INTRODUCTION

Gastrointestinal (GI) bleeding in older adults is associated with increased morbidity and mortality than in the young, in part attributable to increased co-morbid illnesses, and greater medication use of ulcerogenic medications such as aspirin and other non-steroidal anti-inflammatory drugs (NSAIDs). The goal of this paper is to discuss the various etiologic factors, clinical presentations and management options of upper and lower GI bleeding in older adults. For the purpose of this paper, an “older” adult is defined as age greater than 60. The various terms used in the description of GI bleeding, although well known, are tabulated (Table 1).

UPPER GASTROINTESTINAL BLEEDING (UGIB)

Epidemiology

In spite of considerable advances in clinical medicine, UGIB is common and continues to be associated with a significant morbidity and mortality. The annual incidence of hospitalizations for UGIB has been estimated to be 100–150 per 100,000 population resulting in over 300,000 hospitalizations (1,2). UGIB is approximately five times more common than lower gastrointestinal bleeding (LGIB) and is more common in men and older adults (1,3). Over the last 50 years there has been a striking increase in the proportion of older patients presenting with UGIB. It is estimated that 35%–45% of all patients presenting with UGIB are over the age of 60 (4,5). This number is expected to grow as more Americans live longer, a fact that can be further appreciated as we review data from the US census bureau. Current estimates show that 12.4% or 36 million of the total population of 293 million Americans are over the age of 65. The number is projected to increase to nearly 70 million or approximately 20% of the entire population by 2030.

Etiologic Factors for UGIB

Peptic ulcer disease (PUD) remains the most common cause of UGIB in older adults along with erosions and varices accounting for more than 90% of hospitalizations for UGIB (Table 2). In 1981, Antler and Pitchumoni reported that peptic ulcer disease was more common where as Mallory-Weiss tears were less frequent in older patients when compared to the young (6). These results have been recently reproduced by Segal and Cello (7). The incidence of alcohol abuse is lower in the elderly accounting for a decreased incidence of esophageal varices and Mallory-Weiss syndrome. Over the last 20 years there has been a significant increase in the number of patients presenting with UGIB while on NSAIDs or aspirin (8). Musculoskeletal disor-
Gastrointestinal Bleeding in Older Adults


ders are amongst the most common causes of morbidity in the elderly with NSAIDs often used to alleviate pain; aspirin is commonly used for primary and secondary prevention of cardiovascular or cerebrovascular disease in this age group. It is estimated that more than 50% of older adults are either using an NSAID or aspirin when they present with a bleeding episode (8–10). Mortality secondary to NSAID related gastrointestinal toxicity has an annual relative risk of 4.1 when compared to NSAID non-users (11). Introduction of selective cyclooxygenase-2 inhibitors (COX-2) has been shown to decrease gastrointestinal ulcers and bleeding in randomized control trials (12–14). Following these reports, use of selective COX-2 inhibitors has soared over the last 5 years. In 2000, they accounted for one third of all United States NSAID prescriptions and 60% of NSAID costs (15). They account for a world wide sale of approximately $10 billion (16). The incidence of NSAID induced GI lesions may rise if the use of selective COX-2 inhibitors diminishes as a result of recent reports of increased incidence of myocardial infarction and the use of non-selective anti-inflammatory drugs increases (16–18).

**Presentation**

Most older patients with acute UGIB present with either hematemesis or melena or both. Cutaneous as well as visceral pain sensitivity has been shown to decrease with age (19). NSAID use partly decreases the pain from PUD and combined with decreased visceral sensitivity may be the reason why up to one-third of elderly patients with endoscopically detected ulcers

**Table 1**

Terms and their significance

**Hematemesis:** Vomiting of blood. It indicates a source of bleeding proximal to the ligament of Treitz. It may consist of bright red blood indicative of active bleeding or coffee ground material from bleeding in the recent or remote past.

**Melena:** Passage of black, tarry, foul smelling stools as a result of degradation of blood to hematin. It takes the presence of at least 50 cc of blood in the UGI tract to present as melena. The source of bleeding may be the UGI tract, distal small bowel or right colon (early and slow bleeding).

**Hematochezia:** Rectal passage of bright red blood with or without stool. The source often is the lower GI tract but brisk UGI bleeding can cause hematochezia too.

