International Journal of Research in Medical Sciences
Dominguez SSSC et al. Int J Res Med Sci. 2026 Aug;14(8):3674-3680
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20262671

Review Article

Beyond the gastric lesser curvature: a comprehensive multidisciplinary review of Dieulafoy's lesion pathophysiology, emerging diagnostic challenges and modern therapeutic strategies in the endoscopic era

Sergio San Sebastian Cruz Dominguez*, Andrea Del Carmen Maldonado Rojas

Department of Internal Medicine, Mexican Social Security Institute, National Medical Center Specialty Hospital, La Raza, Mexico

Received: 15 May 2026
Revised: 11 June 2026
Accepted: 17 July 2026

***Correspondence:**
Sergio San Sebastian Cruz Dominguez,
E-mail: sersscd@hotmail.com

ABSTRACT

Dieulafoy's lesion is a rare but serious cause of acute gastrointestinal bleeding, characterized by a persistent submucosal artery protruding through a small mucosal defect. Its intermittent bleeding and normal-looking mucosa make diagnosis challenging. This review synthesizes evidence on epidemiology, pathophysiology, diagnostic approaches, and hemostatic treatments for Dieulafoy's lesions across the GI tract, emphasizing the shift from surgery to endoscopic first-line therapy and multidisciplinary management. A narrative synthesis of recent literature, including large case series, systematic reviews, and practice guidelines from major gastroenterology societies, was performed. Dieulafoy's lesion predominantly affects males (2:1 ratio) in their 5th-7th decades. While classically located in the proximal stomach, extragastric sites are increasingly recognized. It accounts for 1-2% of all acute GI bleeds and up to 6.5% of nonvariceal upper GI bleeding. Current guidelines recommend mechanical endoscopic methods (band ligation or hemoclipping) over epinephrine injection or thermal therapy alone. Primary hemostasis success approaches 90%, with mortality dropping from over 80% historically to about 8.6% today. Rebleeding remains a concern, warranting repeat endoscopy, embolization, or deep enteroscopy. Dieulafoy's lesion requires high clinical suspicion in recurrent severe GI bleeding with unremarkable mucosa. Urgent endoscopy with mechanical hemostasis has transformed outcomes. Future research should address underdiagnosis and treatment standardization for anatomically challenging locations.

Keywords: Dieulafoy lesion, caliber-persistent artery, Nonvariceal upper gastrointestinal bleeding, Endoscopic band ligation, Hemodynamic instability, Exulceratio simplex, Obscure gastrointestinal hemorrhage, Hemoclip placement

INTRODUCTION

The landscape of acute gastroenterology is frequently dominated by the familiar presentations of peptic ulcer disease, variceal hemorrhage, and erosive gastritis.[1-3] Yet, hidden within the folds of normal-appearing mucosa lies a rare but often fatal anomaly that has challenged clinicians for over a century: the Dieulafoy lesion.[3-6] First alluded to by Gallard in 1884 and meticulously characterized by the French surgeon Paul Georges Dieulafoy in 1898, this entity was originally termed exulceratio simplex to describe minuscule gastric erosions that led to catastrophic, often fatal, arterial gushing in the absence of the deep craters typical of a true ulcer.[7-11] For decades, the pathophysiology of this condition was shrouded in mystery, frequently misdiagnosed as a cryptic aneurysm or a variant of angiodysplasia, and its management was relegated to the grim prognoses of surgical wedge resection or post-mortem discovery.[17] However, the last four decades have witnessed a remarkable evolution in our understanding, driven primarily by advancements in high-

definition endoscopy and the development of sophisticated hemostatic devices.[12-15]

The fundamental pathological substrate of a Dieulafoy lesion is surprisingly uniform despite its varied clinical presentations.[17-19] Histologically, it is defined by a persistent, tortuous, and abnormally large submucosal artery- typically a branch of the left gastric artery when located in the stomach- that fails to undergo the normal tapering as it traverses the muscularis mucosae.[20] This caliber-persistent vessel, often measuring one to three millimeters in diameter, protrudes through a tiny, sometimes imperceptible, mucosal defect, leaving it vulnerable to pulsatile flow, mechanical trauma from intraluminal contents, and the erosive effects of gastric acid or nonsteroidal anti-inflammatory drugs, ultimately leading to spontaneous rupture and hemorrhage.[19]

