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Letter to the Editor

Impact of *Helicobacter pylori*-related Metabolic Syndrome and Gastroesophageal Reflux Disease on the Risk of Acute Myocardial Infarction

TO THE EDITOR: In their recent study, Eisa et al[1] concluded that gastroesophageal reflux disease (GERD) with concomitant metabolic syndrome (MetS) parameters is a risk factor of acute myocardial infarction (AMI) though this risk may be clinically insignificant.

In this regard, there is increasing evidence for association between *Helicobacter pylori* infection (*Hp*-I) and insulin resistance (IR) or MetS and related morbidity, including GERD and cardiovascular disease (CVD);[2,3] the prevalence of MetS is higher in *Hp*-positive people;[2] *Hp*-linked MetS is a risk factor of GERD and its eradication exhibits a positive effect against GERD in certain populations;[3] AMI, a potentially fatal CVD complication, is closely linked with MetS;[2] and *Hp* is a risk for acute coronary syndrome (ACS) including AMI,[2] thus further investigation is warranted to estimate whether *Hp* eradication affects AMI occurrence.

Specifically, both *Hp*-I and MetS are highly prevalent worldwide and epidemiological studies as well as meta-analyses have shown that obesity induces inflammation (especially abdominal, visceral obesity) and drives to MetS, thereby being an indirect risk factor for GERD.[3] In this respect, the conventional claim that declining *Hp* prevalence has led to a rise in GERD requires to be better studied since, for instance, the current global prevalence of *Hp*-I varies from 39.9% to 84.2% whereas the comparable picture for GERD is quite less varying from 2.5% to 51.2%.[4] Moreover, several data indicate that *Hp* may contribute to GERD pathogenesis by several mechanisms and its eradication results in adequate control of GERD symptoms and improves esophagitis.[3,5]

A recent meta-analysis also indicated that *Hp*-I increases the risk of CVD adverse events, particularly AMI;[6] there is a link between *Hp*-related CagA cytotoxin and ACS and the odds ratio of AMI is twice as greater in *Hp*-positive patients. Likewise, MetS is a major risk factor for AMI,[7] increases the risk of CVD adverse events more than 2-fold, whereas its recovery significantly decreases the risk for major adverse cardiovascular events including AMI. Moreover, *Hp*-I is an independent risk factor for atrial fibrillation (AF),[8] which remains a frequent arrhythmia in AMI and is closely linked with augmented subsequent cardiovascular mortality; coronary artery disease is a risk factor for AF and coronary embolism due to AF is a cause of AMI; and *Hp*-related non-alcoholic fatty liver disease, the hepatic component of MetS, is a risk of AF.[8] Finally, *Hp*-I–related MetS may contribute to the pathophysiology of CVD including AMI by several mechanisms,[9,10] thereby signifying eradication therapy as AMI prevention strategy.

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