Admission and 30-day mortality: Both PHT and NVL were independently predictive, with PHT having the stronger effect and with no significant interaction (table 1).

For transfusion, PHT was strongly predictive but there was a highly significant interaction (P<0.001) such that NVL was associated with increased odds of transfusion in patients without PHT and reduced odds in those with PHT (table 1). The percentages requiring transfusion were: no PHT/NVL 20%, NVL alone 33%, PHT alone 57%, PHT+NVL 44%.

Conclusions There is a mismatch between the transfusion needs and clinical outcome in patients with both PHT and NVL, which may indicate that the bleeding is in some cases attributable to the NVL alone while the clinical outcome is related to the combined risk factors.

Abstract PTU-069 Table 1 The clinical details and outcomes of NVUGIB in users of Antiplatelets (APs), Anticoagulants (ACs), and controls

<table>
<thead>
<tr>
<th></th>
<th>Control n=1976</th>
<th>Antiplatelets n=1039</th>
<th>Anticoagulants n=218</th>
<th>P value Overall</th>
<th>P value AP vs AC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years, median</td>
<td>54 (39–70)</td>
<td>75 (66–82)</td>
<td>75 (66–83)</td>
<td>&lt;0.001</td>
<td>0.92</td>
</tr>
<tr>
<td>Blatchford score</td>
<td>4 (1–8)</td>
<td>7 (4–11)</td>
<td>8 (4–11)</td>
<td>&lt;0.001</td>
<td>0.073</td>
</tr>
<tr>
<td>Endoscopy score</td>
<td>1 (0–2)</td>
<td>1 (0–2)</td>
<td>1 (0–2)</td>
<td>0.002</td>
<td>0.11</td>
</tr>
<tr>
<td>Admitted</td>
<td>1463</td>
<td>889 (85.6%)</td>
<td>203 (93.1%)</td>
<td>&lt;0.001</td>
<td>0.002</td>
</tr>
<tr>
<td>Transfused</td>
<td>436</td>
<td>408 (39.6%)</td>
<td>114 (52.5%)</td>
<td>&lt;0.001</td>
<td>0.001</td>
</tr>
<tr>
<td>Re-bleeding</td>
<td>186 (9.5%)</td>
<td>158 (15.3%)</td>
<td>54 (24.9%)</td>
<td>&lt;0.001</td>
<td>0.001</td>
</tr>
<tr>
<td>Died ≤ 30 days</td>
<td>61 (3.1%)</td>
<td>50 (4.8%)</td>
<td>13 (6.0%)</td>
<td>0.015</td>
<td>0.50</td>
</tr>
</tbody>
</table>

PTU-070 NON-SPECIFIC UPPER GI MURAL THICKENING ON CT – IS IT JUST FROM PERISTALYSIS?


Introduction BSG published guidance on the indications for diagnostic endoscopy in April 2013, including abnormal or suspicious findings on CT imaging. Non-specific upper GI mural thickening on CT is a common abnormal finding raising the suspicion of upper GI malignancy. The correlation between CT mural thickening in the upper GI tract and endoscopic diagnosis of malignancy is not clearly known.

Methods A retrospective single centre study of patients referred for gastroscopy with the indication of ‘abnormal imaging’ (n=147) was performed. Data was collected using the endoscopy software audit tool over a 3-year period (2016 to 2018). Patients with a CT reported finding of ‘mural thickening’ were included for analysis (n=59). Statistics were performed using Welch’s t-test.

Results 59 patients underwent gastroscopy for CT reported mural thickening: oesophageal 20 (34%), GOJ 9 (15%), gastric 23 (39%), pyloric 4 (7%), duodenal 5 (8%) and jejunal 1 (2%). Median time from CT to endoscopy was 21 days (IQR 12–54). Median age was 77 (IQR 62–83). Initial indication for CT scan included: weight loss 16 (27%), abdominal pain 14 (24%), possible malignancy 6 (10%) and dysphagia 3 (5%).

11 (19%) patients had a normal gastroscopy, 24 (41%) showed inflammatory changes (oesophagitis or gastritis), 20 (34%) had evidence of a hiatus hernia, and 5 (8%) had benign polyps.

5 (9%) had a histological diagnosis of gastric adenocarcinoma, 4 (7%) of Barrett’s oesophagus and 1 (2%) of squamous dysplasia. The 5 patients with adenocarcinoma could not be reliably identified by indication for imaging (2 for abdominal pain, 1 for weight loss, and 2 for non GI or systemic related symptoms).

The mean haemoglobin for the patients with malignancy was 104 g/L vs 125 g/L for the overall study group (p=0.13, NS). The mean albumin for the patients with malignancy was
37.6 g/L vs 38.4 g/L for the overall study group (p=0.81, NS).

