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THE FEATURES OF ENDOVASCULAR SURGERY FOR DUODENAL ULCER BLEEDING

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ABSTRACT

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KEYWORDS

Endovascular Surgery, Duodenal Ulcer, Gastrointestinal Bleeding, Embolization. Aim of the research was to study improvement of the results of surgical treatment of duodenal ulcer bleeding based on the use of endovascular embolization. Gastrointestinal bleeding is a relatively common condition with a wide range of underlying causes. In most cases, this acute bleeding is effectively managed by conservative, medical or endoscopic procedures. However, the proportion of endoscopically unrecognized or controlled non-variceal gastrointestinal bleeding still requires alternative, sometimes surgical, treatment. The current S2k guideline Gastrointestinal Bleeding gives importance to interventional radiology in considering its minimally invasive endovascular interdisciplinary therapy options, guideline-oriented endovascular treatment of Gastrointestinal bleeding by embolization and implantation of covered stents is a treatment approach with good technical and clinical success rates and low complication rates. Gastrointestinal bleedings (GIBs) are pathological conditions associated with significant morbidity and mortality. Embolization without angiographic evidence of contrast media extravasation is proposed as an effective procedure in patients with clinical and/or laboratory signs of bleeding. The purpose of this systematic review is to define common clinical practice and clinical and technical outcomes of blind and preventive embolization for upper and lower gastrointestinal bleeding. Knowledge of vascular anatomy is essential to achieve adequate hemostasis. Endovascular embolization dramatically reduces the mortality rate in high-risk patients who require open surgery after failed endoscopy, further studies are needed to fully address these objectives.

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Introduction.

Treatment of peptic ulcers varies depending on the etiology and clinical presentation (see Guidelines). The initial management of a stable patient with dyspepsia differs from the management of an unstable patient with upper gastrointestinal (GI) hemorrhage. In the latter scenario, failure of medical management not uncommonly leads to surgical intervention.

Treatment options include empiric antisecretory therapy, empiric triple therapy for H pylori infection, endoscopy followed by appropriate therapy based on findings, and H pylori serology followed by triple therapy for patients who are infected. Breath testing for active H pylori infection may be used [1-2].

Endoscopy is required to document healing of gastric ulcers and to rule out gastric cancer. This usually is performed 6-8 weeks after the initial diagnosis of peptic ulcer disease. Documentation of H pylori cure with a noninvasive test, such as the urea breath test or fecal antigen test, is appropriate in patients with complicated ulcers [3-4].

Given the current understanding of the pathogenesis of peptic ulcer disease, most patients with peptic ulcer disease are treated successfully with cure of H pylori infection and/or avoidance of nonsteroidal anti-inflammatory agents (NSAIDs), along with the appropriate use of antisecretory therapy. Computer models have suggested that obtaining H pylori serology followed by triple therapy for patients who are infected is the most cost-effective approach; however, no direct evidence from clinical trials provides confirmation [5-6].

Endoscopy should be performed early in patients older than 45-50 years and in patients with associated so-called alarm symptoms, such as dysphagia, recurrent vomiting, weight loss, or bleeding. Age is an independent risk factor for the incidence and mortality from bleeding peptic ulcer, with the risk increasing in persons older than 65 years and increasing further in those older than age 75 years. In one study, at least two risk factors (previous duodenal ulcer, H pylori infection, use of acetylsalicylic acid (ASA)/NSAID, and smoking) were present in two thirds of persons with acute gastroduodenal bleeding [7-8].

The indications for urgent surgery include failure to achieve hemostasis endoscopically, recurrent bleeding despite endoscopic attempts at achieving hemostasis (many advocates surgery after two failed endoscopic attempts), and perforation. Many authorities recommend simple oversewing of the ulcer with treatment of the underlying H pylori infection or cessation of NSAIDs for bleeding peptic ulcer disease. Additional surgical options for refractory or complicated peptic ulcer disease include vagotomy and pyloroplasty, vagotomy and antrectomy with gastroduodenal reconstruction (Billroth I) or gastrojejunal reconstruction (Billroth II), or a highly selective vagotomy [9-12].

The principles of management of bleeding peptic ulcers outlined below are equally applicable to both gastric and duodenal ulcers.

