Angiodysplasia of the gastrointestinal tract

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INTRODUCTION

Aberrant blood vessels are frequently found in the gastrointestinal (GI) tract, where they are probably more common than anywhere else in the body. Some are present from birth or develop as part of inherited syndromes, but the vast majority are acquired later in life. The reasons for the distortion of vascular structures observed with advancing age are poorly understood.

Inconsistent taxonomy of vascular anomalies in the GI tract, along with a common presentation of GI bleeding from different vascular abnormalities, has led to significant confusion in the medical literature [1]. The diagnosis of a vascular anomaly can be based upon endoscopic findings, histologic characteristics, or association with systemic diseases.

Vascular anomalies can be divided into three broad categories:

- Vascular tumors or angiomas, which can be benign (such as hemangiomas) or malignant (such as Kaposi sarcoma or angiosarcoma).
- Vascular anomalies associated with congenital or systemic diseases such as blue rubber bleb nevus syndrome, Klippel-Trénaunay-Weber syndrome, Ehlers-Danlos syndrome, the CREST variant of scleroderma, and hereditary hemorrhagic telangiectasias (Osler-Weber-Rendu syndrome).

- Acquired and sporadic lesions such as angiodysplasias, gastric antral vascular ectasias, radiation-induced vascular ectasias, and Dieulafoy lesions.

Angiodysplasias are the most common vascular anomalies encountered in the GI tract. The pathology, clinical manifestations, diagnosis, and treatment of angiodysplasia will be reviewed here. The evaluation of occult and suspected small bowel bleeding, as well as Dieulafoy lesions and gastric antral vascular ectasias, are discussed separately. (See "Evaluation of occult gastrointestinal bleeding" and "Evaluation of suspected small bowel bleeding (formerly obscure gastrointestinal bleeding)" and "Causes of upper gastrointestinal bleeding in adults".)

CLASSIFICATION

The terms angiodysplasia, arteriovenous malformation, angiectasia, and vascular ectasia have been used synonymously. Angiodysplasias are usually distinguished from telangiectasias, which, while anatomically similar, are usually referred to in the context of systemic or congenital diseases. In addition, some authors use angiectasia as a generic term, reserving the term angiodysplasia for colonic lesions.

Since most vascular abnormalities are detected during endoscopy, a taxonomy based upon endoscopic appearance has been proposed [2]. The classification system recognizes the location, size, and number of angiodysplasias (table 1). With the advent of newer endoscopic techniques, there has been an attempt to classify the lesions found in the small bowel according to size, overt bleeding, and surrounding vessels [3]. However, these classifications are not widely used. (See "Wireless video capsule endoscopy" and "Overview of deep small bowel enteroscopy".)

EPIDEMIOLOGY

Gastrointestinal (GI) angiodysplasia is most often detected in patients older than 60 years [4-6], although presentation in patients in their 30s has been described [7]. The prevalence of GI angiodysplasia in the overall population is not well known. In a report that pooled three prospective studies of colonoscopic screening for neoplasia in healthy asymptomatic adults (older than 50 years, mean age 62 years), angiodysplasia was detected in only 8 of 964 patients (0.8 percent) [8]. The prevalence may be higher with more sensitive techniques such as injection studies in resected colon specimens.

The prevalence is increased in patients with end-stage kidney disease, von Willebrand disease, and possibly aortic stenosis. (See 'Conditions associated with angiodysplasia' below.)

Approximately 40 to 60 percent of patients have more than one angiodysplasia [5,9]. While they are usually in the same portion of the GI tract, synchronous lesions elsewhere in the GI tract are found in approximately 20 percent [10]. In one series of patients thought to be bleeding from angiodysplasia of the colon, small bowel angiodysplasia was detected in 23 percent, either at the original laparotomy or during investigation for recurrent bleeding [10]. This illustrates the point that a given angiodysplasia can only be confidently considered the source of GI blood loss if it is seen to be actively bleeding.

Conditions associated with angiodysplasia — The prevalence of angiodysplasia is increased in older populations, in patients with GI bleeding, and in patients with certain predisposing conditions such as end-stage kidney disease, von Willebrand disease, and possibly aortic stenosis.

End-stage kidney disease — Angiodysplasia account for approximately 20 and 30 percent of episodes of upper and lower GI bleeding, respectively [11], and for approximately one-half of recurrent episodes of upper GI bleeding in these patients [12]. They can occur anywhere along the GI tract and are usually multiple [12]. (See "Unique aspects of gastrointestinal disease in patients on dialysis".)

The reason for the increased prevalence among patients with end-stage kidney disease is unknown. One possible explanation is that the lesions are not more common, but are instead detected more frequently because of the increased risk of bleeding associated with uremia-induced platelet dysfunction leading to a higher number of endoscopic exams [11]. (See "Uremic platelet dysfunction".)

von Willebrand disease — An association between angiodysplasia and congenital or acquired von Willebrand disease has been reported [13]. However, similar to end-stage kidney disease, this association may reflect an increased tendency for angiodysplasia to become clinically evident because of an underlying coagulopathy. (See "Acquired von Willebrand syndrome", section on 'Consequences of reduced VWF'.)

Aortic stenosis — Bleeding from angiodysplasia in patients with aortic stenosis (AS) has been called Heyde syndrome [14]. While it has been repeatedly reported, some aspects remain controversial [15-17].

In support of the relationship of angiodysplasia with AS is the observation that clinically significant bleeding improves in most patients after aortic valve replacement (AVR) [18-21]. One report, for example, evaluated 91 patients with AS and suspected chronic small bowel bleeding, presumably from angiodysplasia [18]. Sixteen of these patients underwent AVR for AS during

follow-up of 8 to 12 years, with cessation of chronic GI blood loss in all but one. (See "Clinical manifestations and diagnosis of aortic stenosis in adults", section on 'Bleeding tendency'.)

A separate issue is whether the prevalence of AS is increased in patients with angiodysplasia. The results of small case-control and retrospective studies have been equivocal:

- One report compared echocardiographic findings in 40 patients with angiodysplasia with 37 matched controls [22]. None of the patients in either group had evidence of aortic stenosis.
- A similar observation was made in a retrospective series of 59 patients with angiodysplasia, only one of whom had echocardiographic evidence of aortic stenosis [23].
- By contrast, a third report of 73 patients with angiodysplasia who had undergone echocardiography did suggest an association with AS [24]. In that report, the prevalence of AS was 32 percent, which was significantly higher than the prevalence among the general population of patients who undergo echocardiography (approximately 14 percent).

Left ventricular assist devices — GI bleeding is common among patients with left ventricular assist devices (LVADs) and is often related to angiodysplasia [25-29]. The pathophysiology is thought to be related to acquired von Willebrand disease. (See "Management of long-term mechanical circulatory support devices", section on 'Bleeding'.)

As an example, in a series of 172 patients with LVADs, GI bleeding developed in 32 (19 percent) [25]. Among those with GI bleeding, angiodysplasia accounted for 31 percent of the bleeding episodes. Similarly, in a series of 391 patients with LVADs, 62 patients (16 percent) developed GI bleeding [29]. The bleeding was attributed to vascular malformations in 27 percent.

