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# Dieulafoy's Lesion in the Esophagus Causing Gastrointestinal Bleeding: A Concise Review


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REVIEW ARTICLE

# Dieulafoy's Lesion in the Esophagus Causing Gastrointestinal Bleeding: A Concise Review

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**Abstract**

**Dieulafoy's lesion (DL) is an uncommon cause of life-threatening gastrointestinal (GI) bleeding. It can occur in any part of the GI tract, including the stomach, duodenum, colon, and esophagus. Dieulafoy's lesion in the esophagus (DLE) is an exceedingly rare entity, with only 23 case reports/series (27 patients) reported to date. We performed a systematic search of published case reports on DLE in PubMed, Google Scholar, and Embase, from inception through January 2024. The search terms for the review were "Dieulafoy's lesion," "esophagus," "oesophagus," "hematemesis," "melena," "hematochezia," and "gastrointestinal bleeding." Articles were eligible for inclusion in the study if they were published in English, described a case of DLE noted on endoscopy or histopathology, and were available as full text. Our literature search yielded 23 articles consisting of 27 patients. Of the 27 patients, eight (30%) were female and 19 (70%) were male. The patients' age ranged from 13 years to 87 years, with an average age of 54.1 years. Twenty-five patients (92.6%) presented with hematemesis and melena as chief complaints. DLE was found in the distal esophagus in 19/27 (70.4%) of the patients and the mid-esophagus in 6/27 (22.2%) of the patients. Two patients had no information about the lesion site. Endoscopic hemoclips were employed in 12 (44.4%) patients, endoscopic band ligation (EBL) in six and electrocautery was used in one patient. Early diagnosis and prompt treatment are crucial for managing DLE, as they can cause recurrent bleeding and potentially lead to complications or death.**

*Keywords:* Dieulafoy's lesion, Dieulafoy's lesion in the esophagus, Gastrointestinal bleeding, Hematemesis, Melena, Therapeutic endoscopy

## 1. Introduction

Dieulafoy's lesion (DL) was first reported by Gallard in 1884 as "military aneurysms of the stomach".[1–8] In 1898, a French pathologist and clinician, Dieulafoy dubbed these lesions *exulceratio simplex* as he believed they were early stages of peptic ulceration interrupted by a bleeding diathesis.[1,2,4,5,7,9] In earlier reports, DL was also known as a cirsoid aneurysm, submucosal arterial malformation, gastric aneurysm, gastric arteriosclerosis, caliber-persistent artery, and Dieulafoy ulcer.[1,3,4,6] It was thought to be an anatomic variant, congenital defect, or an acquired vascular malformation.[1–3,10–12]

DL is an extremely rare cause of acute gastrointestinal (GI) bleeding, which can range from mild to life-threatening. Prompt and accurate diagnosis is crucial because massive bleeding from this lesion can be fatal. These pathological lesions can occur in any part of the GI tract, including the stomach, duodenum, jejunum, ileum, colon, cecum, appendix. rectum, anal canal, and esophagus.[2,13–16] DL are characterized by the presence of a larger-caliber artery that protrudes through the mucosa and can lead to massive hemorrhage.[8,12,17] Diagnosis is typically made through endoscopy, which may require repeated procedures due to the small size of the lesion.[18] Treatment options include thermal

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electrocoagulation, local epinephrine injection, sclerotherapy, banding, and hemoclipping.[19,20]

DLs can be challenging to manage and diagnose, particularly when they are not visualized in standard locations. Dieulafoy's lesion in the esophagus (DLE) is an exceedingly rare cause of upper GI bleeding and is associated with significant morbidity and mortality.[7,11,13,21,22] Owing to its rarity, current data are retrospective in the form of case reports and conference abstracts. To date, only 23 case reports/series have been reported, comprising of 27 patients. Here, we review the clinical features, pathophysiology, diagnosis, and management of DLEs based on reported cases.

