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Gastrointestinal bleeding in the elderly

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Gastrointestinal bleeding in elderly individuals is a frequent cause of consultation with a physician and of hospital admissions. Co-morbidity and greater medication use in this steadily growing patient group influence the clinical course and adversely affect outcome. Clinical presentation is often predictable and guides subsequent patient management. Due to a surprising lack of prospective controlled data in the area of gastrointestinal bleeding, the selection of diagnostic and therapeutic manoeuvres often depends more on local expertise and availability than on an algorithmic approach. Advances in endoscopic, medical, radiological and surgical treatment modalities offer promising new diagnostic and therapeutic tools, particularly in concerted applications. Outcome studies on the appropriate sequence and linking of these modalities are urgently needed. This chapter will address clinical presentation, aetiology, diagnosis and treatment of both upper and lower gastrointestinal bleeding in the elderly.

Key words: gastrointestinal bleeding; haemorrhage; elderly; oesophagitis; oesophageal varices; peptic ulcer disease; angiodysplasia; diverticulosis; non-steroidal anti-inflammatory drugs; endoscopy; angiography; radionuclide scan; transcatheter embolization; interventional endoscopy; intra-operative endoscopy.

INTRODUCTION

Both upper and lower gastrointestinal (GI) haemorrhage are common and potentially life-threatening clinical events.[1] In the elderly population, the incidence of GI bleeding is quoted as being 500 episodes/100 000 people per year, compared to 100/100 000 per year in the average population.[2,3] Co-morbidity is considerable in this patient group, explaining the persistently high mortality rate of approximately 10% throughout recent decades.[4] Research in the area of GI bleeding is hampered by a significant lack of prospective and randomized, controlled studies or any data based on scrupulous meta-analysis.[1] This lack of clarifying clinical research is particularly obvious for patients from the older generation.[4,5] This chapter will review clinical presentation, aetiology, diagnosis and treatment of upper and lower GI bleeding in the elderly.

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CLINICAL PRESENTATION

As in younger patients, medical history (e.g. aspirin use for peptic ulcer disease or prior radiation therapy for radiation proctocolitis) and clinical symptoms have a high predictive value for the source of GI haemorrhage in elderly patients.[6,7] In upper GI bleeding (UGIB), haematemesis (50%) prevails, as opposed to a combination of heamatemesis and melaena (20%) or melaena alone (30%).[2,8] However, patients > 60 years of age have less dyspepsia or alcohol consumption, irrespective of non-steroidal anti-inflammatory drug (NSAID) use, than patients aged < 60 years.[2] They less frequently report abdominal pain, although clinical presentation is sometimes confounded by impaired visual and cognitive abilities. Passage of bright red blood, stool with blood clots or maroon stool/rectum is usually attributed to a bleeding source within the lower GI tract. However, in up to 11% of patients with a GI haemorrhage, which is termed haematochezia, the lesion responsible can be identified within the upper GI tract.[9,10] Conversely, 19% of patients with lower GI bleeding (LGIB) reported melaena, which is traditionally thought to occur only in UGIB cases.[11] Haemorrhage may be acute (10%), overt (85%) or occult (5%), the latter being more common in elderly patients. The clinical spectrum of acuity and severity of UGIB differs from that of LGIB. Based on a member survey of the American College of Gastroenterology (ACG), patients with UGIB were more likely to present to the physician with signs of haemodynamic instability than were patients with LGIB (e.g. shock/orthostasis 35% versus 19%; blood transfusions 64% versus 36%; anaemia 84% versus 61%).[11]

Incidence

Incidence rates of UGIB have remained unchanged during the last decades, ranging from 67–172/100 000 people per year.[11–13] These relatively stable figures could be explained by an increased risk of acute bleeding in the expanding group of elderly people. Indeed, age is associated with a steep rise in the incidence of upper GI haemorrhage, the rate for patients older than 70 years of age being 20–30 times higher than that for patients aged 30 years or less.[13] In a UK study, 27% of patients with acute UGIB were aged over 80 years.[13] For LGIB, an incidence rate of 21/100 000 per year has been reported in a population-based study from a health maintenance organization.[3] Male patients outnumbered female patients (24 versus 17) with a > 200-fold rise in bleeding events from the third to the ninth decade of life. This progressive incidence rate may again reflect the change in disease pattern with ageing. A survey study from the ACG confirmed these demographic data, with 24% of all bleeding events occurring in the lower GI tract.[11]

Course

The hospital course of elderly patients in the USA with UGIB appears to be similar to that of younger patients with regard to the need for intensive care, the frequency of endoscopy or surgery, transfusion requirements and the duration of hospital stay.[2] However, older patients are more likely to die during hospitalization than younger patients;[1] mortality rates for those aged over 60 years are 12–25%, compared with under 10% for patients aged less than 60 years. Peptic ulcer haemorrhage, which is the most frequent cause of UGIB in the elderly, ceases spontaneously in approximately 80% of cases.[6] However, death rates for patients younger than 60 years of age are less than 10% but nearly 35% in those over 80 years of age.[14] This may be due to the fact that the great majority of deaths result from co-morbid disease rather than from uncontrolled bleeding.[3,13] LGIB in the elderly is also associated with greater morbidity and mortality than in younger patients due to the natural ageing process, significant co-morbidity and higher medication use.[3] Mortality varies between 2% and 4%.[3,9,15] All-cause post-hospital mortality rate during a follow-up of 34 months was 19%, although no patient died from GI haemorrhage.[3]

Prognostic factors

Preliminary data suggest that clinical prognostic criteria could be used to separate low- from high-risk patients with acute upper and lower GI haemorrhage.[16] The BLEED classification (i.e. ongoing **b**leeding, **l**ow systolic blood pressure, **e**levated prothrombin time, **e**rratic mental status and unstable co-morbid **d**isease) suggests independent predictors for inhospital complications such as recurrent GI haemorrhage, surgery to control the source of bleeding, or hospital mortality. High-risk patients (i.e. at least one BLEED criterion fulfilled) were more likely to suffer from inhospital complications than were low-risk patients with either UGIB (31% versus 4%) or LGIB (38% versus 12%). The importance of age and co-morbid disease as independent risk factors is also expressed in the Rockall-score for risk assessment of death and rebleeding after acute upper GI haemorrhage.[13] This scoring system, which is based on clinical (e.g. age < 60 years, 60–79 years, > 80 years) rather than endoscopic variables, has now been prospectively validated for stratifying patients into high- and low-risk categories for mortality.[17] Scoring systems are increasingly applied in order to streamline the management of patients with GI haemorrhage (e.g. outpatient versus inpatient care): the mean age of patients deemed suitable for outpatient care (52 years) was lower than that of patients selected for inpatient management (63 years).[18]