**Occult bleeding:** Bleeding not apparent to the patient. Usually detected on stool guiac.

**Obscure bleeding:** Source of bleeding is difficult to pin point on routine endoscopic examination.

<table>
<thead>
<tr>
<th>Study</th>
<th>Peptic Ulcer(%)</th>
<th>Erosions (gastitis/ esophagitis/ duodeni) (%)</th>
<th>Varices (gastric/ esophageal) (%)</th>
<th>Mallory-Weiss tear (%)</th>
<th>Neoplasms (%)</th>
<th>Other/ unknown (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cooper 1988 (84) N = 103 Age &gt; 80</td>
<td>47</td>
<td>27</td>
<td>2</td>
<td>2</td>
<td>4</td>
<td>18</td>
</tr>
<tr>
<td>Segal 1997 (7) N = 100 Age &gt; 60</td>
<td>73</td>
<td>18</td>
<td>11</td>
<td>3</td>
<td>1</td>
<td>N/A</td>
</tr>
<tr>
<td>Thomopoulos 2004 (8) N = 636 Mean age 62.9</td>
<td>59</td>
<td>8.5</td>
<td>13.2</td>
<td>4.9</td>
<td>3</td>
<td>11.4</td>
</tr>
</tbody>
</table>

(continued on page 19)
do not report abdominal pain (7,20). The absence of pain is more obvious in the subset of elderly females using NSAIDs. Nearly 50% of these patients with a proven ulcer did not report abdominal pain as a symptom (21). The absence of abdominal pain is clinically significant because it delays the diagnosis until the development of complications such as hemorrhage, perforation or pyloric stenosis, which are associated with high mortality rates (22,23). Concurrent anticoagulation therapy, often used for thromboembolic prophylaxis in the geriatric population, increases the risk of bleeding from PUD.

As expected, the elderly are more likely to have comorbid illnesses such as cardiovascular disease, chronic obstructive pulmonary disease and renal insufficiency at presentation (7). After hemorrhage, presence of serious concurrent illness is the second most important factor in predicting mortality among patients with UGIB (22). Additionally, the diagnosis may be further complicated due to lack of clinical history secondary to impaired cognitive function in the very elderly. Signs to assess the severity of bleeding, such as orthostasis, may be difficult to elicit in bed-ridden patients. Further, orthostasis may

(continued from page 16)

Table 3
Antibiotic prophylaxis for patients with UGIB

<table>
<thead>
<tr>
<th>Patient condition</th>
<th>Procedure planned</th>
<th>Prophylaxis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>High risk conditions</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prosthetic cardiac valves</td>
<td>Stricture dilation, Variceal sclerotherapy</td>
<td>Recommended</td>
</tr>
<tr>
<td>Previous bacterial endocarditis</td>
<td></td>
<td></td>
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<tr>
<td>Surgically created systemic pulmonary shunts/conduits</td>
<td></td>
<td></td>
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<tr>
<td>Synthetic vascular graft less than 1 year old</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Intermediate risk conditions</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Most other congenital abnormalities</td>
<td>Stricture dilation, Variceal sclerotherapy</td>
<td>Optional</td>
</tr>
<tr>
<td>Acquired valvular dysfunction (e.g. Rheumatic heart disease)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertrophic Cardiomyopathy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mitral valve prolapse with regurgitation or thickened leaflets</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Low Risk conditions</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other cardiac conditions (CABG, repaired septal defect or patent ductus arteriosus, mitral valve prolapse without valvular regurgitation, isolated secundum atrial septal defect, physiologic/functional/innocent heart murmurs, rheumatic fever without valvular dysfunction, pacemakers, implantable defibrillators)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Cirrhosis</strong></td>
<td>All other endoscopic procedures</td>
<td>Recommended</td>
</tr>
<tr>
<td><strong>Prosthetic joints</strong></td>
<td>All other endoscopic procedures</td>
<td>Not recommended</td>
</tr>
</tbody>
</table>

Recommended regimens include amoxicillin 2.0 gm PO 1 hour or IV 30 minutes prior to procedure. For penicillin allergic patients clindamycin 600 mg PO 1 hour or IV 30 minutes before procedure is recommended.

Data modified from ASGE guidelines for antibiotic prophylaxis for GI endoscopy (86).

(continued on page 23)
not be a reliable measure of volume loss in many diabetic patients with autonomic dysfunction, patients with neurologic disorders and those taking beta-blockers. For these patients, orthostasis may lose its value as a sign of significant blood loss, thereby misleading the clinician to underestimate the severity of UGI hemorrhage.