PATHOPHYSIOLOGY

Classically described in the colon, angiodysplasia can occur throughout the gastrointestinal tract. There may be multiple angiodysplasias in one gastrointestinal region, or they may coexist in several different gastrointestinal locations.

Morphology — Angiodysplasias are composed of ectatic, dilated, thin-walled vessels that are lined by endothelium alone or endothelium along with small amounts of smooth muscle. Studies in which casts of angiodysplasias were made by injecting a silicone material demonstrated that the most prominent feature in angiodysplasias is the presence of dilated, tortuous submucosal veins.

Small arteriovenous communications are also present and are due to incompetence of the precapillary sphincter. Enlarged arteries may be seen in larger angiodysplasias and may be associated with arteriovenous fistulas, which explains why bleeding can be brisk in some patients (image 1).

Histologic confirmation is often difficult. When obtained, it shows dilated vessels in the mucosa and submucosa, sometimes covered by only a single layer of surface epithelium (picture 1) [30].

Venous obstruction — The pathogenesis of angiodysplasia is not well understood. However, a proposed theory is that angiodysplasia develop due to intermittent, recurrent low-grade obstruction of submucosal veins at the level of the muscularis propria (figure 1). Over years, the obstruction results in dilatation and tortuosity of the draining areas (ie, submucosal vessels, venules, and superficial capillaries).

The venous obstruction hypothesis is consistent with the observation that angiodysplasia often occurs in the right colon where wall tension is highest. This increased wall tension selectively compresses thin-walled veins, while allowing normal flow through the thicker-walled higher-pressure arterioles.

Angiogenic factors — Increased expression of angiogenic factors has been found in human colonic angiodysplasias [31]. Investigations have suggested a possible role of angiopoietin 1 and 2, as well as TNF in the development of lesions in the small bowel [32,33]. However, the relationship of this observation to pathogenesis is uncertain.

Acquired von Willebrand disease — A possible mechanism by which aortic stenosis (AS) may lead to the development of angiodysplasia is through the development of an acquired form of von Willebrand disease [21,34]. (See "Acquired von Willebrand syndrome".)

Acquired von Willebrand disease is thought to result from mechanical disruption of von Willebrand factor multimers during turbulent passage through the narrowed valve and from a von Willebrand factor interaction with platelets that triggers platelet clearance [21,35]. This raises the possibility that the increased prevalence of angiodysplasia in patients with AS is due to increased detection because these patients are more likely to present with bleeding. (See "Clinical manifestations and diagnosis of aortic stenosis in adults", section on 'Bleeding tendency'.)

CLINICAL MANIFESTATIONS

Angiodysplasia may be found during evaluation of gastrointestinal (GI) bleeding, or it may be discovered incidentally during an endoscopic evaluation being performed for other reasons. If bleeding occurs, the bleeding tends to be recurrent and chronic. However, marked acute bleeding causing orthostasis or hypotension can occur but is less common.

GI bleeding can occur at a multiple sites within the GI tract, including the colon, small intestine, and stomach.

Colon — Colonic lesions are found most often in the right colon. The following distribution was noted in a series that included 59 patients with colonic angiodysplasia (lesions were single in 34 patients and multiple in 25 patients), 47 of whom were asymptomatic [36]:

- Cecum – 37 percent
- Ascending colon – 17 percent
- Transverse colon – 7 percent
- Descending colon – 7 percent
- Sigmoid colon – 18 percent
- Rectum – 14 percent

Other series of patients with angiodysplasia have found even higher rates of right-sided lesions (up to 89 percent) [6,37]. The risk of subsequent bleeding in patients who are found to have nonbleeding colonic angiodysplasia is not well established. The number of lesions and the presence of coexisting coagulopathies or platelet dysfunction may be important determinants. Patients who have bled from colonic angiodysplasia are at increased risk for subsequent bleeding [38]. (See "Etiology of lower gastrointestinal bleeding in adults".)

Small intestine — Angiodysplasia can be found throughout the small intestine. It is frequently detected when patients undergo evaluation for GI bleeding of unclear etiology after unrevealing upper endoscopy and colonoscopy. (See "Evaluation of suspected small bowel bleeding (formerly obscure gastrointestinal bleeding)".)

Approximately 5 percent of patients presenting with GI hemorrhage do not have a source identified by an initial upper endoscopy or colonoscopy; a potential source will be found in the small intestine in three-quarters of these patients [39]. Small intestine vascular lesions are responsible for approximately 40 percent of episodes of small intestinal bleeding in patients over the age of 40 years.

Stomach and duodenum — Angiodysplasia of the stomach or duodenum has been incriminated as the cause of blood loss in 4 to 7 percent of patients with GI bleeding [4,5,9].

Such patients may present with either occult bleeding or overt bleeding. In addition, angiodysplasia may be detected as an incidental finding.

The frequency of clinical manifestations was evaluated in a study of 41 patients with gastric or duodenal angiodysplasia found during upper endoscopy performed for a variety of indications [40]. The lesions were associated with overt bleeding in 11 patients (27 percent) and with occult bleeding in 9 (22 percent) [40]. The remaining 21 patients (51 percent) did not have a history of occult or overt bleeding, and the angiodysplasia was considered to be incidental finding. The risk that incidentally found gastric or duodenal angiodysplasia will subsequently bleed is uncertain.

DIAGNOSIS

Angiodysplasia is usually diagnosed by endoscopy being done to evaluate gastrointestinal (GI) bleeding, but in some cases, radiographic imaging or surgery may be required for detection. The aggressiveness of the diagnostic approach should be individualized depending upon the clinical circumstances. As an example, further evaluation is probably not necessary in a patient with a negative upper and lower endoscopy and negative wireless video capsule endoscopy, unless the patient is bleeding severely enough to require transfusions. (See "Evaluation of suspected small bowel bleeding (formerly obscure gastrointestinal bleeding)".)

Endoscopy — Endoscopic options for the diagnosis of angiodysplasia include upper endoscopy, colonoscopy, wireless video capsule endoscopy, and deep small bowel enteroscopy (eg, single and double balloon enteroscopy). Because angiodysplasia can be located throughout the GI tract, a combination of endoscopic techniques may be necessary.

Angiodysplasias characteristically appear as small (2 to 10 mm), flat, cherry-red lesions with a fern-like pattern of arborizing, ectatic blood vessels radiating from a central vessel (picture 2A-B). The characteristic appearance may be more evident in the colon. Small intestinal lesions are often smaller than those seen in other parts of the GI tract. The fern-like pattern should be specifically looked for since other erythematous mucosal lesions or normal blood vessels can be mistaken for angiodysplasias.

The sensitivity of colonoscopy for the endoscopic detection of angiodysplasia is unknown because most patients do not undergo angiography, which is the gold standard for diagnosis. However, it is estimated to exceed 80 percent [6]. Angiodysplasia may be difficult to visualize during colonoscopy in patients who do not have an optimal bowel preparation or when lesions are situated behind a haustral fold. In addition, transiently decreased mucosal blood flow due to

the administration of opioid sedation or air insufflation may make angiodysplasia less visible. Therefore, the administration of an opioid antagonist or the withdrawal of air during the examination may improve their detection.

