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CASE SERIES

Multiple Organ Gastrointestinal Diseases and Management of Vascular Lesions of the Gastrointestinal Tract Arterio-venous Deformations as Angiodysplasia and Dieulafoy's Lesion, Venous Ectasias, Telangiectasia's

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Abstract

Vascular lesions of the gastrointestinal (GI) tract include arterio-venous deformations as angiodysplasia and Dieulafoy's lesion, venous ectasias (multiple phlebectasias and haemorroids), telangiectasia's which can be associated with heritable hemorrhagic teleangiectasia (HHT), Turner's pattern and systemic sclerosis, hemangiomas', angiosarcoma's and diseases of connective towel affecting blood vessels as pseudoxanthoma elasticum and Ehlers-Danlos's complaint. As a group, they're fairly rare lesions that still may be a major source of upper and lower gastrointestinal bleeding. Clinical donation is variable, ranging from asymptomatic cases over iron insufficiency anaemia to acute or intermittent bleeding that may be life- hanging. Likewise, cases may present with other symptoms, e.g. pain, dysphagia, odynophagia, the presence of a palpable mass, intussusception, inhibition, haemodynamic problems performing from high cardiac affair, lymphatic abnormalities with protein loosing enteropathy and ascites, or dermatological and physical features in syndromal cases. Opinion can generally be made using endoscopy, occasionally with fresh vivisection. Barium radiography, angiography, intraoperative enteroscopy, tagged red blood cell checkup, CT- checkup and MRI- checkup may offer fresh information. Treatment can be characteristic, including iron supplements and transfusion remedy or causal, including remedial endoscopy (ray, electrocautery, heater inquiry or injection sclerotherapy), remedial angiography and surgery. The mode of treatment is of course depending on the mode of donation and other factors similar as associated disorders. However, pharmacological remedy may be warranted, if endoscopic or angiographic remedy is insolvable and surgical intervention not indicated. Good results have been reported with different medicines, albeit utmost of them haven't been tested in large trials.

Keywords: Gastrointestinal Tract, Vascular Lesions, GI bleeding, Angiography, Colonoscopy, CT and MRI

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Introduction

Vas and its derivative vascular are Latin words that imply "vessel"; angeion is the Greek equivalent. As diagnostic modalities become more sophisticated, vascular lesions and GI tract disorders are being more accurately documented. Vascular lesions are a common cause of GI bleeding. Vascular lesions can be solitary or multiple; benign or malignant; isolated, grouped, or diffuse; part of a syndrome or systemic condition; or related to an anatomic anomaly of the vasculature; or occur as a result of treatment [1,2].

Primary Vascular Lesions

Colonic Angioectasia

It is the most common GI vascular anomaly and probably the most common cause of recurrent or chronic lower intestine bleeding in individuals over age 60. Angioectasias (AEs) tend to be acquired as people age, and there does not appear to be a gender difference [3]. AEs are almost always limited to the cecum or ascending colon, are frequently multiple rather than single, and are usually less than 10 mm in diameter. Angiography, colonoscopy **(Figure 1)**, and helical CTA can be used to diagnose them. The roles of CT and MRI for all types of vascular lesions are evolving but are certain to increase as these advanced techniques of diagnosis become more widely available; it is also obvious that conventional angiography is now more important for therapy than diagnosis.

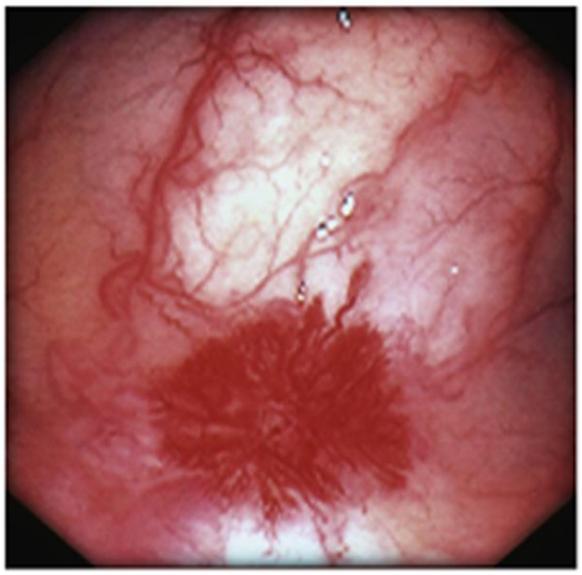

Figure 1: Endoscopic Image of an AE in the Ascending Colon

Pathology

Multiple AEs were common, and they were all found in the cecum and ascending colon. On microscopic examination, mucosal AEs consist of ectatic, distorted, thin-walled venules, capillaries, and arterioles. The presence of dilated, tortuous, submucosal veins, often in areas where overlying mucosal vessels appear normal, is the earliest abnormality. More advanced lesions show an increasing number of dilated and deformed vessels traversing the muscularis mucosa and involving the mucosa.

