**Management of acute bleeding upper gastrointestinal ulcers in the era of endoscopic and intravenous proton pump inhibitor therapy**

Alexandra Ilnyckyj MD FRCPC, George Mathew MD, FRCPC

**BACKGROUND:** Despite advances in therapy, the morbidity and mortality of gastrointestinal bleeding remains high. A review of current practice patterns was undertaken in St Boniface General Hospital, Winnipeg, Manitoba to assess the standard of care provided.

**OBJECTIVE:** To determine whether upper gastrointestinal bleeding (UGIB) attributable to a single acute ulcer is treated appropriately with respect to local therapy, acid suppression and Helicobacter pylori status.

**METHODS:** A retrospective chart review identified patients with consecutive acute UGIB attributable to a single gastric or duodenal ulcer presenting to a tertiary care centre over a six-month period. The lesions were classified as high- or low-risk based on endoscopic appearance. Local care of the ulcer, acid suppressive therapy and management of *H. pylori* were determined by reviewing pertinent chart materials. Appropriate care was defined for all three end points a priori using literature-supported standards of care.

**RESULTS:** Fifty-five patients who met study criteria were identified. Twenty-six of 55 (47%) and 29 of 55 (52%) lesions were considered to be low- and high-risk respectively. Of the high-risk lesions, two of 29 received no local therapy, 24 of 29 received adrenaline injection and two had an endoclip placed. Of the 27 patients whose lesions were treated, 16 received thermal coagulation. Twenty-five of 29 (88%) received intravenous proton pump inhibitors. Thirty-three of 55 (55%) patients did not have *H. pylori* status considered in their management.

**CONCLUSION:** Weaknesses in the management of UGIB were identified, particularly with respect to addressing the role of *H. pylori*. Fragmentation and compartmentalization of patient care may be important contributing factors.

**Key Words:** Management; Peptic ulcer disease

**REVIEW OF GIB MANAGEMENT**

**Local therapy**

Endoscopic treatment is not required in clean-based ulcers (5-7). Intervention on lesions with active bleeding, a visible vessel or adherent clot should be undertaken. Treatment of active bleeding has been shown to be well treated with combination therapy of adrenaline injection (diluted with saline 1:10000) and thermal coagulation (8,9). However, in the treatment of...
Helicobacter pylori

All patients presenting with an ulcerative lesion of the upper GI tract should be examined for the presence of Helicobacter pylori infection. The most sensitive and specific testing is accomplished through histological examination of a gastric biopsy taken at the time of endoscopy. The sample can be tested for urease production if H. pylori is not identified by the pathologist. A less desirable approach consists of measuring antibody levels against H. pylori. All patients who test positive for H. pylori in the setting of an acute ulcer presentation should receive eradication therapy at some point.

Acid suppression therapy

Studies support the use of intravenous proton pump therapy in the high-risk lesion that has been endoscopically treated (14-19).

*Helicobacter pylori*

Lesions with a visible vessel, combination treatment has not been shown to be more effective than using thermal coagulation alone (8,9). There is ongoing debate regarding the ideal management of lesions with an adherent clot (10-13).

**Acid suppression**

Studies support the use of intravenous proton pump therapy in the high-risk lesion that has been endoscopically treated (14-19).

**METHODS**

There are two tertiary teaching hospitals in Winnipeg, Manitoba (population 750,000). Both centres have a dedicated GI bleed service which provides 24 h consultation and endoscopic intervention for patients presenting with acute GIB. The service is staffed by gastroenterologists and general surgeons. Patients presenting with GIB requiring hospitalization are admitted under the care of a clinical teaching unit, either medical or surgical. The GI bleed service functions in the capacity of a consultative team.

A retrospective chart review for a six-month period dated January to June 2002 was undertaken. **International Classification of Diseases, Ninth Revision (ICD-9)** (20) codes on patient discharge summaries were used by the medical records department to extract charts. The ICD-9 codes extracted included GI bleed (578), peptic ulcer disease (536), gastric ulcer (531) and duodenal ulcer (532).

Charts were reviewed by a senior GI Fellow and data were collected using a standardized method. The Fellow reviewed the entire hospital chart with specific attention to the GI bleed team consultation, the endoscopy report, emergency room record if present, doctors orders, chart notes made by the GI bleed team, medication records, discharge summary, discharge recommendations and pathology and microbiology reports.

To meet entry criteria, the endoscopy report had to document an acute gastric or duodenal ulcer as the source of the bleeding. Patients whose source of bleeding were diagnosed as a nonulcer source, multiple sources or unknown source were excluded.

**AIM**

The aim of the present study was to determine whether treatment of UGIB attributable to acute ulcers was consistent with contemporary management practices (4).

**RESULTS**

Two hundred eighty charts were identified by medical records which fulfilled the previously stated ICD-9 codes during the study interval. A review of these charts identified 55 patients that met entry criteria; specifically, an acute GI bleed referred to the GI bleeding service which was attributable to a gastric or duodenal ulcer. Of the 55 patients, 42 were treated by a gastroenterologist and 13 by a surgeon.

