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appear to occur at the GOJ or distal 5 cm of the oesophagus. Sampling the GOJ and the distal 5 cm of the oesophagus in the absence of visible lesions may be adequate for surveillance.

Abstract OTU-016 Figure 1 The timeline of recurrent BO (overall; dysplastic; and HGD/cancer) following CRI

PTU-011 PREDICTORS OF MORTALITY AND REBLEEDING OUTCOMES AFTER PEPTIC ULCER BLEEDING

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Introduction Peptic ulcer disease (PUD) accounts for 25%–56% of acute upper gastrointestinal bleeding (AUGIB) cases and is associated with high rates of mortality and rebleeding. We aimed to assess the rates and factors associated with 1 year mortality and rebleeding in patients with bleeding PUD.

Methods This was a single-centre study of patients with AUGIB and endoscopic confirmation of PUD between November 2012–2014. All patients received at least 1 year of retrospective follow-up after endoscopy. Electronic records were scrutinised for outcomes of mortality and bleeding, with time-to-event analyses performed using a Kaplan-Meier plots and Cox-regression.

Results 91 patients (median age 78.4, 65.9% male) were included. 63.7% were admitted with AUGIB and 36.3% bled during their inpatient stay. Mortality at 30 days and 1 year were 12.1% and 34.1% respectively, with 1 year mortality comprising: cardiovascular (20.0%), bleeding (20.0%), other (33.3%), unknown (26.7%). On univariate analysis, predictors of 1 year mortality included inpatient bleeding (hazard ratio [HR] 2.38, 95% CI: 1.18–4.83, $p=0.016$) [Figure 1], age (HR 1.036 per increase, 95% CI 1.009–1.065, $p=0.009$), Forrest classification (HR 2.22 for class 1 and 2 vs. 3, $p=0.04$), Rockall Score (HR 1.64 per increase, 95% CI 1.28–2.10, $p<0.001$), Charlson index (HR 1.34 per increase, $p<0.001$), aspirin use (HR 3.05, $p=0.003$), rebleeding (HR 5.52, $p<0.001$). The effect of inpatient bleeding on mortality was not significant ($p=0.19$) after adjusting for Charlson index. *H. pylori* was positive in 35.7%; eradication was associated with reduced mortality even after adjusting for Forrest classification and age (HR 0.30, $p=0.007$). Multivariable analyses to

account for age are shown in table 1. The 1 year rebleeding rate was 7.8%. Higher haemoglobin on discharge (HR 0.940 per 10 g/dL increment, $p=0.04$), Forrest 3 ulcers (HR 0.18, $p=0.02$) and *H. pylori* eradication (HR 0.214, $p=0.02$) were significantly associated with reduced rates of rebleeding.

Abstract PTU-011 Figure 1 Survival Following Ulcer Bleeding

Abstract PTU-011 Table 1

Factor	Comparison	Hazard Ratio	95% CI	p-value
Inpatient AUGIB	Admitted with AUGIB	1.91	0.93–3.93	0.077
Forrest 1–2	Forrest 3	2.81	1.28–6.15	0.010*
Rockall Score	Per increase [§]	1.67	1.26–2.21	<0.001*
Charlson Index	Per increase [§]	1.30	1.07–1.58	0.008*
Aspirin on admission	No aspirin	2.07	0.92–4.67	0.081
<i>H. pylori</i> eradication	Not ascertained	0.26	0.11–0.61	0.002*

Conclusion Increasing age, higher Rockall and Charlson scores, Forrest 1 or 2 lesions, inpatient bleed, and rebleeding were factors associated with mortality in bleeding PUD. Higher rates of inpatient mortality may be explained by age and co-morbidities. Eradication of *H. pylori* was associated with improved outcomes and should be considered in all cases of bleeding PUD.

PTU-012 BIOPSY AVOIDANCE STRATEGY IN ADULT COELIAC DISEASE

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Introduction Paediatric ESPGHAN guidelines support a diagnosis of coeliac disease (CD) when immunoglobulin-A anti-tissue transglutaminase (IgA tTG) antibody titres are >10 times the upper limit of normal (ULN) and combined with supportive criteria. This study examines whether serological testing alone could be sufficient for diagnosis in adult patients, thus avoiding the need for duodenal biopsies.

Methods We performed a prospective analysis of CD patients diagnosed in a University hospital. Symptoms of CD, villous atrophy (VA) on biopsy, IgA-endomysial (IgA-EMA) antibodies, tTG and Human Leukocyte Antigen (HLA) genotype were used for analysis. We then compared the tTG antibody level against small bowel histology.

