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A STUDY ON DIEULAFOY'S LESION: PATHOGENESIS, DIAGNOSIS, TREATMENT

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ABSTRACT

Dieulafoy's lesions (DLs) are rare and cause gastrointestinal bleeding resulting from erosion of dilated submucosal vessels. The most common location for DL is the stomach, followed by duodenum. Challenges for diagnosis and treatment of Dieulafoy's lesions include the rare nature of the disease, asymptomatic patients, bleeding symptoms often requiring rapid diagnosis and treatment, variability in the diagnosis and treatment methods resulting from different lesion locations, and the risk of re-bleeding. For these reasons, there is no universal consensus about the diagnosis and treatment approach. Endoscopic therapy is usually successful in achieving primary hemostasis, with hemostasis success rates reaching 75% to 100%. Although various therapeutic endoscopic methods are used to control bleeding in Dieulafoy's lesions, the best method for endoscopic intervention is not clear. Combination endoscopic therapy is known to be superior to monotherapy because of a lower rate of recurrent bleeding. In addition, mechanical therapies including hemostatic clipping and endoscopic band ligation are more effective and successful in controlling bleeding than other endoscopic methods. Advances in endoscopic techniques have reduced mortality in patients with Dieulafoy's lesion from 80% to 8% and consequently, the need for surgical intervention has been reduced. Currently, surgical intervention is used for cases that fail therapeutic endoscopic or angiographic interventions.

KEYWORDS: Dieulafoy lesion, GI bleeding, Endoscopy, Angiography, Gastrointestinal tract.

INTRODUCTION

Dieulafoy's lesion (DL), also known as calibre persistent artery/ submucosal arterial malformation/ solitary exulceratio simplex.[1] This is a rare condition that results in gastrointestinal bleeding due to erosion of dilated submucosal vessels.[2] If left untreated, DL can be life-threatening.[3] A submucosal artery protruding from a small mucosal defect (less than 3 mm) with normal surrounding mucosa or active arterial bleeding without an ulcer base in the GI tract[1] (FIG.1). The effects of DL can range from anaemia to hypovolemic shock.[4] The most common site is the proximal stomach, with occurrences in the small intestine, duodenum, and especially the ileum being extremely uncommon.[1]

As a result, there is no global suggested as an effective way to diagnose and treat the condition. It is difficult to accurately determine the true incidence in the general population because they are silent until they present, and even then, diagnosis can be difficult. The lack of knowledge about the Dieulafoy's lesion contributes to its 'rareness,' increased morbidity, and previously reported mortality rates of up to 80%. Whether rare or not, their precarious nature necessitates their inclusion in the differential diagnosis of any acute GI haemorrhage.[5]

Although Gallard first described DL in 1884, it was quite precisely described 14 years later by French surgeon Georges Dieulafoy, who reported fatal GIH in three patients caused by large, actively haemorrhaging arteries in the stomach due to slight ulcers, which he mistook for small peptic ulcers and named "exulceratio simplex." Several cases of DL have been reported in the medical community since these initial reports. Prior to the invention of endoscopy, the death rate due to haemorrhage in DL was high, the only treatment options for DL bleeding were surgical ligation of the aberrant vessel or subtotal (or total) gastrectomy. As a result, the advancement of hemostatic endoscopic techniques provided a novel and effective therapeutic approach, with a mortality rate of around 10%.[6]

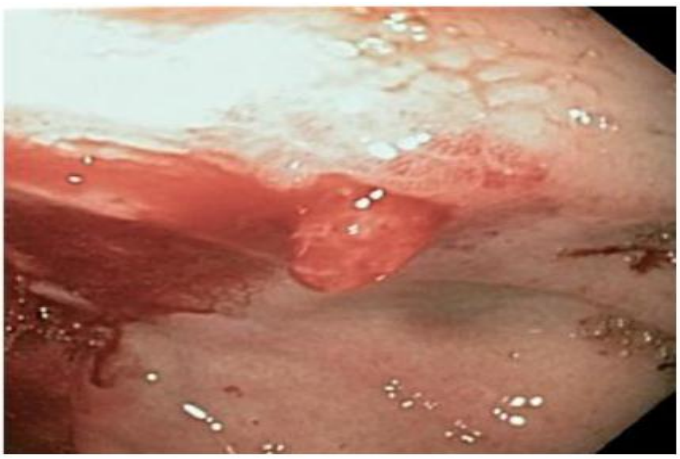

Fig. 1: Gastric Dieulafoy lesion at the anterior wall of the proximal corpus.[6]

Signs and Symptoms

The DL is usually asymptomatic, based on the site of affected and bleeding, Symptoms include blood in the vomit (hematemesis), sticky, dark-colored stools (melena), fresh blood in the stool (hematochezia), and coughing up blood (hemoptysis).[7] In approximately 28% of patients, hematemesis without melena occurs. Repetitive hematemesis with melena has been seen in around 51% of patients with Dieulafoy's lesion; melena without hematemesis was shown in about 18% of patients with Dieulafoy's lesion.[8] Upper abdominal pain, most commonly in the right upper quadrant or epigastric region, is one of the rare symptoms associated with Dieulafoy's lesions in the gallbladder. Dieulafoy's lesions in the gallbladder are frequently associated with anaemia, but they rarely cause excessive bleeding (hematemesis, hematochezia, melena, etc.[8]

Aetiology and Pathogenesis

The incidence and aetiology are still uncertain, They are twice as common in males than females and can affect any age group. However, they do present more commonly in the elderly population.[5] Studies on the history of comorbid diseases have described that most patients have a history of drug abuse, rheumatic fever, congestive heart failure, hypertension, cirrhosis, diabetes, permanent pacemaker, aortic stenosis, cerebral vascular accident, obstructive sleep apnea, chronic obstructive pulmonary disease, hyperlipidemia, coronary artery disease, atrial fibrillation, renal failure, and/or pulmonary hypertension[2], excessive use of alcohol have been described in almost 90% of the patients. These factors suggest that perhaps the final rupture of the vessel results from the compound effects of the vascular ectasia, mucosal atrophy, and possible ischemic injury related to aging and cardiovascular diseases, which weaken an inherently vulnerable point. Despite a lack of direct evidence that

the presentation of DL is caused by aspirin, coumadin, or nonsteroidal anti-inflammatory drugs, the use of these medications has been reported in more than 50% of patients. There is currently no evidence of any relationship between Helicobacter pylori infection and DL.[9] There are cases in the literature of newborns being affected, while rare, this could support the suggestion that these lesions are in fact congenital in nature.[5]