Administration of an opioid antagonist was evaluated in a series of 144 patients older than 60 years of age who underwent colonoscopy for occult or overt bleeding or iron deficiency anemia [41]. Angiodysplasia was detected in 12 patients (8 percent). After reaching and inspecting the cecum, patients were given naloxone hydrochloride (0.4 to 0.8 mg intravenously). Initially inapparent angiodysplasia became visible in an additional four patients.

However, it is unclear if this approach would lead to significant clinical benefit, given the uncertainty that a nonbleeding angiodysplasia is the cause of bleeding during standard colonoscopy, much less one that has been augmented by giving naloxone. Furthermore, reversal of opioid analgesia may cause an uncomfortable subsequent examination due to lack of analgesia and can precipitate acute narcotic withdrawal in patients who use narcotics chronically.

Radiographic imaging — Imaging, such as computed tomography (CT) angiography or magnetic resonance angiography, may provide another method to diagnose angiodysplasia. The accuracy of CT angiography was estimated in a study that included 26 patients suspected of having colonic angiodysplasia who underwent standard colonoscopy, CT angiography, and standard angiography [42]. Compared with the results of colonoscopy and standard angiography, the sensitivity and specificity of CT angiography were 70 and 100 percent, respectively. Further studies are needed to better define the role of this technique in the management of patients with GI bleeding.

In cases of recurrent GI bleeding or persistent iron deficiency anemia where no source has been identified with less invasive means (eg, endoscopy and CT angiography), angiography may establish the diagnosis and possibly permit therapy (image 1 and image 2). The evaluation of patients with suspected small bowel bleeding is discussed in detail separately. (See "Evaluation of suspected small bowel bleeding (formerly obscure gastrointestinal bleeding)".)

Intraoperative enteroscopy — In some patients with a negative endoscopic and radiographic evaluation, the bleeding will be significant enough to require further evaluation. One approach is intraoperative enteroscopy. During intraoperative endoscopy, the segment of bowel being evaluated should be carefully evaluated before pleating the bowel over the endoscope since pleating of the bowel often causes mucosal abrasions that may be confused with angiodysplasia [43]. (See "Evaluation of suspected small bowel bleeding (formerly obscure gastrointestinal bleeding)", section on 'Intraoperative enteroscopy'.)

As in the colon, small bowel angiodysplasia may be very difficult to see in patients with acute GI bleeding, especially when there is significant anemia, hypotension, or when opiates were given during the endoscopic examination.

TREATMENT

Several issues must be addressed with regard to the treatment of angiodysplasia:

- How should incidentally found lesions be managed?
- In patients being evaluated for occult or overt gastrointestinal (GI) bleeding, should nonbleeding lesions found on endoscopy be treated?
- What is the efficacy of endoscopic treatment?
- Which patients require surgery?
- Is there a role for treatment with medical therapy?

Incidentally found lesions — Angiodysplasia that is detected during screening colonoscopy should **not** be treated (as long as there is no history of GI bleeding or unexplained iron deficiency anemia). As discussed above, the risk of subsequent bleeding is unknown, but is probably low given that angiodysplasia is often found in asymptomatic patients (see 'Colon' above). In addition, a patient found to have a single angiodysplasia during a routine endoscopic examination probably has additional undetected lesions in the colon or in other portions of the GI tract. (See 'Epidemiology' above.)

Nonbleeding angiodysplasias in patients with GI bleeding — While it is agreed that actively bleeding lesions require treatment, the management of nonbleeding lesions is less clear since it frequently cannot be determined whether an identified nonbleeding angiodysplasia was the cause of either occult or overt bleeding.

Occult bleeding — Despite endoscopic evaluation, the cause of occult GI bleeding remains unclear in 10 to 40 percent of patients [44]. The proportion of patients with an unclear source of bleeding may be even higher since it is often uncertain whether specific findings (such as small polyps, gastritis, or esophagitis) are actually the cause of bleeding. (See "Evaluation of suspected small bowel bleeding (formerly obscure gastrointestinal bleeding)".)

Similar uncertainty exists for angiodysplasia detected during evaluation of occult bleeding or iron deficiency anemia since angiodysplasia is a common finding in patients without GI bleeding. Angiodysplasia is more likely to be the cause of occult bleeding in patients who have ongoing occult bleeding, multiple lesions, or a bleeding diathesis. As a result, a graduated approach to treatment is reasonable in these patients.

We suggest treating angiodysplasia found during upper endoscopy or colonoscopy in patients with occult bleeding or unexplained iron deficiency anemia, even if the lesions are not bleeding at the time of the endoscopy. If needed, patients should also be started on iron replacement therapy. If the anemia persists despite these measures, more intensive diagnostic and therapeutic options can be considered, such as capsule endoscopy followed by deep or intraoperative enteroscopy for treatment of any angiodysplasia that are found (algorithm 1). (See "Evaluation of suspected small bowel bleeding (formerly obscure gastrointestinal bleeding)".)

Overt bleeding — It can be difficult to determine if a nonbleeding angiodysplasia is the cause of overt bleeding in a patient who has both angiodysplasia and diverticula. This clinical scenario is common since both occur in the same age group [45]. (See "Colonic diverticular bleeding".)

Overt GI bleeding is more likely to be due to angiodysplasia rather than diverticula if:

- There are multiple recurrent episodes of overt bleeding
- The patient has a condition that predisposes to angiodysplasia, such as end-stage kidney disease (see 'Conditions associated with angiodysplasia' above)
- The bleeding appears to be venous in origin

On the other hand, bleeding from the left colon is more likely to be diverticular in origin. Diverticula are less common in the right colon, but when present are more prone to bleeding than those in the left colon. Bleeding from angiodysplasia is more commonly from the cecum or ascending colon, as the right colon is the most common site of angiodysplasia. (See 'Colon' above.)

Despite the fact that it can be difficult to determine if overt bleeding is due to diverticula or angiodysplasia, if nonbleeding angiodysplasia is found during the evaluation of overt bleeding, we suggest that it be treated unless definitive bleeding from a diverticulum is identified.

Endoscopic treatment — A variety of endoscopic treatments can be used to treat angiodysplasia, with approaches employing cautery being the most widely used. The approach that is chosen depends upon the location and mode of access to the lesion, the experience of the endoscopist, and the availability of equipment. Endoscopic therapy should be performed cautiously in the right colon, which is thin-walled and more likely to perforate during treatment than other sections of the GI tract. It has been estimated that following endoscopic therapy, approximately one-third of patients with angiodysplasia will experience rebleeding after a mean

of 22 months [46]. However, a higher rebleeding rate has been noted in the subgroup of patients with small bowel angiodysplasia (45 percent).

Argon plasma coagulation — Argon plasma coagulation (APC) uses high-frequency energy transmitted to tissue by ionized gas. This technique has been used for a variety of bleeding lesions, including angiodysplasia [47]. APC is safe with proper noncontact technique and is the most common and most successful method used to treat angiodysplasia, especially in the right colon [48]. Its popularity is due to its ease of use (especially for large superficial lesions), low cost, and limited depth of coagulation, although the depth of coagulation may be deeper than is commonly reported. (See "Argon plasma coagulation in the management of gastrointestinal hemorrhage".)

