THE MANAGEMENT OF ACUTE UPPER GASTROINTESTINAL BLEEDING: A COMPARISON OF CURRENT CLINICAL GUIDELINES AND BEST PRACTICE

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ABSTRACT

Acute upper gastrointestinal bleeding (AUGIB) is the most common GI emergency, responsible for up to 70,000 hospital admissions in the UK and around 4,000 deaths. The latest UK national audit highlighted inconsistencies in both the management and service provision. Several national and international professional bodies have produced evidence-based recommendations on the management of AUGIB. We carried out a review of the guidance documentation published by four expert bodies including the National Institute of Clinical Excellence, the Scottish Intercollegiate Guidelines Network, the American College of Gastroenterology, and those published in the Annals of Internal Medicine. Consensus is still yet to be reached for initiating blood products in the emergency situation, with some evidence suggesting that liberal transfusion could exacerbate bleeding severity, although there is a lack of large randomised trials. It is widely agreed that prompt endoscopy within 24 hours improves outcomes, but evidence suggests that lowering this threshold confers no additional benefit. Use of proton pump inhibitors both pre and post-endoscopy for non-variceal bleeds is also advocated by professional bodies, with substantial evidence that it reduces the risk of re-bleeding. For patients with suspected oesophageal or gastric variceal bleeding, prophylactic antibiotics and vasopressin analogues are recommended, although guidelines vary on specific regimens. Recent UK and international guidelines provide a useful framework to guide management of patients who present to the emergency department with suspected AUGIB; however, their advice varies in some key areas due to a lack of large randomised trials as supporting evidence.

Keywords: Upper gastrointestinal bleeding, transfusion, endoscopy, proton pump inhibitors, non-variceal bleeding, variceal bleeding, antibiotics, vasopressin.

INTRODUCTION

Acute upper gastrointestinal bleeding (AUGIB) is the most common acute GI emergency and can potentially lead to serious haemodynamic compromise and mortality.[1] Consequently, several national and international guidelines have been developed to promote safe risk stratification and timely management of patients at the emergency department. Anatomically, AUGIB is defined as a frank blood loss from within the GI tract, originating proximal to the ligament of Treitz, i.e. from the oesophagus to the third part of the duodenum.[2,3] Symptomatically, AUGIB presents as haematemesis in the form of fresh blood or 'coffee-ground' vomitus with/without the presence of melaena.[4] AUGIB can also present as haematochezia and would be indicative of an extremely brisk blood loss.[5] Aetiologically, bleeding from the upper GI tract can be categorised into variceal and non-variceal, with 80-90% being secondary to non-variceal causes.[1] The latter include: peptic ulcer disease (20-50%), gastroduodenal erosions (8-15%), oesophagitis (5-15%), Mallory-Weiss tears (8-15%), and arteriovenous malformations/gastric antral vascular ectasia (5%). Other causes, such as highly vascularised tumours of the upper GI tract, make up the remainder.[3,4,6] Variceal bleeding (VB) originates

from gastric or oesophageal varicosities, most commonly in the context of portal hypertension.

The incidence of AUGIB in the UK is estimated at 84-172 per 100,000 patients, equivalent to 50-70,000 hospital admissions, and 4,000 deaths annually.[7,8] The substantial health-economic burden and the impact of this emergency on health services has been explored extensively in the literature.[9] The latest UK national audit carried out by the National Blood Service and the British Society of Gastroenterology (BSG) highlighted inconsistencies in service provision throughout the UK.[10] The 'Scope for improvement' report was published by the Association of Upper GI Surgeons, the BSG, Royal College of Nursing, Royal College of Physicians, and Royal College of Radiologists, and called for the development of services in order to address the heterogeneous management of patients presenting with AUGIB.[11]

METHODS

We carried out a PubMed search and identified several guidelines addressing the management of AUGIB. We have selected four expert key bodies that published guidance on this emergency and reviewed their recommendations. These include the National Institute of Clinical Excellence (NICE), the Scottish Intercollegiate Guidelines Network (SIGN), the American College of Gastroenterology (ACG), and those published in the *Annals of Internal Medicine*. Our aim was not to perform a rigorous systematic review of each recommendation, but to compare and contrast the available guidance. Where appropriate, pivotal studies, and the clinical importance of their findings, were also explored.

