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Angiodysplasia of the Colon

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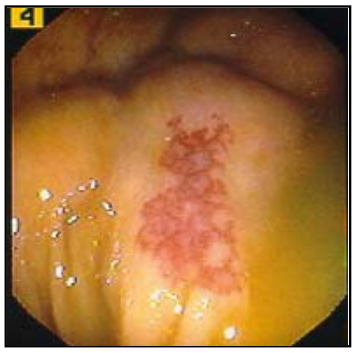
Introduction

Background

Angiodysplasia is the most common vascular lesion of the gastrointestinal tract, and this condition may be asymptomatic, or it may cause gastrointestinal (GI) bleeding.[1] The vessel walls are thin, with little or no smooth muscle, and the vessels are ectatic and thin (see image below).



Angiodysplasia identified on cecum wall during colonoscopy.

Phillips first described a vascular abnormality that caused bleeding from the large bowel in a letter to the *London Medical Gazette* in 1839. During the 1920s, neoplasms were considered the major source of GI hemorrhage. However, in the 1940s and 1950s, diverticular disease was recognized as an important source of bleeding. In 1951, Smith described active bleeding from a diverticulum visualized through a sigmoidoscope. An association between colonic angiodysplasia and aortic stenosis was described by Heyde in 1958.[2]

Vascular abnormalities as a source of active bleeding were controversial. In 1960, Margulis and colleagues identified a vascular malformation in the cecum of a 69-year-old woman who presented with massive bleeding.[3] This diagnosis was accomplished with operative mesenteric arteriography.

Galdabini first used the name angiodysplasia in 1974; however, confusion about the exact nature of these lesions resulted in a multitude of terms that included arteriovenous malformation, hemangioma, telangiectasia, and vascular ectasia. These terms have varying pathophysiologies, with a common presentation of GI bleeding.

Angiodysplasia is a degenerative lesion of previously healthy blood vessels found most commonly in the cecum and proximal ascending colon. Seventy-seven percent of angiodysplasias are located in the cecum and ascending colon, 15% are located in the jejunum and ileum, and the remainder is distributed throughout the alimentary tract. These lesions typically are nonpalpable and small (<5 mm).

Angiodysplasia is the most common vascular abnormality of the GI tract. After diverticulosis, it is the second leading cause of lower GI bleeding in patients older than 60 years. Angiodysplasia may account for approximately 6% of cases of lower GI bleeding. It may be observed incidentally at colonoscopy in as many as 0.8% of patients older than 50 years. The prevalence for upper GI lesions is

approximately 1-2%.

Small bowel angiodysplasia may account for 30-40% of cases of GI bleeding of obscure origin. Retrospective colonoscopic analyses have shown that 12.1% of 642 persons without symptoms of irritable bowel syndrome (IBS), and 11.9% of those with IBS had colonic angiodysplasia.[4]

Angiodysplasia may present as an isolated lesion or as multiple vascular lesions. Unlike congenital or neoplastic vascular lesions of the GI tract, this lesion is not associated with angiomatous lesions of the skin or other viscera.

Clinical presentation in patients with angiodysplasia is usually characterized by maroon-colored stool, melena, or hematochezia. Bleeding is usually low grade, but it can be massive in approximately 15% of patients. In 20-25% of bleeding episodes, only tarry stools are passed. Iron deficiency anemia and stools that are intermittently positive for occult blood can be the only manifestations of angiodysplasia in 10-15% of patients. Bleeding stops spontaneously in greater than 90% of cases but is often recurrent.

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Pathophysiology

The exact mechanism of development of angiodysplasia is not known, but chronic venous obstruction may play a role.[5,6]This hypothesis accounts for the high prevalence of these lesions in the right colon and is based on the Laplace law. The Laplace law relates wall tension to luminal size and transmural pressure difference in a cylinder, whereby the wall tension is equal to the pressure difference multiplied by the radius of the cylinder. In the case of the colon, wall tension refers to intramural tension, the pressure difference is that between the bowel lumen and the peritoneal cavity, and cylinder radius is the radius of the right colon. Wall tension is highest in bowel segments with the greatest diameter, such as the right colon.

This theory involving chronic venous obstruction suggests that repeated episodes of colonic distention are associated with transient increases in lumen pressure and size., which results in multiple episodes of increasing wall tension with obstruction of submucosal venous outflow, especially where these vessels pierce the smooth muscle layers of the colon. Over many years, this process causes gradual dilation of the submucosal veins and, eventually, dilation of the venules and arteriolar capillary units feeding them. Ultimately, the capillary rings dilate, the precapillary sphincters lose their competency, and a small arteriovenous communication forms. This accounts for the characteristic early-filling vein observed during mesenteric angiography.

The developmental theory of angiodysplasia accounts for several clinical and pathologic features, including occurrence in older individuals, location in the cecum and proximal right colon, and prominent submucosal veins that dilate after traversing the muscularis propria. In addition, it also accounts for the lack of pathologic changes in arterioles supplying vascular ectasias and the absence of any mucosal lesion associated with them. Previous studies demonstrating that colonic motility, increased tension in the bowel wall, and increased intraluminal pressure can diminish venous flow lend further support to this theory. Dilated submucosal veins have been one of the most consistent histologic findings and may represent the earliest abnormality in colonic angiodysplasia. This histologic feature supports the theory of chronic venous obstruction in the genesis of angiodysplasia.

Of note, the aforementioned pathophysiologic mechanisms responsible for the development of cecal lesions are unlikely to apply to lesions in the upper GI tract, despite being morphologically identical.

A link between a deficiency of high molecular-weight multimers of von Willebrand factor, aortic stenosis, and colonic angiodysplasia has been proposed.

Frequency

There may be an association between colonic angiodysplasia and true diverticula.[7] Portal hypertension colopathy, a form of colonic angiodysplasia, has been described.[8]Vascular ectasia of the entire GI tract has been reported in a patient receiving high-dose chemotherapy and autologous stem cell transplantation for relapsing Hodgkin disease.[9]

United States

The prevalence of angiodysplasia is 0.8% in healthy patients older than 50 years who are undergoing screening colonoscopy.

Foutch et al noted the prevalence of angiodysplasia to be 0.83% from 3 prospective studies in which screening colonoscopies were performed in 964 asymptomatic individuals (mean age, 62 y).[10]

Patients with von Willebrand disease may have an increased incidence of GI bleeding from colonic angiodysplasia.[11,12,13,14,15,16,17]

International

No widespread studies to determine the international incidence of angiodysplasia have been conducted, but the incidence probably is similar to that in the United States.

Mortality/Morbidity

- Bleeding from angiodysplasia is usually self-limited, but it can be chronic, recurrent, or even acute and life threatening.
- Approximately 90% of bleeding angiodysplasias spontaneously cease bleeding, presumably because of its venous nature.
- Mortality is related to the severity of bleeding, hemodynamic instability, age, and the presence of comorbid medical conditions.

Race

No racial predilection exists in cases of angiodysplasia of the colon.

Sex

Angiodysplasia of the colon occurs with equal frequency in men and women.

Age

Most patients found to have angiodysplasia are older than 60 years; of these patients, most are older than 70 years. However, case reports exist of occurrence in young people.

Clinical

History

Many patients with angiodysplasia are asymptomatic, and the lesions are incidentally found, such as with screening colonoscopy. Clinical presentation and physical examination are related to GI bleeding or its consequences.

- The estimated incidence of active GI bleed in patients with angiodysplasia is less than 10%. However, because these lesions may be located throughout the GI tract and because the rate of bleeding may be variable, the clinical presentation ranges from hematemesis or hematochezia to occult anemia. Bleeding is usually chronic or recurrent and, in most cases, low grade and painless because of the venous source.
- Angiodysplastic lesions are often present in more than one location within the GI tract, and the presentation may vary during a patient's clinical course.
    - GI bleeding from small bowel lesions has occurred in as many as 22% of patients in whom angiodysplasia of the colon was the presumed index source of bleeding.
    - In 40-60% of patients with gastric and duodenal angiodysplasia, multiple lesions are observed at endoscopy. Colonic lesions will be associated in 15-20% of these patients. In addition, angiodysplastic lesions in the colon are more frequently multiple than single. To diagnose and treat patients with suspected angiodysplasia, the diffuse location of lesions and the propensity for multiplicity must be considered. A possible association of true colonic diverticula and angiodysplasia has been proposed and should be kept in mind.
- Hematemesis is frequently observed in patients with angiodysplasia of the upper GI tract. Presentation with hemodynamically well-compensated, chronic bleeding is typical and often suggests the diagnosis. Patients with upper tract lesions may have had bleeding from days to years.
- Bleeding from colonic lesions is most often chronic and low grade, but as many as 15% of patients present with acute massive hemorrhage. Patients with colonic angiodysplasia may present with hematochezia (0-60%), melena (0-26%), hemoccult positive stool (4-47%), or iron deficiency anemia (0-51%).

- Melena occurs in at least one fourth of patients with colonic bleeding.
- Spontaneous cessation of bleeding (occurring in 90% of patients) is the rule for angiodysplastic lesions located in any part of the GI tract.

Physical

Physical examination in a patient suspected of having angiodysplasia should include assessment of their hemodynamic stability and the likely origin of blood loss.

- Extracolonic angiodysplasias occur in 17% of persons with colonic lesions.[18]
- Vital signs may demonstrate tachycardia, hypotension, and postural changes based on the amount of blood loss.
- Stool is typically guaiac positive. Because bleeding may be intermittent, alternating positive and negative guaiac stools can be found.
- In most cases, bleeding presents as bright red blood, but it can also be maroon in color or melena.
- A microcytic hypochromic anemia, reflecting iron deficiency, is observed in 10-15% of cases.
- Hemodynamic instability may occur if bleeding is massive. This is observed in 15% of cases.