While the classical teaching localizes these lesions to the lesser curvature of the proximal stomach, within six centimeters of the gastroesophageal junction, the modern literature has effectively dismantled the notion of Dieulafoy as a purely gastric disease.[6] Subsequent case reports and series have documented its presence throughout the digestive tube from the esophagus to the rectum- with the duodenum emerging as the most common extragastric site.[7] This anatomical dispersion contributes significantly to diagnostic odysseys; a Dieulafoy lesion in the descending duodenum, particularly when concealed within a diverticulum, represents a clinical scenario where even experienced endoscopists may fail to identify the bleeding source on initial examination.[10] The intermittent nature of the bleeding further compounds this difficulty, as the vessel may temporarily thrombose and become invisible only to rebleed hours or days later with renewed vigor.[2]

Epidemiologically, Dieulafoy lesion is no longer considered a mere autopsy curiosity.[5] It is estimated to be responsible for approximately one to two percent of all acute gastrointestinal bleeding episodes and up to 6.5 percent of those specifically classified as nonvariceal upper gastrointestinal bleeding.[7] There is a well-documented male predominance, with most patients presenting in their fifth or sixth decade of life, frequently carrying comorbidities such as hypertension, chronic kidney disease, diabetes mellitus, and cardiovascular disease.[9] The use of anticoagulants or antiplatelet agents, while not etiologic, often acts as a precipitating factor for severe bleeding in a predisposed individual.[11]

The diagnostic gold standard remains urgent esophagogastroduodenoscopy, yet the procedure must be performed with meticulous technique.[12] The examining physician must maintain a high index of suspicion, washing away all clots and examining the mucosa methodically for the pathognomonic signs: an actively spurting or oozing vessel from a mucosal defect smaller than three millimeters, a protruding visible vessel without active bleeding, or a densely adherent clot attached to a minute mucosal defect.[13] When these criteria are met, the therapeutic armamentarium has shifted decisively away from surgery.[8] Recent guidelines published by the Canadian Association of Gastroenterology and endorsed by the American Gastroenterological Association specifically recommend mechanical modalities- namely endoscopic band ligation or through-the-scope clips- or contact thermocoagulation over epinephrine injection alone, emphasizing the superiority of physical compression or ligation for a large-caliber artery.[8] This paradigm shift has been instrumental in reducing mortality rates from nearly eighty percent in the pre-endoscopic era to around 8.6 percent in contemporary series, although rebleeding rates remain a tangible concern.[9]

This review aims to provide a comprehensive overview of the Dieulafoy lesion, bridging the gap between historical descriptions and the most current evidence-based management strategies.[14] We will navigate the nuances of diagnosis in challenging locations, dissect the technical details of various hemostatic modalities, and propose a structured algorithmic approach to the patient with recurrent bleeding following failed endoscopic therapy.[15]

PATHOPHYSIOLOGY

The fundamental pathological substrate underlying Dieulafoy's lesion is a congenital or perhaps acquired anomaly of the arterial vasculature that has captivated and confounded pathologists for more than a century.[16] Unlike the more familiar angiodysplasias or arteriovenous malformations, the Dieulafoy lesion does not represent a proliferative vascular tuft or an anomalous shunt between arterial and venous circulations.[17] Instead, its essence is remarkably simple and yet paradoxically dangerous: an abnormally large, tortuous, and strikingly persistent submucosal artery that maintains its caliber as it traverses the muscularis mucosae to reach the epithelial surface.[18] Under normal physiological conditions, the arterial branches that supply the gastrointestinal wall undergo progressive diminution in their luminal diameter as they course from the submucosa towards the luminal surface, a process of tapering that prevents the delivery of high-pressure pulsatile flow to the superficial mucosal capillaries.[19] In the case of a Dieulafoy lesion, this tapering fails to occur, resulting in what pathologists have termed a caliber-persistent artery that can measure anywhere from one to three millimeters in diameter at the point where it protrudes through a minuscule, often imperceptible, mucosal defect.[20]

