Features of electrolyte metabolism in patients with gastroesophageal reflux disease [abstract]. Dnipro: Dnipropetrovsk Medical Academy of the Ministry of Health of Ukraine; 2020. 20 p.
17. Kovalyova OM, Chukhrienko ND, Pasiieshvili TM, Pasiyeshvili LM, Zheleznia-kova NM. The state of antioxidant defense system in young persons with gastroesophageal reflux disease and autoimmune thyroiditis. Med Perspektivi. 2020;(4):87-93. doi: 10.26641/2307-0404.2020.4.221237.
18. Reva TV, Reva VB, Trefanenko IV, Shumko HI, Shuper VO. Features of the course of gastrointestinal pathology in patients with gastroesophageal reflux disease on the background of hypothyroidism. Med Biol Sci, Physical Educ Sports. 2018;326(4):125. doi: 10.26693/jmbs06.01.125.
19. Antunes C, Curtis SA. Gastroesophageal Reflux Disease [Internet]. In: StatPearlas. Trasure Island (FL): StatPearls Publishing; 2020. Available from: https://www.ncbi.nlm.nih.gov/ books/NBK441938/.
20. Gyawali CP, Kahrilas PJ, Savarino E, Zerbib F, Mion F, Smout AJPM, et al. Modern diagnosis of GERD: the Lyon Consensus. Gut. 2018;67(7):1351-62. doi: 10.1136/gutjnl-2017-314722.
21. Jonasson C, Wernersson B, Hoff DA, Hatlebakk JG. Validation of the GerdQ questionnaire for the diagnosis of gastro-oesophageal reflux disease. Aliment Pharmacol Ther. 2013;37(5):564-72. doi: 10.1111/apt.12204.
22. Katz PO, Gerson LB, Vela MF. Guidelines for the diagnosis and management of gastroesophageal reflux disease. Am J Gastroenterol. 2013;108(3):308-28. doi: 10.1038/ajg.2012.444.
23. Norder GE, Mjrnheim AC, Finizia C, Ruth M, Bergquist H. The diagnostic value of GerdQ in subjects with atypical symptoms of gastroesophageal reflux disease. Scand J Gastroenterol. 2018;53(10-11):1165-70. doi: 10.1080/00365521.2018.1503708.
24. Sampalli T, Dickson R, Hayden J, Edwards L, Salunkhe A. Meeting the needs of a complex population: A functional health- and patient-centered approach to managing multimorbidity. J Comorb. 2016;6(2):76-84. doi: 10.15256/joc.2016.6.83.
25. Tytgat GN, McColl K, Tack J, Holtmann G, Hunt RH, Malfertheiner P, et al. New algorithm for the treatment of gastro-oesophageal reflux disease. Aliment Pharmacol Ther. 2008;27(3):249-56. doi: 10.1111/j.1365-2036.2007.03565.x.
26. Vakil N, van Zanten SV, Kahrilas P, Dent J, Jones R; Global Consensus Group. The Montreal definition and classification of gastroesophageal reflux disease: A global evidence-based consensus. Am J Gastroenterol. 2006;101(8):1900-20. doi: 10.1111/j.1572-0241.2006.00630.x.
27. Yoshida N, Imamura Y, Baba Y, Baba H. Pathogenesis of acute gastroesophageal reflux disease might be changing. Transl Cancer Res. 2016;5(4):645-7. doi: 10.21037/TCR.2016.10.57.
28. Zulman DM, Asch SM, Martins SB, Kerr EA, Hoffman BB, Goldstein MK. Quality of care for patients with multiple chronic conditions: The role of comorbidity interrelatedness. J Gen Intern Med. 2014;29(3):529-37. doi: 10.1007/s11606-013-2616-9.

Стаття надійшла до редакції 22.05.2025. – Дата першого рішення 27.05.2025. – Стаття подана до друку 03.07.2025