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Idiopathic Gastroparesis

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Aim: to characterize the incidence, clinical features, pathophysiological mechanisms, diagnosis and treatment of patients with idiopathic gastroparesis.
Material and methods. The search for sources was carried out in publicly available databases of peer-reviewed scientific literature RSCI, CyberLeninka, PubMed/MEDLINE, Google Scholar in 1990–2025 using the keywords "idiopathic gastroparesis", "symptoms", "pathophysiology", "diagnosis", "treatment", "consensus".
Results. The Consensus of the United European Gastroenterology and European Society for Neurogastroenterology and Motility on gastroparesis (2021) and the Rome Foundation and International Neurogastroenterology and Motility Societies Consensus on idiopathic gastroparesis (2025) define idiopathic gastroparesis (gastroparesis of unknown etiology) as symptoms of nausea and vomiting, often accompanied by postprandial fullness and early satiety, associated with delayed gastric emptying in the absence of mechanical obstruction. According to expert estimates, the incidence of gastroparesis in the USA is 0.16–4 %. Among the possible pathophysiological mechanisms of idiopathic gastroparesis (dysfunction of *n. vagus* and smooth muscle cells, loss of interstitial cells of Cajal, nNOS, macrophages, expressing heme oxygenase-1, abnormalities of syncytium of PDGFRα$^+$-cells, etc.), autonomic neuropathy with loss of enteric nerves is the most proven. In the diagnosis of idiopathic gastroparesis, 4-hour scintigraphy and 13C breath test are preferred. Consensus recommendations for the idiopathic gastroparesis treatment: nutritional support, from the spectrum of pharmacological and surgical interventions, prokinetics and antiemetics are potentially useful. However, the results of treatment often do not adequately address clinical needs.
Conclusion. Large-scale prospective studies of alternative approaches to diagnosis and to individualized methods of idiopathic gastroparesis treatment are needed.
Keywords: idiopathic gastroparesis, symptoms, pathophysiology, diagnosis, treatment, consensus
Conflict of interest: the author declares no conflict of interest.
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Идиопатический гастропарез

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Цель: дать характеристику частоты, клинических признаков, патофизиологических механизмов, диагностики и лечения пациентов с идиопатическим гастропарезом.
Материал и методы. Поиск источников проводили в общедоступных базах данных рецензируемой научной литературы РИНЦ, CyberLeninka, PubMed/MEDLINE, Google Scholar в 1990–2025 гг. по ключевым словам «идиопатический гастропарез», «симптомы», «патофизиология», «диагностика», «лечение», «консенсус».
Результаты. Консенсусы Объединенного европейского гастроэнтерологического общества международных обществ нейрогастроэнтерологии и моторики по гастропарезу (2021) и Римского фонда по идиопатическому гастропарезу (2025) определяют идиопатический гастропарез как симптомы тошноты и рвоты с часто сопутствующими постпрандиальным переполнением и ранним насыщением, связанные с задержкой опорожнения желудка при отсутствии механической обструкции. По оценкам экспертов, частота гастропареза в США составляет 0,16–4 %. Среди возможных патофизиологических механизмов идиопатического гастропареза (дисфункция *n. vagus* и гладкомышечных клеток, потеря интерстициальных клеток Кахаля, nNOS, макрофагов, экспрессирующих гемоксигеназу-1, аномалии синцития PDGFRα$^+$-клеток и др.), наиболее доказана автономная нейропатия с потерей энтеральных нервов. В диагностике идиопатического гастропареза предпочтительны сцинтиграфия в течение 4 часов и ^{13}C-дыхательный тест. Рекомендации Консенсусов в лечении идиопатического гастропареза: нутрициологическая поддержка, из всего спектра фармакологических и хирургических вмешательств потенциально полезными являются прокинетики и противорвотные средства. Однако зачастую результаты лечения не удовлетворяют клиническим потребностям.

