

Gastroduodenal

PWE-157 UPPER GASTROINTESTINAL BLEEDING IN HOSPITALISED PATIENTS—PROSPECTIVE CONTROLLED ANALYSIS OF THE ROLE OF ASPIRIN AND OTHER ANTITHROMBOTIC DRUGS

doi:10.1136/gutjnl-2012-302514d.157

¹A S Taha,* ¹C Kelly, ²C McCloskey, ²T Craigen, ³W J Angerson. ¹Department of Medicine, University of Glasgow, Glasgow, UK; ²Department of Medicine, University Hospital, Crosshouse, Kilmarnock, UK; ³Department of Surgery, University of Glasgow, Glasgow, UK

Introduction Upper gastrointestinal bleeding (UGIB) developing while in hospital is traditionally thought to be due to stress ulceration affecting critically ill patients. The role of potentially damaging drugs is not clear.

Aims Given the increasing use of low-dose aspirin and other antithrombotic drugs—clopidogrel, dipyridamole, and warfarin—for vascular protection, we aimed to clarify their impact on UGIB affecting hospitalised patients.

Methods Between 2008 and 2009, we prospectively assessed the clinical details and outcomes of all inpatient bleeders at our hospital, and compared these with outpatients admitted because of bleeding. For each inpatient we included three outpatients as controls. Patients were excluded if they had a variceal cause. The Charlson comorbidity score and the complete Rockall score for non-variceal upper gastrointestinal bleeding were also calculated. The χ^2 test, Fisher's exact test, Mann–Whitney U test, and logistic regression analyses with ORs and 95% CIs were used as appropriate.

Results Abstract PWE-157 table 1 summarises the main characteristics of patients who bled while in hospital (inpatient bleeders) vs those who were admitted after bleeding (outpatient bleeders). Inpatient bleeders were older and more likely to be males and to have cardiovascular disease. After adjustment for age and sex, the logistic regression analysis still showed that patients who bled as inpatients were significantly more likely to be taking antithrombotic medication, especially non-aspirin agents, than those who were admitted after bleeding, [OR (95% CIs), 2.15 (1.25 to 3.68); $p=0.006$]. The adjusted odds of death within 30 days of bleeding was almost twice as high in the inpatient group, [OR 1.88 (0.74 to 4.77)]. The endoscopic abnormalities in more than 80% of patients in both groups included erosive oesophagitis, gastric, or duodenal ulcers or erosions.

Abstract PWE-157 Table 1

Covariate	Inpatient bleeders (n = 96)	Outpatient bleeders (n = 274)	p Value
Age, years, (median, IQR)	77 (67–83)	68 (50–79)	<0.001
Males	44/96 (45.8%)	166/274 (60.6%)	0.016
Cardiovascular disease	50/96 (52.1%)	80/274 (29.2%)	<0.001
Rockall 3+	81/96 (84.4%)	182/273 (66.7%)	0.001
Aspirin	44/96 (45.8%)	86/274 (31.4%)	0.013
Other antithrombotics	35/96 (36.5%)	50/274 (18.2%)	<0.001
Aspirin or other antithrombotics	62/96 (64.6%)	122/274 (44.5%)	0.001
NSAIDs	11/96 (11.5%)	49/273 (17.9%)	0.15
Death at 30 days	9/94 (9.6%)	12/272 (4.4%)	0.074

Conclusion The use of antithrombotic drugs is a significant risk factor for UGIB developing in hospitalised patients. The endoscopic lesions, whether caused by these agents or not, are potentially preventable.

Competing interests None declared.

PWE-158 NON-SELECTIVE NON-STEROIDAL ANTIINFLAMMATORY DRUGS, PROTON PUMP INHIBITORS, AND THE INCIDENCE OF NON-VARICEAL UPPER GASTROINTESTINAL BLEEDING AND CLOSTRIDIUM DIFFICILE INFECTION

doi:10.1136/gutjnl-2012-302514d.158

¹A S Taha,* ¹C Kelly, ²C McCloskey, ²T Craigen, ³W J Angerson. ¹Department of Medicine, University of Glasgow, Glasgow, UK; ²Department of Medicine, University Hospital, Crosshouse, Kilmarnock, UK; ³Department of Surgery, University of Glasgow, Glasgow, UK

Introduction It is not clear what impact the decline of COX-2 selective inhibitors has had on the use of non-selective non-steroidal anti-inflammatory drugs (ns-NSAIDs) or on the incidence of non-variceal upper gastrointestinal bleeding (NV-UGIB). Also, proton pump inhibitors (PPIs), recommended for NSAID-ulcer prophylaxis, have been linked to *Clostridium difficile* infection.

Methods We aimed to measure the incidence [cases per 100 000 population per annum] of NV-UGIB and *C difficile* infection as well as the use of ns-NSAIDs and PPIs in a well-defined population served by a single centre. Data were collected over three calendar years, 2007–2009. Significance was assessed using the χ^2 test for trend.

Results As shown in Abstract PWE-158 table 1, the use of COX-2 selective and newer NSAIDs continues to decline, while that of ns-NSAIDs continues to rise. This has been coupled with a steady rise in the use of PPIs. On the other hand, the incidence of both NV-UGIB and *C difficile* infection has been falling.

Abstract PWE-158 Table 1 The incidence of non-variceal upper gastrointestinal bleeding and *Clostridium difficile* infection, and the annual number of prescriptions [per 100 000 population]

	2007	2008	2009	p Value
Total population	255 330	255 840	255 720	–
COX-2 & Newer-NSAID use	15.0×10 ³	14.5×10 ³	14.1×10 ³	<0.001
Non-selective NSAID use	31.7×10 ³	32.4×10 ³	33.4×10 ³	<0.001
Proton pump inhibitors' use	75.1×10 ³	81.8×10 ³	86.6×10 ³	<0.001
NV-UGIB incidence	134.7	125.1	89.6	<0.001
<i>C difficile</i> incidence	122.3	104.4	72.7	<0.001

Conclusion The incidence of NV-UGIB has been falling despite the increasing use of ns-NSAIDs. This could be, at least in part, due to the rise in PPI use. Also, the incidence of *C difficile* infection has been falling despite more PPIs being used: this might suggest a poor link between these two factors, or might reflect the outcome of other measures being deployed against this infection. While these falls appear promising, both NV-UGIB and *C difficile* infection continue to represent significant clinical challenges.

Competing interests None declared.

PWE-159 THE EFFECT OF NATURAL POLYMORPHISM IN THE PROMOTER OF THE HELICOBACTER PYLORI CYTOTOXIN ASSOCIATED GENE, CAGA, ON CAGA TRANSCRIPTIONAL REGULATION

doi:10.1136/gutjnl-2012-302514d.159

A A Memon,* C K Marx, K Robinson, J C Atherton. Centre for Biomolecular Sciences, and Nottingham Digestive Diseases Centre Biomedical Research Unit, Queen's Medical Centre, University of Nottingham, Nottingham, UK

Introduction *Helicobacter pylori* persistently colonises the gastric mucosa of almost half the human population. It is the main causative agent of peptic ulcer disease and an important risk factor for