Gastrointestinal bleedings (GIBs) are pathological conditions associated with significant morbidity and mortality. Embolization without angiographic evidence of contrast media extravasation is proposed as an effective procedure in patients with clinical and/or laboratory signs of bleeding. The purpose of this systematic review is to define common clinical practice and clinical and technical outcomes of blind and preventive embolization for upper and lower gastrointestinal bleeding [13-15].

Objectives.

Aim of the research was to study improvement of the results of surgical treatment of duodenal ulcer bleeding based on the use of endovascular embolization.

Materials and Methods.

The material of the article was the data from scientific publications, which were processed, analyzed, overviewed and reviewed by generalization and systematization. research studies are based on a review/overview assessment of the development of critical visibility and overlook of the modern scientific literature. use the following databases: (for extensive literature searches to identify the features of endovascular surgery for duodenal ulcer bleeding). PubMed, Web of Science, Clinical Key, Tomson Reuters, Google Scholar, Cochrane library, and Elsevier foundations. national and international policies and guidelines were also reviewed and as well as grey literature.

From March 2019 to December 2022, 40 patients were embolized during duodenal bleeding.

These patients were divided into the following groups:

- Massive, active bleeding. \triangleright
- Recurrence of bleeding in clinic and endoscopy was unsuccessful.
- AAAA Unstable hemodynamics and solid hemostasis could not be achieved during endoscopy.
- Failed to evacuate stomach contents and failed to see a bleeding ulcer.
- High risk of bleeding recurrence (Forest classification).
- Elderly and patients burdened with co-morbidities, with whom operative.

Results and Discussion.

Gastrointestinal bleeding is classified according to the anatomical location of the source. It is classified as upper gastrointestinal (GI) if the location is greater than the ligament of Treitz. Upper gastrointestinal bleeding is more common than lower gastrointestinal bleeding. Lower gastrointestinal bleeding is called if the source of bleeding is below the Treitz ligament.

The frequency of gastrointestinal bleeding is 100-125 patients per million people per year. Bleeding from the upper gastrointestinal tract is 4 times more frequent than the lower.

Gastrointestinal tract (20-25 patients per 100,000 people per year for lower gastrointestinal bleeding. Compared to lower gastrointestinal bleeding, upper gastrointestinal bleeding is more common in young adults. Different etiologies of gastrointestinal bleeding are trauma, infection (H. Pylori, Pseudomonas), Bleeding from the gastrointestinal tract depends on its location, severity and caused diseases, tumors and vascular anomalies [16-18].

Gastrointestinal (GI) bleeding is a common condition with many possible causes. The most severe bleeding responds well to conservative, medical and endoscopic therapy. Nevertheless, a certain number of endoscopically undetectable or controlled non-varicose gastrointestinal bleeding requires alternative, sometimes surgical, therapy. The updated S2k guideline "Gastrointestinal bleeding" allows interventional radiology with its minimally invasive endovascular technique.

Technical success in 39 patients (97.5%); Technical failure in 1 patient (2.5%). The following criteria are used to evaluate the effectiveness of endovascular hemostasis: Technical success interruption of blood circulation in the area of embolization. Clinical success - Bleeding relapse correction and hemodynamic stabilization. Unsuccessful embolization- The risk of rebleeding after successful embolization is three times higher in patients with coagulation problems, and for the same reason there is a 10-fold increased risk of death compared to patients with normal coagulation.

Non-variceal gastrointestinal bleeding is often an urgent, potentially life-threatening emergency. In 75-85% of cases, they are located in the upper gastrointestinal tract [2-3]. Treatment generally requires a structured interdisciplinary approach with therapeutic options including conservative, surgical, endoscopic, and endovascular procedures. The indication for endovascular therapy is closely based on the recently published S2k guidelines of the German Society for Gastroenterology, Digestive and Metabolic Diseases (DGVS) [19-20].

Upper gastrointestinal bleeding above the ligament of Treitz. With an incidence of 50 to 100 per 100,000 population, it is a common pathology with an average age of onset of 60-70 years [5]. In 70-75% of cases, upper gastrointestinal bleeding stops spontaneously. Mortality rates range from 3 to 14%, for intensive care patients from 42 to 64%. In about 50% of cases, bleeding from the small intestine is caused by an ulcer disease, such as a stomach ulcer or duodenal ulcer. Other causes include esophageal or gastric tumor bleeding, Mallory-White syndrome, erosive gastritis or duodenal ulcer, reflux esophagitis, angiodysplasia, and iatrogenic or posttraumatic changes [21-22].