Заключение. Необходимы широкомасштабные проспективные исследования альтернативных подходов к диагностике и индивидуализированных методов лечения идиопатического гастропареза.
Ключевые слова: идиопатический гастропарез, симптомы, патофизиология, диагностика, лечение, консенсус
Конфликт интересов: автор заявляет об отсутствии конфликта интересов.
Финансирование: работа выполнена в рамках государственного задания по теме «Изучение молекулярно-генетических и молекулярно-биологических механизмов развития распространенных терапевтических заболеваний в Сибири для совершенствования подходов к их ранней диагностике и профилактике», 2024–2028 гг. (FWNR-2024-0004).

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Introduction

Gastroparesis is a complex of symptoms associated with delayed gastric emptying in the absence of mechanical obstruction. The main symptoms are nausea, vomiting, fullness after eating, early satiety, epigastric pain, bloating in the upper abdomen, and belching. Symptoms must persist for more than three months [1, 2].

The prevalence of gastroparesis is unknown because the diagnosis of gastroparesis requires the determination of gastric emptying in the entire population sample, which has not been done so far, and gastric emptying has been assessed only in individuals who sought medical care [3] or from medical records [4, 5]. For example, the age-adjusted prevalence of gastroparesis estimated by various methods in the USA was 9.6 for men and 37.8 for women per 100,000 person-years [3]. In another epidemiological study the age- and sex-standardized prevalence of gastroparesis was 267.7 per 100,000 adults [4], in a large cross-sectional population study (>43 million medical records) this figure was 0.16 % [5], and according to an estimate by a group of leading experts on gastroparesis, H.P. Parkman et al., this disease may affect 5 million [6] or up to 4 % of the US population [7]. Over the past 16 years, the aggregate charges (i.e., "national bill") for gastroparesis increased by 1026 % to $568.42 million annually [8].

Clinical features of idiopathic gastroparesis

By etiology, gastroparesis is divided into several categories: in the most cited classical study in the world by I. Soykan et al. [2], among patients with gastroparesis, the incidence of idiopathic form is 36 %, diabetic gastroparesis — 29 %, post-surgical gastroparesis — 13 %, gastroparesis in Parkinson's disease — 7.5 %, in collagenoses — 4.8 %, intestinal pseudo-obstruction — 4.1 %, and of mixed causes — 6 %. In an epidemiological study, the incidence of diabetic gastroparesis was 57.4 %, post-surgical — 15 %, drug-induced gastroparesis — 11.8 %, and idiopathic gastroparesis — 11.3 % [4]. In the study of gastroparesis in Olmsted County (USA), 49.4 % of patients were diagnosed with idiopathic gastroparesis and 25.3 % with diabetic gastroparesis [3]. In the Gastroparesis Clinical Research Consortium (GpCRC) registry of 1,400 patients with gastroparesis, almost two-thirds of all cases were idiopathic gastroparesis [9].

Idiopathic gastroparesis (IGP) is a gastroparesis of unknown cause, diagnosed based on the absence of diabetes mellitus, previous gastric surgery, other endocrine, neurological, or rheumatological causes, and the use of medications that can delay gastric emptying (opioids, anticholinergics, calcium channel blockers, tricyclic antidepressants, etc.) [10]. A frequent overlap of symptoms of IGP and functional dyspepsia has been noted [6, 10, 11], with 86 % of patients with IGP meeting the criteria for functional dyspepsia, while 1.2 % meet the criteria for epigastric pain syndrome, and 91.0 % meet the criteria for postprandial distress syndrome [12], the latter being significantly more common than epigastric pain syndrome [13]. This complicates the diagnosis of gastroparesis, so it has been proposed to classify patients with a predominance of nausea and vomiting associated with more pronounced retention of gastric emptying as "gastroparesis", and to define early satiety, postprandial fullness, and pain or burning in the epigastrium as the cardinal symptoms of "functional dyspepsia" [6, 11].

The predominant symptoms in patients with IGP were nausea (34 %), vomiting (19 %), abdominal pain (23 %), bloating (7 %), gastroesophageal reflux (6 %) [12], and postprandial fullness [14]. Overall, 22 % of patients with IGP reported chronic stable symptoms, 29 % had refractory symptoms and were unable to maintain oral feeding; in half of the cases, the onset of IGP symptoms was acute [12]. In another study, patients with IGP were more likely to report nausea (79 %), vomiting (41 %), pain (71 %), bloating (90 %), postprandial fullness (97 %), as well as early satiety (72 %), epigastric burning (62 %), and belching (57 %) [15]. In IGP, increased sensitivity to gastric distension was observed, associated with the nature

and severity of gastroparesis symptoms (higher incidence of early satiety, epigastric pain and weight loss, $p < 0.005$ in all cases) [15].