INITIAL MANAGEMENT

The management and approach to a patient presenting with an AUGIB should initially focus on resuscitative measures in response to a haemodynamic compromise. The circulation can be supported initially with intravenous (IV) crystalloids or colloids; however, prolonged resuscitation with saline should be avoided in patients with VB as this may encourage third-spacing and accumulation of ascites.[2,12] While transfusion of blood products can be life-saving in severe AUGIB, it remains unclear as to what their role is in lesser bleeds.[2] In 2002 the BSG advised transfusion for active haematemesis or the presence of hypovolaemic shock.[13] An AUGIB death is rarely related to the actual haemorrhage, but secondary to coexisting morbidities such as cardiorespiratory disease.[14]

Haemoglobin thresholds for transfusion in AUGIB remain controversial. More evidence is required as to whether a restrictive or liberal transfusion regimen confers the best prognosis.[15,16] A 2010 Cochrane review of three randomised controlled trials (RCTs) concluded that liberal transfusions conferred no benefit to survival and, in fact, there was a trend towards increased risk of re-bleeding and mortality, although this trend was not statistically significant.[17] However, a more recent meta-analysis by Wang et al.[18] analysed the data from four RCTs of restrictive versus liberal transfusion strategies in AUGIB and concluded that restrictive transfusion strategies should be employed.[18] Transfusion of red blood cells in AUGIB is common practice, but it is only the current guidelines on the management of AUGIB from the ACG and *Annals of Internal Medicine* that actually provide haemoglobin cut-offs for the initiation of transfusions. The threshold recommended in non-variceal bleeding (NVB) is a haemoglobin level of <7 g/dL, and for VB <8 g/dL.[12,19,20] The 2012 non-variceal guidelines from the ACG also advise that a higher haemoglobin level may be targeted in patients with significant comorbidities such as coronary artery disease; however, the exact value is debatable and should be tailored to each individual.[12,19,20] Table 1 summarises the current recommendations for the initial management of patients presenting with AUGIB.

Coagulopathy is an interesting area of AUGIB management as it can also be indicative of comorbidities such as liver disease, but there is very little evidence as to how such patients should be managed.[19,21] The guidelines for variceal bleeds from the ACG advise that transfusion of platelets and fresh frozen plasma (FFP) should be considered for patients with a significant coagulopathy, but again no specific thresholds are given (i.e. for international normalised ratio [INR] or platelet count).[12] In contrast, NICE does provide these parameters, but in a prospective national UK audit[22] (in which a coagulopathy was defined as an INR ≥1.5), there was a heterogeneous use of FFP, despite the finding that coagulopathy was associated with a 15% mortality rate.[7,21,23]

Risk Assessment

The presentation of AUGIB can range widely from minor non life-threatening bleeds to tragic

exsanguinations.[24] Initial risk stratification is vital to determine the timing of key interventions such as endoscopy.[25,26] In AUGIB, two main factors determine hospital admission: the need for haemodynamic support and whether endoscopic techniques are required to achieve haemostasis.[27]

Table 1: A comparison of the initial management, risk assessment, and timing of acute upper gastrointestinal bleeding.[2,7,12,20]