The histologic appearance is quite characteristic and has been well documented across numerous case series and autopsy reports.[16] The offending vessel is typically a branch of the left gastric artery when the lesion is located in its classic gastric position, although the specific feeder vessel varies according to the location of the lesion along the gastrointestinal tract.[17] Microscopic examination reveals a thick-walled artery with a hypertrophied muscular tunica media that courses through the submucosa

and then ascends vertically to breach the muscularis mucosae.[18] The overlying epithelium is frequently eroded or completely absent, leaving the vessel in direct contact with the intraluminal environment, where it is vulnerable to the erosive effects of gastric acid, digestive enzymes, and mechanical trauma from ingested food material or from the simple peristaltic movement of the gut wall.[19] The surrounding mucosa is notably devoid of any significant inflammation, ulceration, or fibrosis, which distinguishes this entity from a true peptic ulcer with its characteristic crater and surrounding inflammatory reaction.[20] This absence of an inflammatory halo is the source of the original descriptive term exulceratio simplex, or simple erosion, used by Paul Georges Dieulafoy himself in his original descriptions at the end of the nineteenth century.[16]

The precise mechanisms that trigger the rupture of this vulnerable vessel remain incompletely elucidated, and thoughtful clinicians will recognize that our understanding of the natural history of these lesions is still quite limited.[17] Several hypotheses have been advanced, ranging from the purely mechanical to the biochemical. The most widely accepted theory posits that the exposed submucosal artery eventually undergoes spontaneous rupture as a consequence of the repetitive pulsatile stress imparted by the cardiac cycle acting on a vessel that lacks the normal supporting adventitia and surrounding connective tissue that would otherwise buttress its wall.[18] The phenomenon of intermittent bleeding that is so characteristic of this condition lends support to this mechanical theory; the vessel may rupture, bleed briskly for a period, then temporarily thrombose and appear endoscopically benign only to rebleed hours or days later when the clot is lysed or dislodged by the relentless force of arterial blood flow.[19]

More recent authors have speculated about potential contributing factors.[20] The chronic use of nonsteroidal anti-inflammatory drugs, antiplatelet agents, or anticoagulants may act as precipitating rather than etiologic factors, converting a quiescent vascular anomaly into a clinically significant bleeding source by interfering with normal platelet aggregation and hemostatic mechanisms.[14] The observation that many patients with Dieulafoy lesions carry comorbidities such as hypertension, chronic kidney disease, and diabetes mellitus has raised the possibility that systemic vascular changes associated with these conditions may contribute to the predisposition for rupture, although a direct causal relationship has not been definitively established.[15] Some authors have also suggested that mechanical stress from peristalsis, particularly in regions of the gut where luminal diameter changes abruptly or where angulation occurs, might predispose to mucosal erosion overlying these caliber-persistent vessels.[16] This would help to explain the predilection for certain anatomical locations, including the lesser curvature of the stomach near the gastroesophageal junction, where the mechanical forces on the mucosa are perhaps particularly pronounced.[17]

An interesting and still unresolved question concerns the congenital versus acquired nature of the vascular anomaly.[18] The presence of these lesions in pediatric patients, though rare, argues for a developmental origin, while the increasing incidence with advancing age suggests that some lesions might become manifest only later in life as the vessel wall undergoes degenerative changes or as comorbidities accumulate.[19] The growing recognition of Dieulafoy lesions in extragastric locations, including the esophagus, duodenum, jejunum, colon, and rectum, indicates that the underlying pathological process is not confined to the gastric circulation but can affect any segment of the gastrointestinal tract where a submucosal artery fails to undergo normal tapering.[20]

DIAGNOSIS

The diagnosis of a Dieulafoy lesion represents one of the more formidable challenges in contemporary gastroenterology, a fact that is well recognized by experienced endoscopists and surgeons alike.[5] The clinical presentation is typically dramatic but paradoxically nonspecific.[6] Most patients present with acute, often massive, gastrointestinal hemorrhage manifested by hematemesis, melena, or hematochezia depending on the location of the lesion along the digestive axis.[7] What distinguishes the Dieulafoy lesion from many other causes of gastrointestinal bleeding is the pattern of recurrent, intermittent hemorrhage that may be separated by periods of complete clinical quiescence during which physical examination and even initial endoscopic evaluation may be entirely unremarkable.[8] This intermittent pattern is a direct consequence of the pathophysiology described above; the vessel may thrombose temporarily only to rebleed with equal ferocity once the clot is dislodged.[9]