Gastrointestinal (GI) bleeding is a relatively common condition with a wide range of underlying causes. In most cases, this acute bleeding is effectively managed by conservative, medical or endoscopic procedures. However, the proportion of endoscopically unrecognized or controlled nonvariceal gastrointestinal bleeding still requires alternative, sometimes surgical, treatment. The current S2k guideline "Gastrointestinal Bleeding" gives importance to interventional radiology in considering its minimally invasive endovascular interdisciplinary therapy options, guideline-oriented endovascular treatment of GI bleeding by embolization and implantation of covered stents is a treatment approach with good technical and clinical success rates and low complication rates. Knowledge of vascular anatomy is essential to achieve adequate hemostasis [23-24].

According to the guideline, hemodynamically unstable patients with non-variceal gastrointestinal bleeding should receive intensive medical care, and should be evaluated immediately (< 12 hours) after stabilization using an esophagoduodenoscope (strong recommendation, strong consensus). In hemodynamically stable patients, endoscopy should be performed within the first 72 hours of continuous vital signs monitoring (open recommendation, strong consensus). As severe sequelae of LGIB are less common and mortality and bleeding-related mortality are low, outpatient treatment is often possible [14]. Severe lower GI bleeding with hemodynamic compromise should prompt colonoscopy after drug stabilization (recommendation, strong consensus). In these situations, the source of bleeding can only be identified in approximately 42% of cases due to inadequate bowel preparation in emergency management and limited visibility of the colon and proximal bowel [25-26].

Depending on the type and origin of bleeding, endoscopic hemostasis can be achieved by various mechanical methods: Rubber ligation, hemoclips and thermal procedures (electrocoagulation), injection therapy (eg with adrenaline) and the use of hemospray (open recommendation, strong consensus). If attempts at endoscopic hemostasis remain unsuccessful, endoscopy can still facilitate accurate planning of endovascular intervention and facilitate superselective catheterization of the appropriate bleeding vessel through clip marking of the detected bleeding source, especially if active contrast extravasation is not detected angiographically [27-28].

In case of disappointing identification of the source of bleeding during endoscopy, further diagnostic procedures are possible, depending on the dynamics of the bleeding and the availability of other diagnostic procedures, provided that the affected patients are hemodynamically stable or stabilized. Detection using contrast-enhanced multiline computed tomography can be performed at bleeding rates of 0.5 ml/min and above. In multiphase technology, CT as a non-invasive image provides rapid diagnosis with good sensitivity and specificity and can facilitate the planning of further therapy regimens in addition to the localization of bleeding. According to current data, due to the high spatial resolution, digital subtraction angiography (DSA) allows the identification of the source of bleeding with a bleeding frequency between 0.5 and 1 ml/min [21]. There is great variability in the literature regarding the sensitivity and specificity of catheter angiography [29-30]. The invasive nature of DSA, which can be considered a disadvantage, is opposed by the possibility of simultaneous treatment of gastrointestinal bleeding. In direct comparison with CTA, several studies have shown its superiority with respect to the source of bleeding and the sensitivity of detecting the cause of bleeding [22, 23]. In summary, it can be recommended that patients, after a frustrating endoscopic search for the source of bleeding, should first be referred for multiphase CT diagnosis in case of hemodynamic stability, since important information can be obtained regarding the cause. Bleeding, possible vascular anomalies and variants. This may contribute to the accurate planning of subsequent (endovascular) therapy and the consequent reduction of intervention time [31-32]. Furthermore, this approach is consistent with the current S2k guideline recommendations. A blood cell scintiscan using 99 mTclabeled red blood cells with a sensitivity of 93% and a specificity of 95% allows localization of intermittent GI bleeding with a bleeding rate as low as 0.2 ml/min [20].

In cases of hemodynamic instability, hypovolemia should be balanced with erythrocyte concentrates, crystalloids or colloids, and catecholamine or vasoconstrictor therapy before intervention. In addition, appropriate coagulation parameters (INR, PTT) should be determined and, if necessary, optimized, as mechanical embolic agents often cause sufficient vessel occlusion only if the coagulation cascade is intact. The use of glucagon or buscopan may be considered to reduce intestinal peristalsis [33-34].