IGP is more common in women — according to various data, 69 % [7], 75 % [12, 16], 91 % of cases [17]. It has been proven that in normal young premenopausal women, gastric emptying occurs more slowly than in men [18]. Women have more severe symptoms of gastroparesis [7], they are more often hospitalized for gastroparesis, but in men, clinical outcomes of gastroparesis are significantly worse than in women [19]. 46 % of patients with IGP were overweight [10]. Among women with IGP, compared to men, a lower frequency of overweight and smoking was noted [12]. In patients with IGP, body weight is directly associated with upper abdominal pain ($p = 0.01$) and vice versa — with the severity of abdominal bloating ($p < 0.001$) [20]. Depression was noted in 23 % of patients with IGP, gastroesophageal reflux — in up to 40 % of patients [2].

The clinical severity of gastroparesis can be assessed using the American Neurogastroenterology and Motility Society (ANMS) scale for the treatment of gastroparesis: Grade 1 — mild gastroparesis (symptoms are controlled with a normal diet); Grade 2 — compensated gastroparesis (symptoms are partially controlled with diet therapy and medications); Grade 3 — gastroparesis with gastric failure (refractory symptoms) [21, 22]. Severe Grade 3 IGP were associated with the use of antiemetics (odds ratio (OR) — 4.06; $p = 0.004$), use of total/parenteral support (OR = 6.41; $p < 0.001$), and nausea severity (OR = 1.45; $p = 0.006$), while postmenopause was associated with mild IGP of Grade 1 (OR = 0.30; $p = 0.004$) [12].

Compared with diabetic gastroparesis, patients with IGP were on average 13 years younger at the onset of symptoms, had lower body weight, less often required inpatient treatment for gastroparesis [23], and had less severe vomiting, both in terms of severity and the number of episodes [24]. Vomiting was less common in IGP (57 %) compared with diabetic gastroparesis (81 %, $p = 0.004$) [25], whereas patients with IGP had more severe early satiety and postprandial fullness subscores [23]. Nausea, which worsens during or after meals, was generally more common in IGP (52.4 %) compared with diabetic gastroparesis (32.7 %) [25]. Upper abdominal pain was more common in IGP than in diabetic gastroparesis (76 % — IGP, 60 % — type 1 diabetes mellitus, 70 % — type 2 diabetes mellitus; $p = 0.01$) [23]. According to H.P. Parkman et al. [23], IGP may have more pronounced sensory and/or accommodation dysfunction with a predominance of abdominal pain and fullness, while diabetic gastroparesis may be more likely to be caused by motor dysfunction with a predominance of vomiting and delay of gastric emptying.

The consensus of the Rome Foundation and the International Societies of Neurogastroenterology and Motility on IGP (2025) defined nausea and vomiting as the cardinal symptoms of IGP, often accompanied by symptoms of early satiety and postprandial fullness [26].

Pathophysiological mechanisms of idiopathic gastroparesis

The main links in the pathogenesis of IGP are still not fully understood; it is believed that immune-mediated mechanisms are associated (Fig. 1):

— with abnormal function of the enteric nervous system — a decrease in the number of enteric nerve fibers in IGP by 14 % [16] or even 69 % [27];

— with loss of vagal function — a higher sympathovagal balance at rest (LFa/RFa), associated with a decrease in parasympathetic activity, leads to impaired coordination of antropyloroduodenal motility of the stomach and is therefore associated with more severe upper gastrointestinal symptoms in patients with IGP, but not with diabetic gastroparesis [17];