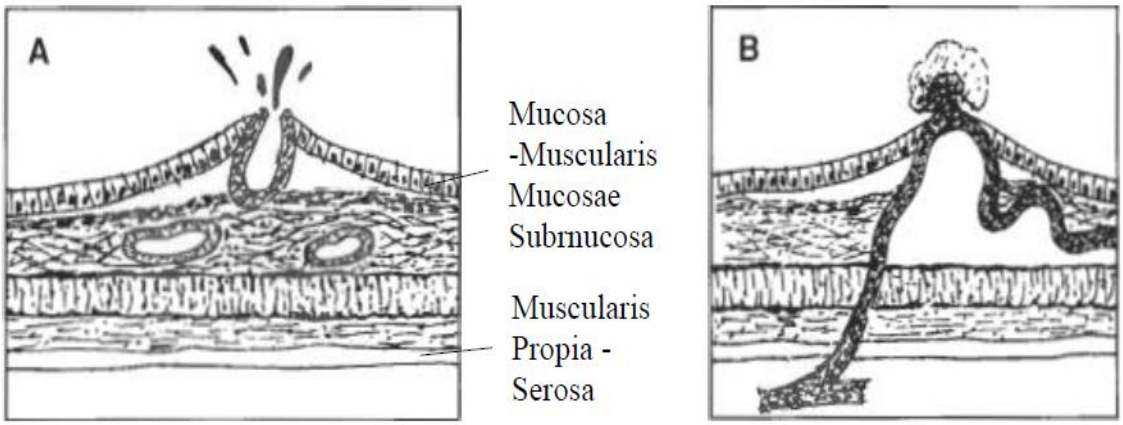

Fig. 2: Graphic illustration of pathologic artery in Dieulafoy's lesion. Bulging of lesion into the lumen of GI tract with arterial Spurting (A), and Sentinel clot or adherent clot (B).[3]

Solid bowel content can contribute to mucosal stercoral ulceration in the colon, but there is no specific pathogenesis for DL. Senile atrophy is another factor involved in DL rupture and hemorrhage.[2] The developement of mucosal defect and pathogenesis of bleeding is still unclear and may be multifactorial.[3] Dieulafoy' s lesion in any location has the same pathology. Characteristic lesion consists of two components. (1) A large tortuous subrnucosal artery usually 1-3 mm in diameter, and (2) Minute mucosal defect usually 2-.5 mm in diameter and solitary. When exposed artery ruptures acute GIB will suddenly occur.[3] DLs are different from typical peptic ulcers because the mucosal defect in DLs is not surrounded by inflammatory cell infiltration, and the exposed artery has a large diameter. The surrounding mucosa is normal. There are no aneurismal, arteriosclerotic, or vasculitis changes in these dilated, tortuous, submucosal arteries. A locally protruding blood vessel in the submucosal layer causes mucosal blood circulation disturbances, and the stress load (such as erosion or atrophy) causes a reduction in blood flow in the overlying mucosa.[2] (FIG.2). Pathologic arteries were significantly larger than normal arteries at level of' muscularis mucosae rather than submucosal level.[3] Pathological reports have failed to associate

Dieulafoy's lesion to aneurysms, arteriosclerosis, elastic tissue abnormalities, or signs of vasculitis.[10]

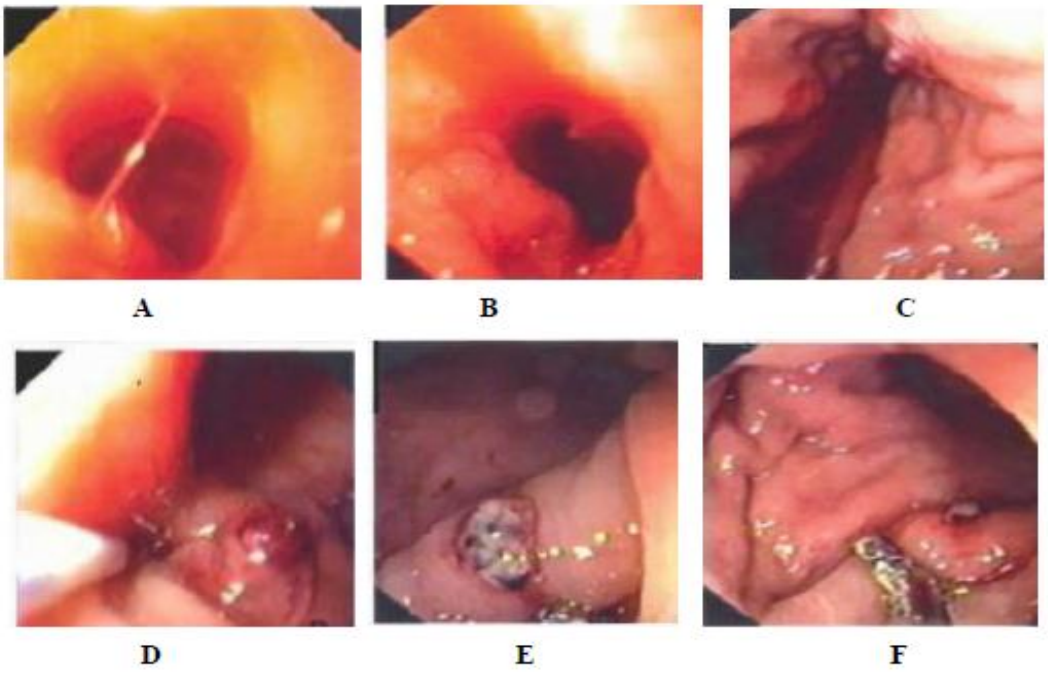

Fig. 3: Endoscopic visualization of stigmata of recent hemorrhage.[3]

Duodenal lesion: A = Active arterial spurting, B = Non-pigment protuberance of non-bleeding visible vessel (NBVV).

Gastric lesion: C = Pigmented protuberance of NBVV in tangential view, D = Pigmented protuberance of NBVV or sentinel clot in surface view seen by retroflexion of endoscope.

