Scientific Research Publishing

Open Journal of Internal Medicine, 2026, 16(1), 110-119
https://www.scirp.org/journal/ojim
ISSN Online: 2162-5980
ISSN Print: 2162-5972

Guideline-Directed Combination Endoscopic Therapy for Severe Forrest Ib Duodenal Ulcer Bleeding in a Young Adult: A Case Report and Literature Review

Yashar Tolentino Najiagdam

Department of Internal Medicine, Division of Critical Care Medicine, Nairobi West Hospital, Nairobi, Kenya
Email: dryasharnajiaghdam@outlook.com

How to cite this paper: Najiagdam, Y.T. (2026) Guideline-Directed Combination Endoscopic Therapy for Severe Forrest Ib Duodenal Ulcer Bleeding in a Young Adult: A Case Report and Literature Review. *Open Journal of Internal Medicine*, **16**, 110-119.
https://doi.org/10.4236/ojim.2026.161012

Received: February 22, 2026
Accepted: March 17, 2026
Published: March 20, 2026

Abstract

Non-variceal upper gastrointestinal bleeding (NVUGIB) remains a major cause of emergency hospitalization and significant morbidity worldwide. Peptic ulcer disease is the most common etiology. Active ulcer bleeding classified as Forrest Ib (oozing hemorrhage) represents high-risk stigmata requiring urgent endoscopic therapy to reduce rebleeding and mortality. We report a 23-year-old patient presenting with melena and severe anemia (hemoglobin 6 g/dL). Urgent esophagogastroduodenoscopy revealed an actively oozing duodenal ulcer consistent with Forrest Ib classification. Endoscopic therapy consisted of diluted epinephrine injection followed by definitive mechanical hemoclip placement. Complete hemostasis was achieved without rebleeding. Hemoglobin improved to 13 g/dL following transfusion and stabilization. Management adhered to the 2021 ACG and 2021 ESGE guidelines recommending combination therapy for high-risk ulcer bleeding and discouraging epinephrine monotherapy.

Keywords

Upper Gastrointestinal Bleeding, Duodenal Ulcer, Forrest Classification, Forrest Ib, Endoscopic Clipping, Epinephrine Injection, Combination Therapy, Non-Variceal Hemorrhage, Rebleeding Risk, ACG 2021, ESGE 2021

1. Introduction

Acute upper gastrointestinal bleeding (UGIB) remains one of the most common gastrointestinal emergencies globally, with an estimated annual incidence ranging between 50 and 150 cases per 100,000 population [1]. Despite improvements in

endoscopic therapy and pharmacologic management, mortality remains approximately 5% - 10% [2]. Peptic ulcer disease accounts for approximately 40% - 50% of NVUGIB cases [1].

Non-variceal upper gastrointestinal bleeding (NVUGIB) remains a major global healthcare burden. Contemporary epidemiologic studies estimate an annual incidence of 50 - 150 cases per 100,000 population, though regional variation exists [1]. Higher incidence rates are reported in elderly populations and regions with elevated *Helicobacter pylori* prevalence or NSAID consumption [3]. Over the past three decades, mortality rates have modestly declined due to improved endoscopic therapy and proton pump inhibitor (PPI) utilization [4]. However, mortality remains between 5% and 10%, with higher rates observed in elderly patients and those with significant comorbidities [1].

Interestingly, while overall incidence of peptic ulcer disease has declined in Western countries due to *H. pylori* eradication programs, the proportion of ulcer bleeding associated with NSAID and antithrombotic therapy has increased [3]. In younger populations, bleeding ulcers are less common but can present dramatically when underlying etiologic factors are present, including *H. pylori* infection, NSAID use, stress-related mucosal injury, smoking, and undiagnosed hypersecretory states. Rebleeding remains the primary driver of morbidity and mortality [1]. Early endoscopic intervention and durable hemostasis significantly reduce adverse outcomes [4].

The Forrest classification system stratifies bleeding ulcers and guides management decisions. Forrest Ia and Ib lesions are considered high-risk stigmata requiring urgent intervention [5]. Management of patients with ulcer bleeding requires a systematic approach incorporating risk assessment, timely endoscopic intervention, and appropriate pharmacologic therapy [6].