Evaluation and Management
Initial resuscitation efforts can be paramount in the clinical course and outcome of patients with GI bleeding. Following history and focused physical examination, a protected airway and adequate oxygenation should be confirmed. Despite the inconsistencies surrounding orthostasis, circulatory status should quickly be evaluated with measurement of supine and standing pulse and blood pressure, the latter if there are no contraindications. Presence of orthostasis and shock implies a 20% to 40% loss of circulatory volume respectively. Two large bore intravenous catheters should be placed and volume resuscitation initiated with crystalloid solution. Initial studies should include a complete blood count, comprehensive metabolic profile, blood typing and cross matching, cardiac markers, stool for occult blood, electrocardiogram and a chest roenterograph. Patients should be empirically treated with an intravenous proton pump inhibitor (PPI) and need for antibiotic therapy assessed (Table 3). When variceal hemorrhage is suspected, patients should also be treated with intravenous octreotide in addition to PPI and antibiotic therapy. A naso-gastric tube should be inserted and the stomach lavaged with water. Presence of a bright red aspirate or clots that fail to clear after approximately 500 cc of water is a sign of ongoing bleeding and warrants very close monitoring in an ICU setting (Figure 1).

Following initial resuscitation, evaluation of all patients should start with a detailed history and physical examination. This is especially important in geriatric
patients, where the history is clouded or complicated by the presence of visual, auditory and cognitive impairment and other co-morbidities. In such a situation it may be necessary to call the primary provider and/or care giver, and perhaps the pharmacist to help piece together components of the history such as extent of bleeding, duration of symptoms, presence of co-morbid illnesses, prior surgical history, known drug allergies and most importantly recent or current medication usage, especially aspirin, clopidogrel, warfarin and compounds containing NSAIDs. A repeat physical examination should be performed with emphasis on orthostasis, signs of cardiopulmonary compromise, stigmata of chronic liver disease and evidence of coagulopathy. A mini mental status exam as a measure of cognitive function may be indicated on admission or later if feasible.

Esophagogastroduodenoscopy (EGD) is the next step in identifying the source of bleeding once the patient’s general condition is stabilized and informed consent is obtained. The latter may be difficult in the subgroup of patients who suffer from cognitive decline. An individual without capacity cannot sufficiently participate in the informed consent process. With the exception of a true life-threatening emergency, every attempt should be made to obtain consent from the patient or surrogate. In the rare case when a guardian cannot be reached, administrative consent should be obtained if time permits (24).

Preparation for endoscopy in the elderly differs little from that in younger adults. For elective EGD, solids should not be ingested within eight hours but clear liquids can be taken up to 5 hours prior to the procedure. Older patients are more likely to have pacemakers and/or defibrillators given the high incidence of cardiovascular diseases in this age group. Recommendations for management of patients with pacemakers and internal defibrillators are not well defined. Cardiology consultation may be indicated. Pacemaker dependent patients should be driven to automatic pacing by placing a magnet on the skin overlying the device whenever monopolar electrosurgical devices are being used. Those who are not in a continually paced rhythm should be monitored, with a magnet available for continuous pacing if needed. If the status of the patient’s rhythm is not known, a magnet should be used during electrocautery. Intra-cardiac defibrillators should be inactivated prior to the use of electrocautery. This must always be done with the use of continuous rhythm monitoring until the defibrillator is reactivated following the procedure. Alternative means of tissue removal, destruction, or hemostasis can be used to simplify management of patients with defibrillators (Table 4). The universal principle in geriatric pharmacology of “Start low and go slow” also holds true for medications used for conscious sedation.

Table 4
Endoscopic control of UGIB

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Thermal coagulation</td>
<td>Applied directly to bleeding point</td>
</tr>
<tr>
<td>2. Injection of epinephrine/sclerosing agents</td>
<td>Sclerosants such as polidocanol or ethanol are popularly used in USA</td>
</tr>
<tr>
<td><strong>First two procedures may be combined to achieve better hemostasis</strong></td>
<td></td>
</tr>
<tr>
<td>4. Band ligation</td>
<td>Effective in varices, Dieulafoy’s lesion, AV malformations and Mallory Weiss tears</td>
</tr>
<tr>
<td>5. Argon Plasma coagulation</td>
<td>For non-contact coagulation. Ideal for lesions with large surface areas, i.e. watermelon stomach and portal hypertensive gastropathy</td>
</tr>
</tbody>
</table>

(continued on page 27)
during endoscopy. As in younger adults, midazolam and/or narcotics are generally used. Initial dosages should be lower and titration should be more gradual (25). IV sedation guided by ASA criteria and administered by an anesthesiologist is preferred in the elderly. Endoscopic treatment performed early has a great impact on reductions in the length of stay, risk of re-bleeding and surgery (26).