Peri-interventional observation should be carried out with continuous monitoring of blood pressure, electrocardiographic parameters, arterial oxygen saturation and, if necessary, respiratory rate. Depending on the patient's hemodynamic status, the presence of an anesthesia team may be necessary

for monitoring. The condition of the patient ultimately also determines whether the intervention is performed under local anesthesia and analgesia or under general anesthesia [35-36].

Based on the available clinical data, there is a strong consensus and an open recommendation for the treatment of gastrointestinal bleeding. This means that open surgical or X-ray endovascular intervention can be performed a) after technical failure of endoscopic hemostasis, including reserve procedures; b) repeated bleeding after the second endoscopic intervention; and c) in case of endoscopically non-localized source of bleeding. This also includes bleeding that is not endoscopically accessible due to special circumstances (eg, after Billroth II or Whipple surgery). Peripancreatic hemorrhage resulting from acute and chronic pancreatitis or after pancreatic surgery is a critical scenario with a high mortality rate. The release of pancreatic proteolytic enzymes causes vascular damage with subsequent pseudoaneurysm formation and vascular ruptures [26, 27]. Surgical treatment in this context is difficult because of the frequent retropancreatic localization of bleeding and the inflammatory reaction of the environment. High mortality and often the necessity of a radical surgical procedure, e. c. (Hemi) pancreatectomy and splenectomy, is the result. In such situations, endovascular therapy is an effective alternative with a good success rate, whose primary use should be considered in appropriate centers [37-38].

Obscure bleeding includes gastrointestinal bleeding that occasionally causes symptoms perceptible to the patient, such as hematemesis, hematochezia, or melena, but is not detected by endoscopic diagnosis. Occult bleeding is noticeable only due to the presence of iron deficiency anemia or a positive result of the blood culture test. Obscure and occult bleeding can occur in any part of the gastrointestinal tract and is a diagnostic and therapeutic challenge. Further diagnostic procedure depends largely on clinical symptoms, which in turn may lead to repeated endoscopic evaluation including special techniques such as deep enteroscopy and capsule endoscopy. In the case of an unknown source of bleeding and persistent symptoms, angiography may be necessary because there is a minimum probability of identifying it compared to occult hemorrhages. Since angiographic imaging of bleeding requires a bleeding intensity of at least 0.5 ml/min, angiography plays a subordinate role in the diagnosis and treatment of occult gastrointestinal bleeding [39-41].

Contraindications to endovascular therapy – such as contrast agent allergy, hyperthyroidism, pregnancy, sepsis, acute renal failure, and consumptive coagulopathy – must be considered relative, especially in acutely dangerous situations. Depending on the intensity of bleeding, the possible advantage of surgical therapy should be considered.

Typically, endovascular treatment of upper gastrointestinal or lower gastrointestinal bleeding is performed under local anesthesia via a transfemoral access route, although a trans brachial approach may also be considered, especially in the case of an unfavorable angle of descent of the visceral vessels. Depending on the vascular anatomy, it may be helpful to insert a long-reinforced guide sheath to stabilize the catheter. Especially in the absence of pre-interventional imaging, mechanical preparation of the study angiography with a multi-hole pigtail catheter allows assessment of the vascular anatomy for further targeted vessel exploration. The most suspected or identified visceral vessels are explored using the preferred selective catheter and selective angiography is performed. For UGIB, the celiac trunk and then the superior mesenteric artery are examined; In cases of LGIB, the superior and inferior mesenteric arteries were studied. For proper aortography, it is recommended to use approximately 50 ml of 1:1 diluted contrast medium at a rate of 15 ml/s; For selective angiography of the celiac, superior and inferior mesenteric arteries, 30-50 mL of 1:1 diluted contrast medium at a flow rate of approximately 6-7 mL/s is recommended [32]. In cases of rectal bleeding and inaccurate presentation of the inferior mesenteric artery, angiography of the internal iliac artery including the middle and inferior rectal arteries should be performed [33]. In this case, care must be taken to ensure sufficient exposure time to distinguish contrast agent extravasation and venous washout. Angiographic evidence of GI bleeding during the active bleeding interval is presented as contrast extravasation in the arterial phase with pooling in the venous phase. However, indirect signs such as pseudoaneurysms, vascular spasms or - in the case of inflammatory changes - redness and focal hyperemia can also be interpreted as angiographic evidence of (intermittent) gastrointestinal bleeding. Early venous discharge may indicate angiodysplasia [42-44].