In two in vivo animal model studies, injury to the muscularis propria was noted frequently and correlated closely with power, duration, and total energy delivered. There were no perforations seen in either of these small studies [49,50]. However, perforation of the cecum has been reported in clinical settings.

Submucosal saline injection prior to treatment with APC may protect against deep wall injury [51]. In a study of ten colonic angiodysplasias, submucosal injection with a saline/epinephrine solution prior to APC was done easily and quickly [52].

A full colon preparation is required when performing APC in the colon, even in the rectum and sigmoid colon, due to the risk of colonic gas explosion from inadequate preparation [53].

Electrocoagulation — Bipolar or heater probe coagulation is effective for treatment of angiodysplasia in the colon or upper GI tract (table 2) [54]. The risk of perforation with heater probe coagulation may be increased in the colon and small bowel beyond the duodenum. These techniques have generally replaced monopolar coagulation, which may be less effective and is associated with an increased rate of complications because of its deeper cautery penetration.

Mechanical hemostasis — Mechanical hemostatic methods such as endoscopic clips have been described for the treatment of localized lesions. These methods have the advantage of avoiding tissue injury, which may be particularly desirable in patients taking anticoagulants and/or antiplatelet agents, or in patients with coagulation defects. These techniques have only been described for angiodysplasia in case reports [55,56], so their efficacy is not established. Endoscopic clips may be deployed in cases of persistent bleeding after therapy using cautery methods to avoid further use of cautery and deeper extension of cautery injury.

Band ligation is another mechanical method that has been described in case reports. It has been used to treat angiodysplasia of the stomach and small bowel [57-59].

Radiofrequency ablation — Radiofrequency ablation (RFA) has been used for treatment of gastric antral vascular ectasia and bleeding from the small bowel [60,61]. In a case series of 20 patients with bleeding from small bowel angiodysplasia [60], rebleeding was seen in four patients (three of whom had a left ventricular assist device [LVAD]).

Effectiveness — Determining the impact of treatment for angiodysplasia is difficult due to the variable natural history of angiodysplasia and the inconsistent impact that bleeding can have on quality of life. Prospective controlled trials have not been performed.

Results from older studies were mixed. In two initial observational studies of 16 patients with angiodysplasia requiring transfusion and 33 patients with angiodysplasia and unexplained iron deficiency anemia, more than one-half of patients had recurrent bleeding episodes after surgery, endoscopic therapy, or transfusions alone [62].

More promising results were noted in a report of 83 patients thought to have bled from small intestinal angiodysplasia who were followed for approximately 30 months [54]. Fifty-five patients received treatment with electrocautery during push enteroscopy and were compared with 28 patients who were treated conservatively. The treated group had a significantly lower transfusion requirement during follow-up (0.32 versus 2.16 units of blood per month).

In a prospective study of 100 patients with colonic angiodysplasia, treatment with APC led to stabilization of hemoglobin levels in 85 patients with a mean follow-up of 20 months [63]. Only one patient in that study went on to require surgical treatment for angiodysplasia.

Patients with bleeding diatheses have worse outcomes, regardless of the therapy employed, if the underlying coagulation defect cannot be reversed. Because endoscopic electrocoagulation can result in worsening bleeding in patients with a coagulopathy, we favor a mechanical hemostatic method, such endoscopic clips, if the coagulation defect cannot be corrected.

Angiography — Angiography may localize the site of active bleeding and permit embolization or infusion of vasopressin to stop the bleeding. Although embolization with microcoils may be more successful than vasopressin infusion, it is associated with a higher rate of complications. Angiography is generally reserved for patients with life-threatening bleeding who are not surgical candidates, or for localization of lesions prior to surgical resection. (See "Angiographic control of nonvariceal gastrointestinal bleeding in adults".)

Surgery — Surgical resection is definitive therapy for lesions that have been clearly identified as the source of bleeding. However, recurrent bleeding can occur from lesions elsewhere in the GI tract [4,40]. In a series that included 16 patients who underwent right hemicolectomy for bleeding from angiodysplasia, unexplained recurrent bleeding developed in six (38 percent).

The causes of recurrent bleeding after surgery included incomplete resection of the initial angiodysplastic lesion, occult lesions missed on arteriography and left behind at surgery, and the appearance of new lesions after surgery.

Surgery can be considered for patients with bleeding from a clearly identified site who have a large transfusion requirement or life-threatening hemorrhage and who have not responded to and/or are not a candidate for endoscopic or angiographic therapies. Preoperative or intraoperative enteroscopy or angiography may be helpful for localizing lesions. As discussed above, aortic valve surgery may reduce bleeding in patients with angiodysplasia and aortic stenosis. (See 'Acquired von Willebrand disease' above.)

Hormonal therapy — Estrogen (with or without progesterone) has been used to control small bowel bleeding in patients with hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu syndrome), end-stage kidney disease, and von Willebrand disease. The use of hormonal therapy in these disorders is discussed separately. (See "Hereditary hemorrhagic telangiectasia (HHT): Evaluation and therapy for specific vascular lesions", section on 'Gastrointestinal lesions'.)

A crossover study of 43 patients who acted as their own controls suggested a benefit in patients with bleeding from sporadic angiodysplasia [64]. However, this has not been confirmed in other studies [65,66]. The best data come from a multicenter, placebo-controlled trial involving 72 noncirrhotic patients bleeding from documented angiodysplasia; there was no benefit from hormone therapy [65]. Based on these findings, there seems to be little role for hormonal therapy in patients with sporadic angiodysplasia.

Angiogenesis inhibitors — Inhibitors of angiogenesis, such as thalidomide and bevacizumab, may play a role in the treatment of vascular malformations [67]. The successful use of thalidomide has been described in a randomized trial [68], case reports [69,70], and observational studies [71,72]. (See "Hereditary hemorrhagic telangiectasia (HHT): Evaluation and therapy for specific vascular lesions", section on 'Gastrointestinal lesions'.)

Thalidomide — A randomized trial compared treatment with thalidomide with iron supplementation in 55 patients with recurrent or refractory bleeding from GI vascular malformations [68]. Patients were assigned to either receive thalidomide (25 mg four times per day) or ferrous succinate (100 mg four times per day). Patients were treated for a total of four months and then were followed for one year after treatment. A response to treatment was defined by a decrease in bleeding episodes of at least 50 percent within the first year of follow-up. Patients treated with thalidomide were more likely than those treated with iron to have a response to treatment (71 versus 4 percent). In addition, patients treated with thalidomide were more likely to have their bleeding stop (46 versus 0 percent), less likely to be dependent upon

blood transfusions (11 versus 48 percent), and less likely to be hospitalized for bleeding (39 versus 100 percent). The most common adverse events seen in the thalidomide group included:

- Fatigue (32 percent)
- Constipation (25 percent)
- Dizziness (21 percent)
- Peripheral edema (14 percent)
- Abdominal distension (4 percent)

Additional adverse events that were noted in one patient each (four percent) included abdominal distension, leukopenia, thrombocytopenia, bradycardia, numb limb, somnolence, headache, hand trembling, tinnitus, rash, pruritus, dry eye, blurred vision, vaginal discharge, and herpes zoster infection. Adverse events seen in the iron group included fatigue (11 percent), constipation (11 percent), dizziness (7 percent), and abdominal distension (7 percent). No severe adverse events were reported in this small trial, but thalidomide has been associated with venous thrombosis, peripheral neuropathy, liver toxicity, and fulminant hepatic failure, in addition to causing congenital anomalies [73-75].