AETIOLOGY

UGIB is thought to originate in anatomical sites that are proximal to the ligament of Treitz, whereas LGIB occurs distal to the ligament of Treitz. Peptic ulcer disease remains the main culprit of UGIB (Table 1), particularly in the elderly population,[2,8] and up to 91% of hospital admissions for UGIB in patients older than 60 years of age are caused by peptic acid disease.[2] As a result of higher alcohol consumption, Mallory–Weiss tears and gastro-oesophageal varices are more commonly found in the younger population.[11,12] A recent study reported an increase in oesophagitis from 3% in sexagenarians to 21% in patients aged 80 years or more as a main source of UGIB.[19] Colonic diverticulosis and diverse forms of colitis (e.g. ischaemic colitis, infectious colitis) account for most LGIB, reflecting the preponderance of occurrence of these entities in the older generation (Table 2). In the ageing population, the causes of GI bleeding have remained little changed over the past several decades.

Oesophagitis

Anaemia or positive faecal occult blood may be subtle signs of erosive oesophagitis, whereas frank haematemesis points to penetrating oesophageal ulcers usually in the distal oesophagus (Figure 1). These lesions are often caused by severe gastro-oesophageal reflux disease (GORD), which is thought to be more prevalent in elderly patients, albeit with less severe symptoms than in younger patients.[20] Patients

Table 1. Aetiology of upper gastrointestinal bleeding.

	General population		Elderly population	
Aetiology	Vreeburg et al. (1997)[12] No. (%)	Peura et al. (1997)[11] No. (%)	Cooper et al. (1988)[8] No. (%)	Segal & Cello (1997)[2] No. (%)
Oesophagitis	48–143 (5–15)	96 (20)	14 (14)	11 (11)
Varices†	29–219 (3–23)	58 (12)	2 (2)	3 (3)
Neoplasia	29–48 (3–5)	NA	4 (4)	1 (1)
Mallory–Weiss tear	10–105 (1–11)	29 (6)	2 (2)	3 (3)
Gastric ulcer	124–190 (13–20)	125 (26)	82 (20)	35 (35)
Gastritis	86–276 (9–29)	63 (13)	13 (13)	7 (7)
Duodenal ulcer‡	190–228 (20–24)	135 (28)	80 (22)	38 (38)
Miscellaneous§	48–171 (5–18)	63 (13)	NA	NA
Undefined	67–133 (7–14)	NA	19 (18)	NA
Number of patients	951	482	103	100
Age of patients (years)	2–98	NA	> 80	> 60

†Includes oesophageal and gastric varices.
‡includes pyloric channel ulcers.
§ Includes angiodysplasia, stomal ulcers, haemobilia, duodenitis, vascular ectasias, enterovascular fistulas, Dieulafoy's lesions.
NA, not available.

Table 2. Aetiology of lower gastrointestinal bleeding.

Aetiology	Richter et al. (1995)[15] No. (%)	Bramly et al. (1996)[7] No. (%)	Longstreth (1997)[3] No. (%)	Peura et al. (1997)[11] No. (%)	Kok et al. (1998)[49] No. (%)
Diverticulosis	51 (47)	60 (24)	91 (42)	43 (30)	57 (29)
Colitis†	6 (6)	52 (21)	35 (16)	21 (14)	49 (26)
Neoplasia	12 (11)	25 (10)	20 (9)	25 (17)	17 (9)
Angiodysplasia	13 (12)	17 (7)	6 (3)	14 (10)	3 (2)
Proctal lesions‡	3 (3)	22 (9)	10 (4)	2 (1)	15 (8)
Miscellaneous§	7 (7)	11 (4)	31 (14)	20 (14)	10 (5)
Undefined	15 (14)	64 (25)	26 (12)	20 (14)	39 (21)
Patient number	107	251	219	145	190
Mean patient age (years)	70	69	67	NA	63

†Includes inflammatory bowel disease (IBD), radiation colitis, ischaemic colitis, infectious colitis, vasculitis.
‡Includes haemorrhoids, anal fissures, rectal ulcers.
§Includes polypectomy bleeding sites.
NA, not available.

aged > 60 years had more oesophageal mucosal disease than patients < 60 years of age (81% versus 47%). Elderly people routinely ingest medications that have been implicated as a cause of oesophageal ulcerations (e.g. potassium, alendronate, tetracycline, NSAIDs).[4] Pill-induced oesophagitis may develop subsequent to insufficient fluid intake and a decrease in saliva production. Older patients in intensive care units (ICUs) may also bleed from the stress-related erosive syndrome in the oesophagus,[21] or cardia pressure necrosis due to nasogastric tubes. We have received several personal communications of severe oesophageal haemorrhage after thermoablative therapy (e.g. photodynamic therapy) in superficial cancer.

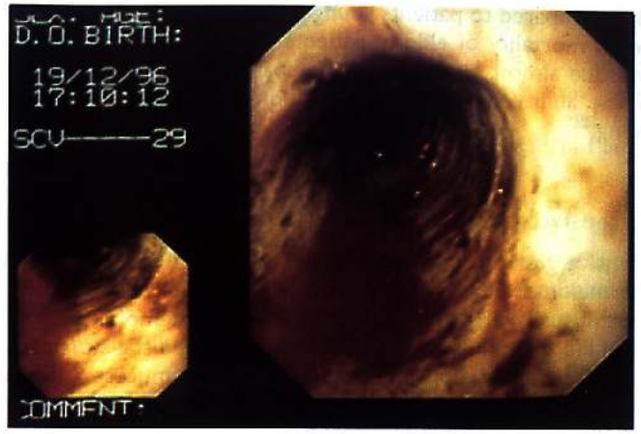

Figure 1. Endoscopic view of ulcerative oesophagitis with overt bleeding.