— with the loss of non-muscle cells of mesenchymal origin within the muscular layer of the gastrointestinal tract, the so-called interstitial cells of Cajal, pacemaker cells [28], the loss of which is associated with a disruption in the generation and propagation of slow waves, which leads to abnormal gastric emptying: in patients with IGP, compared with diabetic form, more diffuse and much more pronounced damage to the ultrastructure of interstitial cells of Cajal was noted in electron microscopic [16] and immunohistochemical studies [29], but the loss of interstitial cells of Cajal did not correlate with the severity of gastroparesis symptoms [30];

— with decreased expression of neuronal nitric oxide synthase (nNOS), detected in 40 % of patients with IGP and in 20 % of patients with diabetic gastroparesis — this leads to deterioration of gastric accommodation and pyloric relaxation [16];

— with increased leukocyte infiltration with immunoreactive CD45 and CD68 in the myenteric plexus, which regulates contraction and relaxation of gastric smooth muscles [16], and in patients with IGP, overall clinical severity and nausea are associated with the presence of myenteric immune infiltrate [30];

— with loss of $CD206^+$ anti-inflammatory macrophages of the antral circular muscles [31];

— with changes in the expression of immune genes in transcriptome profiling of human gastric

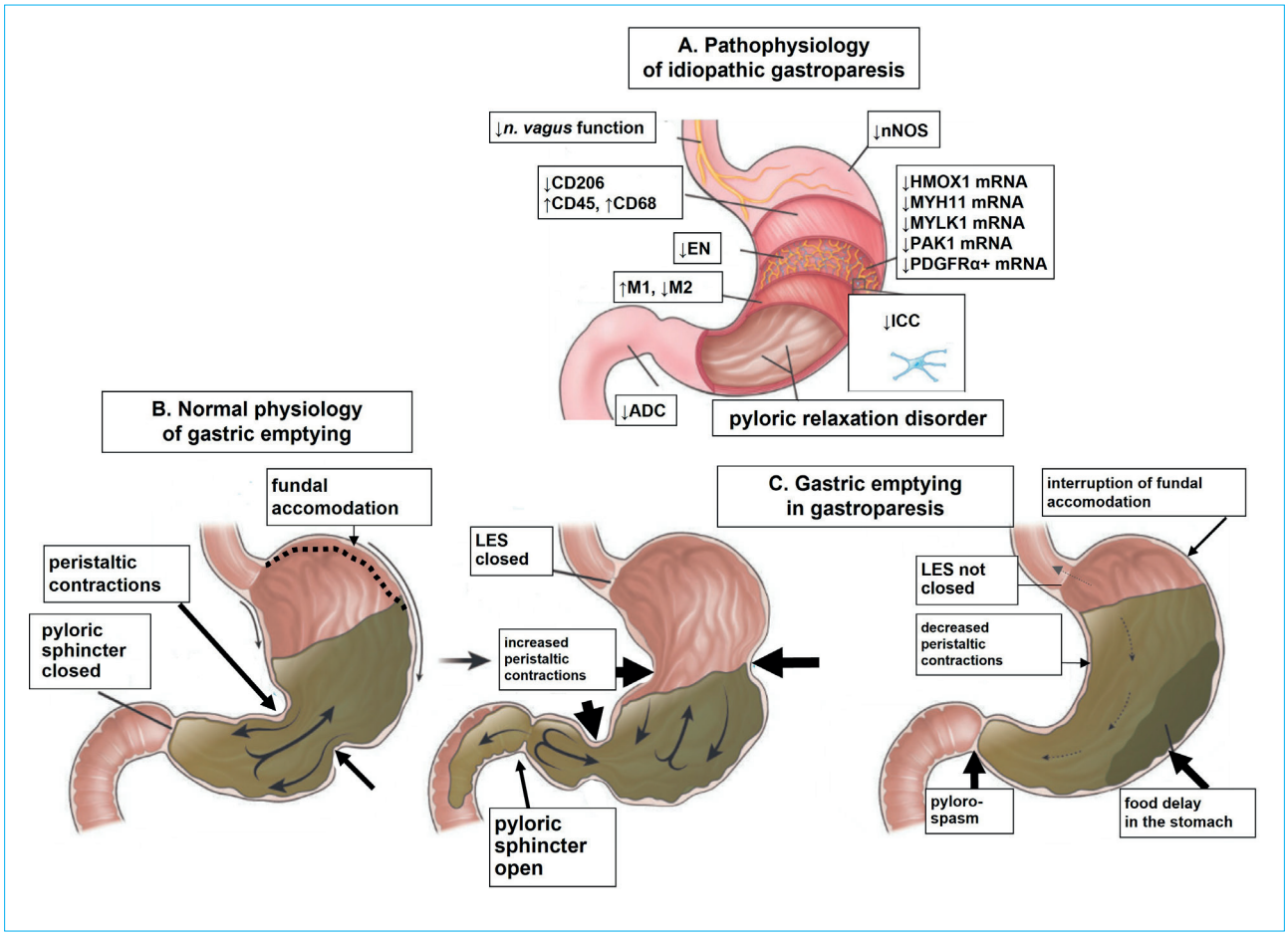

Figure. Pathophysiology of idiopathic gastroparesis (A) and gastric emptying in normal (B) and gastroparetic (C) conditions (adapted from https://patient.gastro.org/gastroparesis/): CD45 and CD68 — general immune cells; CD206 — a membrane protein expressed on anti-inflammatory macrophages; EN — enteric nerves; M1 — pro-inflammatory macrophages; M2 — anti-inflammatory macrophages; ADC — antroduodenal coordination; nNOS — neuronal nitric oxide synthase; HMOX1 mRNA — mRNA encoding the heme oxygenase-1 gene *HMOX1*; MYH11 mRNA — mRNA encoding the smooth muscle myosin heavy chain protein MYH11; MYLK1 