In advanced lesions, where the dilated arteriolar-capillary-venular unit has become a small arteriovenous (AV) fistula due to loss of prearteriolar sphincter function, enlarged arteries and thick-walled veins are occasionally visible. Congenital AVMs, on the other hand, are more likely to have large, thick-walled arteries.

Clinical Features and Associated Conditions

According to recent studies, AEs and diverticulosis are responsible for 3 to 15% and 20 to 65 % of acute lower gastrointestinal bleeding (LGIB) episodes, respectively. Although patients can report massive hemorrhage, AEs usually cause recurrent and low-grade bleeding [2,3]. The nature and degree of bleeding often vary in the same patient with different episodes: Patients may have bright red blood, maroon stools, or melena on separate occasions. This spectrum represents the varying rates of bleeding from ectatic capillaries, venules, and AV communications, which are dependent on the developmental stage of the lesions. In one study, bleeding from AEs was characterized by tarry stools in 20 to 25% of cases, whereas the minority (10 to 15%) of patients had only iron deficiency anemia, with stools that were intermittently positive for occult blood.

According to another study, AEs caused hemodynamically significant LGIB in 21% of cases, while 42% had chronic LGIB or anemia without evidence of acute bleeding. In most patients, AEs are assumed to be asymptomatic or to cause occult obscure GI hemorrhage. In more than 90% of cases, bleeding from AEs stops spontaneously. Heyde, in 1958 described what is still a controversial association: AE, GI bleeding, and aortic stenosis (AS); aortic valve replacement (AVR) had even been recommended for “Heyde syndrome” when bleeding could not be controlled by medical means. Although various analyses and studies have failed to confirm the association, there are numerous reports of Heyde syndrome in the literature. In a retrospective study, the frequency of AS was 31.7% in patients with "AVMs" compared to 14% in the general population, implying the existence of Heyde syndrome.

Diagnosis and Management

Colonoscopy is the primary means of diagnosis and treatment. If the suspected lesion cannot be detected, or if bleeding is massive and colonoscopy cannot be performed, radionuclide scintigraphy with CTA should be done. CTA and 99mTc-labeled red blood cell scintigraphy (RBCS) were compared in a retrospective study for the overall evaluation and management of acute LGIB [4]. Both CTA and RBCs were able to detect active bleeding (38% of cases), but CTA was able to localize the bleeding site in a significantly higher proportion of studies. The risk of performing biopsies of these abnormalities is not justified because pinch biopsy samples acquired during endoscopy from small, nonelevated vascular lesions are frequently nonspecific. Meperidine may diminish the appearance of finer vascular abnormalities; consequently, it should be avoided or, if administered, its effects should be reversed by naloxone so that vascular lesions may be diagnosed precisely; fentanyl does not have this masking effect. Naloxone has been proven to improve the appearance of normal colonic vasculature in about 10% of

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patients who have received meperidine and cause existing AEs to appear or grow in size. For these reasons, naloxone is a useful supplementary medication for patients receiving meperidine and undergoing endoscopic examination for lower intestine hemorrhage. During a colonoscopy, cool water lavage to clean the mucosal surface may cause underlying AEs to vasoconstrict and disappear temporarily.

Angiography is used to determine the location and character of vascular lesions during active bleeding, and it can also detect some vascular lesions after bleeding has stopped. A densely opacified, slowly emptying, dilated, tortuous vein, avascular tuft, and an early-filling vein are the three reliable angiographic signs of AEs. When the rate of bleeding is at least 0.5 mL/min, a fourth sign, extravasation of contrast material, identifies the site of bleeding but is not specific for AE. The treatment of nonbleeding AEs discovered accidentally during a colonoscopy is expected. Endoscopic therapy is not recommended in these circumstances since the risk of bleeding in asymptomatic patients with AEs has been demonstrated to be low (0% in 3 years) in a prospective study, which clearly does not justify the possible hazards of bleeding and perforation with colonoscopic ablation. In most patients, bleeding from AEs can be managed endoscopically or angiographically, avoiding the morbidity and mortality associated with emergency surgery. For the treatment of LGIB, super-selective microcoil embolization has largely replaced intra-arterial vasopressin infusion. Although complications occur in 5% to 9% of instances, such embolization is highly effective and safe. When intestinal vascular lesions are diffuse throughout the bowel or super-selective catheterization is not possible, vasopressin is suggested. Hormonal therapy, which included estrogens and progestins, had been utilized to treat individuals with a variety of GI bleeding vascular lesions. Somatostatin analogs are another therapy option for GI vascular lesions bleeding. Angiogenesis is inhibited, splanchnic blood flow is decreased, vascular resistance is increased, and platelet aggregation is improved with these drugs. Anti-angiogenic agents, such as thalidomide, bevacizumab, and lenalidomide, are novel treatments for AEs and maybe additional vascular lesions in the GI tract. Anti-angiogenic therapy based on VEGF is a potential treatment, but resolving this issue would require a better knowledge of the angiogenic cascade and how anti-angiogenic chemicals interact with it.