Based upon these results, it appears reasonable to treat patients with recurrent or refractory bleeding with thalidomide if they have failed to respond to other therapies. However, until other studies confirm these results, we suggest it only be used as a last resort in transfusion-dependent patients. In addition, due to its teratogenic effects, thalidomide must not be used in women of childbearing potential who are unable to use two reliable forms of birth control for at least one month prior to starting thalidomide and for one month after stopping it.

Bevacizumab — Bevacizumab is a humanized monoclonal antibody against vascular endothelial growth factor that has been used in case reports and small case series to treat refractory bleeding from small bowel angiodysplasia with some success [76-79].

Octreotide — The efficacy of octreotide given subcutaneously (typically 50 to 100 mcg twice per day) in the treatment of angiodysplasia has been evaluated in case reports, a small series, and a meta-analysis in which a response has been observed in some patients [80-87].

In a study of 32 patients receiving 50 mcg twice a day, the actuarial probability of remaining free of rebleeding at one and two years was 77 and 68 percent, respectively [85]. This was higher than the probability seen with patients receiving an oral placebo as part of a separate study (55 and 36 percent, respectively). However, the groups did not differ with regard to the number of bleeding episodes or transfusion requirements, although patients receiving octreotide had a lower requirement for iron.

A meta-analysis summarized the results of three prospective studies with a total of 62 patients who were treated with a somatostatin analog for recurrent bleeding from GI vascular malformations [86]. The analysis found that 76 percent of patients responded to the therapy. In addition, transfusion requirements were significantly lower after patients started long-term treatment compared with baseline. Patients with refractory bleeding and/or inaccessible lesions may benefit from long-term octreotide [88].

A long-acting form of octreotide (octreotide-LAR) is available that can be given intramuscularly once per month. It was studied in 15 patients with recurrent bleeding from GI angiodysplasia [89]. Outcomes were compared between a six-month period while on treatment and the six months prior to starting treatment. The percentage of patients who experienced a bleeding event was lower during treatment than prior to treatment (20 versus 73 percent), median transfusion requirements were lower during treatment (2 versus 10 units), and median hemoglobin levels were higher during treatment (10 versus 7 g/dL).

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Gastrointestinal bleeding in adults" and "Society guideline links: Hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu syndrome)".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topic (see "Patient education: Angiodysplasia of the GI tract (The Basics)")

SUMMARY AND RECOMMENDATIONS

- **Clinical manifestations and diagnosis**
  - Angiodysplasia can remain clinically silent or cause bleeding. Patients who bleed typically present with occult blood loss. (See 'Clinical manifestations' above.)
  - Angiodysplasia is usually diagnosed by endoscopy, but in some cases radiographic imaging or surgery may be required. (See 'Diagnosis' above.)
- **Treatment**
  - Bleeding angiodysplasia require treatment, which can usually be accomplished endoscopically. (See 'Treatment' above.)
  - We suggest that angiodysplasia found incidentally during screening colonoscopy not be treated (**Grade 2C**). (See 'Incidentally found lesions' above.)
  - We suggest treating nonbleeding angiodysplasia when found endoscopically in patients with iron deficiency anemia or evidence of gastrointestinal bleeding (**Grade 2C**). (See 'Nonbleeding angiodysplasias in patients with GI bleeding' above.)
  - Surgery and angiography are alternatives to endoscopic therapy and are typically employed in patients in whom endoscopic therapy has failed. (See 'Angiography' above and 'Surgery' above.)
  - Alternative therapies, such as angiogenesis inhibitors or octreotide, have also been tried, but data supporting their use are limited. These agents are generally reserved for patients who have failed to respond to more traditional therapies. (See 'Angiogenesis inhibitors' above and 'Octreotide' above.)

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Topic 2557 Version 31.0

GRAPHICS

Classification of gastrointestinal angiodysplasia

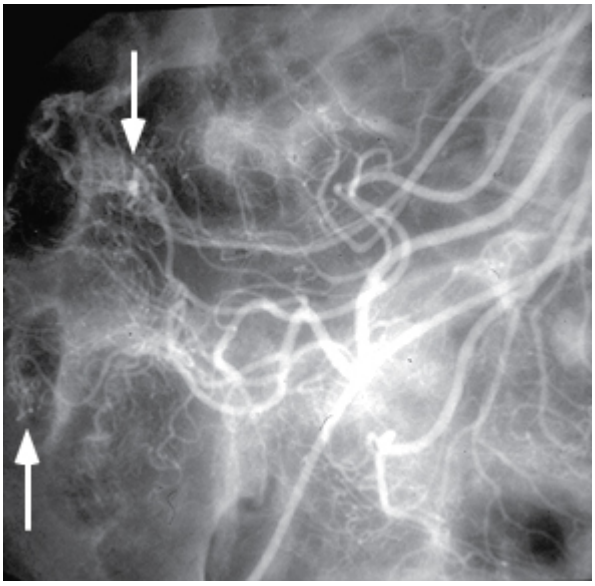
Location
Gastric
Duodenal
Jejunal
Ileal
Colonic
Size
Minute (<2 mm in diameter)
Intermediate (2 to 5 mm)
Large (>5 mm)
Number of lesions
Unique (n = 1)
Multiple (n = 2 to 10)
Diffuse (n >10)

As an example. "J - S2 - N2," signifies multiple angiodysplasias of intermediate size in the jejunum.

Proposed by the European Endoscopy Club in: Schmit A, van Gossum A. Proposal for an endoscopic classification of digestive angiodysplasias for therapeutic trials. Gastrointest Endosc 1998; 48:659.

Graphic 67055 Version 3.0

Angiography of colonic angiodysplasia

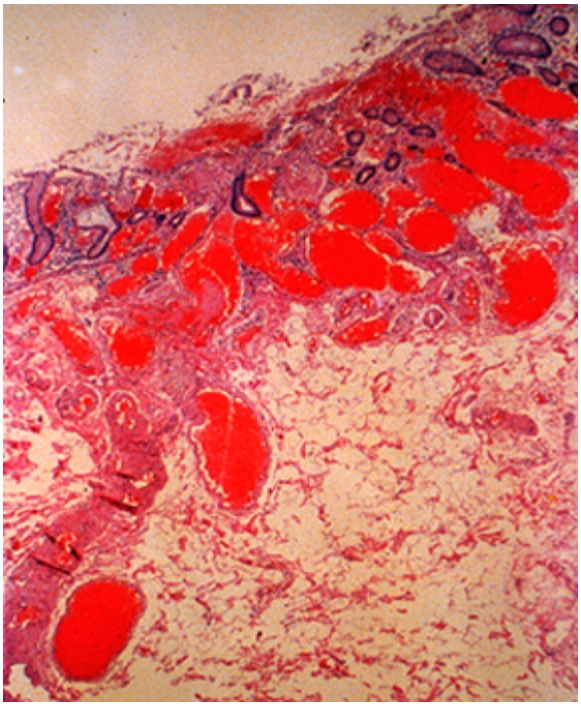


A superior mesenteric arteriogram demonstrates puddling of contrast material in tortuous distended vessels in the cecal wall (arrows).

