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Letter to the Editor

Comment on "The correlation of *Helicobacter pylori* with the development of cholelithiasis and cholecystitis: the results of a prospective clinical study in Saudi Arabia"

Dear Editor,

In their study Guraya et al[1] concluded that the presence of *Helicobacter pylori (Hp)* in bile might indicate a significant risk for cholelithiasis.

By using *Hp* serology, our data indicate presence of past and/or current *Hp* infection *(Hp*-I) in 63 of 123 (51.2%) patients (women 64, mean age 63 years) with calcular biliary and pancreatic diseases (cholecystitis/cholangitis and pancreatitis, respectively)[2]. Moreover, based on histology, the practical gold standard for *Hp*-I diagnosis, our data indicated *Hp* presence in gallbladder tissue in 19.33% of Greek cholecystectomized patients (all women)[2].

Although the pathways of *Hp* penetration into the bile have not been completely elucidated, we considered the possible pathways of *Hp* migration and colonization in the biliary tract and its involvement in inflammatory biliary diseases[2]; the possibilities of *Hp* translocation from the duodenum via Oddi's sphincter and/or its hematogenous spread to the liver and then excretion into the bile were suggested[3,4]. In this regard, the possible influx of activated monocytes infected with *Hp* (due to defective autophagy) from the circulation into the gallbladder might lead to gallbladder-related pathologies ("Trojan horse" pathway, also proposed for the *Hp*-induced brain pathologies)[5].

Hp-I could affect the pathophysiology of gallbladder stone creation and its complication including cholecystitis, cholangitis, pancreatitis and biliary cancer by the following mechanisms: (1) releasing large amounts of proinflammatory and vasoactive substances, such as interleukins (IL) -1, -6, and tumour necrosis factor (TNF)-α involved in a number of inflammatory diseases[6] also including gallbladder disorders[7]; pro-inflammatory IL-1α, L-6 and TNF-α are involved in the pathogenesis of cholelithiasis[8]; (2) producing oxidative stress[6], also involved in gallbladder disease[7]; free radical reactions in the gallbladder wall and in bile can induce gallstone formation[9]; (3) influencing the apoptotic process[6], also involved in chronic cholecystitis and gallbladder oncogenesis[10].

Therefore, *Hp* eradication might display a positive impact on *Hp*-related biliary pathologies.

Conflict of Interest

The Authors declare that they have no conflict of interests.

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