Clinical Course and Outcome

Age is traditionally identified as an independent risk factor for re-bleeding and mortality. It is an integral part of the Rockall scoring system, Baylor bleeding score and the Cedars-Sinai Medical Center index used for predicting the outcome of patients with UGI hemorrhage (27–29). Two independent studies from Europe showed age >70 and 80 to be associated with significantly higher morbidity and mortality (30,31). However, data from Segal and Cello showed no significant difference in hospital course, need for ICU care, transfusion requirements, length of stay or mortality between older adults and their younger counterparts (7). This may be secondary to differences in patient characteristics in Europe and USA. As in many other illnesses, age alone is not a bad prognostic factor, however presence of other co-morbidities is clearly identified to be associated with increased mortality following UGIB. Mortality rates for UGI hemorrhage with concomitant cirrhosis, acute renal failure, ventilator dependent respiratory failure and congestive heart failure range from 25%–65% (4,32–34).

Severity of hemorrhage and recurrent hemorrhage are major causes of death in patients with UGIB. Hemodynamic instability presenting as systolic blood pressure <100 mm Hg, hemoglobin <10 mg/dL, tachycardia with or without orthostasis, bleeding manifesting as hematemesis or hematochezia and failure of blood in gastric aspirate to clear with lavage are early indicators of severe UGIB and adverse outcomes (35). Predictors of recurrent hemorrhage include patient characteristics, size and location of the bleeding lesion and certain typical endoscopic findings. Onset of UGIB in the hospital; especially in the ICU setting, concomitant multi-organ failure and the presence of coagulopathy are some factors indicative of the possibility for re-bleeding. Similarly, endoscopic findings of large ulcers and ulcers in the posterior duodenal bulb have a high probability of re-bleeding. Active bleeding, non-bleeding visible vessel and adherent clot, when seen on endoscopy, also prognosticate re-bleeding risk (Table 5). Flat spots and ulcers with clean base have a very low risk of re-bleeding (36). A recent prospective study of elderly patients with UGIB demonstrated that patients with no early or endoscopic factors predictive of increased risk of re-bleeding and having good social support were successfully managed as outpatients and none re-bled (37).

It is unclear whether the need for surgery for UGIB increases with age. There is evidence supporting decreased mortality and better outcomes for older patients when surgery is performed early rather than

Table 5
Factors predicting mortality and re-bleeding in elderly patients with UGIB

<table>
<thead>
<tr>
<th>Early predictive factors</th>
<th>Risk of re-bleeding (%)</th>
<th>Predicted mortality (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Hemodynamic instability (Systolic blood pressure &lt;100 mm HG, Hemoglobin &lt;10 mg/dL and tachycardia with or without orthostasis)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Presentation as hematemesis or hematochezia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Bloody NG aspirate that does not clear following lavage</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Concomitant liver, renal, cardiac or pulmonary failure (acute or chronic)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Coagulopathy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• In-hospital onset of UGIB</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(The above are some of the indications for ICU admission)</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Endoscopic factors</th>
<th>Risk of re-bleeding (%)</th>
<th>Predicted mortality (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Ulcer base &gt;2 cm in diameter</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Posterior duodenal bulb ulcer</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Endoscopic factors of adverse outcome A, B, C also help in triaging patients to ICU. Data from 36, 87–90
later in the course of UGI hemorrhage (38,39). Therefore, the decision to perform surgery in older patients should be made sooner rather than later. Operative intervention should be considered in patients at high risk of re-bleeding based on criteria identified above and those with bleeding refractory to endoscopic therapy.