Courtesy of Jonathan Kruskal, MD.

Graphic 52754 Version 4.0

Angiodysplasia of the colon

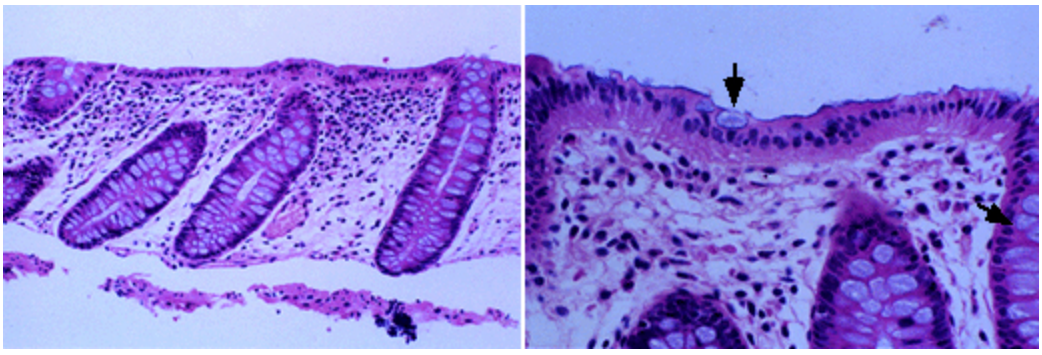


Low power view of a colonic biopsy in angiodysplasia shows mucosal and submucosal vascular dilatation and congestion.

Courtesy of Robert Odze, MD.

Graphic 74301 Version 2.0

Normal colon

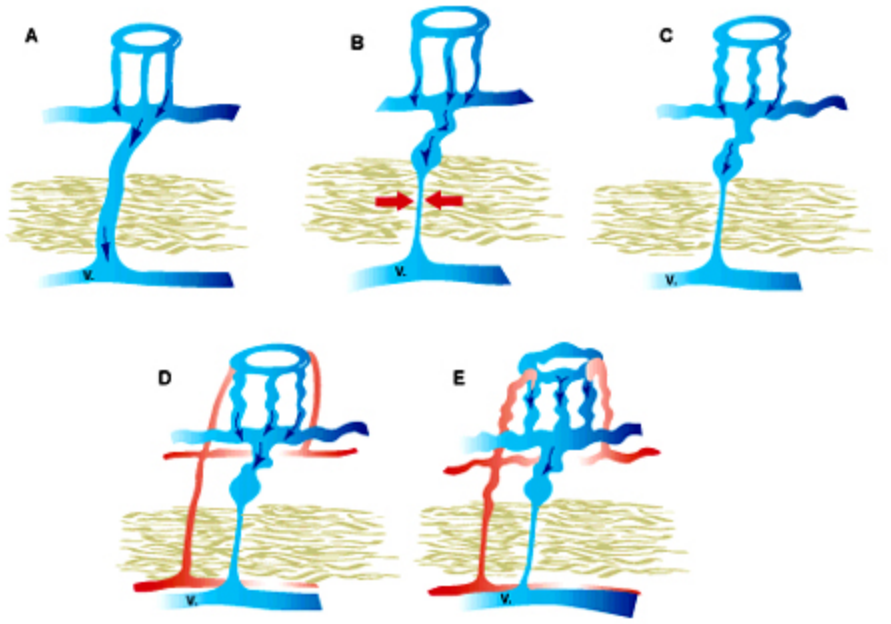


Low (left) and high (right) power views of a biopsy of a normal colon. Low power reveals straight crypts and mild lamina propria mononuclear cell infiltration. High power shows the surface enterocytes with interspersed goblet cells (arrows).

Courtesy of Robert Odze, MD

Graphic 81083 Version 6.0

Proposed mechanism for the development of angiodysplasia

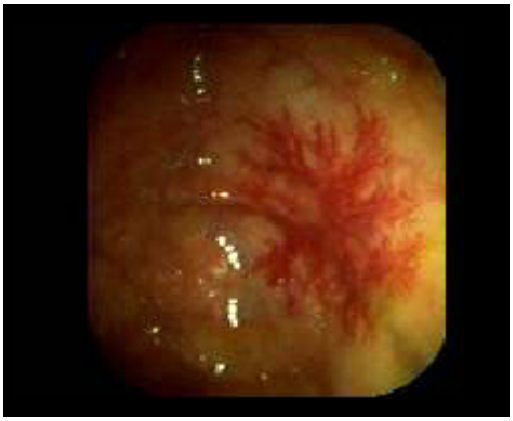


This cartoon depicts one possible explanation for the development of gastointestinal angiodysplasias. Mucosal and submucosal venous drainage is intermittently obstructed by muscular contraction or increased intraluminal pressure (panels A, B, and C). After many years of intermittent obstruction, submucosal veins may become dilated and tortuous (panel D) and involve additional veins and venules draining into the system. Eventually, the capillary ring dilates and the precapillary sphincter becomes incompetent resulting in a small arteriovenous communication lesion.

Boley SJ, Sammartano R, Adams R, et al. On the nature and etiology of vascular ectasias of the colon: Degenerative lesions of aging. Gastroenterology 1977; 72:650.

Graphic 76997 Version 2.0

Endoscopic image of colonic angiodysplasia

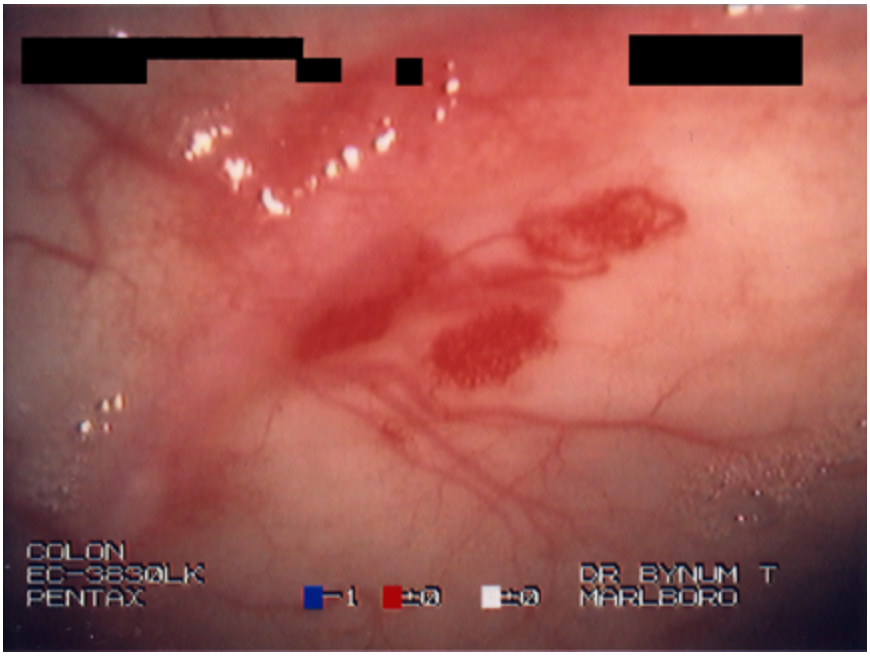


Angiodysplasia appears endoscopically as peripherally expanding dilated capillaries with a central origin measuring between 0.1 to 1.0 cm in diameter.

