Editorial

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Should *H. pylori* screening be integrated into MI care protocols?

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Abstract

Helicobacter pylori is considered a modifiable and exacerbating risk factor in influencing post-myocardial infarction (MI) upper gastrointestinal (GI) bleeding, either due to antithrombotic therapies or other mechanisms. Numerous studies suggest high prevalence of *H. pylori* in post-MI patients, emphasizing the role of screening of *H. pylori* and timely introduction of eradication therapy in infected individual as a cost-effective strategy. *H. pylori* is not only involved in post-MI bleeding but also play a role in triggering myocardial events and its recurrences through plaque destabilization, inflammatory response while its role in dyslipidemia remains inconsistent. This issue is under investigation since 2000 and had shown a significant relationship between involvement of *H. pylori* (especially CagA strains) and MI, role of genetics and environmental factors in triggering *H. pylori* in MI patients; however, most evidence up to date are either observational or cohort studies. Current randomized controlled trials (RCTs) suggest the role of targeted screening in high-risk population and area proved to be beneficial than blanket screening. However, lack of subgroup analysis with RCTs and long-term follow-up is necessary to establish the role *H. pylori* screening in preventing recurrent MI, reducing upper GI bleeding and cardiovascular outcomes. Furthermore, integrating inflammatory biomarkers and genetic factors in screening might help in risk stratification.

The management of post-myocardial infarction (MI) requires dual or triple antithrombotic therapy (aspirin and P2Y12 inhibitor therapy) to reduce recurrence of cardiovascular events, a treatment that inherently increases bleeding risk especially upper gastrointestinal (GI) bleeding. Current acute coronary syndrome (ACS) guidelines, do not include *Helicobacter pylori* infection despite its well-established role in increasing upper GI bleeding[1]. However, *H. pylori* infection is an independent and potentially modifiable risk factor that exacerbates this bleeding risk patients following MI, making its detection and potential eradication clinically relevant. Although the incidence of *H. pylori* in MI patients is relatively common, it varies considerably by region, with studies reporting rates from 20–24% up to 88%. Its prevalence is high in people undergoing coronary angiography as well[2]. A Swedish study found a seroprevalence of 46.5%, while an Egyptian study reported 72%, with higher rates observed in smokers and ST-elevation MI patients. Despite this high prevalence, the role of routine *H. pylori* testing in post-MI care remains a subject of underexplored and debated[3–5].

The growing body of evidence suggests that *H. pylori* is not only involved in increasing bleeding risk in MI patients but also plays a direct role in the pathophysiology of ACS as well. According to a recent Chinese study, this mechanism appears to be caused by systemic inflammatory pathways that induce endothelial dysfunction and a prothrombotic state, which contrast with earlier hypotheses centered on dyslipidemia[6]. Precisely, infection with virulent strains (e.g., CagA-positive) can fasten atherosclerotic plaque destabilization, while chronic infection may worsen metabolic risk profiles. This cumulative pathophysiology suggests that *H. pylori* can also be a potential trigger for initial MI events and may influence long-term recurrence rates. Furthermore, genetic evidence from recent Mendelian randomization studies strengthens this causal link, demonstrating that higher anti-*H. pylori* IgG levels increase the risk of MI, possibly mediated through reductions in high-density lipoprotein cholesterol. Together, these findings suggest that *H. pylori* is not only a modifiable bleeding risk factor but also a potential contributor to the initiation and recurrence of ACS. Also, it highlights that dyslipidemia theory is relatively weak and while inflammatory theories suggest strong mechanistic insights.[6,7]

In the early 2000s, Kowalski *et al* found out no significant association between *H. pylori* infection, and cholesterol while individuals with *H. pylori* especially CagA-positive strains, and coronary artery disease (CAD), had greater coronary artery lumen loss and higher rates of restenosis[8]. In 2012, Eskandarian *et al* demonstrated that *H. pylori* infection increases the short-term risk of recurrent cardiac events after ACS, but not in the long-term[9]. Similarly, Longo-Mbenza *et al* also reported the role of direct bacterial DNA (CagA-positive strains) in atherosclerotic coronary plaques and highlight the risk of threefold increase of developing CAD[10]. In 2020,

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Wärme *et al* conducted cohort study which supported the hypothesis and also suggested in hospital testing of *H. pylori*, while calling for outcome-driven future studies[11]. A Chinese study published in 2024 also reinforced the strong association between *H. pylori* and MI and suggested that genetic factors may contribute to MI risk, while it negates the role of lipids in pathophysiology of *H. pylori* and MI[6]. Currently, Hofmann *et al* conducted a randomized controlled trial (RCT) that demonstrates the role of targeted screening especially in high-risk population and infected patients with anemia or history of GI bleeding[5]. Overall, these studies show the shift from recognizing *H. pylori* as a risk marker to exploring targeted clinical interventions in CAD and AMI.