Rectal lesion: E = Whitish mounds on a large adherent clot indicative of the aged clot, F = Dark red adherent clot was seen after irrigation.[3]

Several mechanisms have been proposed to explain the rupture and subsequent massive hemorrhage. One mechanism focuses mainly on the combined effect of atrophy and ischemia of the mucosa. The pulsation in a thick, large submucosal artery applies pressure to the overlying epithelium and leads to a small erosion and rupture of the vessel towards the lumen. Clinically, Dieulafoy's lesion presents with intermittent and painless GI bleeding. Another theory suggests that thrombosis within the artery and subsequent necrosis of the arterial wall plays a primary role in the ultimate arterial rupture (FIG. 4). The absence of a mucosal inflammatory reaction at the site of the arterial rupture indicates that an acid peptic process is not involved. The stomach is the most common site for Dieulafoy's lesions. Of these lesions, 80% to 95% occur within 6 to 10 cm of the gastroesophageal junction, generally along the

lesser curvature of the stomach. Approximately one-third of lesions are extragastric, located most frequently in the duodenum followed by the colon. They have also been described in the esophagus, jejunum, ileum, rectum, and anal canal.[10] There is subintimal fibrosis of the artery, but there is no true structural aneurysmal change. Importantly, there is an absence of inflammation at the edge of the mucosal defect in contrast to that present in peptic ulcer disease. The tortuous arteries are accompanied by large veins, led to both arterial and venous rupture. Intimal thickening occurs in both veins and arteries with thrombosis formation, but the mechanism of final rupture of the vessel is uncertain.[9]

Bleeding occurs as a result of pressure erosion of the overlying epithelium by the ectatic vessel. These anomalous vessels are abnormally fixed to the muscularis mucosae, instead of being relatively mobile within the submucosal strata, thereby causing abnormal stresses as the tissues move during normal peristalsis. Dysplastic changes evidenced by subintimal fibrosis, loss of elastic fibers adjacent to the necrotic artery wall, and thinning or loss of circular fibers of the artery, which are seen at the point of rupture, support the theory that the final pathologic process is one of slow weakening of the vessel wall with perhaps eventual localized dilation.[9]

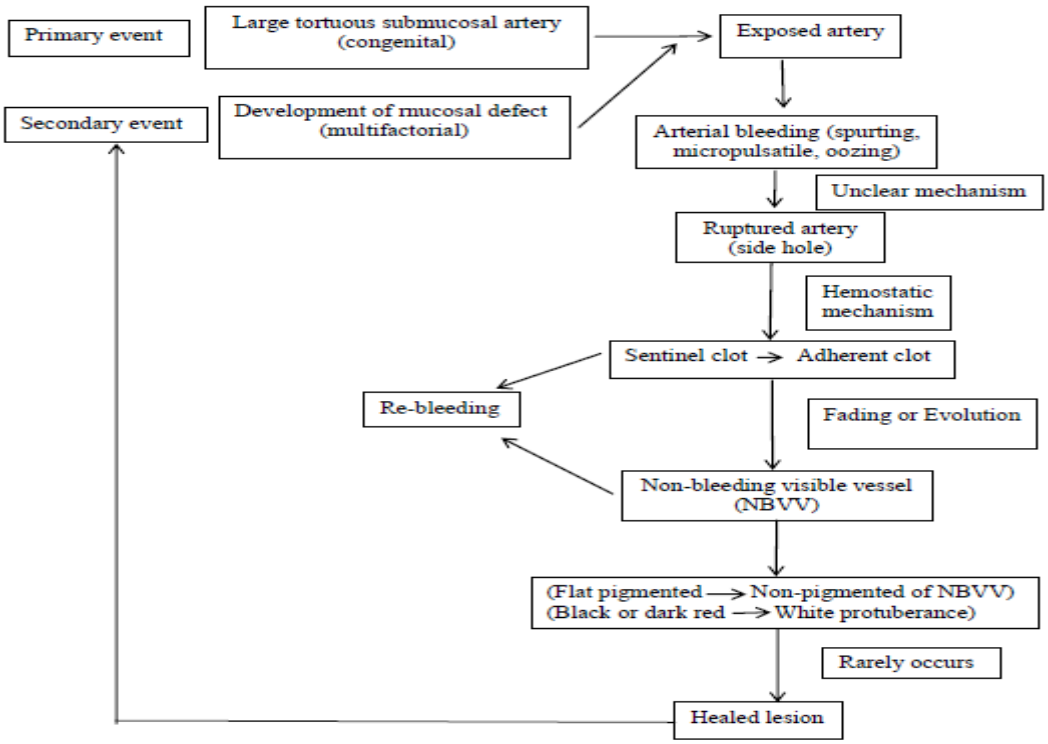

Fig. 4: Summary of the Pathogenesis and Clinical presentation of bleeding in Dieulafoy's lesion.[3]

Diagnosis

Endoscopy

Histologic examination provides definitive diagnosis of Dieulfoy's lesion. Before endoscopic era, diagnosis could be made at urgent laparotomy or at autopsy which had tissue for histologic conformation. Currently endoscopy is the best diagnostic procedure. Selective visceral angiography and scintigraphy are helpful for demonstration and localization if endoscopy is inaccessible or fail.[11,12] Initial endoscopy can identify bleeding lesion in 66-92 percent. Multiple endoscopies are often necessary. Some patients require four endoscopic attempts to delineate the bleeding lesion. In non-bleeding phase, this minute lesion can be easily overlooked. Insufflating air for full distention of stomach because the lesion may be hidden between two mucosal folds. In active bleeding phase all clots must be removed by large bore nasogastric tube irrigation prior to examination. If large blood clots obscure visualization, changing of patient's position can shift away of clots from inspected area or changing of endoscope to a new endoscope with 6 mm accessory channel for rapid removal of clots. Haematin and loose adherent clots must be irrigated with water or water pump for perfect visualization.[13] (FIG.5).