## 2. Methods

With the aid of a medical librarian, we performed a systematic search for published case reports on DLE in PubMed, Google Scholar, and Embase, from inception through January 2024 (Table 1). The search terms for the review were "Dieulafoy's lesion," "esophagus," "oesophagus," "hematemesis," "melena," "hematochezia," and "gastrointestinal bleeding." Semantic Scholar, an artificial intelligence-powered search engine, was used to broaden the search results. Similarly, the reference lists of key articles were manually examined for additional articles. The search results were filtered using the study design, the English language, and human subjects.

The inclusion criteria were as follows: (1) articles published in English, and (2) articles describing cases of DLE noted during endoscopy or pathology. Articles in foreign languages, articles with inadequate information, articles not available as full texts, and non-case report articles were excluded. Twenty-three articles comprising of 27 patients met the inclusion criteria for further review. Two authors independently abstracted data on patient demographics, clinical presentation, lesion site, hemoglobin level, intervention, and outcome. Data analysis was descriptive in terms of means, ranges, proportions, and percentages.

## 3. Results

Overall, our literature search yielded 23 articles consisting of 27 patients. The PRISMA framework represents the search strategy and search results (Fig. 1). There were 20 case reports and three series. Of the 27 patients, eight (30%) were female and 19 (70%) were male. The patients' age ranged from 13 to 87 years, with an average age of 54.1 years. Twenty-five patients (92.6%) presented with upper GI symptoms, such as hematemesis and melena, as their chief complaint, and only two (7.4%) patients experienced hematochezia. DLE was found in the distal esophagus in 19/27 (70.4%) patients and mid-esophagus in 6/27 (22.2%) patients. Two patients had no information about the lesion site. Hemoglobin level was reported in 11/27 (40.1%) of the patients and it ranged from 5.5 g/dL to 14.0 g/dL, with an average of 9.43 g/dL. Information on blood thinner and nonsteroidal anti-inflammatory drug (NSAIDs) use was reported in 5/27 (18.5%) patients, with aspirin being the most common medication. Endoscopic hemoclips were employed in 12/27 (44.4%) patients as the sole therapy or in combination with epinephrine injection sclerosis or hemostatic powder. Endoscopic band ligation (EBL) was used in six patients and electrocautery was used in one patient. Ethanolamine injection sclerotherapy was used in only four patients. A Sengstaken-Blakemore tube was used as salvage therapy in 3/27 (11.1%) patients. One patient died prior to any endoscopic or surgical intervention and DLE was diagnosed during autopsy.

*Table 1. A summary of our search strategy.*

| Items | Specification |
|---|---|
| Search date | Databases searched up to January 31st, 2024 |
| Electronic Databases | PubMed, Embase, Google Scholar |
| Search terms | "Dieulafoy's lesion," "esophagus," "oesophagus," "hematemesis," "melena," "hematochezia," and "gastrointestinal bleeding." |
| Timeframe | From inception to January 2024 |
| Inclusion and Exclusion criteria | Inclusion:<br>- Articles published in English language<br>- Articles describing cases of DLE noted during endoscopy or pathology<br>- Case reports, case series and conference abstracts describing individual cases<br>Exclusion criteria<br>- Non-English language articles<br>- Cases with inadequate information<br>- Articles not available as full-text<br>- Articles not matching the study design |