mRNA — mRNA encoding the smooth muscle myosin light chain kinase MYLK1; PAK1 mRNA — mRNA encoding the p21-activated kinase PAK1; PDGFRα$^+$ mRNA — mRNA encoding the platelet-derived growth factor receptor α-positive (PDGFRα$^+$) on intramuscular fibroblasts; ICC — interstitial cell of Cajal; LES — lower esophageal sphincter

muscle tissue [32, 33] and proteomic analysis [34] — in IGP, a decrease in the expression of smooth muscle cell mRNA encoding specific contractile and regulatory proteins (in particular, myosin MYH11 and kinases MYLK1 and PAK1) and a decrease in the number of intramuscular fibroblast-like cells expressing platelet-derived growth factor receptor α-positive (PDGFRα$^+$), which affect neurotransmission and thereby controls the contractility of the stomach muscles, forming, together with interstitial cells of Cajal and smooth muscle cells, an integrated unit called a syncytium, as well as a decrease in HMOX1 mRNA encoding the hemeoxygenase-1 protein (a cytoprotective enzyme that is expressed in CD206$^+$ macrophages and protects interstitial cells of Cajal from oxidative stress) were detected [32]; an increase in enriched genes of the proinflammatory M1 macrophage phenotype (0.04 ± 0.03 % in patients with IGP vs. 0.004 ± 0.007 % of genes in controls, $p = 0.02$ [33]) and a decrease in the anti-inflammatory M2 phenotype [33, 34]. The authors [32] believe that IGP may be associated to a greater extent with changes in the expression of contractile proteins by smooth muscle cells and the loss of PDGFRα$^+$ fibroblast-like cells than with the loss of interstitial cells of Cajal.

However, among the ESNM experts (2021), no qualified majority was achieved to support the key pathophysiological role of interstitial cells of Cajal loss, vague nerve function, smooth muscle changes,

genetic factors, cytokine release, peptide hormones in susceptibility to gastroparesis and the occurrence of gastroparesis symptoms, but enteric nerve loss is recognized as a pathophysiological mechanism in gastroparesis [11].