Peptic ulcer disease

Elderly people are at an increased risk for suffering from peptic ulcer disease due to the high percentage of *Helicobacter pylori*-positivity and NSAID use in this age group. In addition, the incidence of complications (e.g. haemorrhage, perforation, death) rises progressively with age.[22] In older patients, the risk of serious adverse events, such as peptic ulcer bleeding while taking NSAIDs is 5.5 times that of controls, whereas the risk in younger patients is only 1.5 times.[22] The risk of bleeding is further increased with the intake of higher NSAID doses (10.1 times) and concurrent use of glucocorticoids (4.4 times) or anti-coagulants (12.7 times).[23] Because primary as well as secondary prophylaxis with aspirin and warfarin in cardiovascular and cerebrovascular disease is becoming increasingly commonplace, even more adverse haemorrhagic events are to be expected in the future. Furthermore, a study from Birmingham noted that 45% of admissions for peptic ulcer haemorrhage in patients > 60 years of age were attributable to accessory risk factors such as treatment for heart failure (odds ratio (OR) = 5.9) or diabetes (OR = 3.1), previous peptic lesions (OR = 3.8), oral corticoid intake (OR = 2.7) and a current smoking habit (OR = 1.6).[24] The role of gastroduodenal colonization with *H. pylori* (a prevalence of > 50% in patients > 50 years of age) is more controversial: there is a less prominent role for *H. pylori* in the context of bleeding ulcers, especially in the stomach, and particularly when NSAIDs have been implicated. However, a recent case–control study demonstrated an almost twofold increased risk of ulcer bleeding in the *H. pylori*-positive patients who were taking NSAIDs.[25] *Helicobacter pylori* accounted for about 24% of bleeding peptic ulcers among elderly NSAID users.[25] Stress-related erosive syndrome (SRES) with UGIB is a common observation in older patients under intensive care treatment.[21]

Variceal bleeding

Gastro-oesophageal varices develop in about half of all patients with liver cirrhosis, of whom one-third will experience variceal haemorrhage during their lifetime. However, the relative mortality seems to be significantly lower for patients older than age 50

(OR = 1.3–3.9) compared to patients younger than 50 years of age (OR = 23–31). The increased overall mortality of elderly patients who survived to leave hospital after a first bleed was due almost entirely to non-hepatic causes. There seems to be no age difference in the ability of Child's classification to predict survival at the time of variceal bleeding.[26]

Portal hypertensive gastroenteropathy

Portal hypertensive gastropathy (PHG) and gastric antral vascular ectasia (GAVE, the so-called watermelon stomach), are increasingly recognized as separate nosological entities but both usually present with gastric mucosal haemorrhage.[27] GAVE tends to manifest later in life than PHG, consistently involves the distal stomach and shows the characteristic 'watermelon' appearance of the antral mucosa.[27] Bleeding in both disorders of the stomach in cirrhosis ranges from occult to brisk, necessitating regular blood transfusions or endoscopic treatment.

Angiodysplasia

Angiodysplasia occurs throughout the whole GI tract, preferentially in the right side of the colon, and is often clustered within one intestinal segment.[28] Two–thirds of the patients older than 70 years are affected.[29] The prevalence of angiodysplasia is largely unclear, but has been estimated at 0.8% in 964 healthy individuals on the basis of screening colonoscopies for GI cancer.[28] Haemorrhage from angiodysplasia is frequent, accounting for about 3–12% of all LGIB episodes.[3,15] Synchronous angiodysplasias are thought to occur elsewhere in the GI tract in about 20% of cases. Haemorrhage usually ceases spontaneously, but recurrence is common.

Infectious enterocolitis

Infectious enterocolitis is a common clinical problem in older people. More frequent antibiotic use in this patient group favours pseudomembraneous colitis, which may be accompanied by LGIB.[30] Enterohaemorrhagic *Escherichia coli* (EHEC) strains, most notably 0157:H7, are often reported in nursing home epidemics. They are usually transmitted by ingested undercooked minced beef, are not easily recognized on routine agar and may be complicated by the haemolytic–uraemic syndrome or thrombotic thrombocytopenic purpura.[30] Clinical signs are crampy abdominal pain and tenderness, followed by watery and bloody diarrhoea.

Intestinal vasculopathy

Thromboembolic or non-occlusive mesenteric ischaemia is frequently observed in elderly patients, accounting for 3–9% of LGIB episodes.[3,7] However, occlusive vascular events are only rarely demonstrated. Mesenteric ischaemia preferentially involves the mid-distal colon causing lower abdominal crampy pain followed by LGIB or bloody diarrhoea within a 24-hour time-frame.[21] Non-occlusive mesenteric vasospasm is also seen in ICU patients due to low-flow states from systemic hypotension, and this may cause small-volume bleeding/rectum.[21]

Inflammatory bowel disease

Ulcerative colitis and Crohn's disease manifest in a bimodal fashion, usually around the ages of 20 and 70. Presentation, clinical course and overall prognosis seem to be similar in older and younger patients. Some degree of lower GI bleeding is a frequent symptom in inflammatory bowel disease (IBD). It has been reported in the majority of patients with ulcerative colitis (UC) and in about one-third of patients with Crohn's disease (CD). The localization of diseased bowel seems to be relevant to the risk of bleeding. The Mayo group retrospectively characterized the clinical features and course of major GI bleeding in their patients with IBD over an 8 year period.[31] Thirty-one patients suffered from acute LGIB, representing 1.2% of admissions for CD and 0.1% for UC. An important differential diagnosis in the elderly is between segmental colonic IBD and diverticulitis or localized ischaemic colitis.

Diverticula

Given the high prevalence of diverticulosis in the general population – 50% of people in their eighties are affected – diverticular bleeding accounts for 41–48% of all lower intestinal haemorrhagic episodes.[3,15] Bleeding mostly occurs in the right side of the colon (50–90%).[32] This reflects a propensity for diverticula in the proximal colon to bleed, perhaps because these, mostly pseudodiverticula, have wider necks and domes and, thus, do expose the vasae recta over a greater length to trauma.[33] In the Health Professionals Follow-up Study, the relative risk for diverticular bleeding was 4.6 (1.0–21.7) for regular and consistent NSAID users, and 13.6 (3.5–52.6) for acetaminophen users after adjustment for age, physical activity and diet.[34] The clinical picture in diverticular haemorrhage is of painless but abrupt onset, with spontaneous cessation of bleeding in up to 90% of patients and a recurrence rate of 22–38%.