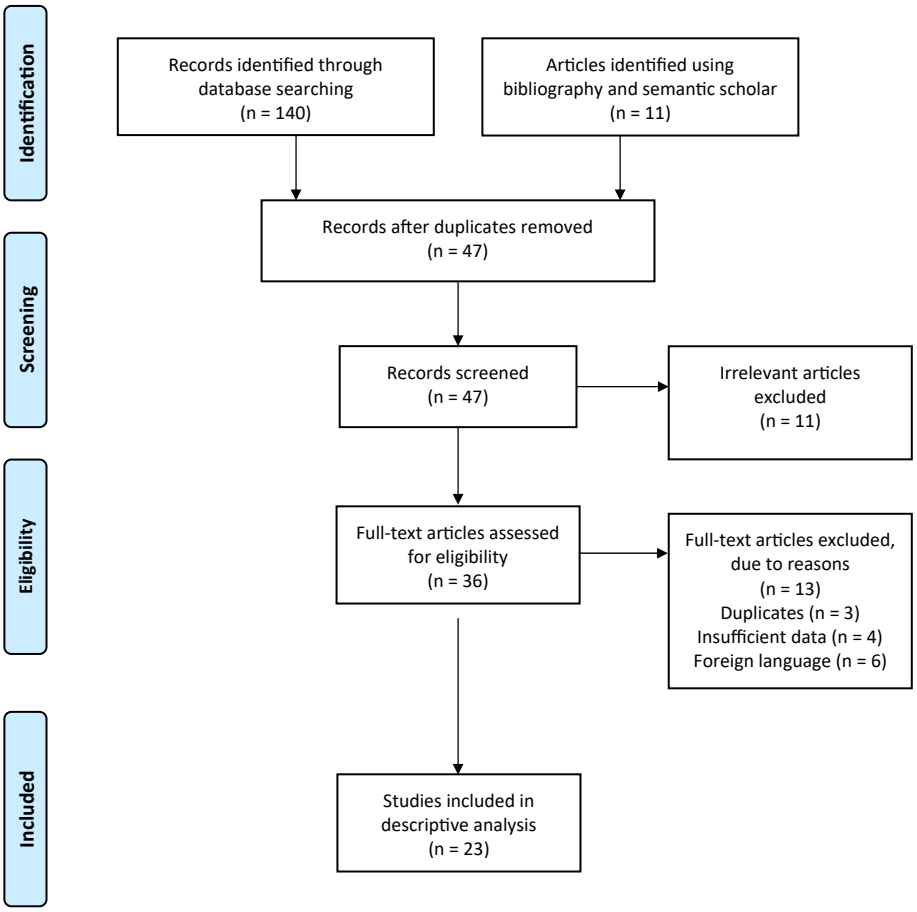


*Fig. 1. PRISMA flow diagram showcasing the search strategy and data abstraction for our study.*

## 4. Discussion

### 4.1. Epidemiology

DL is a unique cause of acute upper GI bleeding accounting for less than 5% of all cases of GI bleeding.[15,18] DLEs are estimated to cause less than 1.5% of upper GI case with most patients presenting with severe hematemesis, melena, and/or hemodynamic compromise.[7,11,22] In the adult population, DLs are twice as common in males compared to females.[6,15] The higher incidence of DLs in males is not observed in children, where there seems to be an equal distribution between genders.[13] The lesions are believed to occur in all age groups; however, they are more frequently observed in the elderly, particularly those in their fifth decade of life.[15] It is speculated that with aging or exposure to certain agents, previously asymptomatic "caliber-persistent arteries" are more prone to bleeding.[1] In our review of DLE cases, 8/27 of the patients were female, whereas 19/27 were male. There was only one pediatric patient with a DLE.

Most DLs occur in the stomach, primarily in the lesser curvature, with an estimated 80–95% of these lesions located within 6 cm of the gastroesophageal junction.[5,7,14] DL tends to be more common in individuals with coexisting conditions such as cardiopulmonary dysfunction and chronic kidney disease.[15,18] The use of certain medications, such as aspirin, warfarin, or NSAIDs, has been observed in around half of the patients experiencing bleeding from these lesions, although a definitive causal link has not been established.[14,15,18] Approximately 10–30% of DLs are extragastric, with a notable presence in the duodenum and colon.[15,23] The etiopathogenesis of extragastric DLs is not yet fully understood owing to their rarity and vague presentation.[15] DLEs are extremely rare, with only 23 cases comprising 27 patients reported to date (Table 2).