2. Case Presentation

2.1. Pre-Bleed Clinical History

A 23-year-old male patient presented with a two-day history of melena and associated fatigue. The patient reported a one-week history of intermittent NSAID use (ibuprofen 400 mg twice daily) for tension headache management. He denied regular aspirin, anticoagulant, or antiplatelet therapy. There was no prior history of peptic ulcer disease, gastrointestinal bleeding, or dyspepsia. Social history was notable for occasional alcohol consumption (1 - 2 drinks per week) and no tobacco use. The patient denied recent stress, corticosteroid use, or known hypersecretory states. Comorbidities were limited to intermittent tension headaches managed with over-the-counter analgesics. Family history was non-contributory for gastrointestinal disorders.

2.2. Initial Presentation and Stabilization

On presentation, the patient appeared pale and fatigued but was hemodynamically stable. Initial vital signs were as follows: blood pressure 98/62 mmHg, heart rate 108 beats/minute, respiratory rate 20 breaths/minute, temperature 36.8˚C, and

oxygen saturation 97% on room air. Shock index was calculated at 1.1 (heart rate/systolic blood pressure), indicating hemodynamic compromise.

Key laboratory studies on admission revealed: hemoglobin 6 g/dL (severe anemia), hematocrit 18%, platelet count 230 × 10^9/L, BUN 28 mg/dL, creatinine 0.9 mg/dL (BUN/Cr ratio 31.1), INR 1.0, and serum lactate 2.1 mmol/L. Liver function tests were within normal limits.

The patient was immediately resuscitated with intravenous crystalloid fluids. Two units of packed red blood cells were transfused during initial stabilization, with appropriate hemodynamic response (blood pressure improved to 110/70 mmHg, heart rate decreased to 92 beats/minute) [7]. Two large-bore peripheral intravenous lines (18-gauge) were secured, and continuous cardiac monitoring was initiated.

2.3. Risk Stratification

Formal risk stratification was performed at presentation. The Glasgow-Blatchford Score was calculated at 15 (indicating high risk requiring urgent intervention), and the pre-endoscopic Rockall Score was 4 (high-risk category) [8] [9]. These scores supported the decision for urgent endoscopic evaluation within 12 hours and informed the need for close monitoring in an intensive care setting [10]. However, given the presence of active bleeding with hemodynamic compromise, urgent endoscopy was indicated regardless of risk score.

2.4. Endoscopic Findings and Diagnostic Work-Up

Urgent esophagogastroduodenoscopy was performed within 10 hours of presentation [11]. Endoscopy demonstrated a duodenal bulb ulcer with active oozing hemorrhage consistent with Forrest Ib classification [5]. The ulcer measured approximately 12 mm in diameter and was located on the posterior wall of the duodenal bulb—an anatomically high-risk region due to proximity to the gastroduodenal artery [12].

Following thorough water jet irrigation and suction clearance of pooled blood, no active spurting vessel was visualized; however, persistent low-pressure oozing from the ulcer base was observed. No large adherent clot requiring extraction was identified.

During the same endoscopic session, gastric mucosal biopsies were obtained from the antrum and body for histopathological evaluation, and a rapid urease test (CLO test) was performed [13]. Histology subsequently confirmed chronic active gastritis with *H. pylori* organisms identified on Giemsa staining. The rapid urease test was also positive for *H. pylori*.

3. Endoscopic Management

Endoscopic hemostasis was achieved using combination therapy as recommended by current guidelines [14] [15].

3.1. Injection Therapy

A 23-gauge sclerotherapy needle was advanced through the working channel. Di-

luted epinephrine (1:10,000) was injected circumferentially around the bleeding site in four quadrants, with a total volume of approximately 8 - 10 mL. The needle was inserted 2 - 3 mm into tissue with slow injection until mucosal blanching was observed. Care was taken to avoid intravascular injection. The goals were temporary vasoconstriction, mechanical tamponade, improved visualization, and reduction in bleeding flow. Following injection, active oozing significantly decreased, allowing clear identification of the bleeding vessel [16].