	National Institute of Clinical Excellence (NICE) 2012	Scottish Intercollegiate Guidelines Network (SIGN) 2008	American College of Gastroenterology 2012 (Non-variceal) and 2007 (Variceal)	*Annals of Internal Medicine* – Clinical Guidelines 2010 (Non-variceal)
Blood transfusion	Transfusion is recommended. No specific cut-offs.	Blood transfusion should be considered after a loss of 30% of the circulating volume.	Non-variceal: Transfuse to target haemoglobin levels of ≥7 g/dL with higher targets in severe blood loss or co-morbidities. Variceal: Transfuse to maintain haemoglobin of 8 g/dL.	Transfuse when haemoglobin levels ≤7 d/dL.
Correction of coagulopathy	FFP can be used in patients with either a fibrinogen level <1 g/L or a PT (INR)/APTT >1.5-times normal. PT complex concentrate can be given in those patients on warfarin and who are actively bleeding.	Not addressed.	Non-variceal: Not addressed. Variceal: Not addressed.	Correction of coagulopathy for patients on anticoagulants.
Transfusion of platelets	Transfuse when actively bleeding and a platelet count of <50.	Not addressed.	Non-variceal: Not addressed. Variceal: Not addressed.	Not addressed.
Risk scoring tools	Blatchford score initially, then complete Rockall score post endoscopy. Consider discharge if Blatchford is 0.	Use abbreviated and full Rockall score. Consider discharge if score is 0. Endoscopy if score is >0. Consider early discharge for patients with complete Rockall score of <3.	Non-variceal: A Blatchford score of 0 can allow the consideration of early discharge of these individuals without an inpatient endoscopy. Variceal: Risk assessment is not addressed with the use of formal scoring systems.	Both Blatchford and Rockall but there is no definitive statement as to which is recommended.
Timing	Immediate endoscopy unstable patients after resuscitation. Endoscopy within 24 hours for all other patients.	Within 24 hours.	Non-variceal: Within 24 hours. Within 12 hours if signs of shock or other high- risk clinical features. Variceal: Within 12 hours.	Within 24 hours.
Secondary care infrastructure	Not addressed.	Management in a dedicated gastrointestinal bleeding unit.	Not addressed.	Not addressed.

FFP: fresh frozen plasma; PT: prothrombin time; INR: international normalised ratio; APTT: activated partial thromboplastin time.

Several risk classification systems have been developed to guide the timing of intervention and predict clinical outcomes.[26] The Blatchford score includes clinical and serum parameters, which are easily available following initial resuscitation.[24] A prospective study undertaken in four UK hospitals by Stanley et al.[25] showed that this measure can identify individuals presenting with an AUGIB that are suitable for outpatient management.[25] A later study by Pang et al.[27] concluded that a score of 0 can predict low-risk patients with high specificity who can be considered for early discharge.[27] In contrast, the Rockall score combines clinical parameters with endoscopic findings to predict the probability of mortality.[28] It has been subsequently modified to exclude endoscopy results, although it appears to be inferior to the Blatchford score for this purpose.[29] Nevertheless, the full Rockall score has an important role in predicting re-bleeding and mortality and, despite its limitations, remains the most widely used system both in the UK and US.[27,30,31]

Timing of Endoscopy

Evidently, oesophagogastroduodenoscopy (OGD) remains key to the management of AUGIB by providing diagnosis and enabling therapeutic intervention.[27] The severity and the suspected underlying aetiology of the AUGIB influence the urgency for endoscopy.[29,32,33] A 1992 meta-analysis showed that prompt endoscopic therapy reduces the risk of death, re-bleeding, and the need for surgery.[34] However, the urgency of endoscopy has been variably defined in the literature ranging from 2-24 hours after initial presentation.[19] A 1993 audit, led the BSG to recommend that high-risk patients should have endoscopy performed within 24 hours of presentation – a consensus reiterated by all guidelines compared in this review article.[2,7,13,20] NICE further propose that endoscopy should be offered immediately in unstable patients, with the ACG recommending that those patients with features of shock or suspected VB undergo endoscopy within 12 hours.[7,12,20]

A review of RCTs and retrospective studies performed in 2009[35] failed to provide evidence that endoscopy, within a few hours of presentation, impacts mortality or reduces the re-bleeding risk, but advocated endoscopy within 24 hours.[35] Similarly, a recent prospective study of 4,478 patients[22] concluded that endoscopy within 12 hours of presentation did not reduce mortality.[22] In order to meet recommendations, endoscopy units require the infrastructure to provide an emergency service 24 hours per day. A nationwide UK audit carried out in 2007 showed that only 50% of OGDs were being performed within 24 hours, increasing to 55% for high-risk patients.[10] One possible explanation is that only 52% of the participating centres had a formal out-of-hours emergency consultant rota. The audit highlighted the need for dedicated GI bleeding units, consisting of experienced nursing staff and evidence-based protocols. Table 1 summarises the current recommendations regarding risk assessment and the timing of endoscopy.