### 4.2. Pathophysiology

The stomach and duodenum maintain a rich vascular supply, and the arteries typically decrease

*Table 2. Literature review of published articles on Dieulafoy's lesion of the esophagus.*

| Author(s) | Year | Patient | Presentation | Lesion size/site | Hgb level on admission | Intervention | Blood thinners |
|---|---|---|---|---|---|---|---|
| Alfroukh et al.[1] | 2023 | 76F | Melena, hematemesis | 5 cm, distal esophagus | 8.5 | Hemoclips | Xarelto, Aspirin |
| Senkaya et al.[2] | 2022 | 72M | Melena | 3 mm, distal | 11.9 | Hemoclips | |
| Bartnicki-Navarrete et al.[3] | 2023 | 48F | Melena, hematemesis | Distal | 13.37 | Endoscopic Band ligation | Etoricoxib |
| Burke et al.[4] | 2021 | 35M | Melena, hematemesis | Mid-esophagus | 8.0 | Hemoclips + hemostatic powder | |
| Nemakayala et al.[5] | 2018 | 55M | Melena, hematemesis, syncope, weakness | Distal | 9.5 | Epinephrine injection sclerosis, gold probe cautery | Aspirin |
| García et al.[6] | 2018 | 63M | Hematemesis, melena, hypotension, | Mid-esophagus | 5.6 | Epinephrine, then hemoclips | |
| Peixoto et al.[7] | 2017 | 66M | Hematemesis, hypotension, tachycardia | Mid-esophagus | | Hemoclips | |
| Benatta et al.[8] | 2017 | 24M | Massive hematemesis | Distal | | Endoscopic Band ligation | |
| Malliaras et al.[9] | 2016 | 55M | Obtunded, hematemesis | Distal | | Epinephrine injection sclerosis, hemoclips | |
| Inayat et al.[10] | 2016 | 53M | Melena | Distal | 10.0 | Epinephrine | |
| | | 68M | Hematochezia | Distal | 8.0 | Hemoclips | |
| | | 80M | Hematemesis | Distal | 14.0 | Hemoclips | |
| Christoffersen et al.[11] | 2012 | 48M | Melena, hematochezia | Distal | | None | |
| Thimmmapuram et al.[12] | 2011 | 38M | Melena, hematemesis | Mid-esophagus | | Epinephrine injection sclerosis, hemoclips | |
| Hajj et al.[13] | 2010 | 61M | Hematemesis | Proximal | | Hemoclips | |
| Turan et al.[14] | 2007 | 63M | Hematemesis | Mid-esophageal diverticulum | | Epinephrine + polidocanol, then hemoclips, then Sengstaken-Blakemore tube | |
| Yanar et al.[15] | 2007 | 25F | Hematemesis | Distal | | Endoscopic Band ligation | |
| | | 76F | Hematemesis | Distal | | Endoscopic Band ligation | |
| Yoshida et al.[16] | 2004 | 74M | Melena, hematemesis | 5 mm, distal | | Ethanolamine oleate, then hypertonic saline-epinephrine, then N-butyl-2-cyanoacrylate injection | |
| Ho et al.[17] | 2004 | 71M | Hematemesis | Distal | 11.9 | Adrenaline injection, bipolar diathermy, then Sengstaken-Blakemore tube | ASA, warfarin |
| Ertekin et al.[18] | 2001 | 25F | Melena, hematemesis | Distal | | Endoscopic Band ligation | |
| Soetikno et al.[19] | 2000 | 47M | Hematemesis | Mid-esophagus | | Endoscopic Band ligation | |
| Usui et al.[20] | 1999 | 22F | Recurrent hematemesis | | | Hemoclips | |
| Scheider et al.[21] | 1994 | 87F | Melena, hematemesis | 2 cm, distal | | 5% Ethanolamine | |
| Anireddy et al.[22] | 1993 | 44M | Hematemesis, Melena, hematochezia | Distal | 5.5 | Epinephrine injection | |
| Rábago et al.[23] | 1993 | 13M | Recurrent hematemesis | Distal | | Ethanolamine sclerosis. Then Sengstaken-Blakemore tube | |
| | | 65F | Hematemesis and melena | Distal | | Adrenaline and ethanolamine sclerotherapy, then transcatheter embolization | |

Key: Hgb − hemoglobin; F − female; M− male; Empty boxes − details missing (not available on the case report/manuscript).