Causes

The exact cause of angiodysplasia is unknown, but theories include the following:

- Degenerative changes of small blood vessels associated with aging (most widely accepted theory)
- Long-term local hypo-oxygenation of the microcirculation from cardiac, vascular, or pulmonary disease
  - Angiodysplasia has been reported to be associated with aortic stenosis. Heyde first reported this association in 1958, describing Heyde syndrome as the combination of calcific aortic stenosis and GI bleeding due to angiodysplasia of the colon.[2]He reported on 10 patients with GI bleeding of unknown origin who had clinical signs of aortic stenosis and speculated that these patients bled from sclerotic GI vessels.[2]One month later, Schwartz et al suggested a similar association.[19]
  - Catell was quoted in a clinicopathologic conference on such a case in 1965. He suggested that these patients bled from a vascular lesion in the ascending colon that the pathologists could not demonstrate. Catell recommended a blind right hemicolectomy, which, in his experience, had resulted in cessation of bleeding in these patients.
  - Mucosal hypoperfusion from cardiac disease was later postulated to be the underlying cause for development of angiodysplasia. Studies using echocardiograms indicated that only a few patients with angiodysplastic lesions had significant valvular heart disease, such as aortic stenosis. More patients had aortic sclerosis than aortic stenosis. Aortic valve replacement or colectomy may be effective in the cessation of recurrent bleeding or after correction of heart failure in hypertrophic subaortic stenosis.[20]
  - In most persons with angiodysplasia, cardiac findings have no importance in the development of angiodysplasia, although in Japan the most prevalent underlying condition in patients with colonic angiodysplasia was cardiovascular disease (56%).[21]Critiques of the literature by Imperiale and Ransohoff found a lack of conclusive evidence to support the association of aortic stenosis, angiodysplasia, and GI bleeding[22]
  - Hypoperfusion or hypo-oxygenation from cardiac or pulmonary disease possibly results in ischemic necrosis of an existing angiodysplastic lesion. The observation that low cardiac output usually is a late occurrence in the course of aortic valve disease has not supported this possibility. In addition, the low cardiac output associated with mitral stenosis is not associated with a propensity for bleeding in angiodysplastic lesions.
  - Cessation of angiodysplastic bleeding after aortic valve replacement in severe aortic stenosis
- Pate et al suggested that Heyde syndrome consists of bleeding from presumably latent angiodysplasia as a result of a hematologic defect, such as a lack of high molecular weight von willebrand factor multimers.[20]

- Bleeding angiodysplastic lesions in the upper GI tract have been found with a high prevalence in patients with chronic renal failure requiring dialysis.[23] However, this has not been a consistent finding. Patients with chronic renal failure are more likely to have coagulopathies that are related to quantitative and qualitative platelet defects and abnormal function and structure of von Willebrand factor.
- Bleeding from angiodysplastic lesions in the upper and lower GI tract has been reported in patients with von Willebrand disease. Because factor VIII complex is synthesized partly in vascular endothelial cells, patients with von Willebrand disease and angiodysplasia have been proposed to have an underlying endothelial defect that may be related to the subsequent development of the 2 disorders. However, as with renal failure, the coagulopathy is more likely responsible for bleeding than for the development of the lesions.
- Roskell et al demonstrated a relative deficiency of collagen type IV in the mucosal vessels in angiodysplasia compared to controls.[24] The authors proposed that this deficiency may be related to the patients' susceptibility to ectasia and hemorrhage.
- In a small study, Junquera et al observed an increased expression of angiogenic factors in human colonic angiodysplasia.[25] This study noted that vascular immunoreactivity for basic fibroblast growth factor was observed in 7 (39%) specimens from patients with colonic angiodysplasia, whereas either very limited or no immunostaining was found in sections from specimens of patients with colonic cancer and healthy margins.
- Patients with scleroderma may also have a higher incidence of angiodysplasia throughout the GI tract, including the colon.

Differential Diagnoses

Colon Cancer, Adenocarcinoma

Colonic Polyps

Diverticulitis

Hemorrhoids

Metastatic Cancer, Unknown Primary Site

Portal Hypertension

Rectal Cancer

Other Problems to Be Considered

- Anorectal fissures
- Diverticulosis
- Enterocolitis (ischemic, infectious, inflammatory bowel, radiation-induced)

Workup

Laboratory Studies

- Complete blood cell (CBC) count: Approximately 10% of patients who bleed from angiodysplasia present with anemia.
- Serum iron level: Iron deficiency is found in 10% of patients with bleeding angiodysplasia.
- Stool for occult blood: As many as 15% of patients with bleeding angiodysplasia will be intermittently positive for occult blood.
- See the algorithm for acute GI bleeding below.
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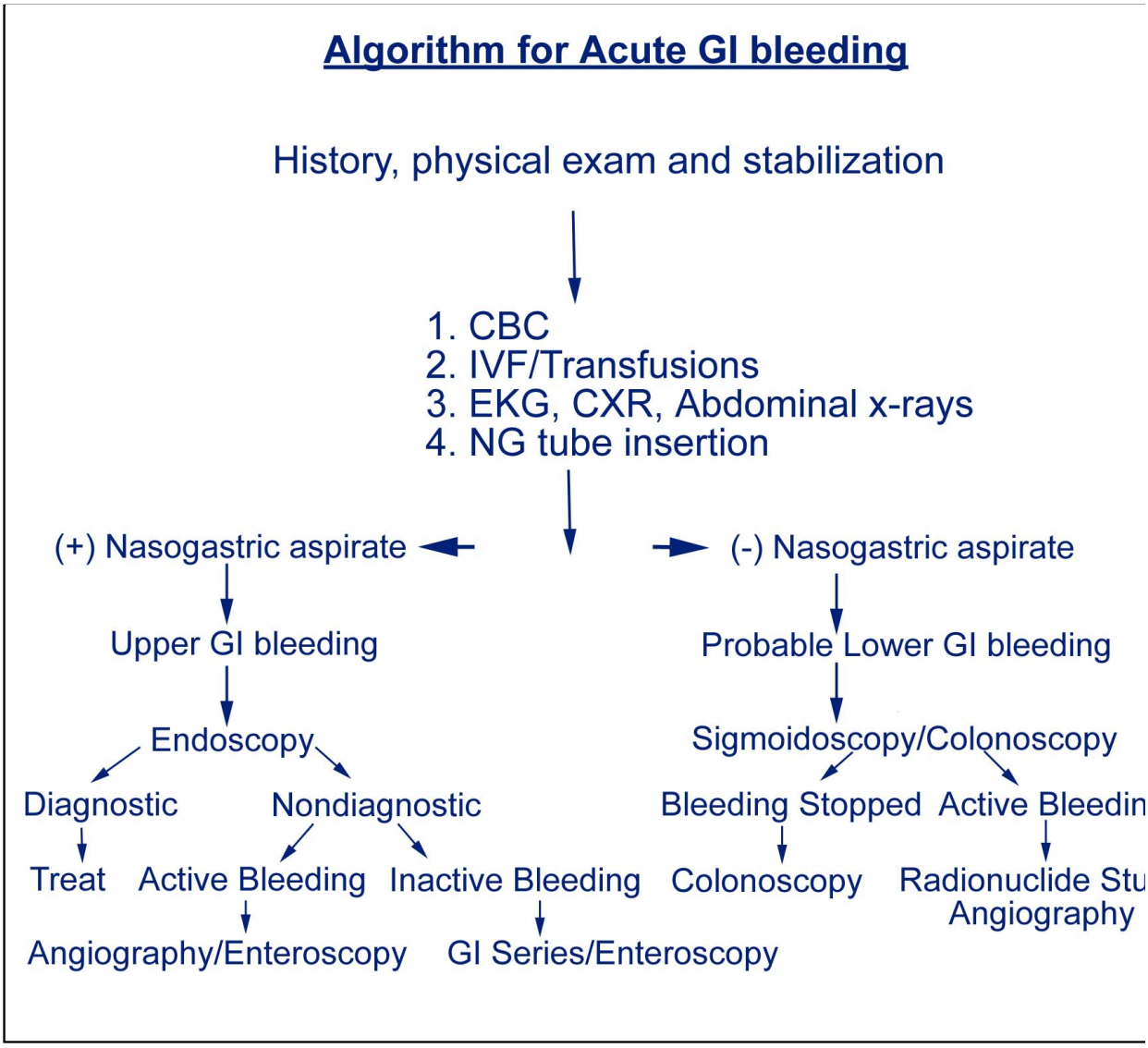
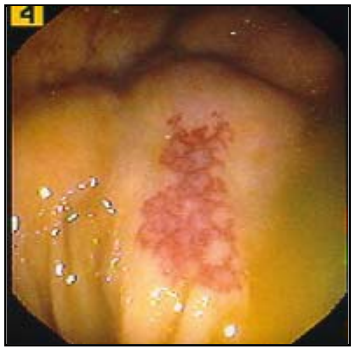


Algorithm for acute gastrointestinal (GI) bleeding. CBC = complete blood cell count; CXR = chest x-ray; EKG = electrocardiography; IVF = intravenous fluid; NG = nasogastric.