Radiation proctocolitis

Certain gynaecological or prostatic malignancies prevail in the elderly population. Radiation therapy of these abdominopelvic cancers can lead to LGIB as either early or late complications of radiation damage.[35] In acute proctocolitis, patients complain of transient diarrhoea, tenesmus and mucoid or bloody discharge per rectum. More worrying are late complications occurring within months or years after radiotherapy in the form of chronic proctocolitis with progressive intramural vasculitis.[35]

Neoplasia

In most instances, neoplastic lesions arising from the GI tract bleed in an occult fashion. Occasionally, bleeding can be exigent or brisk in ulcerative tumours. About 10% of major, and 20% of minor, lower GI haemorrhages in the elderly originate from benign or malignant neoplasia.[11,15] Apparent bleeding from the colon may also occur after endoscopic removal of polyps.[3] Haemorrhage may be immediate, or delayed from a few hours to up to 15 days. Early bleeding may result from a lack of sufficient coagulation of the feeding blood vessel in the polyp stalk, later bleeding from sloughing of the eschar or erosion of a polypectomy ulcer.[36]

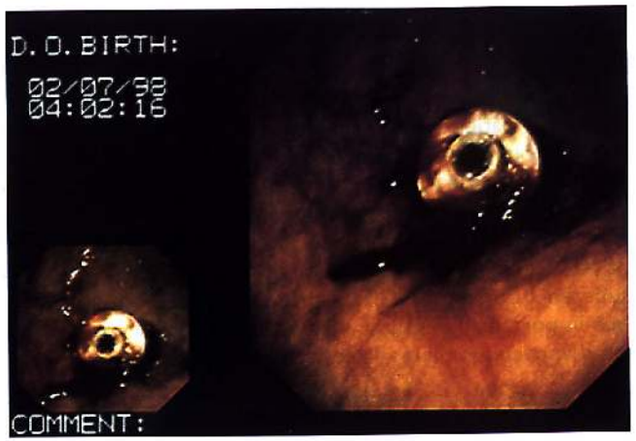

Figure 2. Endoscopic view of the anterior gastric wall: spontaneous bleeding from a percutaneous endoscopic gastrostomy (PEG) button.

Endoscopy-induced haemorrhage

Endoscopic procedures

In this era of increasing application of endoscopy, bleeding complications induced by either diagnostic or therapeutic procedures merit particular attention. Diagnostic upper endoscopy hardly ever caused GI haemorrhage, whereas interventional endoscopic procedures carry a small but significant risk of UGIB (e.g. gastroduodenal polypectomy 0.2–8.0%).[37] Foreign body impaction subsequent to swallowing disturbances in older people may lead to bleeding from pressure necrosis or oesophageal wall laceration. Spontaneous bleeding from a percutaneous endoscopic gastrostomy (PEG) for long-term tube feeding (Figure 2) in geriatric patients has been reported, with a range of 0.2–2.5%.[37] The incidence of bleeding from diagnostic colonoscopy with biopsies has been reported in retrospective reviews as ranging from 0–0.2%.[36] Therapeutic colonoscopy more frequently causes haemorrhage, with a reported incidence of 0.2–3.3% in colonoscopic polypectomy.[36] Risk factors for bleeding events include: advanced patient age, patient coagulopathy, adenomatous polyp > 2 cm in diameter, wide polyp stalk, sessile polyp, the endoscopist's inexperience and anti-coagulant therapy.[36,37]

Anticoagulation

Few data exist on the safety of patients undergoing endoscopy and taking aspirin or other NSAIDs. The risk of minor, self-limited (6.3% intervention group versus 2.1% control group), but not major haemorrhage (< 1.0%) after mucosal biopsy and polypectomy appears to be increased under standard doses of aspirin or other NSAIDs.[37] Patients on anti-coagulation treatment (e.g. warfarin) can be submitted to strictly diagnostic procedures without an increased risk of significant bleeding.[38] Data on the modalities for anti-coagulation therapy that should be stopped before and reinstated after therapeutic endoscopy are not well substantiated, and are based on empirical recommendations. Cessation and reinstatement of anti-coagulation may not be as critical as has been previously thought. In a series of 27 patients who had GI bleeding on warfarin, only one patient suffered from thromboembolism during a median follow-up period of 8 months after cessation of warfarin.

Miscellaneous

Aortoenteric fistulas may occasionally cause severe UGIB in elderly persons after aortic surgery. In any patient with an aortic graft and massive bleeding from the distal duodenum, a high degree of suspicion is warranted for exsanguinating haemorrhage.[39] Haemobilia due to a choledochoarterial fistula (e.g. hepatic artery) after trauma or abdominal surgery can be suspected in recurrent overt UGIB without identification of a bleeding site on upper endoscopy. Dieulafoy's lesions are increasingly reported as a source of haemorrhage anywhere in the GI tract. Particularly in older patients (> 25%), hereditary haemorrhagic teleangiectasia may present with severe GI bleeding.[40] Constipation occasionally causes the solitary rectal ulcer syndrome as a sequela of obstructed defaecation and internal rectal prolapse and stercoral ulcers due to frequent faecal impaction. Both may give rise to massive anorectal haemorrhage in the elderly.

DIAGNOSIS

The diagnostic approach to the patient with GI bleeding does not seem to be age-dependent. As outlined above, eliciting a careful medical history and identifying pertinent risk factors for haemorrhage is pivotal in the establishment of a correct diagnosis in GI bleeding. Important history clues include coagulopathies, NSAID use, previous surgery and co-morbid disease.[11] History taking cannot replace a proper physical examination since visual, auditory and/or cognitive deficits are prevalent in older patients (e.g. perianal inspection and digital rectal examination may be diagnostic in up to 9% of LGIB cases.[7]) Laboratory testing is not a useful diagnostic tool although haemoglobin levels of < 10 g/dl are more likely to be found in UGIB (66%) than in LGIB (40%).[11] A positive nasogastric aspiration (e.g. coffee-ground guaiac-positive material) has been claimed to point to UGIB in approximately 79–93% of cases.[41] However, its negative predictive value is thought to be low, with non-bloody aspirates in 16% of patients with UGIB.[41] The patient's description or the physician's observation of the colour of bloody stools can guide further diagnostic measures.[42] Bright red blood/rectum precludes UGIB in 98.2% unless hypovolaemic shock prevails. Faecal occult-blood testing is the method of choice to confirm suspected, but unidentified, sources of GI haemorrhage.[43]

Endoscopy

Endoscopy is the preferred investigation for both upper and lower GI bleeding because of its accuracy, low rate of complications and potential for therapeutic interventions. However, elderly individuals (0.2–4.9%) are at greater risk of complications than younger ones (0.03–0.13%).[44] Complications may be related to medication use (e.g. adverse reaction to sedatives in older patients), cardiopulmonary events (e.g. aspiration pneumonia), or endoscopic interventions (e.g. perforation).