LOWER GASTROINTESTINAL BLEEDING (LGIB)

Epidemiology
The incidence of LGIB ranges from 20.5 to 27 per 100,000 population per year (3). It is more common in men and the incidence rate increases proportionally with age, with a greater than 200 fold increase from the third to the ninth decade of life (3). This can be explained on the basis of an increased incidence of diverticular disease, colonic neoplasms, angiodysplasia and ischemic colitis in the aging population. Once again, the incidence of LGIB can be expected to rise as more Americans live longer based on the projections of the US census bureau.

Etiologic Factors for LGIB
The prevalence of various etiologies of LGIB vary widely based on the population studied and the diagnostic modalities used in individual studies. For instance, when colonoscopy was used as the primary diagnostic tool, a small bowel source of hemorrhage was identified in only 1%-9% of cases. However, when angiography was used, up to one-third of the hemorrhagic sources were localized in the small bowel. Despite these variations, diverticular disease, colitis (inflammatory, radiation, ischemic and vasculitic), neoplasms and angiodysplasias are responsible for greater than 60% of all cases of LGIB (40,41) (Table 6). It is worth noting that approximately 10%–15% of patients presenting with hematochezia may have an UGIB source of hemorrhage (42).

Diverticulosis
A diverticulum is a sac-like protrusion of the colonic wall. The prevalence of diverticuli increases from less than 5% at age 40 to 65 % by age 85 (43). Although most patients with diverticulosis are asymptomatic, approximately 3%–5% develop acute hematochezia (44). In the elderly, diverticular hemorrhage carries a morbidity and mortality of 10%–20%. Risk factors for hemorrhage include, aspirin or NSAID use, lack of dietary fiber, constipation and age (45,46).

Meyers, et al first described the pathogenesis of diverticular hemorrhage. As a diverticulum herniates, the vasa recta responsible for the wall weakness get draped over the dome of the diverticulum, “and is thus separated from the lumen of the diverticulum by only mucosa and a few strands of attenuated muscle fibers (47).” If a vas rectum ruptures secondary to injurious factors such as NSAIDs or hard stools, intra-luminal...
hemorrhage ensues. In western countries about 90% of diverticuli are left sided, however it is frequently quoted that 50%–90% of bleeding occurs from right colonic lesions (48). Right-sided diverticuli have wider domes and necks exposing the vasa recta to injury over a greater length of the vessel. This may explain the increased frequency and severity of right-sided diverticular hemorrhage.

Most diverticular bleeding ceases spontaneously and less than 1% of patients require greater than 4 units of transfused blood (44). For persistent bleeding, angiographic treatment with vasopressin infusion or embolization of the bleeding vessel is successful in about 90% of cases. However, up to 50% of patients re-bleed on cessation of vasopressin and approximately 20% develop intestinal infarction following embolization (49). Colonoscopic hemostasis of diverticular bleeding is possible using bipolar probe coagulation, epinephrine injection, metallic clips and fibrous glue. The UCLA/CURE approach is to do a rapid colonic purge followed by urgent colonoscopy. Jensen and Machicado reported no re-bleeding during a thirty month follow-up period after endoscopic therapy with the above modalities compared to a 53% re-bleeding rate in patients receiving conservative medical therapy alone (50).

Surgery may be necessary in up to 25% of patients with diverticular disease requiring blood transfusion (51). A positive pre-operative angiogram reduces the risk of re-bleeding (49). Blind segmental resection practiced in the past is not recommended, as it is associated with high re-bleeding rates (47%) and very high morbidity and mortality rates (83% and 57% respectively) (52). Despite improved methods of localization of bleeding and targeted segmental resection, mortality remains as high as 10% (49).

**Vascular Ectasias (Angiodysplasias or Arteriovenous Malformations)**

Vascular ectasias are degenerative lesions of previously normal blood vessels in the cecum and proximal ascending colon. They are unrelated to much less common vascular lesions such as telangiectasias of Osler Weber Rendu disease (hereditary hemorrhagic telangiectasias), hemangiomas and congenital arteriovenous malformations. Vascular ectasias on careful histopathologic examination are noted to be estatic, distorted veins, venules and capillaries mostly lined only by endothelium and occasionally by a small amount of smooth muscle (53,54). Boley and coworkers proposed a pathogenesis concept for the exclusive occurrence of vascular ectasias in the right colon, the portion of the colon with the largest initial luminal diameter and the highest resting wall tension. They postulated that repeated episodes of colonic distension are associated with transient increases in both luminal pressure and size, resulting in multiple episodes of increased wall tension with obstruction of sub-mucosal venous outflow, especially where these vessels pierce the muscle layers of the colon. After many years the process leads to dilatation of venules and capillaries (54).