NVB

Advances in endoscopic and pharmacological therapies in the past few decades have reduced recurrent bleeding, the need for surgery, and mortality from upper GI blood loss.[26]

Proton Pump Inhibitors (PPIs) and Prokinetic Agents

Drugs that modify gastric acid secretion have made a large impact upon the prevention of peptic ulcer disease and outcomes in AUGIB.[21] A 2010 Cochrane review of six RCTs, where an IV PPI was administered on admission, demonstrated that there was a significant reduction in high-risk GI lesions found at endoscopy, signalling a reduced need for therapeutic intervention. There was, however, no significant effect on overall mortality, the need for surgery, or rates of re-bleeding.[36] Both NICE and SIGN do not advocate the use of PPIs prior to endoscopy.[2,7,20]

Administration of prokinetics, prior to endoscopy, is thought to aid visualisation and endoscopic diagnostic yield.[5] Barkun et al.[19] suggested that IV erythromycin or metoclopramide, prior to endoscopy for an AUGIB, decreased the requirement of repeat endoscopy for lesion identification.[19] Nevertheless, the use of these agents has not formed part of standard practice due to the lack of evidence regarding the improvement of clinical outcomes, and it has been agreed that they should be restricted to only those patients with a large volume of blood in the stomach.[5,19,20]

Endoscopic Therapy

The modified Forrest classification is commonly used to categorise the appearances of ulcers found in endoscopy to direct appropriate therapy.[37]

Table 2: Summary of the current recommendations for the management of non-variceal bleeding.[2,7,12,20]

	National Institute of Clinical Excellence (NICE) 2012	Scottish Intercollegiate Guidelines Network (SIGN) 2008	American College of Gastroenterology 2012 (Non-variceal) and 2007 (Variceal)	*Annals of Internal Medicine* – Clinical Guidelines 2010 (Non-variceal)
Pre-endoscopy PPI	Do not give PPI or H2 receptor antagonist.	Do not give PPI.	High-dose IV PPI e.g. 80 mg bolus followed by 8 mg/hr infusion).	PPIs can be used to decrease the need for endoscopic therapy.
Prokinetics	Not addressed.	Not addressed.	Consideration of IV erythromycin infusion prior to endoscopy.	Do not use routinely.
Endoscopic therapy – which lesion?	Not addressed.	Actively bleeding lesions, non-bleeding visible vessels and for those with an adherent clot.	Actively bleeding lesions and non-bleeding visible vessels, adherent clots especially in those patients who may be at greater risk of re-bleeding.	Actively bleeding lesions or visible vessels. Adherent clots at endoscopy can be dislodged with irrigation and then appropriately treated.
Endoscopic therapy – type	Adrenaline monotherapy not recommended. Consider co-therapy with clips, thermal coagulation, fibrin, or thrombin.	Adrenaline monotherapy not recommended. Use co-therapy with adrenaline injection (13 ml of 1:10,000) and clips or thermal coagulation.	Adrenaline monotherapy not recommended. Thermal therapy and sclerosant injection and clips are recommended. For actively bleeding lesions, thermal or adrenaline injection with a second modality would be preferred over clips or sclerosant alone.	Adrenaline monotherapy not recommended. Consider co-therapy with clips, thermal coagulation, or sclerosant injection for high-risk lesions.
Post-endoscopy PPI	Give PPIs in patients with stigmata of recent haemorrhage. PPI type not specified.	High-dose PPIs in major bleeding. Give omeprazole or pantoprazole 80 mg bolus and then 8 mg/hour infusion for 72 hours.	High-dose PPIs when active bleeding, visible vessels, or an adherent clot. Give 80 mg bolus and then 8 mg/hour infusion for 72 hours. PPI type not specified. Other lesions in the Forrest classification can receive once-daily oral PPI.	An IV bolus followed by a continuous PPI. PPI type not specified.
Repeat endoscopy	Consider in patients with high risk of re-bleeding, especially if there is doubt that haemostasis has been achieved. Repeat endoscopy for patients who re-bleed. Consider surgical options for failed haemostasis. Interventional radiology for unstable patients who re-bleed after a 'second look' endoscopy and subsequent therapy.	Endoscopy should be repeated within 24 hours if initial treatment was thought not to be sufficient or if subsequent bleeding would likely result in death.	Not recommended unless there is a re-bleed. Interventional radiology or surgery should be considered for patients who re-bleed after a 'second look' endoscopy and subsequent therapy.	Not recommended unless there is a re-bleed.