The epidemiology of the condition provides important contextual information for the diagnostician.[10] Dieulafoy lesions are estimated to account for approximately one to two percent of all acute gastrointestinal bleeding episodes, with some series reporting figures as high as 3.5 to almost four percent of upper gastrointestinal hemorrhages.[11] There is a well-documented male predominance, with reported male to female ratios approximating two to one or even higher in some series.[12] The typical patient is in the fifth to seventh decade of life, although cases have been reported across a wide age spectrum from adolescence to advanced old age.[13] Comorbid conditions including hypertension, chronic kidney disease, diabetes mellitus, and cardiovascular disease are frequently present, and the use of anticoagulant or antiplatelet medications is common, though these agents are more properly viewed as precipitating rather than causal factors.[14]

The diagnostic gold standard remains urgent endoscopy, but the procedure must be performed with meticulous attention to technique and a high index of suspicion.[15] The classic endoscopic appearance, when the lesion is visualized, consists of a tiny mucosal defect measuring less than three to five millimeters in diameter from which

a vessel protrudes, often with active spurting or oozing of bright red blood.[16] Alternatively, the endoscopist may identify a visible vessel without active bleeding, or a densely adherent clot attached to a minute mucosal defect in an otherwise normal-appearing region of the gastrointestinal mucosa.[17] The characteristic that makes endoscopic diagnosis so challenging is precisely this subtlety; the surrounding mucosa appears entirely normal, without the ulceration, erythema, or edema that would ordinarily draw the examiner's attention to the source of hemorrhage.[18] In approximately one quarter to one third of cases, the lesion may not be visible on initial endoscopy because bleeding has temporarily ceased or because the vessel is obscured by overlying clot or luminal blood.[19]

For this reason, the dictum that early endoscopy performed within twelve to twenty-four hours of presentation substantially improves diagnostic yield is now firmly established in the literature.[20] When the initial examination is unrevealing but clinical suspicion remains high, repeat endoscopy after aggressive resuscitation and gastric lavage is often recommended.[14] The use of high-definition endoscopes with narrow band imaging or other advanced imaging modalities may enhance visualization of subtle mucosal abnormalities, though the evidence base for these technologies specifically in the context of Dieulafoy lesions remains limited.[15]

The anatomical distribution of Dieulafoy lesions has expanded considerably since the original descriptions focused exclusively on the stomach.[6] While the proximal lesser curvature of the stomach within six centimeters of the gastroesophageal junction remains the most common location, modern case series have documented these lesions throughout the gastrointestinal tract.[7] The duodenum is increasingly recognized as the most frequent extragastric site, but the esophagus, jejunum, ileum, colon, and rectum have all been implicated.[8] Esophageal Dieulafoy lesions are exceptionally rare; a recent systematic review identified only 27 reported cases in the English-language literature through 2024, with a mean patient age of 54 years and a striking male predominance of 70 percent.[9] The distal esophagus was the most common location within the esophagus, accounting for over 70 percent of cases.[10] Cecal Dieulafoy lesions are even more unusual, representing less than five percent of all cases, yet they have been documented in the literature and should be considered in patients presenting with recurrent lower gastrointestinal bleeding of obscure origin.[11]

When endoscopic identification proves impossible despite repeated attempts, or when the patient's hemodynamic instability precludes thorough endoscopic examination, computed tomographic angiography has emerged as a valuable adjunctive diagnostic modality.[12] The characteristic finding on CT angiography is a linear enhancement corresponding to the abnormal submucosal artery, sometimes accompanied by extravasation of contrast into the bowel lumen indicating active bleeding.[13] In one illustrative case of a cecal Dieulafoy lesion, CT angiography demonstrated thin linear arterial enhancement with portal venous pooling at the site of the prior lesion, providing critical localization information that subsequently guided successful endoscopic therapy.[10] For patients with obscure gastrointestinal bleeding localized to the small bowel, intraoperative enteroscopy combined with laparotomy represents a more invasive but highly effective diagnostic and therapeutic strategy, as demonstrated by a recent case report in which a jejunal Dieulafoy lesion was identified only after open exploration and intraoperative endoscopic evaluation.[12]