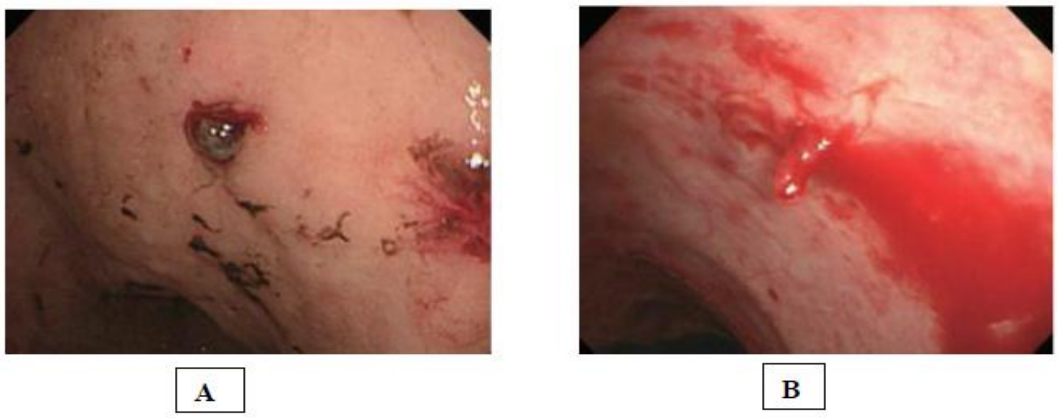

Fig. 5: Endoscopic view of DL in stomach (A) and colon(B).[10]

Endoscopic ultrasonography (EUS) has also been used to aid endoscopic diagnosis by revealing a tortuous submucosal vessel near the mucosal defect. Recently, the usefulness of **double balloon enteroscopy (DBE) or single balloon enteroscopy (SBE)** has been suggested for the detection of a Dieulafoys lesion in the small bowel. In patients who underwent DBE or SBE because of suspected mid-GI bleeding.[14] Emergency endoscopy within 2-4 hr after presentation or immediate after stabilizing hemodynamic status by basic resuscitation is the crucial point in visualization of stigmata of recent hernorrhage. In lower

GIB, prior upper endoscopy should be done to rule out the possibility of upper GI tract lesion then emergency colonoscopy performed by rapid infusion of 3-4 liters of polyethylene glycol electrolyte solution administered via nasogastic tube within a period of 2 hours.[15] The technique for better visualization by colonoscopy in flowing water for irrigation of active bleeding colonic lesion with continuous drainage through another overtube drain. Enteroscopy must be considered in case of blood seen in duodenum and terminal ileum in absence of lesion in stomach and colon. **Push enteroscopy** can visualize proximal jejunum within 40-60 cm from ligament of Treit. It can be done with specific type of push enteroscope or pediatric colonoscope or standard adult colonoscope. **Sonde enteroscopy** can visualize all part of small bowel but it is time consuming and arduous for both of patient and endoscopist and is not suitable for acute situation and therapeutic intervention. **Intraoperative enteroscopy** or **laparosocopic assisted panenteroscopy** is the alternative method for both diagnosis and treatment of bleeding lesion of small bowel. Patient who underwent gastrectomy and developed Dieulafoy's lesion in jejunal loop can be detected by standard upper endoscopy.[16] Currently diagnosis of Dieulafoy's lesion depends upon endoscopic visualization in most cases. Endoscopic criterior used to establish the diagnosis as the followings: (1) active arterial spurting or micropulsatile streaming from a minute mucosal defect or through normal surrounding mucosa; (2) visualization of a protruding vessel with or without active bleeding within a minute mucosal defect or through normal surrounding mucosa; (3) fresh densely adherent clot with a narrow point of attachment to a minute mucosal defect or to normal appearing mucosa. Endoscopic finding of stigmata of recent hemorrhage are arterial spurting or micropulsatile streaming or continuous oozing were seen 57-77 percent in reported of' emergency endoscopy. Protuberance vessel or non-bleeding visible vessel (NBVV) and adherent clots were found in the remaining case.[17] Coincidental endoscopic findings of comorbidity such as isolated varices, peptic ulcer, Mallory - Weiss tear, diverticular disease and tumor may be misintepreted as the cause of bleeding.

Angiography

Angiography is useful when endoscopic methods fail to localise the lesion. It is especially useful for lesions in the colon or rectum where the view could be obscured by active bleeding and poor bowel preparation.[18] There is no specific diagnostic criterion to diagnose a Dieulafoy’s lesion on angiography as typical features are variable, but the diagnosis is suggested on demonstration of a tortuous and ectactic artery. The findings include extravasion of contrast from what is seen as a normal looking blood vesse (FIG.6). For

gastric lesions, the demonstration of tortuous vessels in the territory of the left gastric artery lacking early venous return is suggestive. Lesions in the anorectal region can be missed if they are situated below the region supplied by the inferior mesenteric artery. Internal iliac artery visualisation might be necessary in these cases. The use of computed tomography (CT) angiography has also been described in the literature to locate the source of bleeding that cannot be diagnosed by endoscopic methods.[19] **Selective mesenteric angiography** is useful if multiple endoscopies fail to identify the lesion or in area which endoscopy is difficult to gain access as in post bulbar duodenal lesion, distal small bowel, ileum and right side colon. It demonstrates and locates the lesion only in bleeding phase. If no extravsation is seen in lumen of bowel, angiographic catheter should be temporarily left in situ, as to facilitate repeat examination to be performed immediately after sign and symptom of rebleeding are detected.[20]