3.2. Mechanical Hemostasis

Subsequently, a through-the-scope (TTS) hemoclip device was selected for definitive mechanical hemostasis [17]. The scope was positioned to achieve a perpendicular approach to the vessel axis. Stable scope position was achieved using torque steering and slight withdrawal. The bleeding point was centered in the visual field, and the clip jaws were opened fully prior to deployment.

Critical principles applied included capture of submucosa (not just superficial mucosa), avoidance of tangential placement, and ensuring vessel compression rather than edge approximation only. The clip was deployed directly across the visible bleeding point. Immediate cessation of bleeding was observed. No additional clips were required.

3.3. Hemostasis Confirmation

Hemostasis was confirmed by: 1) absence of active bleeding for more than 2 minutes, 2) no visible arterial pulsation, 3) stable clip positioning, and 4) clear field after irrigation. The patient remained hemodynamically stable throughout the procedure (**Figure 1**).

Figure 1. Endoscopic Images

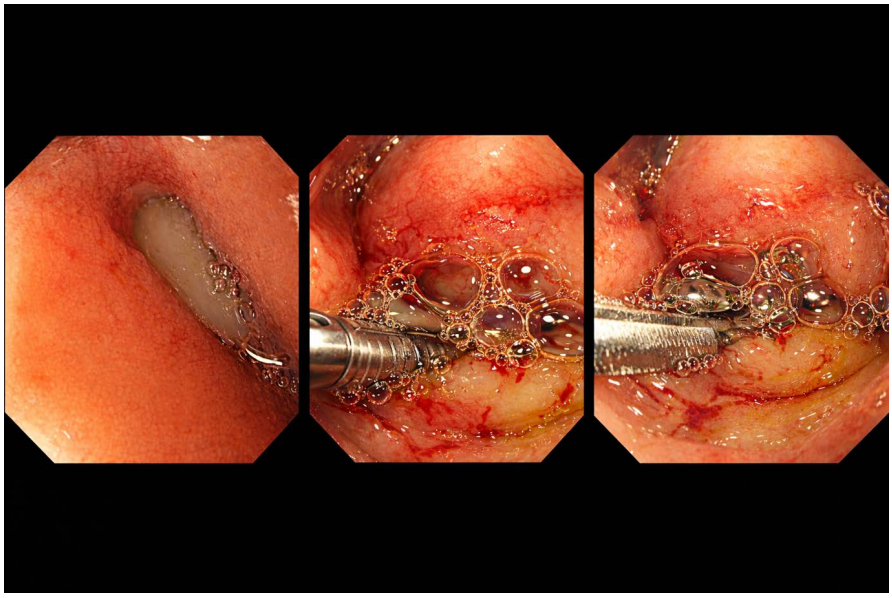

Figure 1. (A) Active oozing hemorrhage from duodenal ulcer (Forrest Ib). (B) Post-hemoclip placement with complete hemostasis.

4. Post-Endoscopic Management

Following successful hemostasis, high-dose intravenous proton pump inhibitor therapy was administered (omeprazole 80 mg bolus followed by 8 mg/hour continuous infusion) for 72 hours in accordance with guideline recommendations [14] [18]. Serial hemoglobin monitoring was performed every 8 hours. A restrictive transfusion strategy was employed, with additional transfusion considered only for hemoglobin less than 7 g/dL or symptomatic anemia.

The patient was monitored closely for recurrent melena, hematemesis, or hemodynamic instability. No rebleeding occurred during hospitalization.

4.1. *H. pylori* Management Plan

Given the positive rapid urease test and histologic confirmation of *H. pylori* infection, eradication therapy was initiated prior to discharge [13]. A 14-day bismuth-based quadruple regimen was prescribed: bismuth subsalicylate 524 mg four times daily, metronidazole 500 mg three times daily, tetracycline 500 mg four times daily, and omeprazole 20 mg twice daily.

Confirmation of eradication was planned via urea breath test at least 4 weeks after completion of antibiotics and 2 weeks after discontinuation of PPI therapy.

If *H. pylori* testing had been negative, the alternative management plan would have included: 1) comprehensive evaluation for ongoing NSAID or aspirin exposure, 2) assessment for Zollinger-Ellison syndrome if clinically suspected (elevated gastrin levels, multiple ulcers, refractory disease), and 3) counseling on avoidance of ulcerogenic medications including alcohol and NSAIDs.