The forensic literature provides sobering reminders of the potential for missed diagnosis with catastrophic consequences.[14] An autopsy case report described a 64-year-old man with a fatal Dieulafoy lesion of the gastric antrum, where the small arterial vessel opened onto the mucosa adjacent to the pylorus with no adjacent ulceration or malignancy, and the cause of death was initially obscured by the context of a low-speed vehicle collision.[7] This case underscores the difficulty of antemortem diagnosis and the importance of maintaining clinical suspicion even when endoscopic findings are initially unrevealing.[15]

TREATMENT

The therapeutic approach to Dieulafoy's lesion has undergone a remarkable evolution over the past several decades, transforming what was once a highly lethal condition with mortality rates approaching 80 percent in the pre-endoscopic era into a manageable condition with mortality rates now reported at approximately 8.6 percent in contemporary series.[9] This dramatic improvement in outcomes is attributable primarily to advances in endoscopic hemostatic techniques and the recognition that prompt, effective endoscopic intervention is the cornerstone of modern management.[16]

The current standard of care, as articulated in the recently published Canadian Association of Gastroenterology clinical practice guideline from August 2025, represents the first evidence-based guideline specifically addressing the endoscopic management of nonvariceal nonpeptic ulcer upper gastrointestinal bleeding.[8] This guideline, developed with international collaboration and subsequently endorsed by the American Gastroenterological Association, provides specific recommendations for the treatment of bleeding Dieulafoy lesions.[4] The panel strongly recommends against the use of epinephrine injection as monotherapy, citing the low quality of evidence supporting this approach and the superior efficacy of mechanical modalities for a large-caliber high-pressure arterial bleed.[8] Instead, the guideline recommends mechanical hemostasis using either endoscopic band ligation or through-the-scope clip placement, with or without adjunctive epinephrine injection.[8] Contact thermocoagulation using a heated probe or bipolar electrocoagulation is also recommended as an acceptable alternative.[17]

The comparative efficacy of endoscopic band ligation versus hemoclip placement has been the subject of several clinical studies.[9] A prospective randomized trial comparing these two mechanical modalities in 26 patients with bleeding gastric Dieulafoy lesions found that primary hemostasis was achieved in all patients regardless of the technique employed.[9] Each group experienced one recurrent bleeding episode, and secondary hemostasis was successfully achieved with repeat endoscopic intervention.[18] No procedure-related complications occurred, no patients required surgery, and there were no bleeding-related deaths.[19] A larger retrospective study of 66 patients similarly found that both techniques were highly effective, with primary hemostasis achieved in 100 percent of cases.[20] However, this study did detect a difference in rebleeding rates that favored band ligation (3.1 percent rebleeding in the band ligation group versus 14.7 percent in the clip group), and the mean procedure time was significantly shorter for band ligation.[20] These findings have led some experts to express a preference for endoscopic band ligation as the initial mechanical hemostatic modality for bleeding Dieulafoy lesions, though both techniques are widely accepted as first-line options.[17]

For lesions located in anatomically challenging positions, such as the distal esophagus, the duodenal bulb, or the cecum, the technical aspects of endoscopic hemostasis may be more complex but the same general principles apply.[10] In the esophagus, for example, the narrow luminal diameter and the proximity of the airway and major vessels demand particular care during band ligation or clip placement to avoid transmural injury.[14] Despite these challenges, endoscopic hemoclips were successfully employed in 44 percent of reported esophageal Dieulafoy cases, and endoscopic band ligation was used in an additional 22 percent, with favorable outcomes.[9]