Oesophagogastroduodenoscopy

Oesophagogastroduodenoscopy (OGD) approaches a diagnostic yield for UGIB of 94%, with a major complication rate of about 0.5% and a mortality rate of 0.1%.[1] The likelihood of detecting an active haemorrhage is increased if diagnostic endoscopy is performed soon after the onset of bleeding.[12,13] Indeed, early upper endoscopy in UGIB was associated with clinically significant reductions in the adjusted risk of recurrent bleeding or surgery (OR = 0.7), and a 31% decrease in the adjusted length of hospital stay, compared with delayed endoscopy.[45] In addition to clinical factors (see Prognostic factors above), endoscopic diagnosis of a lesion and associated stigmata of haemorrhage significantly enhances the ability to predict outcomes (Figure 3).[5] For example, stigmata of recent peptic ulcer heamorrhage appear to represent different phases in the evolution of an actively bleeding vessel through a sentinel clot to a clean ulcer base.[46] A spurting arterial or non-bleeding visible vessel have been associated with a rebleeding rate of 43–55% and a mortality rate of 11%, whereas a clean ulcer base or a flat pigmented spot indicate rebleeding in 5–10% or death in 2–3%.[47] In a study by Longstreth & Feitelberg[18] the only absolute criteria against hospital discharge were endoscopic high-risk findings (e.g. visible vessel), oesophageal varices and PHG. Upper endoscopy has been used variably in the published literature on LGIB. Studies that found upper GI tract examination to be negative had used OGD only in selected patients (< 35%).[3,15] Studies that always performed OGD in the work-up of LGIB detected upper GI bleeding sites in up to 11%.[9]

Enteroscopy

Endoscopy of the small bowel has been introduced into clinical practice primarily for the evaluation of occult gastrointestinal bleeding (OGIB). Evidence is emerging that enteroscopy may be of additional use in both the diagnostic and therapeutic approach to acute or overt GI haemorrhage. Push-enteroscopy allows the additional inspection of approximately 60 cm of the proximal jejunum with a dedicated enteroscope or paediatric colonoscope.[48] Small bowel sources of haemorrhage, not detected during preceding conventional endoscopy, could be identified in up to 38% of LGIB cases.[48] However, the impact of enteroscopy on long-term clinical outcome remains

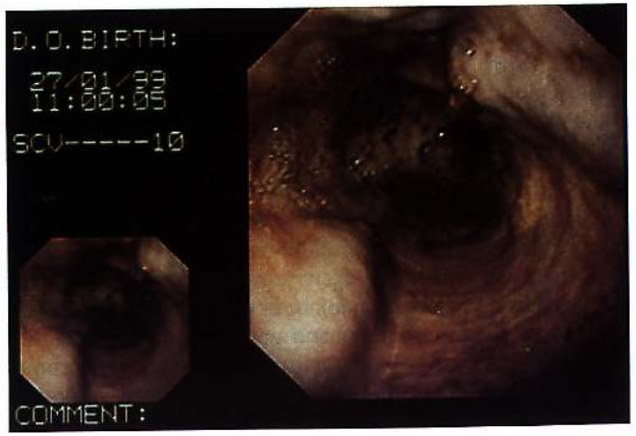

Figure 3. Endoscopic view of prominent oesophageal varices 1 day after UGIB: 'white nipple sign'.

controversial. About 80% of small-bowel lesions so-detected were angiodysplasia, which tend to be profusely distributed and to reoccur spontaneously after treatment. Interestingly, the rate of missed culprit lesions in the upper GI tract during initial OGD approached 42%.[48]

Ileocolonoscopy

Ileocolonoscopy is the most frequently used test in the evaluation of both overt LGIB and occult GI haemorrhage.[1,3] By contrast, ileocolonoscopy was considered to be of no value in acute LGIB, on the basis of fears of poor visibility due to blood and remnant stool, and of complications such as perforation or exacerbation of haemorrhage. The advent of therapeutic options to treat bleeding lesions during early colonoscopy, and of cleansing options for safe bowel preparation by oral purge even in elderly and frail patients, has instigated interest in emergency ileocolonoscopy in recent years.[9] A provisional meta-analysis on the role of ileocolonoscopy as the primary diagnostic method in LGIB quotes 69% (48–90%) of urgent colonoscopic examinations as being positive in detecting the actual or presumptive source of haemorrhage.[42] The criteria for definite or presumed diagnosis of bleeding site have been derived from experiences in UGIB, but they lack validation: active bleeding site, a non-bleeding visible vessel, an adherent clot, fresh blood localized to a colonic segment, or ulceration of a diverticulum with fresh blood in the vicinity.[15,33]

Rectosigmoidoscopy

Rectosigmoidoscopy with anoscopy offers an effective and inexpensive method for the detection of bleeding sites stemming from the sigmoid colon or anorectum.[7] Using this approach, the source of LGIB may be identified in up to 9% of patients.[15] In a recent review, flexible sigmoidoscopy combined with anoscopy could have detected 26% of culprit lesions in LGIB (e.g. radiation teleangiectasia), and would have obviated the need for emergency colonoscopy.[49] However, the potential for synchronous lesions in the proximal bowel necessitates subsequent elective colonoscopy.