Imaging Studies

- Selective mesenteric angiography is a useful diagnostic technique for angiodysplasias, especially in patients with massive bleeding in whom a colonoscopic diagnosis is difficult.
  - The sensitivity of angiography ranges from 58% to 86%. Detection of bleeding depends on the rate of bleeding (as low as 0.5 mL/min), technique, and timing of angiography in relation to the period of bleeding.
  - The above 3 angiographic signs correspond to the development of ectatic changes in vascular lesions. Their prevalence has been recorded less systematically in other parts of the intestinal tract. However, the interpreter must consider all clinical information, because these angiographic findings may be observed in other disorders, such as malignancy.
    - The most frequent and earliest angiographic sign is a densely opacified, dilated, and slowly emptying draining vein within the intestinal wall. This vein is detected during the venous phase of the study and is present in more than 90% of angiodysplastic lesions.
    - As the lesion progresses, a vascular tuft may become apparent during the arterial phase of the study. This is

observed in as many as 70-80% of patients with angiodysplasia. It represents an extension of the dilation process to the mucosal venules.
- The latest sign, an early-filling vein, may be observed in the arterial phase, indicating a more developed arteriovenous communication through the angiodysplastic lesion. It is observed in only 60-70% of cases of angiodysplasia.
- Demonstration of extravasation of contrast dye in the bowel lumen from angiodysplastic lesions is definite evidence of bleeding; however, this is observed in only 6-20% of patients. This low percentage is attributed to the episodic nature of bleeding lesions.

- Angiography of resected specimens has been used to confirm appropriate resection when preoperative studies are equivocal or unsatisfactory. An intraluminal formalin fixation technique on the resected specimen, followed by mucosal dissection, has also been used for documenting correct resection. Vasopressin infusion during angiography can arrest bleeding, but potential complications of vasopressin include bowel infarction, arterial vasospasm, and lower extremity ischemia.
- Radionuclide scanning using technetium-99m (^{99m}Tc)–labeled red blood cells or ^{99m}Tc sulfur colloid is helpful in detecting and localizing active bleeding from angiodysplasia. Scanning can detect bleeding with rates as low as 0.1 mL/min. The intermittent bleeding nature of angiodysplasia has limited the utility of radionuclide studies in this disorder.
  - Red blood cells that are labeled with technetium have a long half-life in the intravascular compartment and are especially useful in patients with intermittent hemorrhage. The usefulness of technetium-labeled red blood cells is attributed to the ability to detect bleeding during the 12 or so hours after a single injection of radiolabeled cells.
  - The reticuloendothelial system rapidly clears ^{99m}Tc sulfur colloid. ^{99m}Tc sulfur colloid has a half-life of only 3 minutes; hence, it is helpful only in patients with active bleeding. The extravasated labeled sulfur colloid easily demonstrates the site of bleeding in the absence of confusing background activity in the circulation, although uptake into the liver and spleen restricts the image area.
  - Nuclear scans lack the specificity of an angiogram in differentiating the nature of bleeding lesions, despite the fact that they are noninvasive and relatively easy to perform. Nuclear scans have proven more useful as an adjunct to angiography by localizing and confirming the presence of bleeding, minimizing the number of angiograms that do not yield meaningful diagnostic information, and allowing rapid selection of the artery to be injected by angiography. Confirm positive findings from a radionuclide study by colonoscopy or angiography if surgical resection is contemplated (see image below).
  - 

    Angiodysplasia identified on cecum wall during colonoscopy.

- Helical computed tomography (CT) angiography can detect extravasation from angiodysplasia and is potentially an important noninvasive test in patients with obscure bleeding sites.
- Capsule endoscopy has been reported to detect cecal angiodysplasias in selected cases. Capsule endoscopy is particularly useful to demonstrate small intestinal lesions, but its role as a diagnostic test for the colon is still experimental.[26]
- Double-balloon endoscopy is useful to detect small bowel lesions, and retrograde double-balloon endoscopy may allow for careful inspection of the cecum and ileocecal valve.[27,28]

Other Tests

- Air contrast barium enema is not recommended during acute GI bleeding. Air contrast barium enemas can help exclude other causes of chronic colonic bleeding; however, they are not useful in detecting angiodysplasia, because the lesions are small and usually do not distort the colonic mucosa. In addition, barium enema can obscure other diagnostic studies.

Procedures

- Endoscopy is the most common method of diagnosing angiodysplasia in both the upper and lower GI tract.
  - Upper endoscopy is used to establish a diagnosis of gastric and duodenal angiodysplasia. Celiac artery and superior mesenteric artery arteriograms frequently fail to demonstrate these lesions, although large lesions with well-formed and enlarged draining veins have been reported in the gastric antrum. Angiography can demonstrate lesions in the more distal small intestine, a region less accessible to endoscopic evaluation. Push enteroscopy has proven to be a successful method of identifying angiodysplasia of the proximal small intestine in patients with obscure bleeding. Other methods of visually evaluating the small bowel are double-balloon enteroscopy, which is still investigational, and capsule enteroscopy, which is currently used in clinical practice.
  - The endoscopic appearance of gastric lesions typically has been described as discrete, flat, or slightly raised (2-10 mm) and bright red. These lesions have fernlike margins or stellate configurations. Proximal small intestinal lesions are the size of a pinpoint, with a similar gross appearance. A surrounding pale rim or halo also characterizes upper tract lesions.
  - Either angiography or colonoscopy may be used to detect colonic lesions. Because colonoscopy is a principal method in the evaluation of GI bleeding, diagnosis of these lesions often results from colonoscopic examination.
  - Comparative studies using selective angiography and colonoscopy indicate that the sensitivity of colonoscopy exceeds 80% when the lesions are located in the area examined by colonoscopy. Most angiodysplastic lesions are located in the right colon, thus, the entire colon must be examined. Angiography has the advantage of detecting additional angiodysplastic lesions not depicted by colonoscopy. In a series by Emanuel et al, 17% of subjects were found to have concomitant colonic and extracolonic angiodysplasia when studied by triple-vessel angiography.[18] Angiography and colonoscopy can play important complementary roles.
  - Skibba et al first described the colonoscopic appearance of angiodysplasia in 1976.[29] Angiodysplastic lesions are often described as discrete and small, with scalloped or frondlike edges and a visible draining vein. They can be flat or slightly raised and can be hidden within mucosal folds. Although angiodysplasia may be detected anywhere in the colon, a strong propensity exists for the cecum and ascending colon.
  - Angiodysplastic lesions encountered as incidental findings are generally small lesions with pale coloration compared with lesions with recent hemorrhage, which are described as extremely bright with elevated centers.
  - The endoscopic appearance of angiodysplasia can be confused with the ectasias associated with systemic diseases, such as hereditary hemorrhagic telangiectasia (HHT), Turner syndrome, Ehlers-Danlos syndrome, blue rubber web nevus syndrome, the CREST variant syndrome (calcinosis, Raynaud phenomenon, esophageal hypomotility, sclerodactyly, and telangiectasia) of scleroderma, gastric antral vascular ectasia (GAVE), portal hypertensive colopathy, and radiation-induced injury. The lack of systemic manifestations distinguishes angiodysplasia from these syndromes.
  - In addition, the endoscopic appearance of angiodysplasia may be difficult to discern from spider angiomata, radiation injury, ulcerative colitis (see image below), Crohn disease (see image below), ischemic colitis, and suction artifacts.
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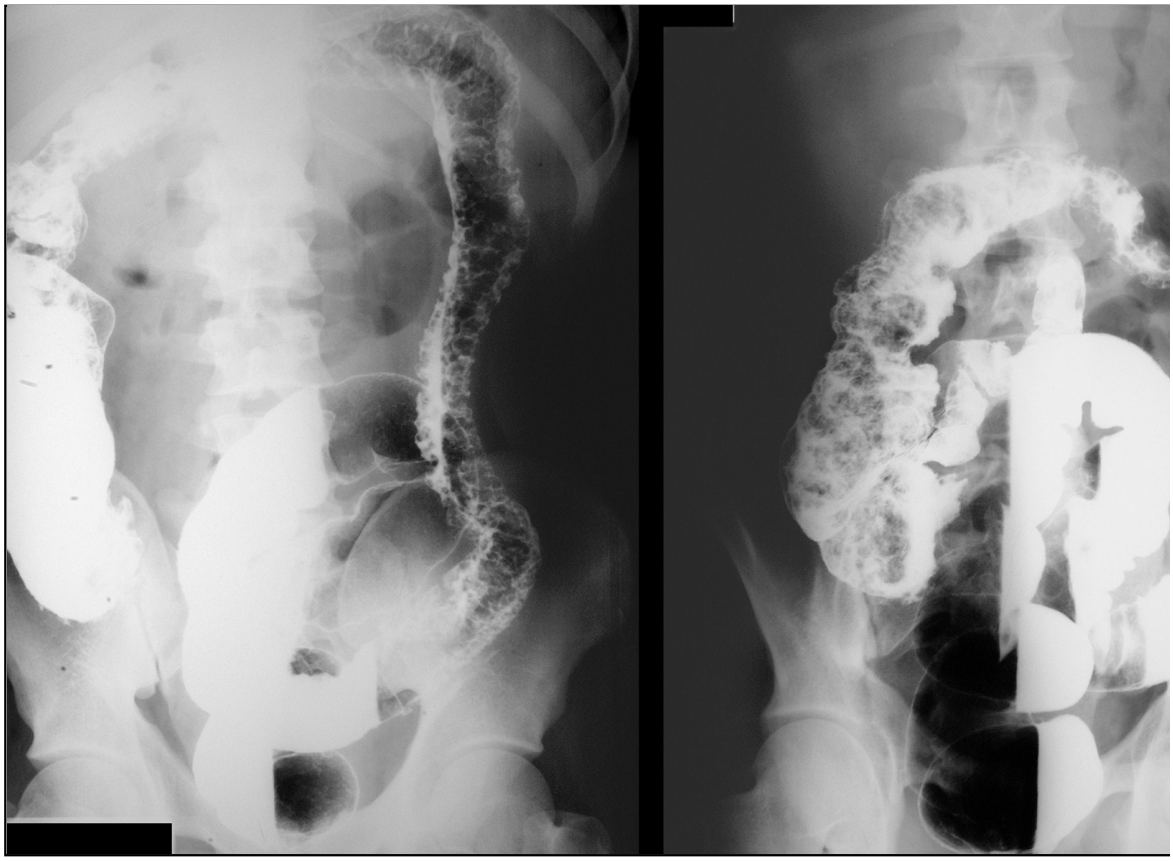
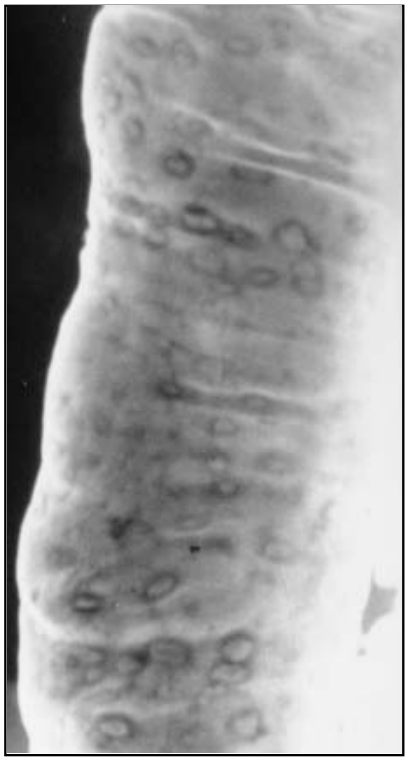