4.2. Outcome and Follow-Up

Time from presentation to endoscopy was 10 hours. Total length of hospital stay was 4 days. Hemoglobin improved from 6 g/dL at admission to 9.2 g/dL at discharge. The reported hemoglobin of 13 g/dL was documented at the 6-week outpatient follow-up visit.

No outpatient rebleeding episodes were reported during the follow-up period. No repeat endoscopy was required. The patient reported full adherence to prescribed medications and completed the *H. pylori* eradication regimen without complications. Follow-up urea breath test at 6 weeks post-treatment confirmed successful eradication of *H. pylori*.

5. Discussion

5.1. Forrest Classification and Risk Stratification

The Forrest classification categorizes ulcers based on endoscopic findings and guides management decisions [5]. Forrest Ib represents active oozing hemorrhage and carries a high risk of rebleeding if untreated. Risk stratification tools such as the Glasgow-Blatchford Score and Rockall Score assist in predicting outcomes and informing triage decisions, though they do not replace urgent endoscopic therapy in actively bleeding lesions [8] [9].

5.2. Pathophysiology

Duodenal ulcers arise from an imbalance between mucosal defense mechanisms and luminal aggressive factors. Protective elements include mucus-bicarbonate barrier, prostaglandin-mediated mucosal blood flow, epithelial restitution, and tight junction integrity. Aggressive factors include gastric acid, pepsin, *H. pylori*-mediated inflammation, and NSAID-induced prostaglandin inhibition.

Helicobacter pylori colonization leads to chronic active gastritis and increased gastrin production, promoting acid hypersecretion [13]. In the duodenal bulb, acid exposure results in gastric metaplasia, allowing *H. pylori* colonization and local inflammatory damage. NSAIDs inhibit cyclooxygenase-1 (COX-1), reducing prostaglandin synthesis, which leads to reduced mucosal blood flow, impaired mucus secretion, and increased susceptibility to acid injury [19]. Concurrent *H. pylori* infection synergistically increases ulcer bleeding risk [3].

Ulcer bleeding occurs when erosion penetrates into submucosal or arterial vessels. In posterior duodenal ulcers, the gastroduodenal artery is particularly vulnerable. However, acidic pH destabilizes clot integrity by impairing platelet aggregation and fibrin polymerization. This explains why high-dose PPI therapy reduces rebleeding risk by maintaining intragastric pH above 6 [18].

5.3. Endoscopic Hemostatic Modalities

Combination therapy significantly reduces rebleeding compared to injection alone [16] [20]. Mechanical clipping provides durable vessel compression and is widely recommended in international guidelines [14] [15]. Injection monotherapy has rebleeding rates of approximately 20% - 30%, while combination therapy reduces this to 5% - 10% [20].

Endoscopic therapy for acute non-variceal upper gastrointestinal bleeding has been extensively studied, with meta-analyses demonstrating significant reductions in rebleeding and mortality [21].

Through-the-scope (TTS) clips are mechanical compression devices deployed via standard working channels [17]. Mechanically, clips function by exerting radial compression across the vessel, collapsing the lumen and interrupting arterial inflow. Key technical principles include perpendicular orientation to vessel axis, adequate submucosal capture, avoidance of superficial mucosal-only grasp, and deployment on non-friable tissue margins.

Over-the-scope clip (OTSC) systems are larger nitinol-based devices mounted externally on the endoscope tip [22]. They provide full-thickness tissue capture, greater compression force, and wider span coverage. Randomized trials show OTSC superiority in recurrent bleeding after standard therapy [22]. Thermal probes achieve hemostasis via coaptive coagulation but carry risks of deep tissue necrosis and delayed perforation. Topical hemostatic powders provide temporary tamponade but do not offer definitive vessel compression.

5.4. Guideline Recommendations

The 2021 American College of Gastroenterology (ACG) guidelines provide strong recommendation for combination therapy for high-risk stigmata (Forrest Ia and Ib), discourage epinephrine monotherapy, recommend high-dose IV PPI for 72 hours following successful hemostasis, and advocate for a restrictive transfusion strategy [14].

