Study of the prevalence of helicobacter bilis colonization in pancreaticobiliary disorders

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List disclosures

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Objectives

- This study investigated the prevalence of H. bilis colonization of the biliary system of patients with pancreaticobiliary maljunction (PBM).
- Discuss its potential role as a causative factor for biliary carcinoma in PBM

Introduction

Helicobacter bilis, a Gram-negative enterohepatic Helicobacter, has been isolated from bile and liver tissue of mice and shown to be a causative factor of chronic hepatitis and liver tumours in mice.

In recent years, H. bilis DNA has been detected in human bile and hepatobiliary tissue by means of polymerase chain reaction (PCR) assays.

Helicobacter bilis is considered to be a causative factor in the pathogenesis of biliary cancer.

Methods

Bile juice and biliary tissue samples were collected from 17 patients with PBM and 27 controls who had benign biliary disease without PBM.

DNA extracted from each biliary sample was subjected to polymerase chain reaction (PCR) analysis for H. bilis and Helicobacter pylori.

Standard univariable statistical tests were used to compare categorical and continuous variables between the study groups, as appropriate: $\chi 2$ test, two-tailed Fisher's exact test and Mann–Whitney U test. P < 0.050 was considered statistically significant.

Results

PCR assays revealed that 12 of the 17 patients with PBM were positive for H. bilis DNA, compared with eight of 27 patients without PBM (P = 0.009).

Among patients with PBM, H. bilis DNA was identified in six of eight children, including a 2-month-old infant, and in six of nine adults.

The high prevalence of H. bilis DNA in the biliary system of patients with PBM was independent of age, sex, common bile duct dilatation, configuration of the pancreatic and bile ducts, and amylase activity in bile.

Table _ Results of polymerase chain reaction assays for Helicobacter bilis and Helicobacter pylorus in bile and biliary tissue samples from patients with pancreaticobiliary maljunction and controls

| Sample source | PBM (n = 17) | | | Controls (n = 27) | | |
|---------------|-------------------------|-------------------------|--------|-------------------|-------------------------|--------|
| | No. of samples examined | No. of positive samples | | No. of samples | No. of positive samples | |
| | | HB DNA | HP DNA | examined | HB DNA | HP DNA |
| Bile | 17 | 11* | 0 | 27 | 6 | 1 |
| Gallbladder | 12 | 5 | 1 | 25 | 4 | 2 |
| Bile duct | 9 | 5 | 1 | 0 | _ | - |
| Total | 17 | 12* | 1 | 27 | 8 | 2 |

PBM, pancreaticobiliary maljunction; HB, Helicobacter bilis; HP, Helicobacter pylori. *P < 0.010 versus controls (two-tailed Fisher's exact test).

Conclusion:

The present finding of a high prevalence of biliary colonization with *H. bilis in adults and children with PBM may point* to a role for chronic sustained biliary colonization with *Helicobacter organisms rendering biliary epithelial cells* susceptible to the development of biliary carcinoma later in life.

The mechanisms by which H. bilis exerts its pathogenetic action on enteric organs are still unknown. H. bilis induces progressive immune reactivity to commensal bacteria that contributes to the development of immune-mediated intestinal inflammation.

➤H. bilis colonization of the biliary system is extremely common in patients with PBM. This may point to a role in the pathogenesis of biliary cancer

>Further elucidation of the relationship between biliary carcinogenesis and H. bilis infection is required.

References

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