Double-contrast barium enema studies in a 44-year-old man known to have a long history of ulcerative colitis. These images show total colitis and extensive pseudopolyposis.



Double-contrast barium enema examination in a patient with Crohn colitis demonstrates numerous aphthous ulcers.

- Evaluation of a patient who may have angiodysplasia requires that the colonoscope be inserted carefully, with minimal use of suction, and that the mucosa be examined more carefully as the instrument is advanced than during withdrawal. Obtaining a biopsy of suspicious lesions may be necessary if classic features are not present.
- Because blood pressure and volume influence the colonoscopic appearance of vascular lesions, angiodysplasia may not be evident in patients who have bled recently. Accurate evaluation of the colon may not be possible until effective fluid resuscitation and blood transfusions.
- Some studies have noted that administering meperidine may diminish the prominence of some vascular abnormalities, and some researchers have advocated reversal by naloxone to aid in the accurate detection of these lesions.

- Angiodysplasia can be detected infrequently by visual inspection of the serosal side of the bowel during laparotomy. Richardson et al made a correct diagnosis of angiodysplasia during surgical exploration in only 1 of 39 cases (2.6%).[30] The remaining patients were diagnosed by angiography or colonoscopy. Fourteen of these individuals had undergone 21 nondiagnostic surgeries before their evaluation.
- Intraoperative enteroscopy can help in localization of distal small bowel lesions. In addition, an angiographic catheter can be placed before surgery into the appropriate feeding vessel supplying the angiodysplastic lesion. The surgeon then can identify the catheter during surgery and explore and resect the appropriate small bowel segment.

Histologic Findings

Endoscopic forceps biopsy has revealed characteristic histopathologic features of angiodysplasia in only 31-60% of specimens. Endoscopic mucosal biopsies for purposes of diagnosis are generally not recommended because of the low diagnostic yield and the risk of provoking hemorrhage.
However, the histologic diagnosis of angiodysplasia is difficult. Acquired lesions such as angiodysplasias must be differentiated from vascular tumors, lesions associated with congenital or systemic disease, or radiation damage.

Angiodysplasias typically are irregularly shaped clusters of ectatic small arteries, small veins, and their capillary connections. They are more often multiple than single. Microscopically, angiodysplastic lesions are dilated, distorted, thin-walled vessels. The amount of

smooth muscle in the vessel wall is variable. The vessel wall can become so thinned that it appears to be composed only of endothelium.

Markedly dilated submucosal vasculature is the most consistent abnormality and the earliest change identified. More advanced lesions involve the mucosa. Because the major portion of the lesion is often submucosal, endoscopic mucosal biopsies are often not diagnostic. Characteristic histopathologic findings of angiodysplasia are identified only in 31-60% of endoscopically obtained biopsies.

In addition, routine pathologic examination usually discovers less than one third of lesions. Injecting the colonic vasculature with silicone rubber and clearing the specimen can be used to identify almost all lesions. In this process, the rubber compound is injected through a catheter placed in one or more of the arteries supplying the colon, after which the specimen is refrigerated for 24 hours to allow the silicone to polymerize. Specimens are then dehydrated in increasing concentrations of ethyl alcohol and cleared with methyl salicylate. The result is a transparent specimen with a filled vascular bed, which is studied through a dissecting microscope using direct light as well as transillumination.

Treatment

Medical Care

Medically manage each patient with angiodysplasia in accordance with the severity of bleeding, hemodynamic stability, and recurrence of symptoms. A conservative approach to patients who are hemodynamically stable is recommended, because most bleeding angiodysplasias will cease spontaneously. Treatment is usually not advocated for asymptomatic patients when angiodysplasias are found incidentally.

Initially, hemodynamically stabilize all patients with active bleeding with intravenous fluid and packed red blood cells as needed. In addition, correct coagulopathies.

When intervention is warranted, institute steps to control hemorrhage. Endoscopic techniques have been employed most frequently.

Gastric and duodenal angiodysplastic lesions have been managed most commonly with endoscopic obliteration techniques. Rebleeding after these techniques has been attributed to other areas of bleeding angiodysplasia rather then failure of obliteration. These techniques include monopolar electrocautery, heater probe, sclerotherapy, band ligation, and argon and neodymium:yttrium-aluminum-garnet (Nd:YAG) lasers.

- Monopolar electrocautery has been used to obliterate angiodysplasia; however, bleeding recurs in approximately 50% of subjects. A reduction in the posttherapy transfusion requirement was not reported to be statistically superior to no therapy. Heater probe or multipolar coagulation devices have more favorable results. Monopolar electrocautery has a higher risk of perforation.
- Sclerotherapy using 0.5-1 mL of 1.5% sodium tetradecyl sulfate has been used to obliterate upper tract angiodysplastic lesions; however, bleeding recurs in half of the subjects. In each case, bleeding arose from another area of angiodysplasia. A significant rate of complications has been reported with sclerotherapy, including perforation, embolism, bacteremia, and stricture. The authors do not recommend the use of sclerotherapy for obliteration of colonic angiodysplasia.
- Argon plasma coagulation (APC) and Nd:YAG lasers are the most successful endoscopic obliterative techniques for upper tract lesions. APC is a no-touch electrocoagulation technique in which high-frequency alternating current is delivered to the tissue through ionized argon gas. A reduction in both the bleeding rate and transfusion requirement has been demonstrated for at least 12 months after laser therapy. However, active bleeding decreases the ablative efficacy of APC by dissipation of the energy, and APC has been associated with colonic perforation.
- Submucosal injection of a saline epinephrine solution followed by the application of APC has been reported.[31]Effectiveness appeared to be reduced in patients with more numerous lesions, those with coagulation disorders, and those who are older. Rebleeding commonly occurred over time.[31]
- New endoscopic techniques such as the Olympus EVIS LUCERA variable indices of hemoglobin chart function have been developed to assess completeness of vascular mucosal ablation.[32]However, their clinical use is still experimental. Argon coagulation appears the best endoscopic option at the moment to control bleeding in these patients with a low rate of adverse effects and complications and relatively lower costs.
- Fifty percent of patients with distal small bowel lesions and no other defined GI bleeding sites have benefited from

enteroscopy and lesion obliteration. In one report, blood replacement requirements for a group of 13 patients decreased by more than 50%, comparing the years before and after endoscopic treatment, and 31% required no further transfusion.[33] This group of patients had small bowel angiodysplastic lesions and unexplained bleeding. New endoscopic techniques to examine the small bowel, such as double-balloon enteroscopy, have been developed but are still time demanding and operator dependent.

- Angiodysplasia of colonic origin has been managed by endoscopic obliteration. Heater probe and multipolar electrocoagulation probe have been more successful than monopolar electrocoagulation. Rebleeding rates for monopolar electrocoagulation have been approximately 50%, with the transfusion requirement resembling that of patients receiving no therapy.
- Super selective embolization of visceral arterial branches is central to the management of patients with lower GI bleeding, including from colonic angiodysplasia.[34] Immediate cessation of bleeding was achieved in 97% of patients with injection of microcoils, polyvinyl alcohol particles, gel form, or by selective vasopressin infusion. Postembolization ischemia occurs in 3%, and overall mortality in high-risk patients is 9%.[34] Selective infusion of vasopressin is less effective than embolization as a definitive therapy because of a high rebleeding rate. Despite the fact that intra-arterial vasopressin can achieve hemostasis for massive lower GI bleeding in 70-91% of patients, bleeding recurs after discontinuation of vasopressin in 22-71% of patients.
    - Endoscopic laser photocoagulation has been successful in controlling bleeding from colonic angiodysplasia, especially right-sided lesions.[31,35] However, complications occur in as many as 15% of patients and are more common when the Nd:YAG laser is used in the right colon. Complications may be attributed to the deeper coagulation of the vascular abnormalities from laser sources, which incidentally has been responsible for more effective bleeding cessation. Patients with colonic angiodysplasia generally have a 60% chance of remaining free of bleeding at 24 months after laser obliteration.
    - Endoclips have been used in anecdotal case reports for bleeding angiodysplasia of the cecum and right colon.[34]
    - Angiodysplasia that presents with acute hemorrhage can be controlled effectively with angiography, although it is seldom needed. Angiography is appropriate in severely ill patients who are not candidates for surgical intervention. In these patients, transcatheter embolization of selected mesenteric arteries has been quite effective. However, the rate of complications is sufficiently high and must be balanced against the risk of surgical resection.
    - Angiography plays a more important role in preoperative localization of small bowel lesions immediately before surgical resection, because intraoperative palpation, endoscopy, and visual inspection through multiple enterotomies are of little value with angiodysplasia.
    - Injection of dyes, such as methylene blue, indigo carmine, and fluorescein, has been used to assist in localization of angiodysplasia before surgical resection.