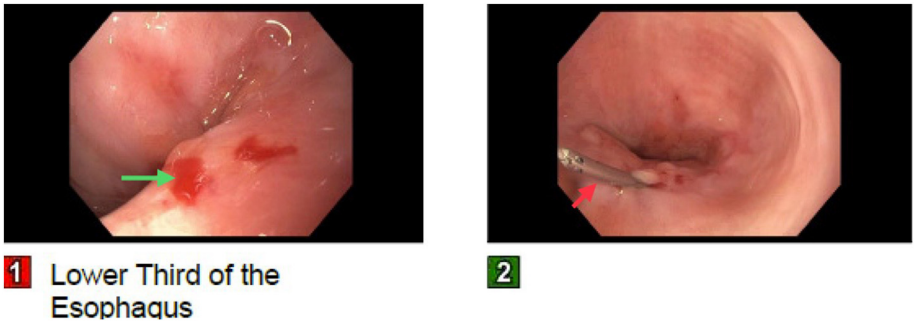


*Fig. 2. Endoscopic image showing a bleeding Dieulafoy's lesion in the distal esophagus (green arrow). The lesion was injected with 4 mL of epinephrine followed by a hemostatic clip placement (red arrow). Image courtesy of Drs. M. Alkomos and K. Amer (St. Joseph's University Medical Center, Paterson, New Jersey).*

in diameter as they reach the mucosa.[14] DL presents as a larger-caliber artery (1–3 mm) that protrudes through the GI mucosa, resulting in significant hemorrhage.[3,11,15,24] This lesion is composed of a normal blood vessel with an abnormally wide diameter, and its etiopathogenesis remains unclear. Damage to the aberrant vessel can result in severe, intermittent arterial bleeding from tiny vessel stumps.[11,15,18]

### 4.3. Clinical presentation

DL is a rare cause of recurrent GI bleeding, and the signs and symptoms depend on the location of the lesion. Clinical features include painless melena, hematochezia, or iron deficiency anemia (IDA).[6,14,15] DLE chiefly presents with hematemesis and melena but can also cause serious bleeding, requiring rapid life-saving hemostatic procedures.[11,25–28] Other symptoms may include fatigue, palpitations, chest pain or syncope.[19,21,29]

### 4.4. Diagnosis

Triage blood tests for upper GI bleeding may reveal acute blood loss anemia, or IDA. White blood cell and platelet counts are typically normal. Additional tests may include coagulation studies, complete metabolic panel, amylase level, and an arterial blood gas analysis. In our review, hemoglobin level was reported in 12 (41.4%) patients and it ranged from 5.5 g/dL to 14.0 g/dL, with an average of 9.43 g/dL. DL is diagnosed through various methods, including esophagogastroduodenoscopy (EGD), endoscopic ultrasound (EUS), capsule endoscopy, and mesenteric angiography.[2,30] EGD is the primary diagnostic tool and involves the visualization of a protruding vessel with or without bleeding, the presence of micro pulsatile streaming from a mucosal defect, and the appearance of a fresh, densely adherent clot with a narrow point of attachment to a minute mucosal defect (Fig. 2).[2,21,30,31] However, initial EGD can be non-diagnostic due to the small size of the lesion or poor visibility.[15,32] In our study, most of the lesions were found in the distal esophagus, followed by the mid and proximal esophagus. The DL site was unspecified in one patient.

In some cases, additional endoscopies may be required for diagnosis. Other diagnostic modalities, such as endoscopic ultrasound and mesenteric angiography, can also be used.[2,6] EUS provides better visualization during endoscopy and aids in identifying arterial flow of the suspicious artery or lesion.[2,23] EUS also helps in assessing the best site for injecting the sclerotherapy agent and evaluating the agent's efficacy based on rate of flow post injection.[2,22,23] EUS has limited use during active bleeding. Squillace et al.[8] were the first to use an EUS to characterize a DL in a geriatric patient who presented with painless melena and severe anemia. The patient was successfully treated with epinephrine injection sclerotherapy. Arteriography or computed tomographic angiography (CTA) can be used to diagnose DLs in the stomach, colon, small bowel, or rectum, using contrast extravasation and arterial tortuosity detection.[11,19,33] Durham et al.[24] reported a case series in which gastric DLs were diagnosed preoperatively with visceral arteriography. Red cell scanning serves as an additional diagnostic tool in cases where endoscopic examination fails to yield a diagnosis.[19]