Radiology

Angiography

Arteriography of the mesenteric artery or the coeliac axis is routinely applied to detect small and large bowel bleeding at rates as low as 0.5–1.0 ml/min.[50] Occasionally, gastric or duodenal sources of haemorrhage are disclosed (e.g. aortoenteric fistula). Significant complications (e.g. contrast reactions, renal failure, femoral artery thrombosis, intestinal vessel laceration) have been reported for visceral angiography in up to 9.3% of examinations.[51] An updated review of 15 retrospective studies on the ability of angiography to localize haemorrhage in acute LGIB reported positive angiograms in 382 out of 839, with a range of 27–77%.[42,52] As in scintigraphy, sensitivity seems to depend on the acuity of bleeding.[53,54] New techniques have been proposed (e.g. helical CT angiography and intravascular contrast three-dimensional magnetic resonance imaging), but these await further clinical evaluation.[50]

Barium studies

Barium enemas do not play any role in the evaluation of acute LGIB. Small bowel follow-through does not have a place in acute LGIB either, but has proven to be diagnostic in approximately 5% of patients with OGIB or iron-deficiency anaemia.[53,55] Enteroclysis increases the sensitivity for small bowel pathology in LGIB, with a reported yield of approximately 10% in retrospective series (e.g. Crohn's disease).[53] There may be an additional diagnostic yield by means of a new technique that combines endoscopy and radiology (i.e. enteroscopy–enteroclysis), but confirmatory studies are needed.[56]

Radionuclide imaging

The preferred field of application of technetium ^{99m}Tc-labelled erythrocyte scans is the evaluation of LGIB or occult GI haemorrhage that exceeds a rate of 0.1 ml/min. Early continuous scanning followed by static scanning delayed by up to 24 hours is routine.[42] The review by Zuckerman & Prakash[42] has been updated to collect 17 retrospective studies for a total of 1523 tagged red cell scans in LGIB, of which 677 were positive and 368 were confirmed by other diagnostic procedures (e.g. angiography). The false-positive rate was 22%, with a surprising range of 6–59%. By comparison, the appearance of the radionuclide spot immediately on scanning predicted a positive angiogram in 61%, while later appearance predicted a positive result in only 7% of cases.[54] There are preliminary data to support scintigraphy as a screening test prior to visceral arteriography in order to increase the diagnostic yield in acute LGIB.[54,57]

TREATMENT

The treatment of GI haemorrhage in the elderly is similar to that in younger patients. However, elderly individuals suffer from a higher all-over morbidity and mortality.[4] Resuscitation may proceed in parallel with the initiation of diagnostic or therapeutic procedures.[5,21,58] Frequently, haemorrhage ceases spontaneously but, if not, may need endoscopic, medical, angiographic or surgical intervention, or any combination of these techniques.

Endoscopy

According to a recent ACG accuracy, endoscopic therapy is applied to 51% of patients with UGIB and 27% of patients with LGIB.[11] There is an array of therapeutic tools available to control both acute bleeding and the stigmata of precedent bleeding. Their feasibility and effectiveness in achieving haemostasis has been proven for injection sclerotherapy, band ligation, mono- or multipolar electrocautery, laser, argon plasma coagulation and clipping devices.[12,37] Randomized clinical trials and meta-analyses have shown convincingly that endoscopic therapy reduces the rate of rebleeding, emergency surgery and mortality in patients with high-risk peptic ulcer lesions (e.g. active bleeding, visible vessel, adherent clot).[6,47] In patients at high risk for recurrent bleeding, the use of early endoscopic therapy to control haemorrhage was associated with reductions in recurrent bleeding or surgery (OR = 0.2) and length of hospital stay (−31%).[45] Whether rebleeding is more common in older compared with younger

patients is still under debate.[6,59] Injection sclerotherapy (e.g. polidocanol), thermal contact treatment (e.g. heater probe) and haemoclips (Figure 4) have so far not been shown to be more effective than epinephrine injection alone in peptic ulcer bleeding.[6] There might be a slight advantage of repeated fibrin glue injection.[60] In variceal haemorrhage, band ligation has been proven to be superior to injection sclerotherapy with regard to rebleeding, local complications and short-term survival.[61] Angiodysplasia of both the small[28] and large[62] bowel are mostly treated by thermal ablation. Uncontrolled studies with small patient numbers describe endoscopic treatment of diverticular haemorrhage.[32,33,63] Post-polypectomy haemorrhage occurs in up to 3.3% of patients[36], but may safely be salvaged by therapeutic endoscopy (Figure 5).[64]

Medical treatment

As in younger individuals, interventional therapeutic endoscopy remains the primary treatment for GI haemorrhage in the elderly. However, additional medical treatment may reduce the rate of rebleeding or prevent the first bleeding episode by directly influencing the pathogenetic mechanisms of haemorrhage.[65] High-risk peptic ulcers (i.e. active bleeding, non-visible vessel, adherent clot) may profit from aggressive intravenous proton-pump inhibitor (PPI) therapy, even after successful endoscopic therapy.[66] In a large placebo-controlled trial from Hong Kong, the rate of 30-day recurrent bleeding was reduced from 23% to 7% in patients on high-dose intravenous omeprazole after initial endoscopic haemostasis. Furthermore, the duration of hospital stay, number of blood transfusions required and, probably, mortality and surgical intervention rates were reduced by supplementary acid suppression.[66] GI bleeding from NSAID use may be prevented by prescribing concomitant PPI prophylaxis in at-risk patients, or by using the newer cyclo-oxygenase-2-specific inhibitors.[67] First bleed and rebleeding from *H. pylori*-induced gastroduodenal ulcers are best prevented by consequent eradication of the culprit bacterium.[65] Primary prophylaxis of variceal bleeding relies on sufficient β-blockade with propanolol, while vasoactive drugs (e.g. octreotide are firmly established in the treatment of acute variceal haemorrhage.[68]

Angiography

Therapeutic angiography may be indicated in the frail and severely ill patient or, increasingly, in patients who continue to bleed after therapeutic endoscopy. Vasopressin therapy has been successfully applied for GI haemorrhage since the early 1970s.[50] High initial haemostasis rates (47–92%) were reported[50], but rebleeding in the short term was common (16–40%). Complication rates were considerable, qualified as minor in about 40% or as major (e.g. myocardial ischaemia in elderly patients) in about 15% of interventions.[44] Therefore, transcatheter embolization with different agents, such as gelatine sponges, coil springs, or polyvinyl alcohol particles became more attractive.[50] A direct comparision with mesenteric vasopressin infusion in GI bleeding found equal initial haemostasis rates (70% versus 71%), but no rebleeding after gelatine sponge embolization (0% versus 25%).[58] Embolization has been successfully used to treat uncommon causes of UGIB (e.g. haemobilia), but the main indication remains LGIB.[50] However, advances in catheter and guidewire design have enabled superselective catheterization and embolization of vasa recta, significantly reducing complication rates. This method is now considered to be the primary radiological approach to GI bleeding in experience hands.[69,70]