Characteristics of the degree of gastric retention in gastric hyperplasia

A generally accepted method for assessing of gastric emptying for the diagnosis of gastroparesis is scintigraphy examination of gastric emptying from solid food labeled with 99mTc with visualization for 4 hours (the criteria for gastric emptying delay are detection of > 60 % of the contents in the stomach after 2 hours, and > 10 % — after 4 hours) [7, 11, 12, 14, 23, 26]. In addition to scintigraphy, ^{13}C breath test, wireless capsule for motility assessment, antropyloroduodenal manometry, electrogastrography [10, 11, 21, 26], transabdominal ultrasound [35], etc. are used to diagnose IGP. According to scintigraphy results, 45 % of patients with IGP had mild delay of gastric emptying (≤20 % of gastric contents) at 4 hours, 27 % had moderate delay of gastric emptying (>20–35 %), and 28 % had severe delay of gastric emptying (>35 %) [12]. The 4-hour delay of gastric emptying rate in scintigraphy in IGP patients with overweight or normal weight was 31 and 26 %, respectively [12].

Compared to IGP, delay of gastric emptying was higher in patients with diabetic gastroparesis [23]. Thus, delay of gastric emptying on 4-hour scintigraphy was confirmed in 16 % of patients with IGP and in 29 % of patients with diabetic form ($p = 0.01$), but in the IGP experienced more symptoms after taking a test breakfast for scintigraphy compared to patients with diabetic gastroparesis [14].

In patients with IGP, histological examination of full-thickness biopsies did not reveal any correlation between the delay of gastric emptying degree on 4-hour scintigraphy and loss of interstitial cells of Cajal or enteric nerves, which, on the contrary, was confirmed in patients with diabetic gastroparesis [30].

Certain studies have identified a moderate correlation between the severity of IGP symptoms and the 4-hour gastric emptying rate ($r = 0.169$; $p = 0.035$) [12, 24]. Conversely, other investigations found no correlation between the overall severity of IGP symptoms and gastric emptying parameters [10, 11, 15, 25]. Based on these latter findings, some authors [15] have concluded that in patients with IGP, the symptom profile is determined by proximal stomach dysfunction rather than by the severity of delayed gastric emptying.

Treatment

Since 64 % of patients with IGP or diabetic gastroparesis have deficiencies in calories, vitamins A, B_6, C, K, iron, potassium, zinc, and minerals [36], dietary modification is useful in the treatment of patients with IGP, and rehydration and nutritional support in the form of enteral or parenteral nutrition may be required in cases of severe weight loss or intractable vomiting [11].

An epidemiological study of IGP in Great Britain showed that 31.6 % of patients with IGP did not receive any formal pharmacological therapy after diagnosis [37]. The Rome Foundation and International Societies of Neurogastroenterology and Motility consensus on IGP recommended diet, nutritional support, and opioid cessation for treatment, and considered antiemetic and prokinetic agents to be potentially useful [26]. The United European Society of Gastroenterology and European Society of Neurogastroenterology and Motility Consensus Panel on gastroparesis agreed on the use of dietary interventions as well as prokinetics as a group and 5-HT4 receptor agonists as a class [11]. There is no consensus on the efficacy of proton pump inhibitors, different types of antiemetics (5HT3 antagonists ondansetron, granisetron, etc.), 5-HT1A agonists (tandospirone, buspirone), ghrelin receptor agonists (relamorelin, ulimorelin, TZP-102), motilin receptor agonists (camicinal, mitemcinal, erythromycin, clarithromycin), neurokinin-1 receptor antagonists (aprepitant, tradipitant), opioid antagonists (naloxone, naloxegol), phosphodiesterase-5 inhibitor (sildenafil), non-selective cannabinoid receptor agonists (tetrahydrocannabinol, dronabinol), tricyclic antidepressants (nortriptyline), selective serotonin reuptake inhibitors, serotonin and norepinephrine reuptake inhibitors, antidepressant mirtazapine, other neuromodulators, herbal medicine (including Iberogast STW-5), acupuncture, or psychological therapy (including hypnotherapy and cognitive behavioral therapy) for gastroparesis. The same applies to invasive treatments: botulinum toxin injections, gastric electrical stimulation, pyloric endoscopic myotomy, or (partial) gastrectomy [11].

In this review, we will only present a few studies of treatment approaches that identify patients with IGP. Metoclopramide, a dopamine-2 receptor antagonist, is the only drug approved in the United States for the treatment of gastroparesis, including IGP [38]. Treatment with domperidone resulted in significant improvement in prospective studies of patients with gastroparesis (63 % of patients with IGP, 32 % — with diabetic gastroparesis, and 5 % — after fundoplication) [39] and up to 70 % of patients with IGP [2].