Colonic vascular ectasias are noted in over 25% of asymptomatic individuals over the age of 60 (55). Bleeding from vascular ectasias is usually subacute and recurrent. They manifest as iron deficiency anemia and occult blood positivity. Nearly 15% of patients have massive hemorrhage. An association of vascular ectasias and aortic stenosis is mentioned in the literature (56). During colonoscopic evaluation, which is the diagnostic procedure of choice, care should be taken to examine the mucosa during endoscope insertion without causing suction injury. Traumatic mucosal lesions may be confused for telangiectasias on endoscopy. It is important for endoscopists to remember that these lesions may not be clear in patients who are volume depleted. Administration of meperidine may also diminish the prominence of these vascular abnormalities.

Definitive treatment for bleeding from vascular ectasias is with heater probe or bipolar methods during endoscopy. In hemorrhagic cases, intra-arterial treatment and trans-catheter embolization may stop bleeding. When bleeding is recurrent and massive, right hemicolectomy is recommended.

**Colitis (Ischemic, Infectious and Inflammatory)**

Colitis is the common response to acute mucosal injury, resulting from the activation of the immune system and inflammatory cascade. Various types of colitis can be indistinguishable based on initial presentation and endoscopic findings. Patients may present (continued on page 33)
with abdominal pain, hematochezia, fever and dehydration. Endoscopically, the mucosa may appear edematous, friable and ulcerated. Diagnosis may therefore require careful interpretation of the histopathologic findings in an appropriate clinical context. Ischemic and infectious colitis are far more common than inflammatory bowel disease in the elderly.

Ischemic colitis is responsible for about 3%–9% of cases of LGIB (3,57). It can result from hypotension, embolic events or anatomic or functional changes in the mesenteric vasculature. However, a precipitating event or vascular lesion often cannot be identified. Colonic atherosclerosis is almost universal in the elderly and has uncertain significance thereby diminishing the role of angiography in the evaluation of suspected colonic ischemia. Patients often present with crampy abdominal pain followed by LGIB or bloody diarrhea. Severe hemorrhage is rarely secondary to ischemic colitis. Ischemic colitis commonly involves the watershed areas i.e. the splenic flexure, right colon or rectosigmoid junction. Initial KUB may show “thumb impressions” on the wall of the colon filled with air. Barium enema is not always required, but when performed may show “thumb impressions.” Sigmoidoscopy may reveal colonic ulcerations with rectal sparing. Histologically, necrosis is evident without much acute or chronic inflammation. Treatment is supportive and most cases resolve spontaneously. Some patients may develop chronic colitis resembling ulcerative colitis but differs in the sense that it is segmental, rectal sparing and unresponsive to standard ulcerative colitis treatment. This may be further complicated by perforation or stricture formation and may necessitate a hemi-colectomy (58).

The elderly are at increased risk for infectious colitis and its complications (59). The mortality from infectious colitis increases with age (60). Common causes of enteric infections in elderly patients are *Campylobacter*, *Salmonella*, *Shigella* and *Escherichia coli O157:H7* (61). *Clostridium difficile* should also be considered in elderly residents of long-term care facilities, hospitalized patients and those recently treated with antibiotics. However, *C. difficile* causes hematochezia in fewer than 10% of cases (62). A history or recent antibiotic ingestion for any reason may be suggestive. Infectious colitis is suspected during outbreaks of bloody diarrhea or following consumption of undercooked hamburger meat. *E. Coli O157:H7* can lead to acute thrombotic thrombocytopenic purpura and death, especially in the elderly. Most common organisms can be identified on stool culture. *E. Coli O157:H7* is identified on MacConkey’s sorbitol agar by its inability to ferment sorbitol. Since other coliforms share this quality, all sorbitol negative colonies are then tested for shiga toxin or with anti-sera to O157:H7. *C. difficile* colitis is usually diagnosed by a stool assay for toxin A and B. Treatment includes a specific antimicrobial regimen based on organism identification in addition to adequate hydration and electrolyte replacement. Metronidazole use to treat *C. difficile* colitis may interfere with oxidation of warfarin and induce excessive anticoagulation.

