



Title	Gastric epithelial expression of trefoil family factor 2 and mucin 6 in normal and Helicobacter pylori-infected subjects
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GIH-03 Role of liver biopsy in the management of liver dysfunction after hematopoietic stem cell transplantation in a hepatitis B virus prevalent patient population

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Introduction: Derangement of liver-function-tests (LFT) is common after hematopoietic stem-cell-transplantation (SCT). The role of liver biopsy in such cases has not been defined in hepatitis B virus (HBV) prevalent patients. The impact of liver biopsy in the management of LFT derangement post-SCT in a HBV-prevalent population was examined.

Materials and methods. Seventy-five liver biopsies, performed for 323 patients with LFT derangement post-SCT (263 allogeneic, 60 autologous), were analysed. The HBV carrier rate was 13.6%.

Results. Significantly more LFT derangements and therefore liver biopsies occurred in allogeneic versus autologous SCT. Prior to biopsy, graft-versus-host disease (GVHD) and HBV reactivation were clinically diagnosed in 70.6% and 25.3% of cases. A definite histopathologic diagnosis was obtained after biopsy in 53 cases, with GVHD, HBV-hepatitis and concomitant GVHD/HBV-hepatitis found in 33%, 21% and 8% of cases respectively. The clinical and histopathologic diagnoses were concordant in 43 cases and discordant in 10 cases. Clinical management was altered in 6/10 discordant cases, 5 of which were due to HBV or hepatitis C virus (HCV) reactivation. Twenty-two biopsies showed non-diagnostic histopathologic features. Twenty of these cases were successfully managed based on clinical diagnoses. The clinical/biochemical features of patients clinically diagnosed to have GVHD did not differ significantly whether or not they were HBV/HCV carriers. However, liver biopsies in HBV/HCV carriers resulted in significantly more treatment alterations as compared with non-carriers.

Conclusions. Clinical diagnoses of LFT derangements post-SCT might be adequate for initiation of treatment, but liver biopsy in HBV/HCV carriers were needed as this might impact on management.

GIH-04 Gastric epithelial expression of trefoil family factor 2 and mucin 6 in normal and *Helicobacter pylori*-infected subjects

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Introduction: The role of trefoil family factors (TFFs), a group of small secretory peptides, and mucins, the major components of the mucous viscous gel covering the surface of epithelium, in mucosal defence and healing has been postulated. *H. pylori* infection is associated with gastric mucosal inflammation and injury. The aim of this study was to determine whether *H. pylori* infection is associated with altered expression of TFF2 and MUC6 at the different gastric sites.

Method: Gastric biopsy specimens taken from gastric antrum, incisura and body were used for the detection of *H. pylori* infection and histological assessment, and the determination of TFF2 and MUC6 expression by immunohistochemistry.

Results: Of the 76 patients recruited, 27 (35.5%) were positive for *H. pylori* infection. Chronic gastritis was present in 26 (96.3%) *H. pylori* positive patients and 7 (14.3%) *H. pylori* negative patients ($P < 0.001$). In all 42 (100%) patients with normal mucosa (i.e. without *H. pylori* and chronic gastritis), TFF2 and MUC6 were coordinately expressed in regenerative zone and deep portion of glands of antral mucosa, and only in the regenerative zone of gastric incisura and body mucosa. However, in patients with *H. pylori* infection, TFF2 and MUC6 expression was detected within the foveola of antral mucosa, incisura and body in 59.3%, 44.4% and 11.1%, respectively (all $P < 0.05$, compared with normal mucosa). Moreover, TFF2 and MUC6 expression was also detected in the glands of incisura and body mucosa in a proportion (96.3% and 14.8%, respectively) of *H. pylori* infected patients.

Conclusion: *H. pylori* infection is associated with extended TFF2 and MUC6 expression in the gastric antral, incisura and body epithelium, which indicates a protective role of these factors in *H. pylori* infection.

Aims: To determine gastric expression of trefoil family factor 2 (TFF2) and MUC6 in *Helicobacter pylori* positive and negative subjects, and its association with antralisation at the gastric incisura. **Methods:** Gastric biopsies from the antrum, incisura, and body of 76 dyspeptic patients without ulcers were used for the determination of *H. pylori* infection, histological changes, and epithelial TFF2 and MUC6 expression. **Results:** In the foveola, the rates of TFF2 and MUC6 immunostaining were greater in *H. pylori* infected ($n = 27$) than in uninfected patients ($n = 49$) at the antrum (59.3% v 4.1% for TFF2 and 63.0% v 4.1% for MUC6; both $p < 0.001$) and incisura (44.4% v 2.0% for TFF2 and 48.1% v 0% for MUC6; both $p < 0.001$). *Helicobacter pylori* attaches to mucin oligosaccharides that are expressed on host gastric epithelium. We used the rhesus macaque model to characterize the effect of *H. pylori* infection on gastric mucin oligosaccharides during acute and chronic infection. Specific pathogen (*H. pylori*)-free rhesus macaques were inoculated with *H. pylori* J166. **Conclusion:** *H. pylori* may affect the expression of MUC5AC, MUC2, MUC5B, and MUC6 in AGS cells via CagA and/or NH₂, but not E-cadherin. **View. Show abstract.** Moreover, *H. pylori*-infected gastric surface epithelium protects itself by the downregulation of MUC5AC and the upregulation of MUC6; this process forms a mucus gel-layer to protect the underlying epithelium [31,32]. *Helicobacter pylori* infection increases the expression of TFF2 and MUC6 in the gastric epithelium. Aberrant TFF2 and MUC6 expression is associated with antralisation of gastric incisura. In the deeper glands, the rates were also greater in *H. pylori* infected than in uninfected patients at the incisura (85.2% v 22.4% for both TFF2 and MUC6; $p < 0.001$). Antral-type mucosa was present at the incisura in 28 of the 76 patients. TFF2 and MUC6 expression in the foveola and deeper glands was significantly associated with antral-type mucosa, independent of *H. pylori* status. **Conclusions:** *Helicobacter pylori* infection increases the expression of TFF2 and MUC6 in the gastric epithelium. Aberrant TFF2 and MUC6 expression is associated with antralisation of gastric incisura.