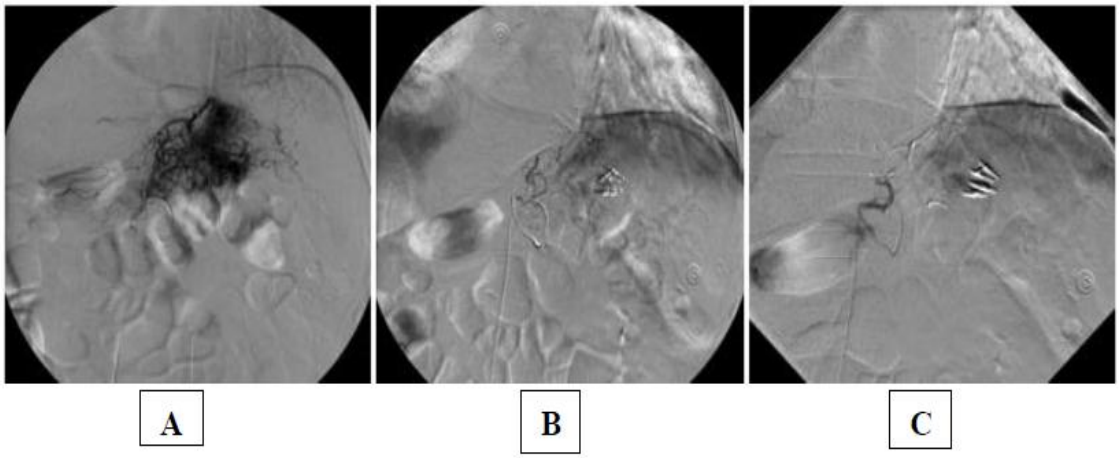

Fig. 6: Angiographic findings of Dieulafoy's lesions in the gastrointestinal tract. (A) There is an extravasation of contrast from a branch of the left gastric artery. (B) Gel-foam embolization was performed. (C) Extravasation of contrast was no longer seen on angiography.[10]

Red cell scanning

Technetium-99m labelled red blood cell scans have also been used to identify the location of bleeding Dieulafoy's lesion when endoscopy had failed. It has the advantage that the threshold for detecting extravasation into the gut is only 20% of that required by angiography.[21]

Differential diagnosis

Duodenal ulcer

Mucosal ulceration that can penetrate into submucosa and muscularis propria, or perforate onto the serosal surface; degree of surrounding fibrosis relative to chronicity of disease. Mucosal defect occurs about 2–4 cm. Bleeding vessel or adherent clot sometimes visible in ulcer base, 50% of UGIB(Upper Gastro Intestinal bleeding) occurs. Symptoms: epigastric abdominal pain, belching, anorexia, haematemesis Risk factors: H. pylori infection, smoking, NSAIDs, steroids, vagal tone (Cushing's ulcer), burns (Curling's ulcer), Zollinger-Ellinson syndrome (rare).[22]

Gastric ulcer

Background mucosal changes depend on aetiology, for example, *Helicobacter pylori* gastritis or NSAID-related reactive gastropathy and other causes. Mucosal defect occurs about 2–4 cm. It has Smooth base with perpendicular borders. Bleeding vessel or adherent clot sometimes visible in ulcer base. 50% of UGIBs occurs in this case. Symptoms will be the same as duodenal ulcer.[22]

Oesophageal varices

Large dilated submucosal veins expanded submucosa with elevation of mucosa above normal tissue, Haemosiderin-laden macrophages, fresh blood, dilated submucosal vessels are the diagnostic features. Commonly in distal oesophagus dilated arteries are small (1–2mm) or large (1–2 cm). It occurs about 5%–10% of UGIBs. Haemorrhage risk: Size of varices, Anticoagulants, Active alcohol use, Systemic infection.[22]

Gastritis

H. pylori gastritis (usually antral but may be pangastric, bacteria highlighted by histochemistry), NSAID-related reactive gastropathy (foveolar hyperplasia, fibromuscular lamina propria expansion, paucity of inflammation); acute gastritis (mucosal oedema, haemorrhage and superficial erosions) Mucosal erythema and oedema, typically associated with friability and superficial mucosal 'breaks' (erosions). It causes 10%–20% of UGIBs. It is a heterogeneous phenomenon. Common aetiologies are H. pylori colonisation, NSAIDs, alcohol, critical illness. Treatment should comprise H. pylori eradication, antacid therapy and cytoprotective agents.[22]

Mallory-Weiss tear

Longitudinal mucosal lacerations of the distal oesophagus/proximal stomach, with surrounding haemorrhage and acute inflammatory reaction Longitudinal mucosal lacerations with or without active bleeding and adherent clot Occasionally healing tears may appear as superficial 'blood blisters' Up to 10% of UGIBs More common in the young. Predisposing factors include alcoholism and hiatus hernia. Haematemesis initiated by severe coughing or retching Non-bleeding tears may be managed conservatively, with acid suppression and antiemetics.[22]

Treatment

There is no consensus on the treatment of Dieulafoy's lesion. Treatment options are dependent on mode of presentation, site of the lesion and available expertise. The evolution of endoscopic methods of haemostasis has markedly reduced the need for surgery in Dieulafoy's lesions.[5]

In the years following the first description of this condition, most patients with a bleeding Dieulafoy's lesion were treated surgically. In 1990, before surgery, the endoscopic treatment of Dieulafoy's lesion should be attempted.[23] With advances in endoscopic techniques, endoscopic therapy has gradually replaced surgery and has emerged as the first option for diagnosing and managing Dieulafoy's lesions.Endoscopic treatment is safe and highly successful in terms of achieving initial hemostasis and hemostasis success rates reach 75% to 100%.[24,25]

With the use of endoscopic measures, the treatment options are divided into three groups: 1) thermal-electrocoagulation, heat probe coagulation and argon plasma coagulation; 2) regional injection-local epinephrine injection and sclerotherapy; and 3) mechanical- banding and hemoclip (FIG.7). Prior to implementing these curative techniques, an attempt needs to be made to make the patient hemodynamically stable. It is vital to focus on volume resuscitation in order to prevent consequences of end-organ damage. Multiple large bore, intravenous lines are inserted and volume resuscitation performed with crystalline solution, whether with normal saline or lactate ringers. Depending on the patient's level of anemia, transfusion of packed red blood cells is often required and in most patients with DL it has been shown to require three or even more units. DL requires hemostatic therapy, as often times re-bleeding can occur and the diagnosis, specifically the source of the bleeding, may not be initially discovered.[26]

1. Injection therapy

Many kinds of sclerosing agent can be used such as epinephrine or adrenalin, polidocanol, absolute alcoho1, sodium tetradecyl sulfate, hypertonic glucose histoacryl, and combination of hypertonic sodium chloride plus epinephrine (HSE solution).[27] Epinephrine and polidocanol are more favorable. Two to four perilesion injection with or without intralesion injection were used by most authors and more than one agent were used for each lesion by some endoscopists. The goal for injection therapy is obliteration of pathologic artery. Epinephrine causes mild focal mucosal damage and mild tissue inflammation without tissue necrosis or vascular thrombosis. Other sclerosing agents cause mucosa, submucosal and muscular necrosis and vascular thrombosis with occasional serositis. Initial hemostasis attributed to volume tamponade and longterm hemostasis affected from vascular thrombosis. Excessive volume injection causes extensive tissue necrosis and perforation. Epinephrine and polidocanol should not be used in excess of 10 ml and ethanol 2 ml.[28,29]

2. Thermal therapy

All thermal modalities generate heat, at 60 degree C heat produces protein coagulation and contraction of treated area. Heating with firm pressure on vessel causes coaptation. Thermal modalities used in Dieulafoy's lesion include the followings.