Approximately 15% of all patients with inflammatory bowel disease (IBD) develop symptoms after the age of 65 (63). Bimodality in age-specific incidence rates for IBD, with a second peak occurring between the ages of 60–70 has been reported (64). Diagnosing IBD in the elderly can be challenging given the vast differential diagnoses for colitis. Although gastrointestinal bleeding is common with IBD, severe hematochezia is infrequent. It accounts for hospitalization in 6% of patients with Crohn’s disease and 1.4%–4.2% of patients with ulcerative colitis (65,66). Therapy for acute exacerbations as well as quiescent disease has not been studied specifically in the older population, however general principles of management hold true in this age group. Older adults on corticosteroids should be screened for osteoporosis and prophylaxed with bisphosphonates and calcium with vitamin D unless contraindicated.

**Neoplasms**

Neoplasms, both benign and malignant, cause lower gastrointestinal bleeding in 10%–20% of cases in the elderly (67). Approximately, 2%–26% of neoplasms present with bleeding as a presenting symptom (68, 69). Bleeding from a neoplasm is neither brisk nor massive.

**Evaluation and Management**

The initial evaluation and resuscitation efforts remain unchanged for both upper and lower GI hemorrhage. However, certain characteristics help narrow the dif-
ferential for LGI bleeding (Table 7). Approximately 10%–15% of patients presenting with hematochezia have an UGI source of bleeding (42). In patients taking NSAIDs, pain may not be a significant presenting complaint (7,20,21). It is therefore prudent to perform NG lavage and confirm bilious, non-bloody aspirate in elderly patients presenting with hematochezia (70). If there is any suspicion of an upper GI source of hemorrhage, upper endoscopy should be performed first (Figure 2). Urgent colonoscopy following rapid purge is the test of choice for evaluation of LGIB once the patient has been hemodynamically stabilized (71). The diagnostic accuracy of colonoscopy in the setting of acute LGI bleeding ranges from 72%–86% with cecal intubation achieved in 95% of patients (42,72,73). In a patient with active bleeding where colonoscopy is not feasible, radionuclide imaging and/or arteriography can help identify the source of bleeding. Radionuclide imaging requires an active bleeding rate of 0.1–0.5 mL/min whereas angiography cannot accurately detect bleeding slower than 1 mL/min (74,75). Accuracy rates of radionuclide imaging and angiography for bleeding localization remain highly variable, ranging from 24%–78% and 27%–77% respectively (76). They are important tools in the diagnostic work up and treatment of patients with LGI hemorrhage who cannot undergo emergent colonoscopy. Identification of the bleeding source on angiography permits intra–arterial infusion of vasopressin or embolization of the culprit vessel. Intra-arterial vasopressin infusion is successful in controlling the bleeding in up to 90% of patients with diverticular disease or angiodysplasia, however, bleeding recurrence is high and the elderly may not tolerate the cardiovascular complications of vasopressin (49,77). Conversely, embolization with poly vinyl alcohol particles or microcoils provide more definitive means of controlling the bleeding. Embolization is complicated by intestinal infarction in up to 20% of patients (78). Patients who fail angiographic or endoscopic therapy have traditionally required surgery. Every effort should be made to identify the bleeding source prior to referral for segmental colectomy. Blind resections are associated with very high re-bleeding and mortality rates and should only be reserved for the very rare exsanguinating colonic bleeding where immediate surgery must be performed (52,79–82).

It is estimated that up to 5% of patients will GI bleeding will have a negative upper endoscopy and colonoscopy. This scenario of an older adult with obscure GI bleeding is important to recognize. Obscure GI bleeding may be further sub-categorized as occult or overt. Obscure-overt bleeding is characterized by persistent and recurrent visible evidence of bleeding per mouth or rectum, whereas obscure-occult GI bleeding is defined as a positive fecal occult blood test after a negative esophagogastroduodenoscopy, colonoscopy and routine small-bowel radiographic studies (83). Radionuclide scanning, arteriography, push enteroscopy and double-balloon enteroscopy are diagnostic studies that may be helpful in the evaluation of elderly patients with obscure GI bleeding. Wireless (continued on page 40)
capsule endoscopy is a new addition to the armamentarium of diagnostic studies used for evaluation of obscure GI bleeding (17).

References


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GERIATRIC GASTROENTEROLOGY, SERIES #16

PRACTICAL GASTROENTEROLOGY • MARCH 2006