Surgical Care

Surgical resection is the definitive treatment for angiodysplasia.

- Partial or complete gastrectomy for management of gastric angiodysplasia has been reported to be followed by bleeding in as many as 50% of patients. Rebleeding was attributed to other angiodysplastic lesions.
- Right hemicolectomy for angiodysplasia is second-line therapy after endoscopic ablation, if repeated endoscopic coagulation has failed, if endoscopic therapies are not available, and for life-threatening hemorrhage.
- The mortality rate associated with surgical resection ranges from 10% to 50%. This is based on the view that surgery carries a much higher risk in elderly patients, who often have multiple coexisting medical problems, including coronary artery disease, coagulopathy, and renal and pulmonary dysfunction.
    - In a study by Meyer et al, right hemicolectomy resulted in 63% of the subjects remaining free of intestinal bleeding (mean follow-up, 3.6 y), and 37% had some degree of recurrent bleeding.[36]
    - Trends toward reduced transfusion requirements have been observed after surgical resection, as well as after electrocoagulation as the only mode of therapy, and in patients who received no specific intervention.
    - Surgical resection is preferred for acute management of severe hemorrhage or for management of recurrent hemorrhage over a relatively short period accompanied by a large transfusion requirement.

Consultations

Consultation with both a gastroenterologist and a surgeon is recommended for cases of angiodysplasia. Interventional radiology often plays a critical role in the management of these patients.

Diet

Withhold oral intake until the diagnosis has been made and treatment has been initiated.

Activity

Restrict activity until hemodynamic stability can be maintained.

Medication

Medical treatment has been used in active and recurrent bleeding from colonic angiodysplasia with controversial results. Hormonal treatment with estrogen and progesterone has been evaluated by randomized trials but remains controversial and is probably not effective. Octreotide, both short and long acting, has been described as effective in a few case reports and case series only. Other agents, such as thalidomide, remain experimental. Desmopressin (DDAVP) has also been used in specific subsets of patients. At the moment, no medical therapy has been proven to effectively treat bleeding from angiodysplasia.

Oral Contraceptives

Only use hormonal therapy for the small subset of patients who are transfusion-dependent from bleeding angiodysplasia refractory to conservative and endoscopic therapy and who are poor surgical candidates. This therapy is not for routine management of bleeding angiodysplasia. No large-scale, randomized, double-blinded studies have demonstrated its effectiveness.

Estrogen-progesterone therapy, previously used to treat bleeding associated with HHT, also has been tried in patients with GI bleeding from angiodysplasia.

Proposed mechanisms by which hormonal therapy might affect bleeding include improvement in coagulation, alterations in microvascular circulation, and improvements in endothelial integrity.

Data from a double-blinded, crossover trial using 0.05 mg ethinyl estradiol and 1 mg norethisterone administered daily to 10 elderly patients with GI ectasia (6 of the patients had HHT) have indicated that the combination significantly reduced bleeding and transfusion requirements. Several other small series with anecdotal success have been described, but one must be skeptical.

A retrospective cohort study of 64 patients by Lewis et al refutes the benefits of hormonal therapy in angiodysplasia.[37] Thirty patients were administered 5-10 mg of norethynodrel with mestranol (0.075-0.15 mg) or with conjugated estrogens (0.625 mg), and the bleeding rates did not differ before and after therapy, and they did not differ from bleeding rates of historical controls or from patients who refused therapy.[37] Treatment adverse effects in this study included vaginal bleeding, fluid retention, and stroke (23% of the treated patients).

Overall, the current data do not support the use of hormonal therapy in patients with colonic angiodysplasia.

Ethinyl estradiol and norethindrone (Ovcon 50)

Suggested mechanisms by which hormonal therapy might affect bleeding include improvement in coagulation, alterations in the microvascular circulation, and improvements in endothelial integrity. One active tab contains ethinyl estradiol 0.05 mg and norethindrone 1 mg.

Dosing

Adult

1 tab PO qd

Pediatric

Not established

Interactions

May reduce the hypoprothrombinemic effects of anticoagulants; estrogen levels may be reduced with coadministration of barbiturates, rifampin, and other agents that induce hepatic microsomal enzymes; an increase in corticosteroid levels may occur when administered concurrently with ethinyl estradiol; use of ethinyl estradiol with hydantoins may cause spotting, breakthrough bleeding, and pregnancy; increase in fluid retention caused by estrogen intake may reduce seizure control

Contraindications

Documented hypersensitivity; thrombophlebitis; undiagnosed vaginal bleeding; pregnancy; estrogen-dependent neoplasia; severe hepatic disease; breast cancer

Precautions

Pregnancy

X - Contraindicated; benefit does not outweigh risk

Precautions

Cigarette smoking increases the risk of serious cardiovascular adverse effects; caution in patients with hepatic impairment, migraine, seizure disorders, cerebrovascular disorders, breast cancer, or thromboembolic disease; associated adverse effects include GI distress, breakthrough bleeding, breast tenderness, weight change, and contact lens intolerance; in males, adverse effects include gynecomastia and decreased libido

Somatostatin Analogues

Somatostatin analogues have been reported to decrease the rate of bleeding from intestinal angiodysplasia. In our experience, these agents are usually well tolerated and may decrease the rate of chronic bleeding. Octreotide should be the first choice in patients with portal hypertension.

Octreotide (Sandostatin)

Mechanism of action in this setting is not fully understood. Used in acute variceal bleeding and for recurrent bleeding after endoscopic therapy.
May reduce the transfusion requirement.

Dosing

Adult

100 mcg SC bid

Pediatric

Not established

Interactions

May reduce the effects of cyclosporine; patients on insulin, oral hypoglycemics, beta-blockers and calcium channel blockers may need dosage adjustments

Contraindications

Documented hypersensitivity

Precautions

Pregnancy

B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals

Precautions

Adverse effects primarily related to altered GI motility, and include nausea, abdominal pain, diarrhea, and increased incidence of gallstones and biliary sludge; because of alteration in counter-regulatory hormones, (insulin, glucagon and GH) hypo- or hyperglycemia may be seen; bradycardia, cardiac conduction abnormalities and arrhythmias have been reported; due to inhibition of TSH secretion, hypothyroidism may also occur; exercise caution in patients with renal impairment; cholelithiasis may occur

Follow-up

Further Inpatient Care

- Admit the patient with colonic angiodysplasia to the intensive care unit (ICU) if the patient is hemodynamically unstable. Monitor for recurrent bleeding and stabilization of the hematocrit. Transfuse as needed.

Further Outpatient Care

- The exact time frame for follow-up colonoscopy in patients with angiodysplasia is controversial. If the patient is asymptomatic, a repeat colonoscopy is not recommended. Outpatient monitoring of hemoglobin and repeated tests for occult blood can be performed. Patients with chronic GI bleeding may need repeated colonoscopies.

Deterrence/Prevention

- No preventive methods for angiodysplasia have been definitely identified at this time. Avoidance of nonsteroidal anti-inflammatory drugs (NSAIDs) is recommended in patients with chronic bleeding.

Complications

- Hemodynamic instability may result from massive bleeding.

Prognosis

- The prognosis in patients with angiodysplasia is favorable because most angiodysplasias spontaneously cease bleeding (90% of cases).
- Richter et al reviewed the clinical course of 101 patients with colonic angiodysplasia.[38]
    - The cases of 15 asymptomatic individuals who had never bled were followed for as long as 68 months (mean, 23 mo), and no patient experienced bleeding during this observation period.[38]Therefore, conservatively manage nonbleeding angiodysplasia that is discovered as an incidental finding.
    - Thirty-one patients with overt bleeding or anemia managed with blood transfusions alone had rebleeding rates at 1 year of 26% and 3 years of 46%. The high rate of rebleeding justifies treatment for angiodysplasia in symptomatic individuals.
- Rebleeding after hemicolectomies occurs in 5-30% of patients, which is much less than that of endoscopic techniques.

Patient Education

- If angiodysplasia is identified incidentally, most patients can be reassured because most remain asymptomatic.
- Preventive treatment with endoscopic obliteration should be decided on a patient-to-patient basis and should not be done routinely.