2.1 Monopolar or Multipolar electrocoagulation (MPEC or BICAP)

2.2 Thermocoagulation (Heater probe)

2.3 Laser photocoagulation.

Heater probe and BICAP are more favorable. They provide the same effectiveness and application. Depth of tissue destruction is less than 3-4 mm. Heat probe application initially made at perilesion and then directly to vessel is recommended if bleeding vessel is larger than 1 mm. Laser photocoagulation using non-contact mode showed successful control of bleeding but has not received popular attention due to its cumbersome nature and expensiveness. Monopolar electrocoagulation is less favorable because it causes deeper tissue destruction and adherence to coagulated tissue requiring frequent cleaning of the probe and high recurrent bleedingrate. Despite the advent of liquid monopolar electrocoagulation or electrohydrothermal probe, they had no advantage over multipolar electrocoagulation or BICAP. However, dry monopolar electrocoagulation is available in most hospitals and can provide good hemostatic effect if bleeding vessel is less than 0.5 mm.[30] Many endoscopists prefer prior epinephrine injection to thermal coagulation with heater probe or MPEC

(BICAP). Prior epinephrine injection is useful to slow or stop bleeding before thermal therapy application. It also prevents excessive tissue destruction and transmural necrosis by thermal therapy. Removal of adherent clot can be done only when endoscopic therapy and surgical back up is readily available.[31]

3. Mechanical therapy

Hemorrhage can also be controlled by mechanical methods such as endoscopic band ligation (EBL) or application of hemoclips, both of which have been considered to be the first option in the management of Dieulafoy's lesions. One of the most widely used endoscopic mechanical therapies is hemoclipping. It is suggested to be useful and more successful than injection therapy in achieving permanent hemostasis for bleeding Dieulafoy's lesions.[32] However, it is difficult to apply a hemoclip when the angle of approach is tangential or when the lesion is located at the difficult sites (particularly, the gastric fundus, the lesser curvature of the stomach, or the posterior wall of the duodenal bulb) or when the lesion has a fibrotic ulcer base (FIG.7). In addition, prior incorrectly deployed hemoclips can hinder the accurate positioning of subsequent hemoclips. Therefore, it is important to apply the hemoclips accurately on Dieulafoy's lesions, particularly the first one.[33] For this reason, endoscopic hemoclip application should be reserved for experienced endoscopists; nonetheless, it is a good therapy option for Dieulafoy's lesions. Band ligation EBL is another mechanical hemostatic option (FIG.8). The advantages of EBL are ease of use, accessibility to difficult sites (particularly, the esophagogastric junction and the posterior wall of the proximal body of the stomach), and low perforation risk. However, it also has limitations such as poor visual field, unfeasibility in fibrotic tissue, the time interval required to prepare the device, and the difficulty of re-intubation due to the presence of the band-ligating device itself. EBL is also associated with some complications such as delayed bleeding in the case of a residual vessel within a necrotic ulcer as well as perforation.[34] Following the first use of EBL for Dieulafoy's lesion in 1994, EBL has proven to be as effective as bipolar coagulation for Dieulafoy's lesions and has been found to be a simple, successful, effective, and inexpensive procedure. Other endoscopic treatment: Over-the-scope clip (OTSC), It has been developed for the closure of small mural defects. A few studies have reported the use of OTSC for management of a Dieulafoy's lesion, and it may find potential application in routine endoscopic therapy of these lesions.[35]

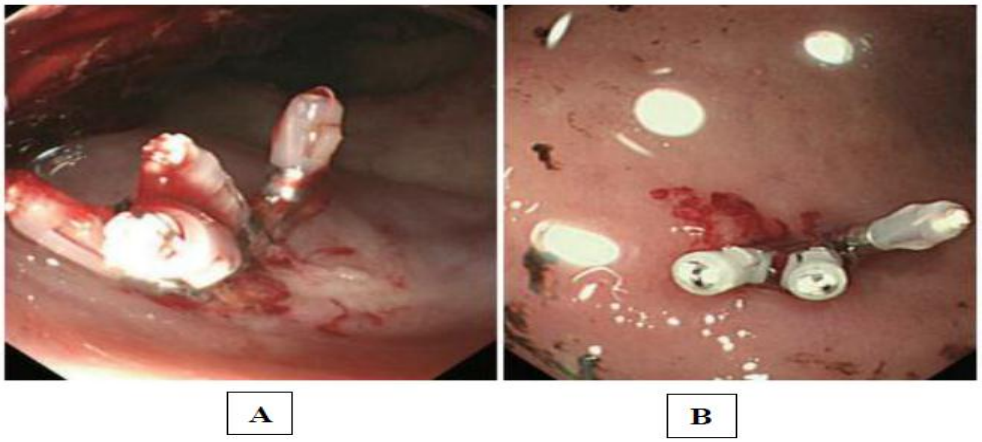

Fig. 7: Endoscopic hemostatic clip application to Dieulafoy's lesions in the Colon (A) and Stomach (B).[10]

Which is the preferred procedure for bleeding dieulafoy's lesions? Each technique has both advantages and disadvantages related to the hemostatic mechanism involved, the technical procedure itself, and varying success rates. **Monotherapy vs. combination therapy:** Combination endoscopic therapy is superior to monotherapy and consists of injection therapy followed by thermal or mechanical therapy, with permanent hemostasis achieved in 95% of all cases. A lower rate of bleeding recurrence has been described with the use of combined endoscopic treatment compared with monotherapy. **Mechanical therapy vs. other methods** Theoretically, mechanical hemostasis leads to less damage to the surrounding tissue than other modalities, and several studies have shown that mechanical therapies including hemostatic clipping and EBL are more effective and successful in achieving hemostasis than other endoscopic methods, such as injection and thermal therapy. These reports suggest that mechanical therapy could be an appropriate first-line approach for the management of a Dieulafoy's lesions.[36]