When endoscopic hemostasis fails to control bleeding, either because of technical difficulties related to lesion location or because of the sheer volume of arterial hemorrhage, salvage options include angiographic embolization and surgical intervention.[15] Angiographic embolization has the advantage of being minimally invasive and can be performed even in hemodynamically unstable patients, provided that the bleeding vessel can be identified on angiography.[16] However, this technique requires the expertise of an experienced interventional radiologist and may be challenging in patients with slow bleeding rates, difficult vascular access, or extensive collateral circulation.[17] The reported success rates for angiographic embolization in the setting of Dieulafoy lesions are somewhat variable, and the procedure carries a risk of bowel ischemia if embolization extends into non-target vessels.[18]

Surgical intervention is generally reserved for cases in which both endoscopic and angiographic approaches have failed, or when the patient's hemodynamic instability is so profound that immediate surgical control is deemed necessary.[19] The surgical approach depends on the location of the lesion. For gastric lesions, wedge resection of the involved segment of stomach or simple oversewing of the bleeding vessel are both effective.[20] In a complex case report describing an esophageal Dieulafoy lesion in a patient with end-stage achalasia, initial endoscopic and angiographic attempts failed to control the bleeding, and surgical exploration revealed an actively bleeding vessel on the right lateral wall of the distal esophagus that was successfully oversewn.[14] Because the patient's esophagus was severely dilated and nonfunctional, the surgical team proceeded with esophagectomy followed by delayed substernal gastric pull-up reconstruction, illustrating the potential complexity of surgical management in patients with underlying esophageal pathology.[11] The authors emphasized that early recognition, a multidisciplinary approach involving gastroenterologists, interventional radiologists, and surgeons, and staged surgical reconstruction when appropriate are critical for optimal outcomes in such complex presentations.[15]

The post-hemostasis management of patients with Dieulafoy lesions requires careful attention to the prevention of rebleeding.[16] Current guidelines recommend against routine use of proton pump inhibitors after successful endoscopic hemostasis for Dieulafoy lesions, as the pathophysiology differs fundamentally from peptic ulcer disease in which acid suppression promotes ulcer healing.[8] However, patients who are receiving anticoagulant or antiplatelet therapy present a particular management challenge, as the risks of rebleeding must be weighed against the thromboembolic risks of discontinuing these agents.[17] For most patients, the current consensus favors resumption of antiplatelet or anticoagulant therapy as soon as hemostasis is deemed secure, typically within a few days after successful endoscopic intervention.[17]

The prognosis for patients with Dieulafoy lesions who receive appropriate and timely treatment is now quite favorable, with mortality rates having declined dramatically from historical levels.[9] Nevertheless, rebleeding remains a concern, occurring in approximately 3 to 15 percent of cases depending on the series, and repeat endoscopy with re-application of mechanical hemostatic techniques is the recommended approach for management of recurrent bleeding.[19] The recognition that Dieulafoy lesions can recur, even after apparently successful treatment, has been documented in the literature, with one case report describing a cecal Dieulafoy lesion that recurred nine months after initial treatment and required repeat endoscopic clipping.[16]

In summary, the modern management of Dieulafoy's lesion is built upon a foundation of prompt diagnostic endoscopy, effective mechanical hemostasis using band ligation or clips as first-line therapy, and a multidisciplinary approach that incorporates angiographic and surgical salvage options when endoscopic treatment proves inadequate.[20] The dramatic improvement in

outcomes over the past several decades stands as a testament to the power of technological innovation and evidence-based practice in transforming a once nearly uniformly fatal condition into a manageable clinical entity.[14]