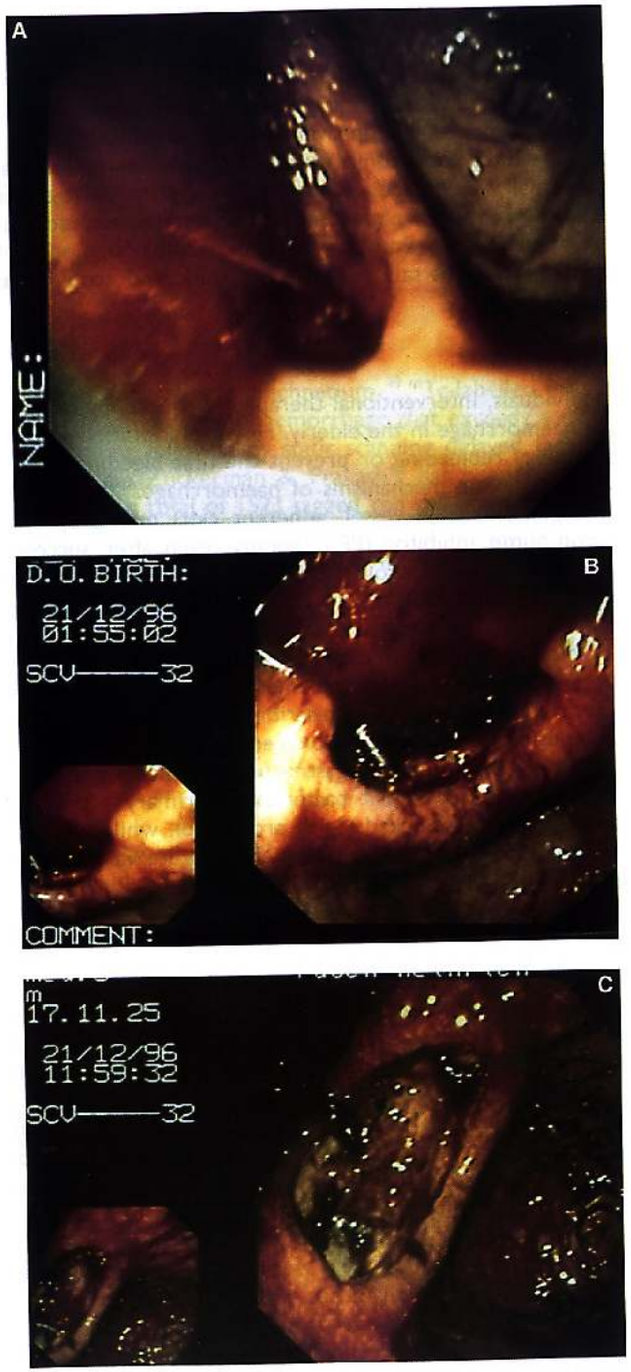

Figure 4. Endoscopic view of the gastric angulus. (A) acutely bleeding giant ulcer, (B) application of a metal clip to the spurting vessel, (C) ulcer base with metal clip after rinsing.

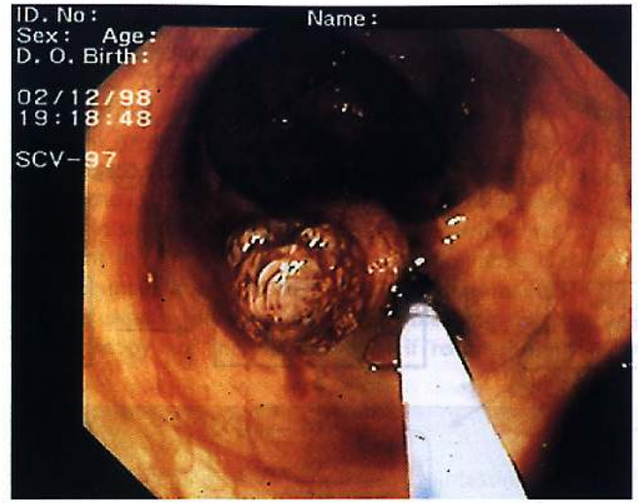

Figure 5. Endoscopic view of a spontaneously bleeding colonic polyp: haemostatic epinephrine injection therapy.

Surgery

Surgery may be required if non-surgical methods fail to arrest the GI haemorrhage. Advanced age, by association with more co-morbid disease, is a significant risk factor for peri-operative morbidity and mortaliy, but should not delay surgery if necessary. Early elective surgery seems to be superior to emergency surgery, with mortality rates of 13% versus 25% in patients aged > 60 years.[4] Indeed, initial haemostasis is most frequently achieved by interventional endoscopy or radiology creating an opportunity for physicians and surgeons alike to confer and decide on the optimal subsequent treatment strategy. In peptic ulcer haemorrhage, identification of high-risk lesions such as large ulcers (> 2 cm in diameter) and severity of index bleed (Forrest I-IIb) may justify early elective surgery.[59] However, a recent study from Hong Kong demonstrated that in patients with a mean age of 65 years, endoscopic retreatment of peptic ulcer bleeding was associated with fewer complications than surgery.[71] Transjugular intra-hepatic portosystemic shunt (TIPS) is now commonplace in the treatment of unrelenting haemorrhage from oesophageal or gastric varices.[39,50] Surgical management of LGIB is ideally undertaken with a reliable knowledge of the cause, acuity and location of the culprit haemorrhage. On the basis of the security of pre- or intra-operative characterization of the bleeding lesion, a segmental intestinal resection to include this lesion can be safely performed.[39] Blind subtotal abdominal colectomy with ileorectal anastomosis may be the treatment of choice in massive unrelenting LGIB without identification of the bleeding site.[58] Blind segmental colectomies carry similar mortality rates (5–33%), but are associated with significantly higher rebleeding rates.[39,58] In recent years, intra-operative endoscopy has gained momentum, mostly in the elective setting.[48] Identification of bleeding sites has been possible in up to 83–100% of cases, and resective surgical or endoscopic treatment has resulted in haemostasis in up to 55–100%.[53] In previous reviews, patients were submitted to surgery for LGIB in 2.7%[3] to 16.0%[49] of cases.