The 2021 European Society of Gastrointestinal Endoscopy (ESGE) guidelines recommend combination therapy (strong recommendation, high-quality evidence), consider OTSC in refractory bleeding, and recommend repeat endoscopy for re-bleeding [15]. The International Consensus Group recommends early endoscopy within 24 hours, combination therapy as preferred approach, and consideration of angiographic embolization before surgery in refractory cases [23]. The Asia-Pacific Consensus emphasizes risk stratification, supports early endoscopy, and advocates *H. pylori* eradication [24]. All major guidelines converge on definitive mechanical therapy as standard of care [25].

Current policies in acute NVUGIB management emphasize a structured approach to diagnosis, risk stratification, and treatment [26].

All major guidelines converge on definitive mechanical therapy as standard of care.

5.5. Management of Rebleeding

After successful endoscopic hemostasis and initiation of high-dose intravenous PPI therapy, routine scheduled second-look endoscopy is generally not indicated in the absence of clinical evidence of rebleeding [15]. Post-procedural management should instead emphasize close clinical monitoring and prompt repeat endoscopy if rebleeding is suspected.

Early repeat endoscopy should be triggered by clinical rebleeding, including recurrent hematemesis or fresh melena, hemodynamic instability (tachycardia, hypotension, rising shock index), hemoglobin drop (commonly ≥ 2 g/dL) or ongoing transfusion requirement, and persistent high nasogastric aspirate blood (if NGT used). Repeat endoscopy is typically performed urgently (same day/within 24 hours) once rebleeding is suspected, after immediate resuscitation.

At re-endoscopy, escalation follows a structured hierarchy: re-clip (additional TTS clips) if anatomy allows, add thermal therapy if clip placement is limited, consider OTSC for fibrotic ulcer base, failed standard clipping, or recurrent bleeding after initial combination therapy [22]. Randomized trials of endoscopic hemostatic techniques have established the superiority of combination therapy over monotherapy [27]. Endoscopic management strategies in peptic ulcer bleeding continue to evolve with advances in device technology and technique [28]. If endoscopic salvage fails, angiographic embolization is preferred before surgery in many settings.

5.6. Key Clinical Principles

This case underscores several critical clinical principles. First, age alone does not

determine risk severity; young patients may present with life-threatening anemia despite preserved hemodynamics. Second, Forrest Ib lesions require definitive therapy despite the absence of spurting hemorrhage; oozing bleeding may represent unstable arterial erosion. Third, injection therapy should be viewed as an adjunct rather than definitive treatment. Fourth, mechanical compression remains the cornerstone of durable hemostasis. Fifth, anatomical awareness of posterior bulb vascular proximity guides therapeutic choice. Sixth, adherence to guideline-directed therapy reduces variability in outcomes [25]. Seventh, structured escalation algorithms reduce delays in definitive management. Eighth, acid suppression is mechanistically essential for clot stabilization. Ninth, *H. pylori* eradication is mandatory to prevent recurrence [13]. Tenth, multidisciplinary coordination between gastroenterology, interventional radiology, and surgery remains critical in refractory cases [29]. Cost-effectiveness analyses demonstrate that preventing rebleeding offsets procedural expense [30].

6. Conclusion

Forrest Ib duodenal ulcer bleeding requires urgent definitive endoscopic therapy. Combination therapy with epinephrine injection followed by mechanical clipping provides superior outcomes and reduces rebleeding risk compared to monotherapy [16] [20] [31]. Recent studies have demonstrated that early endoscopy within 24 hours improves outcomes in patients with upper gastrointestinal bleeding [32]. Early intravenous followed by oral proton pump inhibitor therapy represents an optimal approach for high-risk bleeding peptic ulcers [33]. The decision to continue or discontinue low-dose aspirin in patients with ulcer bleeding requires careful risk-benefit assessment [34]. This case demonstrates successful guideline-concordant management in a young adult, emphasizing the importance of comprehensive pre-bleed history, risk stratification, proper endoscopic technique, and systematic post-procedural care including *H. pylori* testing and eradication.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

References

[1] Lanas, A., Dumonceau, J.M., Hunt, R.H., Fujishiro, M., Scheiman, J.M., Gralnek, I.M., *et al.* (2018) Non-Variceal Upper Gastrointestinal Bleeding. *Nature Reviews Disease Primers*, **4**, Article No. 18020. https://doi.org/10.1038/nrdp.2018.20

[2] Sung, J.J.Y., Kuipers, E.J. and El-Serag, H.B. (2009) Systematic Review of NVUGIB Epidemiology. *Gastroenterology*, **136**, 1603-1613.