Super selective vascular examination and therefore the use of coaxial or triaxial microcatheter techniques are often necessary to detect upper or lower gastrointestinal bleeding. The coaxial

technique involves the use of a microcatheter in a selective catheter; The triaxial technique involves the use of a microcatheter and a selective catheter in a reinforced guide sheath or guide catheter.

The often-intermittent nature of gastrointestinal bleeding can lead to a negative angiographic result, despite recent relevant bleeding. In these cases, repeat angiography at a later stage or provocation of bleeding with selective intra-arterial administration of nitroglycerin, heparin or tPA may be considered [45-46].

In the absence of evidence of active contrast extravasation, blind or empiric embolization can be performed based on endoscopic findings, although prior endoscopic clip labeling may be helpful [16, 36].

The patient should be operated urgently in case of technically frustrating intervention, because of feeding vessels that cannot be explored, or even in case of diffuse bleeding. Until the patient is transported to the OR, balloon occlusion can be used to achieve preoperative hemodynamic stabilization. Additional options to promote, identify, and expedite surgical therapy include catheter-assisted methylene blue staining of the bleeding bowel segment [47-48].

Once identified angiographically, the location (distal vs. proximal) of upper or lower gastrointestinal bleeding determines the endovascular procedure to be used.

A special case of UGIB is acute hemorrhage of the branches or collaterals of the peripancreatic blood vessel, the etiologies of which are often caused by pancreatitis, tumors, and trauma. Bleeding from pancreaticostomy or biliodigestive anastomosis after pancreatic surgery, with a mortality rate of 11-38%, is among the most difficult and difficult to treat complications due to vascular edema. In many cases, such postoperative complications of endovascular therapy are readily available, which can significantly reduce morbidity and mortality [49-51].

Refractory, symptomatic peptic ulcers, though rare after eradication of H pylori infection and the appropriate use of antisecretory therapy, are a potential complication of peptic ulcer disease. Obstruction is particularly likely to complicate peptic ulcer disease in cases refractory to aggressive antisecretory therapy, H pylori eradication, or avoidance of NSAIDs. Obstruction may persist or recur despite endoscopic balloon dilation. Perforation is also a possibility. Penetration, particularly if not walled off or if a gastrocolic fistula develops, is a potential complication. In addition, ulcer bleeding, particularly in patients with a history of massive hemorrhage and hemodynamic instability, recurrent bleeding on medical therapy, and failure of therapeutic endoscopy to control bleeding is a serious complication [52-54].

Patients with gastric ulcers are also at risk of developing gastric malignancy. The risk is approximately 2% in the initial 3 years. One of the important risk factors is related to H pylori infection. H pylori is associated with atrophic gastritis, which, in turn, predisposes to gastric cancer. H pylori infection is associated with gastric lymphoma or mucosa-associated lymphoid tissue (MALT) lymphoma. Normal gastric mucosa is devoid of organized lymphoid tissue. H pylori infection promotes acquisition of lymphocytic infiltration and often the formation of lymphocytic aggregates and follicles from which MALT lymphoma develops. Eradication of H pylori is very important in this group of patients because eradication of H pylori has been shown to cause a remission of MALT lymphoma [55-56].

Malignancy should be strongly considered in the case of a persistent nonhealing gastric ulcer. Endoscopic ultrasound examination may be helpful for assessing mucosal invasion or detecting associated adenopathy in such patients. Surgical resection should be considered if evidence of cancerous transformation is present [57-58].

Acid suppression is the general pharmacologic principle of medical management of acute bleeding from a peptic ulcer. Reducing gastric acidity is believed to improve hemostasis primarily through the decreased activity of pepsin in the presence of a more alkaline environment. Pepsin is believed to antagonize the hemostatic process by degrading fibrin clots. By suppressing acid production and maintaining a pH above 6, pepsin becomes markedly less active. Concomitant H pylori infection in the setting of bleeding peptic ulcers should be eradicated, as this lowers the rate of rebleeding. [59-60].

Two classes of acid-suppressing medications currently were use histamine-2 receptor antagonists (H2RAs) and PPIs.[48] Both classes were available in intravenous and oral preparations. Examples of H2RAs include cimetidine, famotidine, and nizatidine. Examples of PPIs include omeprazole, pantoprazole, lansoprazole, and rabeprazole [61-62].

