PTU-091

**CHANGING TRENDS IN PEPTIC ULCER DISEASE:
THE RISE OF NSAID-INDUCED AND FALL OF
H PYLORI-INDUCED ULCERS**

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Introduction Non-steroidal anti-inflammatory drugs (NSAIDs) and *Helicobacter pylori* are the main causes of peptic

ulcer disease (PUD). Data from the UK from 1997 to 2005 indicated that the aetiology is slowly changing as the prevalence of *H pylori* and use of non-aspirin NSAIDs (NANSAIDs) has been decreasing while the use of low-dose aspirin (LDA; ≤ 325 mg/day) has been increasing.

Methods In order to investigate these changing trends, subjects who presented with endoscopically confirmed PUD at Royal Liverpool Hospital between July 2005 and June 2010 were identified from the endoscopy database and recruited either prospectively or retrospectively. Recruits were interviewed using a structured questionnaire and GPs were contacted to capture data missing from case-notes. Patients were categorised as either NSAID users (those on NSAIDs within 2 weeks) or non-users (those not on NSAIDs within 3 months of endoscopy). Upper GI bleeding (UGIB) was defined as haematemesis, melaena or anaemia (haemoglobin drop ≥ 2 g/dl) and/or endoscopic stigmata of recent bleeding. *H pylori* status was determined by the rapid urease test and/or histology, or serology (IgG, ELISA) where these were negative or not done.

Results Of the 389 patients enrolled, 220 (57%) were using NSAIDs and 169 (43%) were non-users. 29% of the whole cohort were taking LDA alone. 57% of the patients were 65 years or above, comprising 66% of NSAID users and 41% of non-users, with mean ages of 67 and 60 (SD 13.5 vs 16) years, respectively. The mean age of those using LDA alone was 70 (SD 10.6) years. *H pylori* was positive in 41% of ulcers (46% DU, 33% GU). Amongst NSAID users, 51% were on LDA, 30% on NANSAIDs, 15% on both LDA and NANSAIDs and 4% on high dose aspirin. NSAID users had more GU (59% vs. 45%, $p=0.006$), fewer DU (31% vs 48%, $p=0.001$) and were less likely to be *H pylori* positive (34% vs. 49%, $p=0.005$); there was no difference in gender (% males 50 vs. 56, $p=0.318$) or prevalence of UGIB (22% vs 20%, $p=0.697$) between the two groups. Compared to NANSAID users, LDA users were more likely to be *H pylori* positive (43% vs. 23%, $p=0.003$) with a similar prevalence of UGIB (21% vs. 20%, $p=0.987$). 22% of patients with PUD were neither using NSAIDs nor *H pylori* positive.

Conclusion NSAIDs, particularly LDA, were the commonest cause of PUD in this cohort, especially in those over 65 years. Our findings are compatible with the steady decline in the prevalence of *H pylori*-positive PUD and increase in non-NSAID non-*H pylori* PUD over recent years. LDA users were older and more likely to be *H pylori* positive compared to those using NANSAIDs; the significance of this in terms of ulcer pathogenesis needs further study.

Competing interests None.

Keywords Aspirin, *Helicobacter pylori*, non-steroidal anti-inflammatory drugs, peptic ulcer disease.