A recent meta-analysis [40] demonstrated significant improvements in gastric emptying and GCSI scores among patients with both idiopathic and diabetic gastroparesis receiving highly selective 5-HT4 agonists (velusetrag, felcisetrag, prucalopride),

although a previous randomized, placebo-controlled trial using prucalopride found significant improvements in symptoms and gastric emptying only in patients with IGP, but not in patients with diabetic gastroparesis [41].

Neurokinin-1 receptor antagonists can alleviate nausea and vomiting in IGP (aprepitant [42]) and in patients with both idiopathic and diabetic gastroparesis (tradipitant [43]). Erythromycin, a motilin receptor agonist, improves gastric emptying by 40–50 % in both idiopathic and diabetic gastroparesis, but the clinical response declines after 4 weeks of oral erythromycin [44].

Among patients with IGP, 15 weeks of the tricyclic antidepressant nortriptyline was ineffective in alleviating gastroparesis symptoms by altering visceral sensitivity [45].

Intrapyloric injection of botulinum toxin A, which relieves pyloric spasm, improves both gastric emptying and gastroparesis symptoms in patients with IGP [46], but these results were not confirmed in another cohort of patients with 82.6 % IGP [47]. Predictors of success after peroral endoscopic gastric myotomy (G-POEM) were found in a meta-regression analysis to be idiopathic etiology of gastroparesis, previous treatment with botulinum toxin, and gastric electrical stimulation with Enterra device [48]. Gastric electrical stimulation had an immediate and powerful antiemetic effect in 38 patients (mainly with IGP) with drug-refractory gastroparesis [49]. However, idiopathic etiology, compared with diabetic one, was found to be a less favorable prognostic factor for clinical improvement of gastroparesis treatment in gastric electrical stimulation during an 18-month follow-up [50]. More recently, a prospective, double-blind, randomized trial of gastric electrical stimulation in the treatment of chronic vomiting in patients with IGP showed a reduction in vomiting both during the initial 6-week treatment and after 12 months of ongoing course of electrostimulation [51]. Placement of a percutaneous gastrostomy tube to decompress the distended stomach may be useful for symptomatic relief in patients with refractory IGP [52]. Gastrectomy in patients with gastroparesis (43 % — after gastric surgeries, 34 % — with IGP, and 23 % — with diabetic gastroparesis) relieved symptoms of gastroparesis in 69–89 % of cases during a 6-month follow-up [53].

Prognosis

Gastroparesis is not associated with a reduction in life expectancy, but the prognosis depends on the cause of the disease [11]: patients with diabetic gastroparesis have a 1.9-fold higher risk of mortality after diagnosis than patients with idiopathic gastroparesis [37]. After 6 years of follow-up in patients with gastroparesis of various etiologies (36 % — IGP), 74 % of patients required ongoing prokinetic therapy, 22 % of patients were able to discontinue it, 5 % of patients underwent gastrectomy, 6.2 % of patients switched to gastric electric stimulation, and 7 % of patients died [2]. In a prospective 4-year study in patients with idiopathic and diabetic form of the gastroparesis, independent negative predictors of improvement included overweight/obesity, smoking history, use of pain medications, moderate to severe abdominal pain, severe gastroesophageal reflux, and moderate/severe depression [54].

Conclusion

Idiopathic gastroparesis (IGP) is a heterogeneous syndrome characterized by delayed gastric emptying and its associated symptoms in the absence of mechanical obstruction. Patients with IGP exhibit a broad spectrum of neuroimmunological gastric dysfunctions. The therapeutic objectives for IGP involve symptom alleviation, normalization of nutritional status and hydration, and improvement of gastric emptying, primarily through the use of prokinetic and antiemetic agents. However, patients with decompensated IGP demonstrate poor responsiveness to treatment. Consequently, there is a compelling need for well-designed, long-term clinical trials to establish definitive diagnostic and therapeutic strategies for idiopathic gastroparesis.

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