H2RAs are an older class of medications, and in the setting of an actively bleeding duodenal ulcer, their use has been largely superseded by the use of PPIs. Many gastroenterologists assert that intravenous PPI therapy maintains hemostasis more effectively than intravenous H2RA. Thus, intravenous H2RA no longer has a role in the management of bleeding peptic ulcers [63-64].

PPIs have a very good safety profile, although attention must continue to be focused on adverse effects, especially with longterm and/or high-dose therapy, such as Clostridium difficile infection, community-acquired pneumonia, hip fracture, and vitamin B12 deficiency. Long-term use of PPIs is also associated with decreased absorption of some medications. PPIs impair gastric secretion of acid; thus, absorption of any medication that depends on gastric acidity, such as ketoconazole and iron salt, is impaired with long-term PPI therapy. In addition, achlorhydria (absence of intragastric acidity) may be associated with iron deficiency anemia, because the ferric form of iron must be converted to the ferrous form by gastric acid. Most iron absorbed is in the ferrous form [65-67].

Parenteral PPI administration is used after successful endoscopic therapy for ulcers with highrisk signs, such as active bleeding, visible vessels, and adherent clots. Parenteral PPI use before endoscopy is a common practice. Based on intragastric pH data, nonvomiting patients with bleeding ulcers may be treated with oral lansoprazole (120-mg bolus, followed by 30 mg every 3 h). When indicated, intravenous pantoprazole or omeprazole is administered as an 80-mg bolus followed by a continuous 8-mg/h infusion for 72 hours. A study by Chan et al determined that intravenous, standarddose omeprazole was inferior to high-dose omeprazole in preventing rebleeding after endoscopic therapy for peptic ulcer bleeding. This treatment is changed to oral PPI therapy after 72 hours if no rebleeding occurs [68-70].

The standard-dose PPI infusion was found to be as effective as a high-dose regimen in reducing the risk of recurrent bleeding following endoscopic hemostasis of bleeding ulcers. The primary end point was the in-hospital rebleeding rate (determined on repeat endoscopy). Patients with actively bleeding ulcers and those with a nonbleeding visible vessel or an adherent clot were treated with (1) epinephrine injection and/or thermal coagulation, then randomized to receive an intensive regimen of 80-mg PPI bolus, followed by 8 mg/h as continuous infusion for 72 hours, or (2) a standard regimen of a 40-mg PPI bolus daily, followed by saline infusion for 72 hours. After the infusion, all patients were given 20 mg PPI twice daily orally [71-72].

In the intensive PPI regimen group, rebleeding recurred in 11.8%, whereas in the standard regimen group, rebleeding recurred in 8.1%. Most of the rebleeding episodes occurred during the initial 72-hour infusion. The duration of hospital stay was less than 5 days for 37.0% in the intensive regimen group and 47.0% in the standard group. There were fewer surgical interventions in the standard group. Five patients in each treatment group died [73-74].

Research shows some benefit from parenteral PPI in decreasing rebleed rates. No randomized, controlled trial has provided evidence to support the use of parenteral PPI in this setting, but giving oral PPI both before and after EHT for persons with peptic ulcers with signs of recent hemorrhage can be justified on the grounds of cost-effectiveness.

Whether acid suppression improves the therapeutic outcomes of peptic ulcers compared with placebo may be more important than the issues raised above. Many researchers have compared parenteral PPI therapy with placebo, and overall, the results have demonstrated a shorter period of bleeding and a decreased incidence of rebleeding with PPI therapy. Some studies have demonstrated a decreased need for emergency surgery and blood transfusion; however, evidence that parenteral PPI reduces mortality from ulcer bleeding is relatively recent [75-76].

The recommended biochemical and imaging investigations in the diagnosis of perforated peptic ulcer are as follows: Suspected gastroduodenal perforation; Routine laboratory studies and arterial blood gas analysis; Acute abdomen from suspected perforated peptic ulcer- CT scanning; Acute abdomen from suspected perforated peptic ulcer: Chest and abdominal radiography as initial diagnostic assessment in the event CT scanning is not immediately available; Acute abdomen from suspected perforated peptic ulcer if free air is not seen on imaging and perforation remains a concern: Imaging with water-soluble contrast (oral or via nasogastric tube) [77-78]

The recommended targets for resuscitation in unstable patients with a perforated peptic ulcer are as follows: Rapid resuscitation to reduce mortality; Restoration of physiological parameters with a mean arterial pressure 65 mm Hg or higher, urine output of 0.5 mL/kg/h or greater, and lactate normalization; Use of hemodynamic monitoring (invasive or noninvasive) to optimize fluid/vasopressor therapy, with an individualized resuscitation strategy.