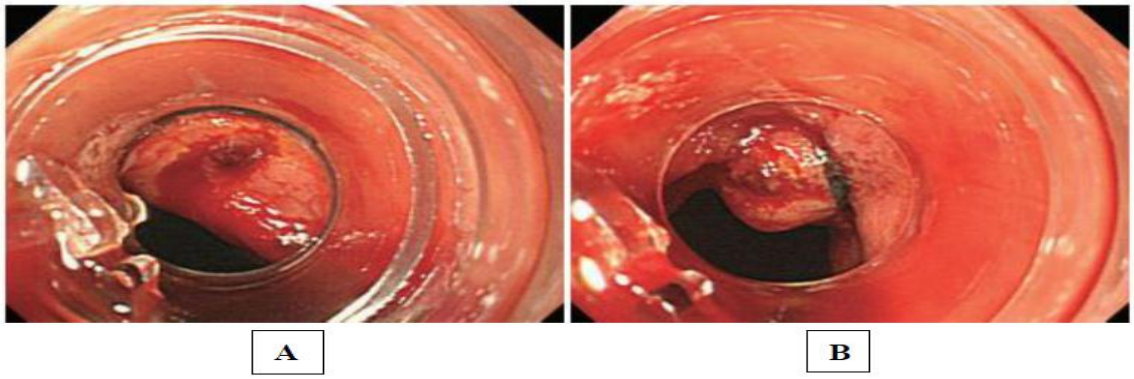

Fig. 8: Endoscopic band ligation to a Dieulafoy's lesion just below the gastroesophageal junction (A, B).[10]

Adjuvant therapy

There are little data as to whether acid suppressive therapy is required after endoscopic therapy, but, in view of the relatively high frequency of concurrent GI pathology, this might seem logical. Proton pump inhibitors (both intravenous and orally administered), H2 receptor antagonists, and sucralfate have been given routinely in some centers.[37]

Angiography and Embolisation

Angiography can be used to embolise actively bleeding Dieulafoy's lesions (FIG.6). This is useful to deal with lesions that fail to respond to endoscopic methods of haemostasis.[38] However, embolisation carries the risk of ischaemia to the area supplied by the relevant artery. If the bleeding lesion is supplied by multiple collaterals, extensive embolisation may be required, which sometimes make it an unsuitable choice of treatment because of the risk of the resultant ischaemia. Selective embolisation may be considered as the treatment of choice in actively bleeding Dieulafoy's lesions in the GI tract. Angiography with embolisation is the preferred treatment for bronchial Dieulafoy's lesions.[39]

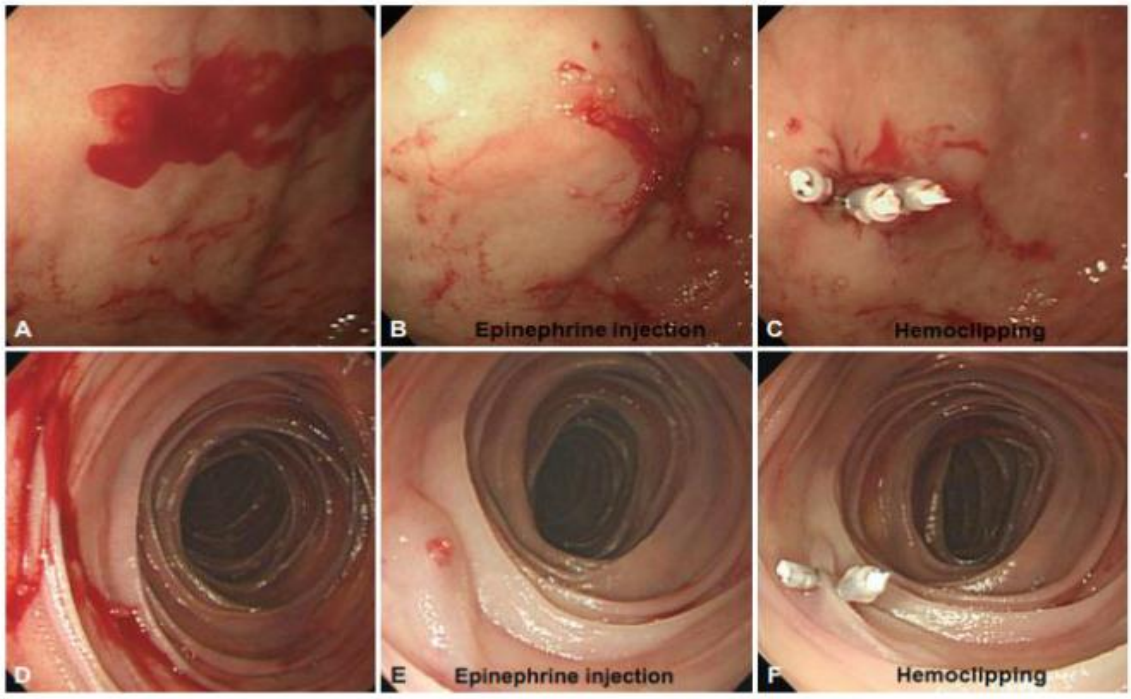

Fig. 9: Combination therapy consists of epinephrine injection therapy followed by hemostatic clipping of Dieulafoy's lesions in the stomach (A-C) and the jejunum (D-F).[10]

Surgical treatment

Surgical resection was historically the first-line treatment of Dieulafoy's lesions, usually taking the form of gastrotomy and wide-wedge resection or gastrectomy.[40] This has now been overtaken by advances in endoscopic procedures. Surgical resection is currently reserved for the 5% of cases that are refractive to endoscopic or angiographic methods. Surgical procedures currently employed include under-running of the lesion or a wedge resection of the affected section of gut. Surgical resection is still preferable, especially in lesions that may be exposed to hard stool, perhaps increasing their likelihood of re-bleeding.[41]

