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KEY CHALLENGES IN DYSPEPSIA MANAGEMENT

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Abstract: Functional dyspepsia is a common gastrointestinal disorder that is further subclassified into postprandial distress syndrome (PDS) and epigastric pain syndrome (EPS). This distinction is based on the predominance of symptoms: PDS is characterized by postprandial bloating and early satiety, whereas EPS is defined primarily by epigastric pain or burning.

The diagnostic evaluation of functional dyspepsia is guided by patient age and the presence of alarm features. Individuals aged 60 years or older, as well as those presenting with red-flag symptoms such as unexplained anemia, warrant prompt assessment with esophagogastroduodenoscopy (EGD) to exclude structural disease. In all patients, Helicobacter pylori infection should be investigated and eradicated if present, given its established role in dyspeptic symptoms.

Therapeutic options for functional dyspepsia include proton pump inhibitors (PPIs), neuromodulators, and prokinetic agents. However, the supporting evidence for these interventions remains limited, and clinical response rates are modest. As such, management often requires a tailored approach, balancing symptom control with patient expectations and ongoing reassessment.

Keywords: Epigastric pain, epigastric burning, early satiety, postprandial fullness, nausea, Helicobacter pylori, peptic ulcer disease

Introduction

Although the exact mechanisms underlying functional dyspepsia remain incompletely understood, its pathophysiology is recognized as complex and multifactorial, with distinct processes contributing to each clinical subtype. Traditionally, disturbances in gastric physiology—both macroscopic and microscopic have been implicated.

Macroscopic physiological mechanisms include gastroesophageal reflux disease, abnormalities of gastric motility such as delayed or rapid gastric emptying, gastric dysrhythmias, and antral hypomotility. Alterations in visceral hypersensitivity also play a role, encompassing a reduced threshold for pain despite normal gastric compliance, aberrant processing of afferent input within the spinal cord or brain, and dysfunction of gastric mechanoreceptors.

Microscopic physiological mechanisms involve impaired mucosal barrier function, particularly heightened sensitivity to duodenal acid or lipids that compromise mucosal integrity. Gastroduodenal inflammation has been observed, characterized by altered lymphocyte populations, increased eosinophils, and mast cell infiltration. Additional contributors include changes in the gut microbiome and the presence of Helicobacter pylori infection. Evidence supporting the link between intestinal inflammation and functional dyspepsia includes the identification of increased small-bowel homing T lymphocytes expressing α4β7-integrin and chemokine receptor 9. These cells are strongly associated with cytokine release, including tumor necrosis factor-α, and have been correlated with symptom severity and delayed gastric emptying, underscoring the duodenum's central role in disease progression. Allergen exposure may further

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exacerbate inflammation by promoting eosinophil recruitment in genetically predisposed individuals.

Psychological factors also contribute significantly to functional dyspepsia. Anxiety and depression have been associated with increased amygdala activation and dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, reflecting altered central processing of visceral stimuli. Stress-induced activation of the HPA axis leads to cortisol release, a phenomenon linked to heightened HPA responsiveness in patients with functional gastrointestinal disorders. Acute stress has been shown to elevate salivary cortisol levels and increase intestinal permeability even in healthy individuals. Moreover, epidemiological data indicate a higher prevalence of functional gastrointestinal disorders among individuals with a history of childhood abuse, highlighting the interplay between psychosocial stressors and gastrointestinal pathophysiology.

Discussion

When lifestyle modifications fail to adequately control symptoms, pharmacologic and behavioral interventions may be required. The choice of therapy is guided by the predominant symptom profile and often involves a combination of medications and supportive behavioral strategies.

Pharmacologic Therapy

Several classes of medications have been employed in the management of functional dyspepsia:

- Gas-relieving agents: Over-the-counter preparations containing simethicone may provide symptomatic relief by reducing intestinal gas. Common examples include Mylanta and Gas-X.

- Acid-suppressing agents: H2-receptor antagonists, such as cimetidine, famotidine, and nizatidine, are available in both non-prescription and prescription formulations. These agents reduce gastric acid secretion and may alleviate epigastric discomfort.

- Proton pump inhibitors (PPIs): PPIs inhibit gastric acid secretion by blocking proton pumps within parietal cells. Non-prescription formulations include lansoprazole, omeprazole, and esomeprazole, with stronger prescription versions also available.

- Antibiotics: In patients with confirmed Helicobacter pylori infection, eradication therapy with antibiotics is indicated, typically in combination with acid-suppressing agents.

- Neuromodulators: Low-dose tricyclic antidepressants and selective serotonin reuptake inhibitors may be prescribed to modulate visceral pain pathways by attenuating neuronal activity.

- Prokinetic agents: These medications enhance gastric emptying and improve antral contractility, thereby reducing postprandial distress and upper abdominal pain.

- Anti-emetics: Agents such as promethazine, prochlorperazine, and meclizine may be used to control nausea and vomiting associated with dyspeptic symptoms.

Behavioral Therapy

Psychological interventions, including cognitive-behavioral therapy and stress management techniques, may complement pharmacologic treatment. These approaches target the central processing of visceral stimuli and are particularly beneficial in patients with comorbid anxiety or depression.

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