Conclusions Upper GI mural thickening on CT cannot be dismissed. Despite oesophaestis, gastritis and hiatus hernia making up most endoscopic diagnoses (75%), it correlated with malignancy, dysplasia or metaplasia in 10/59 (17%) patients in this study. Patients with malignancy could not be accurately differentiated by indication for imaging or by biochemical markers. We conclude that there is good concordance in pathology detection at gastroscopy following findings of thickening on CT scan. We recommend gastroscopy is performed in every case when this abnormality is detected incidentally.

PTU-071 HELICOBACTER PYLORI BREXIT: NICE VS MAASTRICHT A COMPARISON OF ERADICATION GUIDELINES AND RESISTANCE IN LONDON
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10.1136/gutjnl-2019-BSGAbstracts.287

Introduction The Public Health England (PHE) advises HP testing and treatment if positive, in patients without alarm symptoms who have uncomplicated dyspepsia unresponsive to lifestyle change and antacids. However, the suggested antibiotic therapy is incongruent with latest Maastricht V/Florence guidelines which suggest bismuth containing quadruple therapy first line in regions with high metronidazole and clarithromycin resistance and second line routinely. In London, a study showed overall resistance to metronidazole at 59% and clarithromycin at 11% with non-UK birth being main risk factor.2 Between 2016 and 2018 was generated. A list of all gastroscopies with CLO testing over a 2-year period from January 2016 to December 2018 was generated.

Results Between October 2017 and September 2018, the endoscopy unit performed 1375 CLO tests out of 5000 gastroscopies. A review of 100 patients who had CLO testing showed that they were all appropriate.

Conclusion Patients in whom we send HP resistance testing have high resistance rates to conventional first line antibiotics. Given that these patients are likely to have previously failed at least one treatment regime; the results are inevitable skewed towards resistant isolates. While NICE and PHE guidelines are appropriate for some populations, areas of London which are at risk of higher resistance rates should use Maastricht guidelines. Therefore, we recommend a 10-day course of bismuth-containing quadruple therapy as second line.

REFERENCES

PTU-072 A MULTI-CENTRE REVIEW OF ACUTE UPPER GI BLEEDING; CAN BLOOD UREA LEVELS AID DIAGNOSIS?
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10.1136/gutjnl-2019-BSGAbstracts.288

Introduction Risk scoring for acute upper gastrointestinal bleeding (AUGIB) is key when assessing patients for requiring OGD. The Rockall score utilises age, comorbidities and shock. The Glasgow-Blatchford score (GBS), in addition, utilises haemoglobin, melaena, and blood urea levels. Raised blood urea levels can represent digestion of blood from the upper gastrointestinal tract giving rise to melaena; the presence of both gives a high GBS. However, inexperienced health care professionals can misinterpret the absence/presence of melaena, raised urea levels may be due to kidney injury. Nevertheless, Gastroenterologists may use urea to diagnose AUGIB if patients haven’t had overt/witnessed/reliably reported haematemesis or melaena.

It has been shown that a raised urea:creatinine ratio (URCR) can be associated with AUGIB and may be superior to urea alone as it mitigates for kidney injury. However, URCR is not widely used in the UK in the assessment of AUGIB. We aim to assess the association of urea and URCR levels with AUGIB.

Methods A retrospective review at three UK centres (Kettering General Hospital, Queen Elizabeth Hospital Birmingham, and University Hospital Coventry & Warwickshire) was undertaken. Endoscopy reports and blood tests were reviewed of patients undergoing inpatient OGD for suspected AUGIB within 2017/8, data were recorded in an Excel spreadsheet. URCR was calculated by dividing Urea by creatinine, and multiplying by 1000 (normal = 100). Statistics were analysed using SPSS.

Results 357 patients’ records were reviewed (median age = 68), 179 had a plausible AUGIB (50.1%). Receiver operator characteristic (ROC) curves for Urea gave an area under the curve (AUC) = 0.733. For URCR, AUC = 0.789.

Binary logistic regression modelling was performed using age, urea, and URCR. χ² (3, n = 357) = 102.92, p<0.001. 25–34% of the variance in AUGIB is explained by the model. The model URCR value of 97.7 can be used to predict AUGIB, applying this to our data set correctly identifies 124/179 patients with AUGIB (69.3%), and is predicted to correctly identify 74.5%.

Conclusion This pilot study has limitations as bleeding lesions may have not been identified at OGD. Urea and URCR have AUCs of 0.733, & 0.789. Logistic regression modelling suggests a URCR level of 100 would correctly identify ~70% of AUGIB in patients with suggestive symptoms. Outside of firm