PPI: proton pump inhibitor; IV: intravenous.

In this classification, Grade 1 is for active bleeding (1a active spurting, 1b for active oozing), Grade 2 is for those with the stigmata of recent haemorrhage (2a visible vessel, 2b adherent clot, 2c flat pigmented spot), and Grade 3 for lesions without signs of recent haemorrhage.[21,26,38] The size of the ulcer and signs of bleeding have been shown to correlate with the risk of re-bleeding and death.[28] Endoscopic therapy is indicated for Grades 1 and 2a; however, the role in Grade 2b lesions has proved controversial despite the reported 8-36% risk of recurrent haemorrhage.[19,20,38,39] A meta-analysis of five RCTs[40] showed that endoscopic intervention was effective for ulcers with active bleeding or visible vessels, but that the role in those with an adherent clot was uncertain.[40] Conversely, Kahi et al.[39] suggested that endoscopic therapy can prevent re-bleeding in the presence of adherent clots.[39]

A number of modalities for endotherapy can promote haemostasis, including injection, thermocoagulation, and application of mechanical clips.[21,26] The beneficial role of adrenaline injections was demonstrated in the late 1980s with a prospective study[41] which compared injection without other endoscopic therapy, and found that adrenaline significantly improved outcomes.[41] Adrenaline has been popular with clinicians due to its safety profile, ease of use, and cost-effectiveness, but today it is considered inferior to other monotherapies or combination therapies.[39,42-45] A meta-analysis in 2004 of 1,673 patients comparing adrenaline alone with adrenaline and a second endoscopic technique, showed that the additional therapy reduced the re-bleeding rate from 18.4% to 10.6%, and mortality from 5.1% to 2.6%.[42] A more recent Cochrane review further confirmed these findings.[44] A meta-analysis by Yuan et al.[45] suggested that clipping is no more superior to other modalities,[45] and this was also a point made by Laine and McQuaid.[40] This latter study also demonstrated that monotherapy with thermal devices, sclerosants, clips, thrombin, or fibrin glue provides more effective haemostasis than adrenaline alone.

Post-Endoscopy Management in NVB

PPIs

The use of PPIs post endoscopy has been extensively studied, with evidence that a high-dose PPI produces an almost neutral pH within the stomach, favouring haemostasis by enhancing platelet aggregation and clot formation.[1,46] A study comparing high-dose omeprazole (an initial bolus IV injection of 80 mg, followed by an infusion of 8 mg per hour for 72 hours) versus placebo after endotherapy to bleeding peptic ulcers, revealed that PPI substantially reduced the risk of recurrent bleeding.[46] A larger trial using esomeprazole demonstrated a reduction in re-bleeding rates at 72 hours sustained for 30 days.[47] A 2006 Cochrane review of 24 trials also supported the use of PPI therapy after endoscopy. Nevertheless, there remains limited evidence of any reduction in mortality.[48] The standard regimen in the guidelines is the initial bolus of PPI followed by an infusion over 72 hours, and this has been acknowledged in the majority of the non-variceal literature included in this article.