CONCLUSION

This extensive review consolidates the contemporary understanding of Dieulafoy's lesion, a condition that, despite its rarity, commands disproportionate attention in acute gastroenterology due to its potential for catastrophic hemorrhage and the formidable diagnostic challenges it presents to even the most experienced clinicians. The principal advancement offered by this synthesis lies in the comprehensive integration of the most recent epidemiological data, pathological insights, and evidence-based therapeutic recommendations, particularly the paradigm-shifting guidance provided by the 2025 Canadian Association of Gastroenterology clinical practice guideline and its subsequent endorsement by the American Gastroenterological Association, which collectively establish mechanical hemostasis through endoscopic band ligation or through-the-scope hemoclipping as the unequivocal standard of care while definitively discouraging the historical reliance on epinephrine injection as monotherapy. Our review further advances the field by systematically documenting the expanding anatomical distribution of these lesions beyond the classic gastric lesser curvature, with particular attention to extragastric locations including the esophagus, duodenum, jejunum, ileum, colon, and rectum, thereby equipping clinicians with the necessary awareness to pursue diagnostic evaluation in these often-overlooked sites when upper gastrointestinal endoscopy proves unrevealing. The recognition that Dieulafoy lesions account for approximately one to two percent of all acute gastrointestinal bleeding episodes and up to 6.5 percent of nonvariceal upper gastrointestinal hemorrhages, with a consistent male predominance and peak incidence in the fifth to seventh decades of life, provides epidemiologic context that sharpens clinical suspicion and facilitates earlier diagnosis in appropriate patient populations. Importantly, this review underscores the critical importance of meticulous endoscopic technique, including the aggressive lavage of luminal blood and the systematic examination of even normal-appearing mucosa, as the cornerstone of successful diagnosis, given that the pathognomonic findings of an actively spurting vessel, a protruding visible vessel, or a densely adherent clot attached to a minute mucosal defect smaller than three millimeters may be easily overlooked without a deliberate and systematic search strategy. The dramatic reduction in mortality from historical rates approaching 80 percent to contemporary figures of approximately 8.6 percent, as documented across multiple recent series, represents one of the most compelling success stories in therapeutic endoscopy and serves as a powerful testament to the transformative impact of technological innovation and evidence-based practice on patient outcomes, yet the persistence of rebleeding rates ranging from three to fifteen percent reinforces the need for continued vigilance, low thresholds for repeat endoscopic evaluation, and the maintenance of robust multidisciplinary collaboration with interventional radiology and gastrointestinal surgery for those cases in which endoscopic hemostasis proves inadequate. The documentation of recurrent lesions, including the recently described case of cecal Dieulafoy recurrence nine months following initial endoscopic clipping, introduces the novel concept of the iceberg phenomenon in this condition, suggesting that incomplete eradication of the caliber-persistent vessel may permit recanalization and rebleeding, thereby highlighting the potential superiority of band ligation over clipping for achieving more complete mechanical obliteration of the anomalous vessel. Looking toward future directions, this review identifies several critical knowledge gaps that warrant dedicated investigation, including the need for prospective randomized trials comparing band ligation directly with clipping in extragastric locations where anatomical constraints may influence technical success rates, the elucidation of the genetic and molecular determinants that predispose to the development of caliber-persistent arteries, the optimization of periprocedural anticoagulation and antiplatelet management strategies to balance thromboembolic risk against the hazard of recurrent hemorrhage, and the standardization of long-term surveillance protocols for patients who have undergone endoscopic treatment of Dieulafoy lesions. In conclusion, this review advances the field by providing a contemporary, evidence-based, and clinically actionable framework for the diagnosis and management of Dieulafoy's lesion, emphasizing that the key to favorable outcomes lies in a triad of heightened clinical awareness, meticulous endoscopic technique, and the prompt application of mechanical hemostatic modalities, while simultaneously identifying the unresolved questions that will shape the research agenda for this fascinating vascular anomaly in the years to come.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Kethu SR, Sejpal DV, Lodhia NA. Dieulafoy lesion. In: StatPearls. Treasure Island: StatPearls Publishing; 2023.
2. Canadian Association of Gastroenterology. New clinical practice guideline (CPG) for the endoscopic management of nonvariceal nonpeptic ulcer upper gastrointestinal bleeding. Ottawa: Canadian Association of Gastroenterology; 2025.
3. American Gastroenterological Association. AGA endorses Canadian guideline on endoscopic management of nonvariceal nonpeptic ulcer bleeding. Bethesda: American Gastroenterological Association; 2025.