Courtesy of Rome Jutabha, MD.

Graphic 50137 Version 4.0

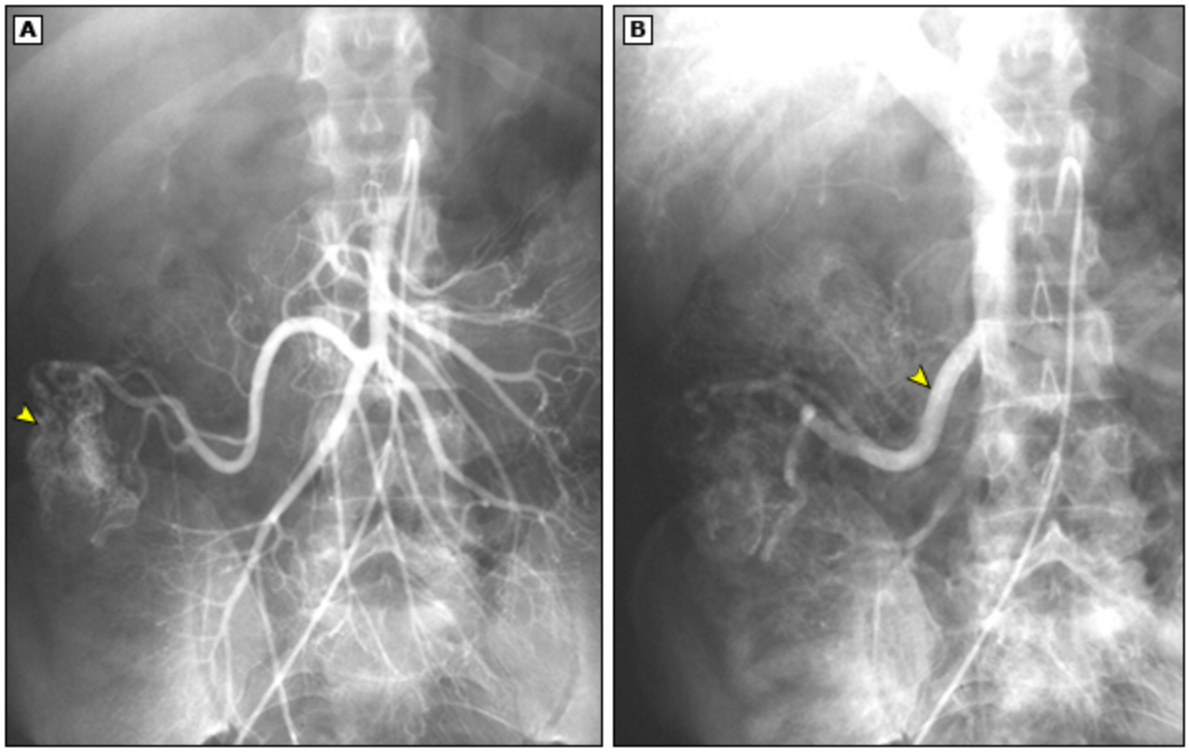
Endoscopic image of colonic angiodysplasia



Courtesy of T Edward Bynum, MD.