Surgical indications and appropriate timing of surgery in patients with perforated peptic ulcer are as follows: In association with significant pneumoperitoneum, extraluminal contrast extravasation, or signs of peritonitis: Operative treatment strongly recommended. Performing surgery as soon as possible, particularly in patients with delayed presentation or those older than 70 years.

The recommended surgical approach (laparoscopic vs open) for perforated peptic ulcer is as follows: Stable patients: Laparoscopic approach, unless equipment and skilled personnel are not available, in which case an open approach is recommended unstable patients- Open surgery.

The recommended antimicrobial and antifungal therapy strategies in perforated peptic ulcer are as follows: Administration of broad-spectrum antibiotics; Microbiological sample collection for analysis for bacterial and fungal pathogens in all patients undergoing surgery, with post analysis antibiotic therapy adjustment as needed; Antifungal agents not suggested as standard empiric therapy; may be appropriate in high-risk patients, such as those who are immunocompromised, have comorbidities, or are of advanced age. The recommended antimicrobial regimen and duration of therapy in perforated peptic ulcer are as follows: Initiation of empiric broad-spectrum antibiotics as soon as possible, targeting gram-negative, gram-positive, and anaerobic bacteria. Short course of 3-5 days or until inflammatory markers normalize [79-80].

The goals of pharmacotherapy are to eradicate H pylori infection, to reduce morbidity, and to prevent complications in patients with peptic ulcers. Acid suppression is the general pharmacologic principle of medical management of acute bleeding from a peptic ulcer, using histamine-2 receptor antagonists (H2RAs) and proton pump inhibitors (PPIs). Both classes are available in intravenous or oral preparations. Discontinuation of NSAIDs is paramount, if it is clinically feasible. For patients who must continue with their NSAIDs, PPI maintenance is recommended to prevent recurrences even after eradication of H pylori.

The recommended primary therapy for H pylori infection is proton pump inhibitor (PPI)– based triple therapy. Antacids or a GI cocktail (typically an antacid with an anesthetic such as viscous lidocaine and/or an antispasmodic) may be used as symptomatic therapy in the ED. Maintenance treatment with antisecretory medications (eg, H2 blockers, PPIs) for 1 year is indicated in high-risk patients. High-risk patients include those with recurrent ulcers and those with complicated or giant ulcers. If H pylori eradication is not achieved despite repeat treatment, maintenance antisecretory therapy should be recommended. Patients with refractory ulcers may continue receiving once-daily PPI therapy indefinitely. In this setting, if H pylori is absent, consider a secondary cause of duodenal ulcer, such as Zollinger-Ellison syndrome.

Primary prevention of NSAID-induced ulcers includes avoiding unnecessary use of NSAIDs, using acetaminophen or nonacetylated salicylates when possible, and using the lowest effective dose of an NSAID and switching to less toxic NSAIDs.

Conclusions.

Gastrointestinal (GI) bleeding is a relatively common condition with a wide range of underlying causes. In most cases, this acute bleeding is effectively managed by conservative, medical or endoscopic procedures. However, the proportion of endoscopically unrecognized or controlled non-variceal gastrointestinal bleeding still requires alternative, sometimes surgical, treatment. The current S2k guideline "Gastrointestinal Bleeding" gives importance to interventional radiology in considering its minimally invasive endovascular interdisciplinary therapy options, guideline-oriented endovascular treatment of GI bleeding by embolization and implantation of covered stents is a treatment approach with good technical and clinical success rates and low complication rates. Knowledge of vascular anatomy is essential to achieve adequate hemostasis. Endovascular embolization dramatically reduces the mortality rate in high-risk patients who require open surgery after failed endoscopy, further studies are needed to fully address these objectives.

Acknowledgments.

Declaration of Interest Statement. We confirm that the manuscript has been read and approved by all named authors and that there are no other persons who satisfied the criteria for authorship but are not listed. We further confirm that the order of authors listed in the manuscript has been approved by all of us.

Conflict of interest: None.

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