The trajectory of research highlights that *H. pylori* and MI are strongly associated and might contribute to its pathogenesis. Therefore, if prophylactic screening of *H. pylori* in MI patients and *H. pylori* screening in post-MI patients should be incorporated as preventive strategy and timely eradication therapy is suggested, this could reduce overall burden of upper GI bleeding, and also prove to be cost effective by preventing complications, reducing hospitalization, and minimizing long-term healthcare expenditures[12].

Nevertheless, several limitations remain. Rather than applying blanket screening, a strategy of targeted testing should be prioritized in high-risk countries and in patients with comorbidities such as anemia, diabetes, or prior GI disease. Furthermore, the usage of proton pump inhibitors (PPIs) should be considered before enrolling participants in future studies as their use can suppress *H. pylori*, leads to false-negative diagnostic tests and underestimation of true prevalence. Thus, future studies must carefully account for PPI use by including washout periods or employ multiple diagnostic modalities.

While current guidelines do not consider *H. pylori* screening mandatory for post-MI patients, emerging data suggest that *H. pylori* can be an aggravating factor in MI patients. While current data highlight the role of targeted screening (high-risk countries and patients with anemia and prior GI bleeding), hold potential in preventing and early diagnosis of upper GI bleeding, and may show better prognosis with eradication therapy and improved cardiovascular outcomes as well, however, its broader role remain underexplored. Therefore, calling for future large RCT with long-term follow-up is necessary to establish the role of *H. pylori* screening in recent guidelines, as current data relies on observational and cohort studies. Studies should also assess dual outcomes that determining whether eradication therapy reduces post-MI bleeding in *H. pylori* patients, but also reduces recurrence of cardiovascular events. Furthermore, the pathophysiological role of *H. pylori* in inducing MI should also be explored, specifically its role in systemic inflammation and plaque destabilization. Since the use of PPIs are very common and leads to suppression of *H. pylori*, false-negative results can be generated with urea breath test and stool antigen test. Hence, before enrolling patients, the history of PPIs usage should be taken and integration of washout periods of 2 weeks should be followed, to avoid underestimating of prevalence. Additionally, considering inflammatory markers (interleukin (IL)-6, C-reactive protein) a part of screening protocol should be considered as it can help in risk stratification[13]. Prospective investigations have to focus on host immune-genetic polymorphisms (e.g., IL-1β, TNF-α) and *H. pylori* virulence factors (e.g., CagA). These host-pathogen interactions are anticipated to impact vascular inflammation, plaque destabilization, and interindividual variance in cardiovascular risk, aiding in the identification of subgroups that may benefit the most from focused screening and eradication[6]. Also, microbiome research may elucidate how *H. pylori* interacts with other gut bacteria and contributes to systemic inflammation and atherosclerotic plaque destabilization, opening up new pathways for precision screening and therapy in post-MI patients. Finally, in conjunction eradication, alternative methods such as optimized antiplatelet or anticoagulant regimens, increased gastroprotective therapy, or combination approaches should be investigated to strike a compromise between cardiovascular protection and bleeding risk reduction.

In conclusion, *H. pylori*-targeted screening has shown great potential in recent investigation, as a preventive strategy to reduce burden of bleeding in post-MI patients, yet highlighted a lot of novel questions that remain unanswered. However, major limitations in these areas hinder our capability to construct firm conclusions, requiring vigorous large RCTs and long-term follow-up. Incorporating targeted screening into post MI-individuals and determining if *H. pylori* directly affects cardiovascular outcomes, this might be a cost-effective approach and is feasible with current acute care are required to prove its validity.

This editorial adheres to the Transparency in the Reporting of Artificial Intelligence in Research (TITAN) guideline[14]. During the preparation of this manuscript, generative artificial intelligence (AI) tools (DeepSeek, DeepSeek Inc., June 2025 version; temperature parameter = 0.7) were used only as auxiliary aids to assist in formatting standardization, organization of background information, and language consistency checks.

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