DLE is an important consideration in cases of recurrent, unidentifiable, and hemodynamically compromising GI bleeding. Other possible causes of esophageal bleeding include early esophageal tumors, Mallory-Weiss syndrome, esophageal varices, Cameron lesions, erosive ulcers, aorto-esophageal fistula, and esophageal necrosis.[2,7,9,11,29,34–36] Mallory-Weiss is a disease in which massive

hemorrhage may occur due to tearing of the esophagus due to continued vomiting. Cameron lesions are seen in those with hiatal hernias, and can cause acute upper GI, which can be life-threatening. Angiodysplasia is another cause of upper GI bleeding, although mostly seen in the stomach, it can also present in the esophagus as a "vascular tuft" on endoscopic evaluation.[34]

### *4.5. Treatment and prognosis*

As noted above, endoscopy is both diagnostic and therapeutic for DLEs. Endoscopic therapeutic options include injection sclerotherapy, mechanical hemoclipping or ligation, or electrocoagulation.[14,37,38] An injection of 2.5 mL of epinephrine (diluted at 1:10,000) is an inexpensive, simple, and effective intervention that can be repeated as needed.[15] Although this modality aids visualization and improves hemostasis success rates, it carries a high risk of rebleeding.[13,15,37] In our review, injection sclerotherapy was often followed by hemoclipping, EBL, or Sengstaken-Blakemore tube salvage therapy due to rebleeding.[7,21,23,37,39,40] Epinephrine alternatives include ethanol, ethanolamine oleate, B-Butyl-2-cyanoacrylate, and polidocanol.[14,39,41,42]

Endoscopic hemoclipping is a superior monotherapy for the management of an actively bleeding DLE and has lower rebleeding rates.[13,15,38] It is, however, difficult to employ at certain angles to achieve hemostasis.[6,14] EBL is an alternative therapy for lesions in difficult anatomic sites, but it poses difficulty in re-intubation and is a time intensive.[14,15,43] Although less effective, endoscopic thermal therapies remain an alternative for hemostasis, especially in the stomach and small bowel. Contact thermocoagulation can be employed using bipolar or heater probes. Argon plasma coagulation (APC) is a non-contact form of thermocoagulation.[13,15,26,44] Angiography with embolization is effective in 40–89% of cases of non-diverticular bleeds.[15] Sengstaken-Blakemore tube placement is a last-resort intervention in actively bleeding DLE refractory to endoscopic therapies. DLs in other parts of the GI tract can be managed surgically when all other options have failed.[13,15,27,28,45]

## 5. Conclusion

DL is a rare cause of GI bleeding, accounting for 1–5.8% of cases. It is characterized by an aberrant submucosal artery with an abnormally wide diameter that protrudes into the mucosa, causing a small-wall defect with fibrinoid necrosis at the base of the lesion. These lesions are often found in the stomach, particularly along the lesser curvature of the stomach. They can also occur in the duodenum, colon, and distal esophagus. The exact pathophysiology of DL is not fully understood; however, it is believed to involve a combination of vascular malformation, arterial hypertension, and mucosal fragility. Early diagnosis and proper treatment are crucial for managing these lesions, which may require repeated endoscopy for diagnosis and endoscopic therapy for treatment. In some cases, intravascular embolization or surgical intervention may be necessary.

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## Author contributions

LB conceptualized the idea of this review article. JR, DA, MM, TW, NM, and RY assisted with literature review, curation of sources, and drafting and revision of the manuscript. KA, YC, and WB edited and proofread the final draft. All authors approved the final version of this manuscript.

## Data availability statement

The authors declare that data supporting the findings of this study are available within the article.

## Conflicts of interest

No conflicts of interest to declare.

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