Results 443 CD patients (66.8% female, median age 41 years, range 15–84 years) were diagnosed between 2008 and 2016. 56.9% (n=252, 95% CI=52.12–61.53) had a tTG value of greater than 10 times the ULN. 292 fulfilled ESPGHAN guidelines for features of malabsorption (diarrhoea=157, weight loss=45 and anaemia=190). Of these symptomatic patients, 70.4% (n=179, 95% CI=64.86–76.08) had a tTG value 10 x ULN. The proportion reaching the 10 x tTG threshold was 55.4% (n=87, 95% CI=47.64–63.19) for diarrhoea, 60.0% (n=27, 95% CI=45.69–74.31) for weight loss, and 74.2% (n=141, 95% CI=67.99–80.43) for anaemia. Of the 151 patients who did not experience malabsorptive features, 49.0% met the 10 x ULN tTG level (n=74, 95% CI=41.03–56.98). The sensitivity of tTG antibodies and EMA antibodies for predicting VA was 93.2% (95% CI=90.89–95.57) and 90.7% respectively (95% CI=88.05–93.44). Combined tTG and EMA was 98.6% (95% CI=97.67–99.72). All patients had compatible HLA typing, thereby failing to add any further diagnostic value.

Conclusions 56.9% of patients would have been correctly diagnosed with CD and avoided a duodenal biopsy using an IgA tTG threshold of >10 times the ULN. Symptoms and HLA typing did not add any supportive information. This study provides evidence that a biopsy avoidance strategy may be implemented into adult gastroenterological practice.

PTU-013 IS SEROLOGY PREDICTIVE OF PERSISTING VILLOUS ATROPHY IN PATIENTS WITH ESTABLISHED COELIAC DISEASE (CD)?

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Introduction Recent work has suggested that an IgA-tissue transglutaminase (tTG) <1.2 U/mL may predict mucosal healing in those with established CD. This study examines whether the combination of serological markers may be used as a surrogate marker for the detection of villous atrophy (VA) in known CD patients.

Methods We undertook a prospective analysis of known Coeliac disease (CD) patients diagnosed in a University hospital. All patients underwent a gastroscopy, with four biopsies taken from the second part of the duodenum, and one from the duodenal bulb. Serological markers were assessed at the time of endoscopy tTG, IgA-endomysial antibodies (EMA), IgA-anti-gliadin antibody (IgA AGA) and IgG-anti-gliadin antibody (IgG AGA), and their performances in isolation and in combination compared to histological outcomes.

Results 107 patients (67.3% female, median age 53 years (20–81 years)) that were on a gluten-free diet for

a median duration of 6 years were included. The performance of the different serological markers to detect VA, both in isolation and in combination are shown in table 1. The performance of tTG using the previously used cut-off of <1.2 u/ml produced a sensitivity of 38.5%, a specificity of 73.8%, a

positive predictive value of 64.9% and a negative predictive value of 43.7% to detect VA.

Abstract PTU-013 Table 1 Ability of serological markers to detect VA

	Sensitivity	Specificity	Positive Predictive Value	Negative Predictive Value
tTG	39.0% (CI 29%–48%)	97.0% (CI 89%–99%)	88.9% (CI 64%–98%)	70.8% (CI 61%–81%)
EMA	40.5% (CI 26%–57%)	96.9% (CI 88%–99%)	89.5% (CI 65%–98%)	71.6% (CI 61%–80%)
IgA AGA	47.5% (CI 32%–64%)	95.5% (CI 87%–99%)	86.4% (CI 64%–96%)	75.3% (CI 65%–84%)
IgG AGA	37.5% (CI 23%–54%)	90.0% (CI 80%–96%)	68.2% (CI 45%–85%)	71.6% (CI 61%–80%)
EMA+tTG	33.3% (CI 20%–50%)	98.5% (CI 91%–100%)	93.3% (CI 66%–100%)	30.4% (CI 21%–41%)
IgA+IgG AGA	28.6% (CI 16%–45%)	100% (CI 93%–100%)	100% (CI 70%–100%)	68.4% (CI 58%–77%)
IgA or IgG AGA+EMA	28.6% (CI 16%–45%)	100% (CI 93%–100%)	100% (CI 70%–100%)	68.4% (CI 58%–77%)
IgA or IgG AGA+tTG	31.0% (CI 18%–47%)	100% (CI 93%–100%)	100% (CI 72%–100%)	69.1% (CI 59%–78%)

Conclusions This study is the first study to evaluate the combination of serological markers to detect VA in patients with established CD. Our findings oppose recent work that serology may be used as a surrogate marker of mucosal healing in known CD.

PTU-014 EFFECTS OF HELICOBACTER PYLORI INFECTION ON IRON METABOLISM GENES IN PATIENTS WITH IRON DEFICIENCY ANAEMIA

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Introduction There is a strong association between *Helicobacter pylori* (HP) infection and iron deficiency anaemia (IDA). The balance between iron uptake, storage and reutilisation is maintained by the liver hormone hepcidin. Paediatric studies have shown that HP disrupts iron function by inducing hepcidin. However, the effects of HP on iron metabolism and tissue hepcidin levels in adults remain controversial. Previously, we have shown by immunohistochemistry that the iron transport protein, ferroportin, was localised to the cytoplasm in the duodenal tissues from the HP-IDA group compared with the IDA group (70% versus 40% respectively) whereas in the control group ferroportin immunoreactivity was mainly localised to the membrane borders.¹ Hence, in this study, we aimed to investigate the tissue expression of hepcidin at the mRNA level and the effects of HP on iron metabolism in duodenal and gastric tissues from IDA patients and controls.

Methods Patients with HP-IDA, IDA and control groups (n=14/group) participated in this study, with ethics approval and informed consent. Duodenal and gastric biopsies were