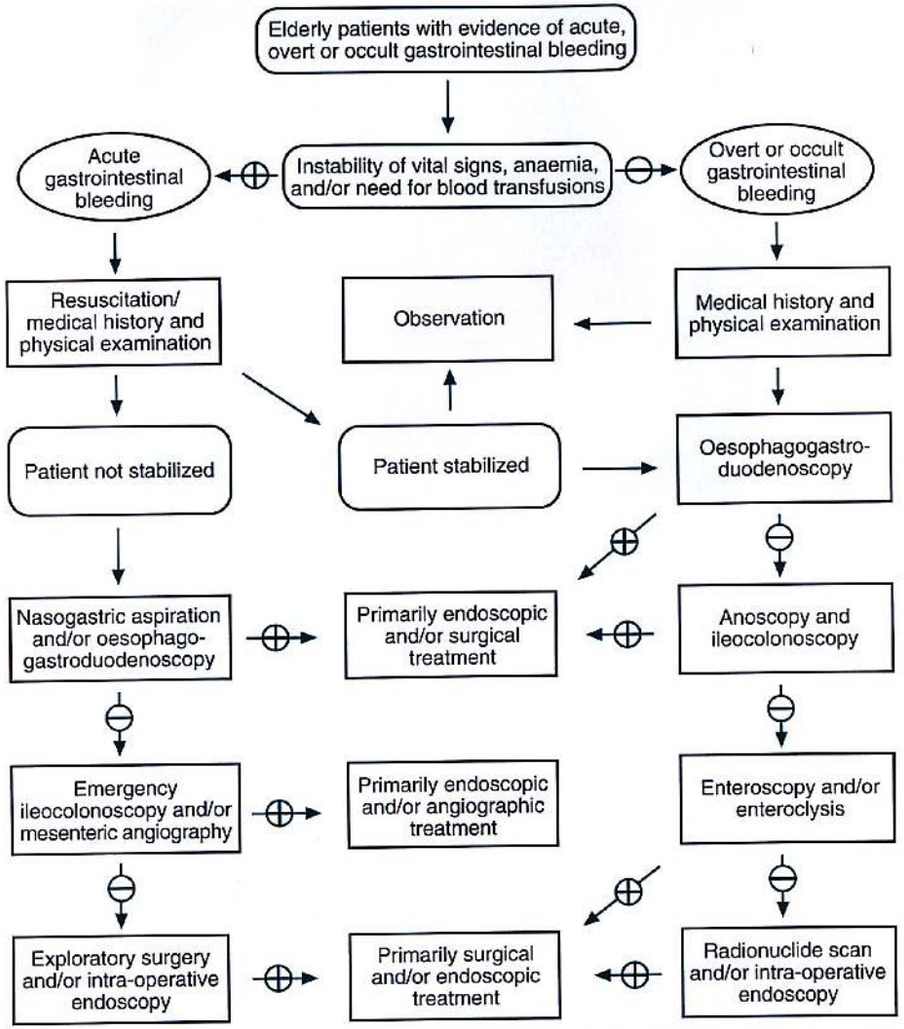

Figure 6. Algorithm for literature-based management of GI bleeding in the elderly. ⊕, positive response to question; ⊖, negative response to question.

SUMMARY

Figure 6 summarizes the management of elderly individuals with acute, overt or occult haemorrhage of the upper or lower GI tract. The proposed algorithm is literature- rather than evidence-based, in the face of a paucity of contemporary randomized controlled trials in the reviewed literature. Thus, a selection bias may exist, which is related to the authors' personal experience in this area. Outcome and cost-effectiveness studies are urgently needed. The aetiology of GI bleeding has not changed much throughout recent decades, but does decisively influence clinical presentation, diagnostic approach, therapeutic intervention and, eventually, prognosis. The initial examination of the patient should focus on signs of haemodynamic instability and ongoing haemorrhage. Subsequent comprehensive medical history, particularly concerning coagulopathies and co-morbid disease, should guide further diagnostic measures. Upper and lower endoscopy has evolved as the essential diagnostic tool, even in the emergency setting. Continued advances in endoscopic technique and equipment justify the synchronous performance of diagnostic and therapeutic procedures in a single session. Additional medical treatment may reduce the rate of rebleeding or prevent the first bleeding episode. Angiography is more appropriate in the acute setting. The major role of nuclear medicine scans may lie in the identification of potential bleeding sites in persons with occult bleeding. Exploratory surgery and intra-operative endoscopy are increasingly used if routine methods have failed to

Practice points

- the proportion of patients presenting with gastrointestinal (GI) haemorrhage who are elderly has markedly increased in recent years
- advanced age is associated with significant co-morbitidy and greater medication use
- mortality in GI haemorrhage of old age is mainly caused by extra-intestinal disease
- haemorrhage from the upper GI tract is 4–5 times more frequent than from the lower GI tract
- the aetiology of GI bleeding has not changed much in recent decades
- medical history has a high predictive value for the origin of bleeding
- elderly patients with acute GI bleeding need immediate and vigorous resuscitation
- coagulopathies should be corrected and NSAID/aspirin ceased as initial measures in GI bleeding
- oesophagogastroduodenoscopy is the primary diagnostic as well as therapeutic option in acute, overt and occult GI bleeding
- ileocolonoscopy and enteroscopy have evolved as therapeutic tools even in the emergency setting

Research agenda

- population-based prospective controlled studies on GI bleeding are needed
- risk assessement is required of elderly individuals presenting with GI haemorrhage
- the appropriate sequence of diagnostic measures in acute, overt or occult bleeding needs to be developed
- the prognostic value of endoscopic bleeding stigmata should be investigated
- differential therapeutic approaches to bleeding lesions in the GI tract need to be evaluated
- the development of an evidence-based algorithm for the management of GI haemorrhage is needed
- cost-effectiveness studies in this era of financial constraints in the health system should be carried out

detect and treat the source of GI bleeding. However, improved survival from advances in treatment has been offset by the rising number of high-risk elderly patients.

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