[3] Lanas, A., Carrera-Lasfuentes, P., Arguedas, Y., *et al.* (2015) Risk of Upper and Lower GI Bleeding Associated with NSAIDs and Antiplatelets. *Gastroenterology*, **149**, 927-939.

[4] Sung, J.J.Y., Ching, J.Y.L., Wu, J.C., *et al.* (2009) Early Endoscopy Reduces Mortality in UGIB. *Gastroenterology*, **137**, 117-124.

[5] Forrest, J.H., Finlayson, N.D.C. and Shearman, D.J.C. (1974) Endoscopy in Gastro-

intestinal Bleeding. *The Lancet*, **304**, 394-397.
https://doi.org/10.1016/s0140-6736(74)91770-x

[6] Laine, L. and Jensen, D.M. (2012) Management of Patients with Ulcer Bleeding. *American Journal of Gastroenterology*, **107**, 345-360.
https://doi.org/10.1038/ajg.2011.480

[7] Villanueva, C., Colomo, A., Bosch, A., *et al.* (2013) Restrictive vs Liberal Transfusion in UGIB. *The New England Journal of Medicine*, **368**, 11-21.

[8] Blatchford, O., Murray, W.R. and Blatchford, M. (2000) A Risk Score to Predict Need for Treatment for Upper-Gastrointestinal Haemorrhage. *The Lancet*, **356**, 1318-1321.
https://doi.org/10.1016/s0140-6736(00)02816-6

[9] Rockall, T.A., Logan, R.F., Devlin, H.B. and Northfield, T.C. (1996) Risk Assessment after Acute Upper Gastrointestinal Haemorrhage. *Gut*, **38**, 316-321.
https://doi.org/10.1136/gut.38.3.316

[10] Stanley, A.J., Laine, L., Dalton, H.R., Ngu, J.H., Schultz, M., Abazi, R., *et al.* (2017) Comparison of Risk Scoring Systems for Patients Presenting with Upper Gastrointestinal Bleeding: International Multicentre Prospective Study. *British Medical Journal*, **356**, i6432. https://doi.org/10.1136/bmj.i6432

[11] Laursen, S.B., Leontiadis, G.I., Stanley, A.J., *et al.* (2017) Relationship between Timing of Endoscopy and Outcomes in UGIB. *Gastroenterology*, **152**, 528-536.

[12] Moore, K.L., Dalley, A.F. and Agur, A.M. (2013) Clinically Oriented Anatomy. 7th Edition, Lippincott Williams & Wilkins.

[13] Gisbert, J.P., Khorrami, S., Carballo, F., *et al.* (2004) *H. pylori* Eradication in Bleeding Ulcers. *Alimentary Pharmacology & Therapeutics*, **19**, 617-629.

[14] Laine, L., Barkun, A.N., Saltzman, J.R., *et al.* (2021) ACG Clinical Guideline: Upper Gastrointestinal and Ulcer Bleeding. *The American Journal of Gastroenterology*, **116**, 899-917.

[15] Gralnek, I.M., Stanley, A.J., Morris, A.J., *et al.* (2021) ESGE Guideline: Nonvariceal Upper GI Hemorrhage. *Endoscopy*, **53**, 300-332.

[16] Vergara, M., Calvet, X. and Gisbert, J.P. (2014) Epinephrine Injection vs Combination Therapy. *Cochrane Database of Systematic Reviews*, **10**, CD005584.

[17] Sung, J.J.Y., Tsoi, K.K.F., Lai, L.H., *et al.* (2007) Endoscopic Clipping vs Injection and Thermal Therapy. *Gastrointestinal Endoscopy*, **66**, 904-911.

[18] Lau, J.Y., Sung, J.J., Lee, K.K., *et al.* (2000) IV Omeprazole Reduces Recurrent Bleeding. *The New England Journal of Medicine*, **343**, 310-316.