Repeat endoscopy

Despite endotherapy, re-bleeding is common in patients with AUGIB (between 15-20% of patients).[49] Guidelines focus upon whether a repeat endoscopy should be performed prophylactically to ensure that adequate haemostasis has been achieved or whether it should be only utilised following a confirmed re-bleed. Marmo et al.[49] found a significant reduction in re-bleeding compared to a control group who did not undergo repeat endoscopy;[49] however, it should be noted that the trials included in this particular review were published between 1990 and 2000, prior to the routine use of high-dose PPI post procedure, which confers a reduction in re-bleeding in its own right. Nonetheless, a more recent trial[50] comparing re-endoscopy and surgery in re-bleeding found that further endotherapy reduced the need for surgery and was associated with fewer complications.[50] UK guidelines advocate the 'second look' endoscopy in those patients at high risk of a further bleed or if there is potentially inadequate haemostasis at initial endoscopy.[2,7] However, non-variceal guidelines from the US confine its use to those patients who re-present with subsequent haemorrhage.[12,20]

VB

Variceal haemorrhage is due to oesophageal or gastric varices, secondary to portal hypertension conferred by liver cirrhosis. Indeed, oesophageal varices are present in approximately 30-40% of patients with cirrhosis,[51] and bleeding from varices

occurs at an annual rate of 5-15%.[1] Despite the high mortality rate from variceal haemorrhage, there has been a reduction over recent years, most likely precipitated by the use of antibiotic prophylaxis, portal antihypertensives, and effective endoscopic therapy.[51]

Antibiotic Prophylaxis

Bacterial infection is another frequent complication in cirrhotic patients with an AUGIB, present in 25-65% of patients on admission or during their hospital stay,[12,52] and it is believed to promote VB.[53]

Table 3: Summary of the recommendations for the management of variceal haemorrhage.[2,7,12]

	National Institute of Clinical Excellence (NICE) 2012	Scottish Intercollegiate Guidelines Network (SIGN) 2008	American College of Gastroenterology 2012 (Non-variceal) and 2007 (Variceal)	*Annals of Internal Medicine* – Clinical Guidelines 2010 (Non-variceal)
Prophylactic antibiotics	Recommended (No preferred type)	Recommended (No preferred type)	Short-term antibiotic prophylaxis should be given to every patient with cirrhosis. Oral norfloxacin 400 mg BD or IV ciprofloxacin is the recommended regimen. IV ceftriaxone may be used in advanced liver disease or if there are high rates of quinolone resistance.	N/A
Pharmacological therapy	Terlipressin recommended.	Terlipressin recommended.	Pharmacological therapy (somatostatin, terlipressin, octreotide) should be commenced as soon as variceal haemorrhage is suspected and continued for 3-5 days.	N/A
Endoscopic intervention	Band ligation should be used for oesophageal varices. Endoscopic injection of N-butyl-2-cyanoacrylate should be used for gastric varices. TIPS should be considered if variceal bleeding is not controlled by the above measures.	Band ligation should be used for oesophageal varices. Endoscopic injection of N-butyl-2-cyanoacrylate should be used for gastric varices. TIPS should be considered if variceal bleeding is not controlled by the above measures. Balloon tamponade can be considered as a temporary measure if bleeding is failed to be controlled.	Band ligation or sclerotherapy should be used for oesophageal varices. Endoscopic injection of N-butyl-2-cyanoacrylate should be used for gastric varices. TIPS should be considered if variceal bleeding is not controlled by the above measures. Balloon tamponade can be considered as a temporary measure (maximum 24 hours) if bleeding is failed to be controlled.	N/A

IV: intravenous; TIPS: transjugular intrahepatic portosystemic shunt.