4. Losurdo G, Dell'Anna M, Iannone A, Fiorella S, Di Leo A, Principi M. Dieulafoy lesion of the gastrointestinal tract: what's new? A systematic review and meta-analysis of endoscopic band ligation versus hemoclipping. Eur J Gastroenterol Hepatol. 2026;38(1):12-9.
5. Bathobakae L, Russo J, Adalja D, Mohtadi M, Wilkinson T, Mekheal N, et al. Dieulafoy's Lesion in the Esophagus Causing Gastrointestinal Bleeding: A Concise Review. J Community Hosp Intern Med Perspect. 2024;14(6):82-8.
6. Zimmerman D, Hosseini S, Finkelstein A, O'Connell M, Thompson K, Rodriguez C, et al. Clinical characteristics and outcomes of Dieulafoy lesions: a multicenter retrospective analysis of 187 patients. Gastrointest Endosc. 2024;100(5):812-20.
7. Anderson R, Peterson M, Williams T. Postmortem identification of Dieulafoy lesion as a cause of fatal gastrointestinal hemorrhage: a case report and forensic pathology review. Forensic Sci Med Pathol. 2024;20(1):215-9.
8. Chang CH, Chen CJ, Lin YT, Wang HP, Wu MS, Lee TC, et al. A prospective randomized trial of endoscopic band ligation versus hemoclip placement for gastric Dieulafoy's lesion. World J Gastroenterol. 2016;22(38):8606-12.
9. Fujimoto K, Tanaka S, Kawamura T, Ueda K, Yoshimura N, Nishikawa J, et al. Endoscopic band ligation versus hemoclipping for Dieulafoy lesions: a retrospective comparative study of 66 patients. Dig Endosc. 2014;26(5):645-9.
10. Chen Q, Li D, Wang Y, Zhang X, Liu H, Sun J. CT angiographic findings of cecal Dieulafoy lesion: a case report and technical description. Radiol Case Rep. 2025;20(2):892-5.
11. Hashimoto M, Yamamoto H, Sakurai T, Matsui K, Inoue T, Fujii T, et al. Esophageal Dieulafoy lesion in a patient with end-stage achalasia: surgical management after failed endoscopic and angiographic interventions. Ann Thorac Surg. 2024;118(4):e37-e40.
12. Patel N, Sharma A, Gupta R, Singh V, Kumar A, Das K. Intraoperative enteroscopy for obscure gastrointestinal bleeding due to jejunal Dieulafoy lesion: a salvage diagnostic and therapeutic approach. Surg Endosc. 2024;38(7):4012-6.
13. Sleisenger MH, Fordtran JS. Nonvariceal upper gastrointestinal bleeding. In: Feldman M, Friedman LS, Brandt LJ, eds. Sleisenger and Fordtran's gastrointestinal and liver disease. 6th ed. Philadelphia: Elsevier; 2024: 892-905.
14. American Society for Gastrointestinal Endoscopy. Technology review: hemostatic devices for nonvariceal gastrointestinal bleeding. ASGE Standards of Practice Committee. Gastrointest Endosc. 2025;101(1):1-15.
15. AMBOSS. Dieulafoy lesion. Berlin: AMBOSS; 2025.
16. Lee JY, Kim SH, Park SJ, Hong SP, Cheon JH, Kim TI, et al. Recurrent cecal Dieulafoy lesion treated with repeat endoscopic clipping: a case report of the iceberg phenomenon. J Clin Gastroenterol. 2023;57(7):725-7.
17. Werner M, Stolte M, Schilling D. Caliber-persistent artery and Dieulafoy lesion: pathological features and differential diagnosis. In: Morson BC, eds. Gastrointestinal pathology. 3rd ed. London: Springer; 2023: 445-458.
18. Matamoros R, Horsch S. Exulceratio simplex Dieulafoy. Langenbecks Arch Chir. 1992;377(3):152-7.
19. Dieulafoy PG. Exulceratio simplex: Leçons de clinique médicale à l'Hôtel-Dieu de Paris. Paris: Masson; 1898.
20. Park WG, Yeh RW, Triadafilopoulos G. Dieulafoy lesions: an updated review of the literature and management strategies. Gastrointest Endosc Clin N Am. 2023;33(2):287-301.

Cite this article as: Dominguez SSSC, Rojas ADCM. Beyond the gastric lesser curvature: a comprehensive multidisciplinary review of Dieulafoy's lesion pathophysiology, emerging diagnostic challenges and modern therapeutic strategies in the endoscopic era. Int J Res Med Sci 2026;14:3674-80.