[19] Chan, F.K. and Graham, D.Y. (2004) Review: Prevention of NSAID-Related Ulcer Complications. *Gastroenterology*, **127**, 947-957.

[20] Marmo, R., Rotondano, G., Piscopo, R., *et al.* (2007) Dual Therapy vs Monotherapy in High-Risk Bleeding Ulcers: Meta-Analysis. *Gastrointestinal Endoscopy*, **66**, 920-927.

[21] Cook, D.J., Guyatt, G.H., Salena, B.J. and Laine, L.A. (1992) Endoscopic Therapy for Acute NVUGIB: Meta-Analysis. *Gastroenterology*, **102**, 139-148.
https://doi.org/10.1016/0016-5085(92)91793-4

[22] Schmidt, A., Gölder, S., Goetz, M., Meining, A., Lau, J., von Delius, S., *et al.* (2018) OTSC Superior in Recurrent Ulcer Bleeding. *Gastroenterology*, **155**, 674-686.
https://doi.org/10.1053/j.gastro.2018.05.037

[23] Barkun, A.N., Almadi, M., Kuipers, E.J., Laine, L., Sung, J., Tse, F., *et al.* (2019) Management of Nonvariceal Upper Gastrointestinal Bleeding: Guideline Recommenda-

tions from the International Consensus Group. *Annals of Internal Medicine*, **171**, 805-822. https://doi.org/10.7326/m19-1795

[24] Sung, J.J.Y., Chiu, P.W.Y., Chan, F.K.L., *et al.* (2018) Asia-Pacific Consensus on NVUGIB. *Gut*, **67**, 1757-1768.

[25] Barkun, A.N., Bardou, M., Kuipers, E.J., Sung, J., Hunt, R.H., Martel, M., *et al.* (2010) International Consensus Recommendations on NVUGIB. *Annals of Internal Medicine*, **152**, 101-113. https://doi.org/10.7326/0003-4819-152-2-201001190-00009

[26] Holster, I.L. and Kuipers, E.J. (2012) Management of Acute NVUGIB: Current Policies. *Gut*, **61**, 821-831.

[27] Jensen, D.M., Kovacs, T.O.G. and Jutabha, R. (2006) Randomized Trial of Endoscopic Hemostatic Techniques. *Gastrointestinal Endoscopy*, **63**, 211-218.

[28] Tsoi, K.K. and Sung, J.J. (2019) Endoscopic Management Strategies in Peptic Ulcer Bleeding. *Best Practice & Research*: *Clinical Gastroenterology*, **42**, Article 101609.

[29] Lau, J.Y., Barkun, A., Fan, D., Kuipers, E.J., Yang, Y. and Chan, F.K. (2013) Challenges in the Management of Acute Peptic Ulcer Bleeding. *The Lancet*, **381**, 2033-2043. https://doi.org/10.1016/s0140-6736(13)60596-6

[30] Spiegel, B.M., Ofman, J.J., Woods, K. and Vakil, N.B. (2001) Cost-Effectiveness of Endoscopic Therapy. *Gastroenterology*, **120**, 1276-1287.

[31] Calvet, X., Vergara, M., Brullet, E., Gisbert, J.P. and Campo, R. (2004) Addition of a Second Endoscopic Treatment Improves Outcome. *Gastroenterology*, **126**, 441-450. https://doi.org/10.1053/j.gastro.2003.11.006

[32] Bai, Y., Chen, D., Wang, R., *et al.* (2022) Early or Delayed Endoscopy for Patients with Upper Gastrointestinal Bleeding. *The New England Journal of Medicine*, **386**, 1297-1308.

[33] Lau, J.Y., Yu, Y., Tang, R.S., *et al.* (2023) Early Intravenous Then Oral Proton Pump Inhibitors versus Standard Treatment in Patients with High-Risk Bleeding Peptic Ulcers. *Gut*, **72**, 457-464.

[34] Sung, J.J.Y., Lau, J.Y.W., Ching, J.Y.L., Wu, J.C.Y., Lee, Y.T., Chiu, P.W.Y., *et al.* (2010) Continuation of Low-Dose Aspirin in Ulcer Bleeding. *Annals of Internal Medicine*, **152**, 1-9. https://doi.org/10.7326/0003-4819-152-1-201001050-00179