Miscellaneous

Medicolegal Pitfalls

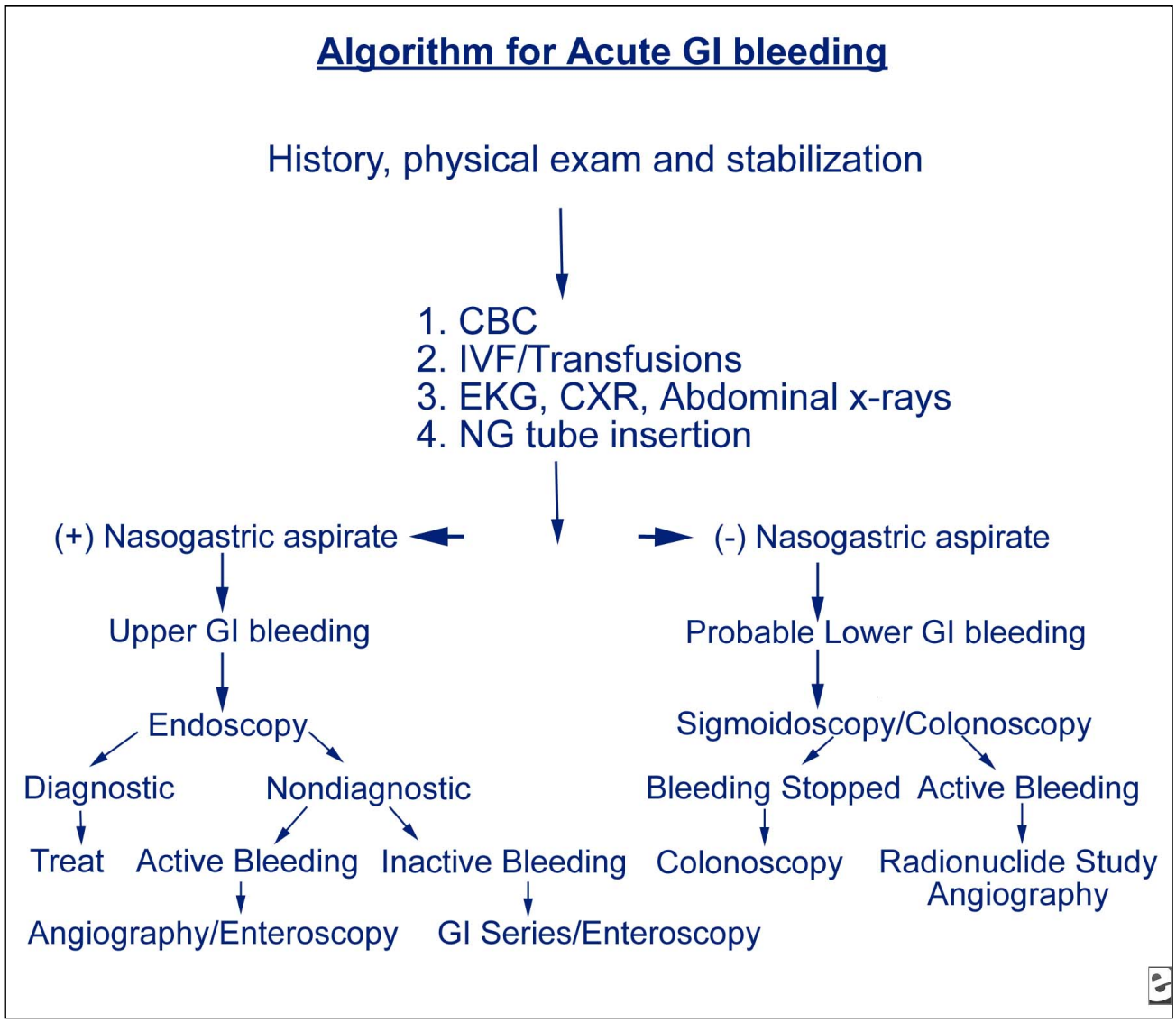
- No intervention is needed for nonbleeding angiodysplasia found incidentally, unless the patients will need to be anticoagulated.

- The presence of left-sided diverticular disease does not alter the extent of colonic resection if the angiogram demonstrates right-sided angiodysplasia with or without extravasation, because as many as 80% of bleeding diverticula are known to occur on the right side of the colon. The risk of the left colon becoming a source of future bleeding if left behind is relatively low compared with the increased morbidity and mortality of subtotal colectomy.

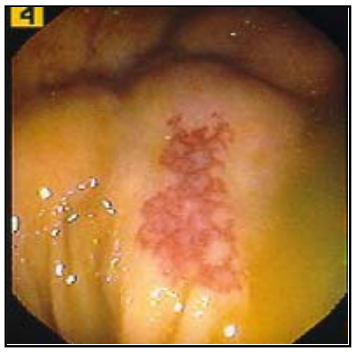
Special Concerns

- Obliteration of angiodysplasia in the cecum should be done with extreme care due to a higher risk of perforation when compared with lesions in the left colon.

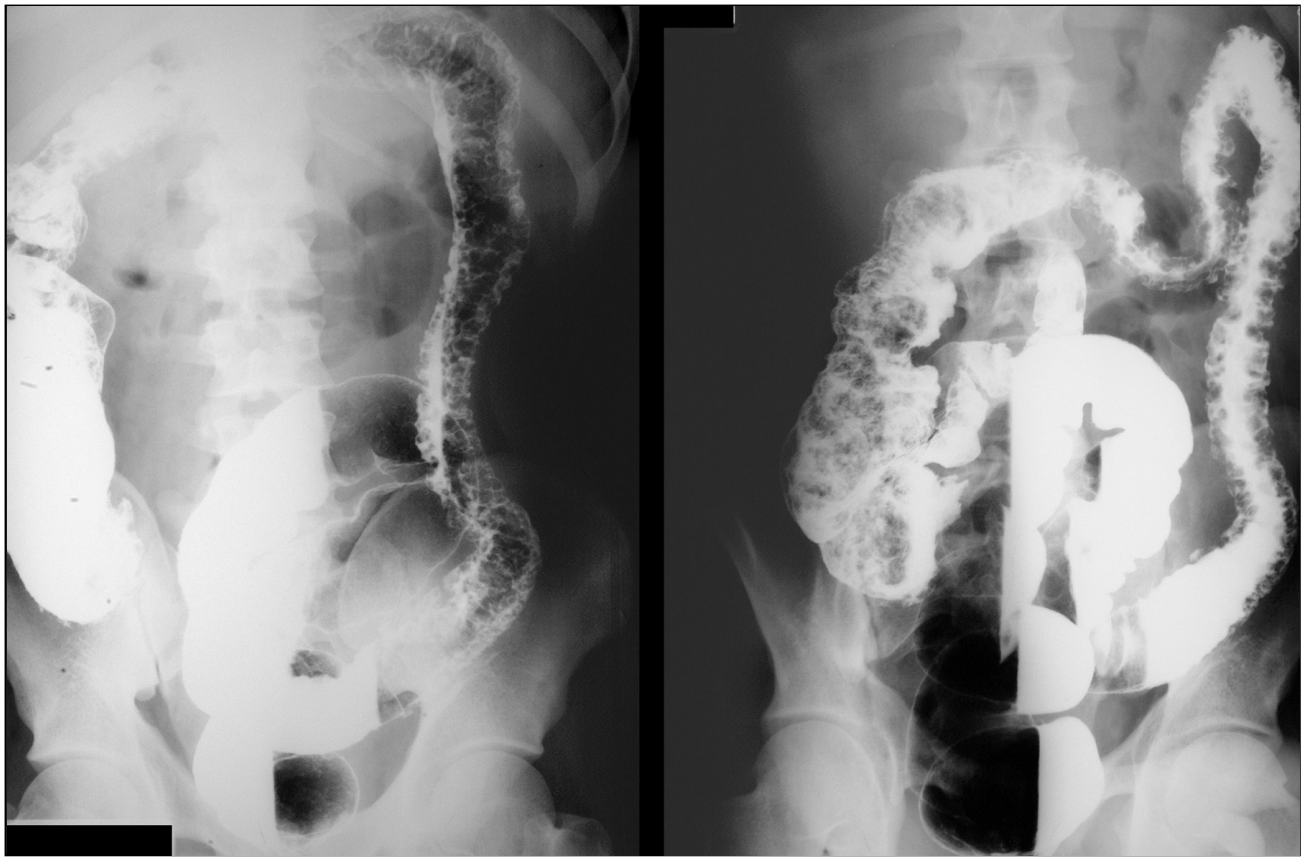
Multimedia



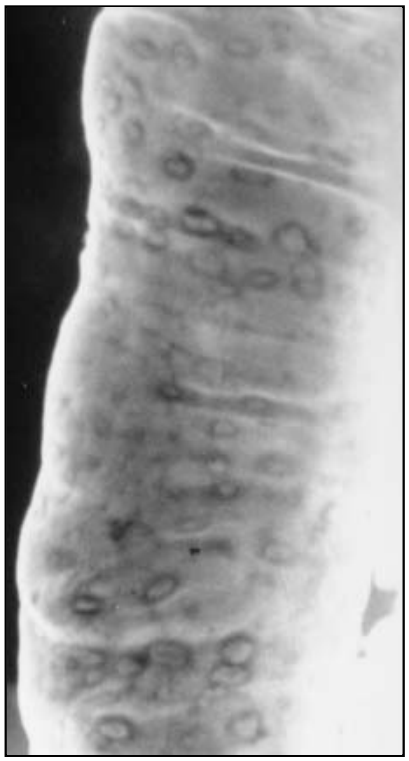
Media file 1: Algorithm for acute gastrointestinal (GI) bleeding. CBC = complete blood cell count; CXR = chest x-ray; EKG = electrocardiography; IVF = intravenous fluid; NG = nasogastric.



Media file 2: Angiodysplasia identified on cecum wall during colonoscopy.



Media file 3: Double-contrast barium enema studies in a 44-year-old man known to have a long history of ulcerative colitis. These images show total colitis and extensive pseudopolyposis.



Media file 4: Double-contrast barium enema examination in a patient with Crohn colitis demonstrates numerous aphthous ulcers.

References

1. Regula J, Wronska E, Pachlewski J. Vascular lesions of the gastrointestinal tract. *Best Pract Res Clin Gastroenterol*. 2008;22(2):313-28. [Medline].

2. Heyde EC. Gastrointestinal bleeding in aortic stenosis. *N Engl J Med*. 1958;259:196.

3. Margulis AR, Heinbecker P, Bernard HR. Operative mesenteric arteriography in the search for the site of bleeding in unexplained gastrointestinal hemorrhage: a preliminary report. *Surgery*. Sep 1960;48:534-9. [Medline].

4. Akhtar AJ, Shaheen MA, Zha J. Organic colonic lesions in patients with irritable bowel syndrome (IBS). *Med Sci Monit*. Sep 2006;12(9):CR363-7. [Medline].