Seminal work in 1985 by Rimola et al.[54] first showed that the prophylactic use of non-absorbable, oral antibiotics can significantly reduce the incidence of concomitant infection in cirrhotic patients with AUGIB.[54] A 2002 review concluded that short-term antibiotic use decreased both the rate of infection and mortality – this was evident regardless of the presence of ascites.[55] An updated 2010 Cochrane review further supported the use of antibiotics.[56] Norfloxacin is a poorly absorbed quinolone that was shown to be successful in preventing bacterial infections in cirrhotic patients with GI haemorrhage, and has subsequently been standard for this purpose.[57] However, a study[52] comparing oral norfloxacin with IV ceftriaxone found that the latter was a more effective prophylactic agent in patients with advanced cirrhosis.[52] Regardless of the agent, short-term antibiotic prophylaxis is recommended in the NICE and SIGN guidelines, together with those from the ACG.[2,7,12]

Pharmacological Therapy

Other pharmacological therapies used in VB act to lower the portal pressure and thus reduce the blood flow to the varices. They do not, however, replace the need for endotherapy.[51] Vasopressin is a potent vasoconstrictive agent, but may also affect the blood supply to the myocardium; the high risk for cardiac complications has limited its use in reducing portal pressures. Terlipressin is a synthetic analogue of vasopressin and is less potent. A systematic review of the use of terlipressin in acute variceal haemorrhage found that this particular agent not only controls blood loss, but uniquely reduces mortality.[58] As in-hospital mortality rates from variceal haemorrhage are between 20-50%,[58] it can be seen that terlipressin would appear to be the most appropriate choice as the first-line pharmacological agent. Octreotide (a synthetic analogue of somatostatin) has also been shown to be effective in controlling bleeding, and some authors found that it can be superior to its other vasoactive counterparts in oesophageal bleeding.[59]

A meta-analysis found that the efficacy of endotherapy was significantly improved when used in synergy with vasocontrictors.[60] A later review from D'Amico et al.,[61] comparing sclerotherapy with vasoactive drugs, found no difference in efficacy when looking at the controlling of bleeding, and concluded that they can be safely used as initial therapy prior to endoscopy.[61] The NICE and SIGN guidelines advocate the use of terlipressin in any patient with a suspected variceal bleed, but the guidelines from the ACG specify that either terlipressin, octreotide, or somatostatin should be initiated rapidly in the acute setting.[2,7,12] The differences between the USA and UK in this regard may be due to the differences in drug pricing or licensing between the two countries.[62]

Endoscopic Therapy

Endoscopy remains at the forefront of the current management in VB. The two endoscopic methods available to treat bleeding oesophageal varices are band ligation and sclerotherapy. Endoscopic sclerotherapy has been shown to be a highly effective method of controlling an initial bleed and can halt blood loss in up to 90% of patients.[63] A study of variceal banding versus sclerotherapy reported that banding was superior in terms of the control of bleeding and re-bleeding risk, and also reduced mortality rates.[64] Consequently, band ligation has been recommended universally as the first choice for oesophageal varices in all of the current guidelines, but those from the ACG make the addition that sclerotherapy can be used when banding is not technically feasible.[2,7,12] Gastric varices can be managed by banding, sclerotherapy, or endoscopic injection of the tissue adhesive N-butyl-2-cyanoacrylate. A recent trial comparing banding versus cyanoacrylate injection found that glue injection was more effective at controlling the initial haemorrhage and reducing re-bleeding rates.[65] A similar study showed no difference in terms of the initial haemorrhage control, but found that cyanoacrylate reduced the long-term re-bleeding risk.[66] The endoscopic injection of this adhesive has been adopted as first-line practice on both sides of the Atlantic.[2,7,12]

DISCUSSION

Guideline consensuses for the management of AUGIB still present some uncertainties. With regards to initiating blood products, some evidence suggests that liberal transfusion could exacerbate bleeding severity, although there is a paucity of large RCTs. Conversely, it is clear that prompt endoscopy (within 24 hours) improves outcomes, but evidence suggests that lowering this threshold (e.g. to 12 hours) confers no additional benefit. The use of PPIs, both pre and post endoscopy, for non-variceal bleeds is also advocated by professional bodies, with substantial evidence that it reduces

the risk of re-bleeding. For patients with suspected oesophageal or gastric VB, prophylactic antibiotics and vasopressin analogues are recommended. In summary, recent UK and international guidelines provide a useful framework to guide management of patients who present to the emergency department with suspected AUGIB; however, their advice varies in some key areas due to the lack of large RCTs.

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