Twenty-four of the 26 patients diagnosed with clean-based (low-risk) ulcers received no endoscopic intervention; two received endoscopic therapy (injection) (Table 1). Twenty-four of the 29 patients diagnosed with high-risk lesions were treated with regard to the three issues relevant to the management of UGIB; specifically:

- local treatment (source document: endoscopy report);
- acid suppressive therapy (source documents: physician order sheets and drug administration sheets); and
- *H. pylori* testing and eradication (source documents: GI consultation record, pathology report, microbiology report, drug administration sheets, chart progress notes, discharge summary and discharge recommendations to primary care physician).

High-risk lesions were defined as lesions with adherent clot, visible vessel or actively bleeding ulcer. Low-risk lesions included clean-based ulcers or ulcers with heme stains.

**TABLE 1**

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Total (n=55)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low risk, n (%)</td>
<td>26 (47)</td>
</tr>
<tr>
<td>No treatment</td>
<td>24 of 26 (92)</td>
</tr>
<tr>
<td>Injection adrenaline</td>
<td>2 of 26 (8)</td>
</tr>
<tr>
<td>High risk, n (%)</td>
<td>29 (52)</td>
</tr>
<tr>
<td>No treatment</td>
<td>2 of 29 (7)</td>
</tr>
<tr>
<td>Injection adrenaline</td>
<td>24 of 29 (83)</td>
</tr>
<tr>
<td>Thermal coagulation</td>
<td>16 of 27 (55)</td>
</tr>
<tr>
<td>Endoclip</td>
<td>2 of 29 (7)</td>
</tr>
</tbody>
</table>

**TABLE 2**

**Acid suppression**

<table>
<thead>
<tr>
<th>Acid suppression</th>
<th>Total (n=55)</th>
</tr>
</thead>
<tbody>
<tr>
<td>IV PPI for high-risk ulcer, n (%)</td>
<td>25 of 29 (88)</td>
</tr>
<tr>
<td>IV PPI for low-risk ulcer, n (%)</td>
<td>5 of 26 (19)</td>
</tr>
<tr>
<td>PPI therapy total (oral or IV), n (%)</td>
<td>53 of 55 (96)</td>
</tr>
</tbody>
</table>

*IV PPI Intravenous proton pump inhibitor*
inhibitor therapy intravenously (Table 2). The intravenous dosage was a bolus of 80 mg of pantoprazole followed by an intravenous infusion of 8 mg/h. The duration of the infusion was not reviewed.

The all-cause mortality in the present study was 11% (six of 55 patients). All deaths occurred in patients who suffered with multiple medical comorbidities. The mean age of these six patients was 78 years. The mean time to death was 42 days after admission (range two to 115 days).

**DISCUSSION**

The present review overwhelmingly demonstrates that physicians are treating UGIB attributable to an acute ulcer appropriately with respect to local therapy and acid suppression. Unfortunately, _H pylori_ testing and treatment is not maximized. Failure to undertake biopsy acquisition for _H pylori_ has been described by other workers (21). In this national database of 8000 patients with acute upper gastrointestinal ulcers, 33% did not have biopsies taken for _H pylori_. Of note, the database included only patients with nonbleeding acute ulcers, whereas all of our patients were presenting with bleeding ulcers. This may explain the higher rate of nonacquisition in our study.

The omission of _H pylori_ biopsy acquisition is especially disappointing because eradication of _H pylori_ is the greatest advancement in peptic ulcer disease management to date. We speculate that this may be attributable to compartmentalized care. During presentation, UGIB management is focused on volume repletion and stabilization of the patient. During endoscopy, local therapy is foremost; acquisition of the biopsy for _H pylori_ may be undermined. The disparity between the national database review and our own data support this speculation. Thereafter, hospitalization may not afford opportunities to review the issues pertinent to UGIB. Either short or prolonged hospital stay may detract from the importance of _H pylori_ testing and treatment if _H pylori_ biopsy was not obtained during the gastroscopy. As well, fragmentation of care among the endoscopist, the admitting physician and the family physician may further contribute to omission of _H pylori_ testing and treatment.

**CONCLUSIONS**

Despite the management of acute GIB by a dedicated bleed service, our review revealed significant weaknesses in the management of this common and costly disease. High-risk lesions are not uniformly treated with endoscopic interventions. Of greater magnitude is the failure in acquiring an _H pylori_ biopsy, or undertaking serological testing or advising empirical therapy in those in whom biopsy was not obtained. Compartmentalization and fragmentation of care may contribute to oversight. Regardless, the GI bleed consultant must be vigilant to ensure _H pylori_ status and treatment is addressed because the organism is integral to the pathogenesis of peptic ulcer disease.

**REFERENCES**

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