5. Clouse RE. Vascular lesions: ectasias, tumors, and malformations. In: Yamada T, Alpers DH, Laine L, Owyang C, Powell DW, eds. *Textbook of Gastroenterology*. Vol 2. 3rd ed. Philadelphia, Pa: JB Lippincott; 1999:2564-78.

6. Ming SC, Goldman H. Vascular abnormalities of the gastrointestinal tract. In: Ming SC, Goldman H, eds. *Pathology of the Gastrointestinal Tract*. 2nd ed. Baltimore, Md: Williams & Wilkins Company; 1998:272-4.

7. Mudhar HS, Balsitis M. Colonic angiodysplasia and true diverticula: is there an association?. *Histopathology*. Jan 2005;46(1):81-8. [Medline].

8. Biss T, Hamilton P. Myelofibrosis and angiodysplasia of the colon: another manifestation of portal hypertension and massive splenomegaly?. *J Clin Pathol*. Sep 2004;57(9):999-1000. [Medline]. [Full Text].

9. Schmidmaier R, Bittmann I, Gotzberger M, et al. Vascular ectasia of the whole intestine as a cause of recurrent gastrointestinal bleeding after high-dose chemotherapy. *Endoscopy*. Sep 2006;38(9):940-2. [Medline].

10. Foutch PG, Rex DK, Lieberman DA. Prevalence and natural history of colonic angiodysplasia among healthy asymptomatic people. *Am J Gastroenterol*. Apr 1995;90(4):564-7. [Medline].

11. Rosborough TK, Swaim WR. Acquired von Willebrand's disease, platelet-release defect and angiodysplasia. *Am J Med*. Jul 1978;65(1):96-100. [Medline].

12. Cass AJ, Bliss BP, Bolton RP, Cooper BT. Gastrointestinal bleeding, angiodysplasia of the colon and acquired von Willebrand's disease. *Br J Surg*. Sep 1980;67(9):639-41. [Medline].

13. Duray PH, Marcal JM Jr, LiVolsi VA, et al. Gastrointestinal angiodysplasia: a possible component of von Willebrand's disease. *Hum Pathol*. Jun 1984;15(6):539-44. [Medline].

14. Gostout CJ, Bowie EJW, Balm R, Fischer PK. Is angiodysplasia associated with von Willebrand's disease?. *Gastroenterology*. 1990;98:A172.

15. Fressinaud E, Meyer D. International survey of patients with von Willebrand disease and angiodysplasia. *Thromb Haemost*. Sep 1 1993;70(3):546. [Medline].

16. Makris M. Gastrointestinal bleeding in von Willebrand disease. *Thromb Res*. 2006;118 suppl 1:S13-7. [Medline].

17. Hirri HM, Green PJ, Lindsay J. Von Willebrand's disease and angiodysplasia treated with thalidomide. *Haemophilia*. May 2006;12(3):285-6. [Medline].

18. Emanuel RB, Weiser MM, Shenoy SS, Satchidanand SK, Asirwatham J. Arteriovenous malformations as a cause of gastrointestinal bleeding: the importance of triple-vessel angiographic studies in diagnosis and prevention of rebleeding. *J Clin Gastroenterol*. Jun 1985;7(3):237-46. [Medline].

19. Schwartz J, Rozenfeld V, Habot B. Cessation of recurrent bleeding from gastrointestinal angiodysplasia, after beta blocker treatment in a patient with hypertrophic subaortic stenosis--a case history. *Angiology*. Mar 1992;43(3 pt 1):244-8. [Medline].

20. Pate GE, Chandavimol M, Naiman SC, Webb JG. Heyde's syndrome: a review. *J Heart Valve Dis*. Sep 2004;13(5):701-12. [Medline].

21. Ueno S, Nakase H, Kasahara K, et al. Clinical features of Japanese patients with colonic angiodysplasia. *J Gastroenterol Hepatol*. Aug 2008;23(8 pt 2):e363-6. [Medline].

22. Imperiale TF, Ransohoff DF. Aortic stenosis, idiopathic gastrointestinal bleeding, and angiodysplasia: is there an association? A methodologic critique of the literature. *Gastroenterology*. Dec 1988;95(6):1670-6. [Medline].

23. Kaaroud H, Fatma LB, Beji S, et al. Gastrointestinal angiodysplasia in chronic renal failure. *Saudi J Kidney Dis Transpl*. Sep 2008;19(5):809-12. [Medline]. [Full Text].

24. Roskell DE, Biddolph SC, Warren BF. Apparent deficiency of mucosal vascular collagen type IV associated with angiodysplasia of the colon. *J Clin Pathol*. Jan 1998;51(1):18-20. [Medline]. [Full Text].

25. Junquera F, Saperas E, de Torres I, Vidal MT, Malagelada JR. Increased expression of angiogenic factors in human colonic angiodysplasia. *Am J Gastroenterol*. Apr 1999;94(4):1070-6. [Medline].

26. Kovacs M, Pak P, Pak G, Feheer J, Huttl K. [Multiple angiodysplasias diagnosed by capsule endoscopy] [Hungarian]. *Orv Hetil*. Dec 23 2007;148(51):2435-40. [Medline].

27. Jarbandhan S, van der Veer WM, Mulder CJ. Double-balloon endoscopy in the diagnosis and treatment of hemorrhage from retrovalvular angiodysplasias. *J Gastrointestin Liver Dis*. Sep 2008;17(3):333-4. [Medline]. [Full Text].

28. Monkemuller K, Fry LC, Neumann H, Rickes S, Malfertheiner P. [Diagnostic and therapeutic utility of double balloon endoscopy: experience with 225 procedures] [Spanish]. *Acta Gastroenterol Latinoam*. Dec 2007;37(4):216-23. [Medline].

29. Skibba RM, Hartong WA, Mantz FA, Hinthorn DR, Rhodes JB. Angiodysplasia of the cecum: colonoscopic diagnosis. *Gastrointest Endosc*. Feb 1976;22(3):177-9. [Medline].

30. Richardson JD, Max MH, Flint LM Jr, et al. Bleeding vascular malformations of the intestine. *Surgery*. Sep 1978;84(3):430-6. [Medline].

31. Suzuki N, Arebi N, Saunders BP. A novel method of treating colonic angiodysplasia. *Gastrointest Endosc*. Sep 2006;64(3):424-7. [Medline].

32. Hurlstone DP, Karageh M, Sanders DS. The Olympus EVIS LUCERA variable indices of haemoglobin chart function: a novel technique for establishing the completeness of vascular mucosal ablation in colonic angiodysplasia. *Endoscopy*. Jan 2006;38(1):102. [Medline]. [Full Text].

33. Vakil N, Huilgol V, Khan I. Effect of push enteroscopy on transfusion requirements and quality of life in patients with unexplained gastrointestinal bleeding. *Am J Gastroenterol*. Mar 1997;92(3):425-8. [Medline].

34. Pishvaian AC, Lewis JH. Use of endoclips to obliterate a colonic arteriovenous malformation before cauterization. *Gastrointest Endosc*. May 2006;63(6):865-6. [Medline].

35. Polese L, Angriman I, Pagano D, et al. Laser therapy and surgical treatment in transfusion-dependent patients with upper-gastrointestinal vascular ectasia. *Lasers Med Sci*. Sep 2006;21(3):140-6. [Medline].

36. Meyer CT, Troncale FJ, Galloway S, Sheahan DG. Arteriovenous malformations of the bowel: an analysis of 22 cases and a review of the literature. *Medicine (Baltimore)*. Jan 1981;60(1):36-48. [Medline].

37. Lewis BS, Salomon P, Rivera-MacMurray S, et al. Does hormonal therapy have any benefit for bleeding angiodysplasia?. *J Clin Gastroenterol*. Sep 1992;15(2):99-103. [Medline].

38. Richter JM, Hedberg SE, Athanasoulis CA, Schapiro RH. Angiodysplasia. Clinical presentation and colonoscopic diagnosis. *Dig Dis Sci*. Jun 1984;29(6):481-5. [Medline].

39. Alavi A, Ring EJ. Localization of gastrointestinal bleeding: superiority of 99mTc sulfur colloid compared with angiography. *AJR Am J Roentgenol*. Oct 1981;137(4):741-8. [Medline]. [Full Text].

40. Alvarez L, Puleo J, Balint JA. Investigation of gastrointestinal bleeding in patients with end stage renal disease. *Am J Gastroenterol*. Jan 1993;88(1):30-3. [Medline].

41. Apostolakis E, Doering C, Kantartzis M, Winter J, Schulte HD. Calcific aortic-valve stenosis and angiodysplasia of the colon: Heyde's syndrome--report of two cases. *Thorac Cardiovasc Surg*. Dec 1990;38(6):374-6. [Medline].

42. Bhutani MS, Gupta SC, Markert RJ, et al. A prospective controlled evaluation of endoscopic detection of angiodysplasia and its association with aortic valve disease. *Gastrointest Endosc*. Nov 1995;42(5):398-402. [Medline].

43. Boley SJ, Sammartano R, Adams A, et al. On the nature and etiology of vascular ectasias of the colon. Degenerative lesions of aging. *Gastroenterology*. Apr 1977;72(4 pt 1):650-60. [Medline].

44. Boley SJ, Sprayregen S, Sammartano RJ, Adams A, Kleinhaus S. The pathophysiologic basis for the angiographic signs of vascular ectasias of the colon. *Radiology*. Dec 1977;125(3):615-21. [Medline].

45. Brandt LJ, Mukhopadhyay D. Masking of colon vascular ectasias by cold water lavage. *Gastrointest Endosc*. Jan 1999;49(1):141-2. [Medline].

46. Britt LG, Warren L, Moore OF 3rd. Selective management of lower gastrointestinal bleeding. *Am Surg*. Mar 1983;49(3):121-5. [Medline].

