GASTROINTESTINAL ACID-RELATED DISEASES AND ISCHEMIC HEART DISEASE: PATHOPHYSIOLOGICAL LINKS AND CLINICAL IMPLICATIONS

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Annotation. This review summarizes key evidence on the interrelations between gastroesophageal reflux disease, peptic ulcer disease, liver disorders, inflammatory bowel diseases, and ischemic heart disease. It highlights both common and specific risk factors, as well as mechanisms of comorbidity, including those linked to standard treatments. Based on meta-analyses and large-scale population studies, the review aims to support the improvement of current clinical guidelines for managing comorbid conditions.

Keywords: Comorbidity, gastroesophageal reflux disease, ischemic heart disease, dual antiplatelet therapy, anticoagulants, aminosalicylates.

Modern medicine is dynamic and constantly evolving. However, clinical studies that form the basis of guidelines, algorithms, and protocols often consider comorbidity as an exclusion criterion. In real-world clinical practice, comorbid conditions are common and complicate both diagnosis and patient management, particularly when one disease limits the use of essential treatments for another. While the proposed use of multiple or cumulative risk models helps manage such cases, it does not address many of the practical challenges faced by clinicians.[1]

Gastroesophageal Reflux Disease and Ischemic Heart Disease. There is now little doubt about the association between gastroesophageal reflux disease (GERD) and ischemic heart disease (IHD). These conditions share common risk factors, including male sex, obesity, diabetes, hypertension, smoking, and alcohol consumption. Myocardial ischemia has been described as a

result of coronary artery spasm triggered by acidic reflux, reduced lower esophageal sphincter pressure, and sympathetic activation. In patients with both GERD and IHD, episodes of heartburn have been shown to coincide with myocardial ischemia and cardiac arrhythmias. A rare phenomenon has also been reported—mechanical compression of the left atrium by paraesophageal hernias, which reduces cardiac blood supply and may lead to ischemia, angina, and arrhythmias.[2]

A nationwide population-based cohort study demonstrated an association between GERD and increased risk of IHD. The overall incidence of IHD was 82% higher in the GERD cohort compared to those without GERD (11.8 vs. 6.5 per 1,000 person-years), with an adjusted hazard ratio (aHR) of 1.49 (95% CI: 1.34–1.66). This association remained significant after adjusting for age, sex, hypertension, diabetes, hyperlipidemia, alcohol-related diseases, stroke, COPD, asthma, gallstones, anxiety, depression, chronic kidney disease, and cirrhosis.[3]

Conversely, cardiac pathology can influence GERD through various mechanisms, including reduced regional esophageal blood flow and hypoxia due to endothelial dysfunction, as well as upper GI dysmotility in IHD. Additionally, standard IHD treatments—such as calcium channel blockers, nitrates, beta-blockers, and antiplatelet agents—may negatively affect lower esophageal sphincter tone.[4]

Proton pump inhibitor (PPI) therapy improves GERD symptoms and may indirectly benefit IHD by reducing esophago-cardiac reflex activity. However, prolonged PPI use has been associated with increased risks of atherosclerosis and potential impairment of cardiac contractility.[5]

Conclusion: Comorbid conditions, though often excluded from clinical trials, are frequently encountered in real-world practice and complicate patient management. Gastroesophageal reflux disease (GERD) and ischemic heart disease (IHD) share common risk factors and show a significant bidirectional relationship. GERD may contribute to myocardial ischemia through reflex mechanisms and

structural effects, while IHD and its treatments can exacerbate GERD symptoms. Population-based studies confirm a significantly increased risk of IHD in GERD patients. While proton pump inhibitors (PPIs) may alleviate GERD symptoms and indirectly benefit cardiac function, long-term use may pose cardiovascular risks, highlighting the need for careful therapeutic strategies in comorbid patients.

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