Role of minimally invasive surgery

While the majority of patients requiring surgery would have undergone laparotomy, more recently laparoscopic surgery for removal of these lesions.[42] However, successful laparoscopic resection relies on accurate localisation of the bleeding. Laparoscopic transgastric resection of Dieulafoy's lesion involving an anterior gastrotomy and resection of the lesion, without the need for endoscopy, has been performed. However, such procedures involve an unnecessary gastrotomy. Accurate intra-operative localisation of these lesions could represent a challenge.[43] Several methods have been described to ensure precise localisation. This was first tackled by intra-operative endoscopy that allowed for real-time localisation. Following induction of pneumoperitoneum, upper GI endoscopy was then performed. The laparoscopic light was then dimmed or turned off. The gastroscope was used to visualise and locate the bleeding lesion. The area was marked by clips or suture laparoscopically and resection was then performed. The use of combined endoscopy and laparoscopy to identify and ligate the artery that feeds the bleeding Dieulafoy's lesion without the need for resection. Laparoscopic wedge resection is reported to have lower re-bleeding rates when compared with oversewing the Dieulafoy's lesion. However, wedge resection may not be feasible for lesions situated within 6 cm of the gastrooesophageal junction; in these circumstances, oversewing the lesion or laparoscopic clip placement on the vessel feeding the lesion may be a better option. Pre-operative localisation of the lesion by both tattooing with India ink and using clips, thus allowing accurate localisation whilst alleviating the need for intra-operative endoscopy. The most frequent difficulty with clip placement is that it could fall out prior to surgical intervention. Clips used for pre-operative localisation of the lesion(s) have not been known or reported to interfere with the stapling devices or to cause disruption of the staple line during wedge resection.[44]

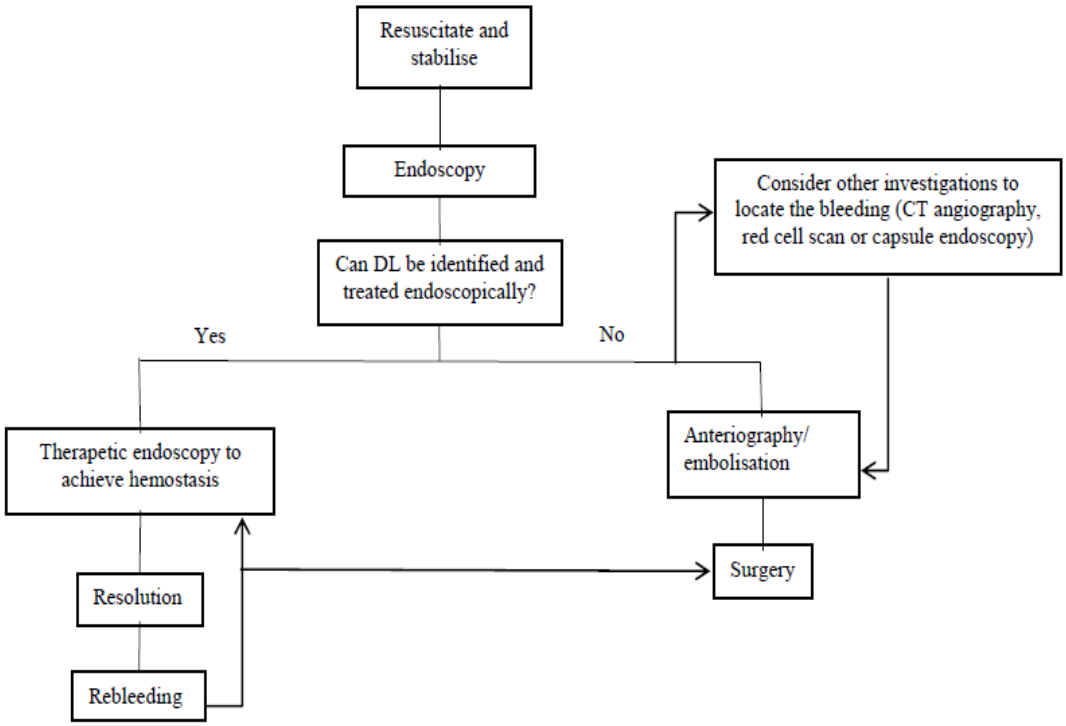

Fig. 10: Treatment algorithm for Dieulafoy's lesion.[5]

Recurrent bleeding

Short-torm, recurrent bleeding is common, occurring in 6% to 28% of cases. In series in which rates of recurrent bleeding at 72 hours could be compared, mechanical (EBL or hemoclip) or combined injection with coagulation appear bettor than injection monotherapy.[45] Single-modality therapy had a higher rate of recurrent bleeding (up to 50%). Repeated endoscopic treatment is recommended as hemostasis can be achieved in almost all patients. Because the lesion is small and may not be found easily after the initial endoscopy, tattooing of the site is advocated to facilitate future endoscopic or surgical localization. Long term, rates of recurrence and recurrent bleeding appear low once the DL is completely treated. In two studies, during a mean follow-up of 3 years after endoscopic treatment of DLs, there was no recurrence of bleeding.[46]

Prognosis

Advances in endoscopy have increased the detection rate of Dieulafoy's lesions and have significantly decreased the mortality from 80% to 8.6%. Improvement in the prognosis might also be explained by the increasing use of endoscopic rather than surgical intervention to control the bleeding which is reported to be effective in more than 90% of patients. Outcome

of acute GI bleeding due to Dieulafoy's lesion has more favourable outcome when compared to acute bleeding from gastric or duodenal ulcer.[47]

Mortality

Before the era of endoscopic therapy, patients oftenn required surgery and the mortality rate was as high as 80%. In the most recent series, mortality rates were significantly below 20%. Mortality after intervention was found to be associated with either advanced age or pre-existing systemic diseases and not directly from continued GI bleeding.[48]

CONCLUSION

In conclusion, DL is an uncommon disorder, Dieulafoy's lesion is increasingly being identified as a cause of major GI bleeding. Prompt endoscopic examination and possibly multiple repeated endoscopies may be needed to make the diagnosis. Endoscopic treatment can achieve hemostasis in greater than 90% of cases. Multimodality therapy has been shown to be more effective than single modality in preventing recurrent bleeding, and mechanical therapies, including EBL and hemoclip application, have been demonstrated to be at least as effective in achieving hemostasis and preventing further bleeding. Currently, surgical intervention is used only after failure of therapeutic endoscopic and angiographic interventions and it should be guided by preoperative localization. The mortality related to DL has significantly decreased.

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