47. Browder W, Cerise EJ, Litwin MS. Impact of emergency angiography in massive lower gastrointestinal bleeding. *Ann Surg*. Nov 1986;204(5):530-6. [Medline]. [Full Text].

48. Chalasani N, Cotsonis G, Wilcox CM. Upper gastrointestinal bleeding in patients with chronic renal failure: role of vascular ectasia. *Am J Gastroenterol*. Nov 1996;91(11):2329-32. [Medline].

49. Chou CC, Dabney JM. Interrelation of ileal wall compliance and vascular resistance. *Am J Dig Dis*. Dec 1967;12(12):1198-208. [Medline].

50. Cottone C, Disclafani G, Genova G, et al. Use of BICAP in a case of colon angiodysplasia. *Surg*

Endosc. 1991;5(2):99-100. [Medline].

51. Duchini A, Sessoms SL. Gastrointestinal hemorrhage in patients with systemic sclerosis and CREST syndrome. *Am J Gastroenterol*. Sep 1998;93(9):1453-6. [Medline].

52. Ettorre GC, Francioso G, Garribba AP, et al. Helical CT angiography in gastrointestinal bleeding of obscure origin. *AJR Am J Roentgenol*. Mar 1997;168(3):727-31. [Medline]. [Full Text].

53. Flynn CT, Chandran PK. Renal failure and angiodysplasia of the colon. *Ann Intern Med*. Jul 1985;103(1):154. [Medline].

54. Foutch PG. Angiodysplasia of the gastrointestinal tract. *Am J Gastroenterol*. Jun 1993;88(6):807-18. [Medline].

55. Foutch PG. Colonic angiodysplasia. *Gastroenterologist*. Jun 1997;5(2):148-56. [Medline].

56. Ghosh J, Speake D, Benbow EW, Watson AJ. Co-existence of true colonic diverticula with angiodysplasia. *Colorectal Dis*. Sep 2005;7(5):530-2. [Medline].

57. Greason KL, Acosta JA, Magrino TJ, Choe M. Angiodysplasia as the cause of massive lower gastrointestinal hemorrhage in a young adult. Report of a case. *Dis Colon Rectum*. Jun 1996;39(6):702-4. [Medline].

58. Gupta N, Longo WE, Vernava AM 3rd. Angiodysplasia of the lower gastrointestinal tract: an entity readily diagnosed by colonoscopy and primarily managed nonoperatively. *Dis Colon Rectum*. Sep 1995;38(9):979-82. [Medline].

59. Hemingway AP. Angiodysplasia as a cause of iron deficiency anaemia. *Blood Rev*. Sep 1989;3(3):147-51. [Medline].

60. Ho S, Jackson J. The angiographic diagnosis of colonic carcinoma. *Clin Radiol*. May 1998;53(5):345-9. [Medline].

61. Hochter W, Weingart J, Kuhner W, Frimberger E, Ottenjann R. Angiodysplasia in the colon and rectum. Endoscopic morphology, localisation and frequency. *Endoscopy*. Sep 1985;17(5):182-5. [Medline].

62. Howard OM, Buchanan JD, Hunt RH. Angiodysplasia of the colon. Experience of 26 cases. *Lancet*. Jul 3 1982;2(8288):16-9. [Medline].

63. Katsinelos P, Paroutoglou G, Beltsis A, et al. Recurrent colonic Dieulafoy's lesion associated with bizarre vascular malformations and abnormal von Willebrand factor. *Acta Gastroenterol Belg*. Oct-Dec 2005;68(4):443-5. [Medline].

64. Kheterpal S. Angiodysplasia: a review. *J R Soc Med*. Oct 1991;84(10):615-8. [Medline]. [Full Text].

65. Kitiyakara T, Selby W. Non-small-bowel lesions detected by capsule endoscopy in patients with obscure GI bleeding. *Gastrointest Endosc*. Aug 2005;62(2):234-8. [Medline].

66. Krevsky B. Detection and treatment of angiodysplasia. *Gastrointest Endosc Clin N Am*. Jul 1997;7(3):509-24. [Medline].

67. Moparty B, Raju GS. Role of hemoclips in a patient with cecal angiodysplasia at high risk of recurrent bleeding from antithrombotic therapy to maintain coronary stent patency: a case report. *Gastrointest Endosc*. Sep 2005;62(3):468-9. [Medline].

68. Noer RJ, Derr JW. Effect of distention on intestinal revascularization. *Arch Surg*. Sep 1949;59(3):542-9. [Medline].

69. Orsi P, Guatti-Zuliani C, Okolicsanyi L. Long-acting octreotide is effective in controlling rebleeding angiodysplasia of the gastrointestinal tract. *Dig Liver Dis*. May 2001;33(4):330-4. [Medline].

70. Potter GD, Sellin JH. Lower gastrointestinal bleeding. *Gastroenterol Clin North Am*. Jun 1988;17(2):341-56. [Medline].

71. Reinus JF, Brandt LJ. Vascular ectasias and diverticulosis. Common causes of lower intestinal bleeding. *Gastroenterol Clin North Am*. Mar 1994;23(1):1-20. [Medline].

72. Roberts PL, Schoetz DJ Jr, Coller JA. Vascular ectasia. Diagnosis and treatment by colonoscopy. *Am Surg*. Jan 1988;54(1):56-9. [Medline].

73. Sabanathan S, Nag SB. Angiodysplasia of the colon: a post-mortem study. *J R Coll Surg Edinb*. Sep 1982;27(5):285-91. [Medline].

74. Scheffer SM, Leatherman LL. Resolution of Heyde's syndrome of aortic stenosis and gastrointestinal bleeding after aortic valve replacement. *Ann Thorac Surg*. Oct 1986;42(4):477-80. [Medline].

75. Semba T, Fujii Y. Relationship between venous flow and colonic peristalsis. *Jpn J Physiol*. Aug 1970;20(4):408-16. [Medline].

76. Serin E, Paydas S, Yildizer K, Seyrek N, Sagliker Y. von Recklinghausen's disease associated with angiodysplasia of colon and end-stage renal disease. *Nephron*. 1995;71(2):243. [Medline].

77. Sharma R, Gorbien MJ. Angiodysplasia and lower gastrointestinal tract bleeding in elderly patients. *Arch Intern Med*. Apr 24 1995;155(8):807-12. [Medline].

78. Szilagyi A, Ghali MP. Pharmacological therapy of vascular malformations of the gastrointestinal tract. *Can J Gastroenterol*. Mar 2006;20(3):171-8. [Medline]. [Full Text].

79. Tedesco FJ, Waye JD, Raskin JB, Morris SJ, Greenwald RA. Colonoscopic evaluation of rectal bleeding: a study of 304 patients. *Ann Intern Med*. Dec 1978;89(6):907-9. [Medline].

80. Thelmo WL, Vetrano JA, Wibowo A, et al. Angiodysplasia of colon revisited: pathologic demonstration without the use of intravascular injection technique. *Hum Pathol*. Jan 1992;23(1):37-40. [Medline].

81. Welch CE, Athanasoulis CA, Galdabini JJ. Hemorrhage from the large bowel with special reference to angiodysplasia and diverticular disease. *World J Surg*. Jan 1978;2(1):73-83. [Medline].

82. Wright HK, Pelliccia O, Higgins EF Jr, Sreenivas V, Gupta A. Controlled, semielective, segmental resection for massive colonic hemorrhage. *Am J Surg*. Apr 1980;139(4):535-8. [Medline].

83. Zuckerman G, Benitez J. A prospective study of bidirectional endoscopy (colonoscopy and upper endoscopy) in the evaluation of patients with occult gastrointestinal bleeding. *Am J Gastroenterol*. Jan 1992;87(1):62-6. [Medline].

84. Zuckerman GR, Cornette GL, Clouse RE, Harter HR. Upper gastrointestinal bleeding in patients with chronic renal failure. *Ann Intern Med*. May 1985;102(5):588-92. [Medline].

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- Embolization, Hemorrhage [in the Radiology section]
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- Transfusion and Autotransfusion [in the Hematology section]
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- Diagnostic Evaluation of Obscure Gastrointestinal Bleeding
- An Economic Evaluation of Capsule Endoscopy for Obscure-Occult Gastrointestinal (GI) Bleeding
- Efficacy and Safety Study on Nasogastric (NG) Tube in Patients With Upper Gastrointestinal Bleed
- Thalidomide Reduces Arteriovenous Malformation Related Gastrointestinal Bleeding
- Transfusion Requirements in GI Bleeding

National Guideline Clearinghouse

- ACR Appropriateness Criteria® treatment of acute nonvariceal gastrointestinal tract bleeding. American College of Radiology - Medical Specialty Society. 2006. 6 pages. NGC:005537
- ASGE guideline: the role of endoscopy in acute non-variceal upper-GI hemorrhage. American Society for Gastrointestinal Endoscopy - Medical Specialty Society. 2004 Oct. 8 pages. NGC:004062
- ASGE guideline: the role of endoscopy in the patient with lower-GI bleeding. American Society for Gastrointestinal Endoscopy - Medical Specialty Society. 2005 Nov. 5 pages. NGC:004584
- Consensus recommendations for managing patients with nonvariceal upper gastrointestinal bleeding. Canadian Association of Gastroenterology - Medical Specialty Society. 2003 Nov 18. 15 pages. NGC:003441
- Management of acute upper and lower gastrointestinal bleeding. A national clinical guideline. Scottish Intercollegiate Guidelines Network - National Government Agency [Non-U.S.]. 2008 Sep. 57 pages. NGC:006730
- Occult blood. Laboratory medicine practice guidelines: evidence-based practice for point-of-care testing. National Academy of Clinical Biochemistry - Professional Association. 2006. 10 pages. NGC:005644