Graphic 73541 Version 2.0

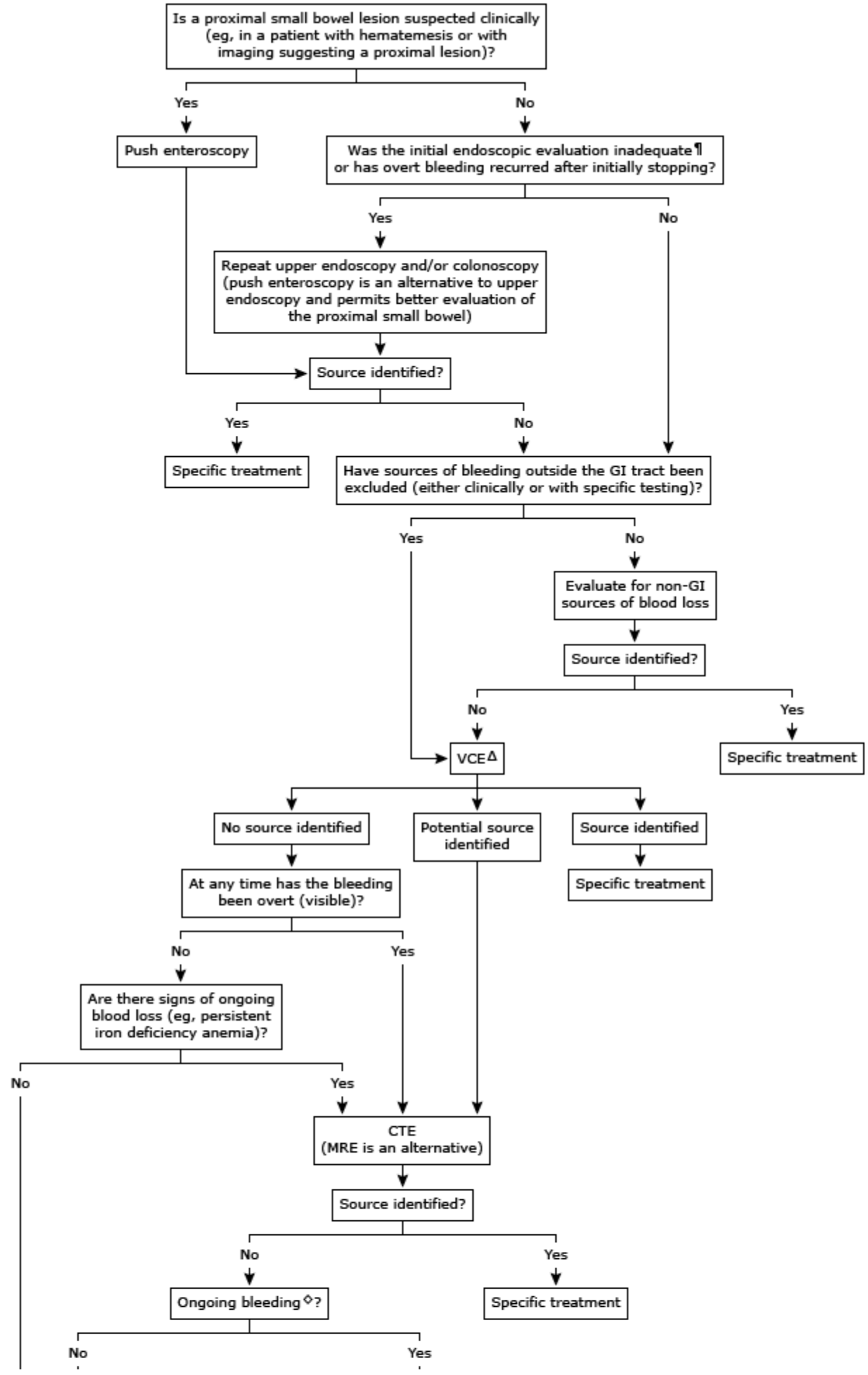
Angiodysplasia on arteriography

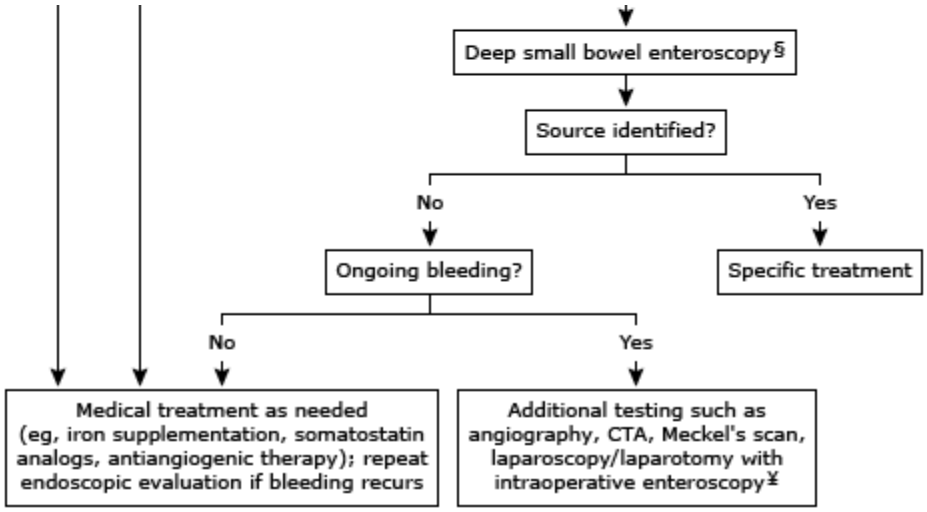


A superior mesenteric angiogram (A) shows the arterial phase of an extensive arteriovenous malformation of the ascending colon (arrowhead). Image B shows the venous phase and a large early draining vein (arrowhead).

Graphic 93362 Version 1.0

Evaluation of suspected small bowel bleeding in hemodynamically stable patients*


Is a proximal small bowel lesion suspected clinically (eg, in a patient with hematemesis or with imaging suggesting a proximal lesion)?
Yes
No
Push enteroscopy
Was the initial endoscopic evaluation inadequate¶ or has overt bleeding recurred after initially stopping?
Yes
No
Repeat upper endoscopy and/or colonoscopy (push enteroscopy is an alternative to upper endoscopy and permits better evaluation of the proximal small bowel)
Source identified?
Yes
No
Specific treatment
Have sources of bleeding outside the GI tract been excluded (either clinically or with specific testing)?
Yes
No
Evaluate for non-GI sources of blood loss
Source identified?
No
Yes
Specific treatment
VCEΔ
No source identified
Potential source identified
Source identified
Specific treatment
At any time has the bleeding been overt (visible)?
No
Yes
Are there signs of ongoing blood loss (eg, persistent iron deficiency anemia)?
No
Yes
CTE (MRE is an alternative)
Source identified?
No
Yes
Ongoing bleeding◇?
Specific treatment
No
Yes



GI: gastrointestinal; VCE: video capsule endoscopy; CTE: computed tomographic enterography; MRE: magnetic resonance enterography; CTA: computed tomographic angiography.

* Small bowel bleeding should be suspected in patients with signs of GI bleeding who have had a negative initial endoscopic evaluation (typically upper endoscopy and colonoscopy). The evaluation of hemodynamically unstable patients is discussed in the context of the specific bleeding manifestations (eg, hematemesis). Refer to UpToDate topic reviews on the evaluation and management of GI bleeding for details.

¶ For patients with risk factors for hemobilia or hemosuccus pancreaticus, the upper endoscopy should have included evaluation with a side-viewing duodenoscope. Patients with risk factors for an aortoenteric fistula should also have undergone CTA. If the initial upper endoscopy and/or colonoscopy was inadequate (eg, fair or poor visualization, failure to reach the cecum), repeat examination should be considered before initiating an evaluation for small bowel bleeding.

Δ VCE should be done as close to the acute bleeding episode as possible to increase diagnostic yield. Patients at risk for capsule retention should undergo small bowel imaging (eg, CTE) or a patency capsule study prior to VCE.

◇ In patients with significant comorbid illnesses with slow rates of blood loss, it may be reasonable to stop the evaluation and treat with iron repletion and/or transfusions as needed.

§ Push enteroscopy is an alternative if not already done and if deep small bowel enteroscopy is not available. Intraoperative enteroscopy is an alternative if there are contraindications to deep small bowel enteroscopy, such as dense intra-abdominal adhesions.

¥ The choice of test will depend on the rate of bleeding, patient characteristics, and the degree of suspicion for a small bowel lesion. A Meckel's scan should be performed in younger patients with overt bleeding. Angiography or CTA can be obtained if there is active bleeding. Surgical exploration is appropriate if no other studies have revealed a source and significant bleeding continues or if there is high suspicion for a small bowel neoplasm. If the evaluation is still negative, non-GI sources of blood loss should be reconsidered.

Graphic 95608 Version 4.0

Endoscopic therapy of lower intestinal bleeding

	Probe diameter¶	Coaptive pressure	Electrogenerator settings*		Desired outcome	Speci featur
			Heater probe¶	Bipolar probe¶		
Angiodysplasia	Large$^{\Delta}$	Light	10-15 J	10-15 W, 1 second pulses	Bleeding stops/angiodysplasia obliterated	Periphery lesion treated before center; caution w right-side lesions
Diverticulosis	Large$^{\Delta}$	Light to moderate	10-15 J	15-20 W, 1-2 second pulses	Bleeding stops/visible vessel flat	Coaptive pressure may depe upon location (dome vs neck of diverticul
Postpolypectomy	Large$^{\Delta}$	Light to moderate	10-20 J	15-20 W, 1-2 second pulses	Bleeding stops	Snaring (tampona of polyp stalk with current n be adequ for early bleeds

J: Joules; W: Watts

* Preinjection with epinephrine solution can be considered before endoscopic coagulation.

¶ Probe size, power settings, and coaptive pressure will necessarily vary depending on clinical experience and location of lesion. Repeated cautery to the same point increases risk of perforation.

Δ Large probe preferred if therapeutic endoscope used.

Adapted with permission from: Zuckerman G, Prakash C. Gastrointest Endosc 1999; 49:231.

Graphic 71455 Version 3.0

Contributor Disclosures

John R Saltzman, MD, FACP, FACG, FASGE, AGAF No relevant financial relationship(s) with ineligible companies to disclose. **Lawrence S Friedman, MD** Other Financial Interest: Elsevier [Gastroenterology]; McGraw-Hill [Gastroenterology]; Wiley [Gastroenterology]. All of the relevant financial relationships listed have been mitigated. **Anne C Travis, MD, MSc, FACG, AGAF** No relevant financial relationship(s) with ineligible companies to disclose.

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