The neodymium yttrium-aluminum-garnet laser, endoscopic sclerosis, monopolar and bipolar electrocoagulation, and heater probe have been used in the past to ablate various vascular lesions throughout the GI tract and reduce active bleeding. Hemoclips, in combination with cautery, endoscopic band ligation, and argon plasma coagulation (APC), have been employed for this purpose more recently. The heater probe and APC are most typically utilized for AEs [5]. Aspirin and aspirin-containing medicines, other NSAIDs, anticoagulants, and antiplatelet agents, should be avoided for at least several days before endoscopic ablation of vascular lesions, depending on the agent. Because the colon wall is not so thinned with a smaller-diameter lumen, aspiration of some intraluminal gas right before thermal therapy adds a measure of safety. When AE is detected by colonoscopy or angiography, and therapy with any or both of these modalities fails, cannot be performed, or is unavailable, and the patient has persistent or recurrent LGI bleeding, a right hemicolectomy is recommended. If the site of the bleeding and its cause cannot be determined and the bleeding continues, a subtotal colectomy (STC) is recommended.

Angiodysplasia

Angiodysplasias are most typically identified in the stomach, and small intestine of patients with chronic kidney disease but are also seen in about 10% of patients with colonic AEs. The blue rubber bleb, hemangioma, angioma, Dieulafoy lesion, and portal hypertensive enteropathy are the other vascular lesions that can occur in the small intestine. Although SBE and DBE are used to diagnose and treat many of these lesions, VCE is currently the mainstay for diagnosing and evaluating patients with obscure and occult GI bleeding because it is noninvasive, easily performed, and allows for a thorough examination of the entire small intestine. In patients who undergo VCE for obscure occult GI bleeding, vascular lesions are the most frequently identified culprit lesion, especially in those older than 65 years of age.

SBE and DBE can be used to evaluate the small intestine anterogradely or retrogradely, allowing for endoscopic therapy to be started at the time of diagnosis. Small intestinal vascular lesions (i.e., angiodysplasias, telangiectasias, blue rubber blebs, and Dieulafoy's) were identified as suspected causes in 51% of patients in a recent study that investigated long-term outcomes in patients undergoing DBE for obscure GI bleeding; these lesions were successfully treated by APC in 97% of patients, with a cumulative rebleeding rate of 46% at 36 months. After endoscopic therapy, about 34% of patients with angiodysplastic lesions and about 45% of patients with isolated small bowel angiodysplastic lesions rebled, according to a systematic review and meta-analysis. The meta-analysis also included four studies that looked at the influence of octreotide analogs on rebleeding rates in patients who were refractory to endoscopic therapy and found that both rebleeding rates and transfusion requirements were significantly reduced. The use of thalidomide for refractory bleeding was studied in two studies, and both showed a reduction in rebleeding rates and transfusion requirements.

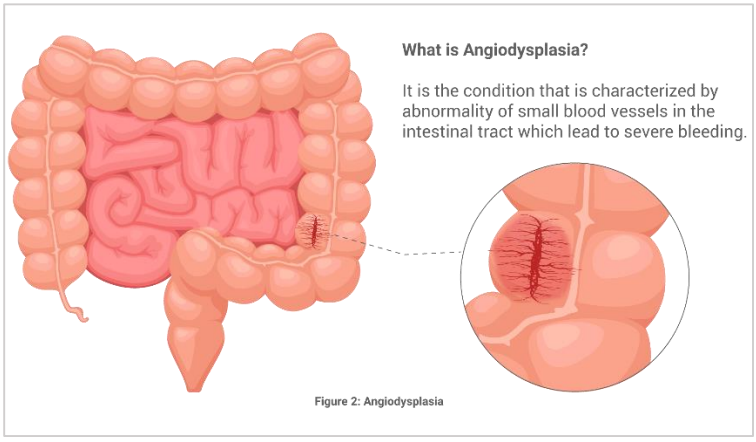

Figure 2: Angiodysplasia

Dieulafoy Lesion

This vascular lesion, which can occur anywhere in the GI tract from the esophagus to the rectum, is an unusual cause of massive GI hemorrhage. It is twice as common in males as in females and

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presents at a mean age of 52 years. The presence of a persistently large-caliber artery in the submucosa and, in some cases, the mucosa, usually with a small, overlying mucosal defect, is the anomaly. Dieulafoy called the lesion *"exulceratio simplex"* because he assumed it was the initial stage of a gastric ulcer. This lesion is also known as an atherosclerotic aneurysm, which is an incorrect phrase because the artery's walls are uniform in caliber throughout and show no remarkable degree of arteriosclerosis. The overlying mucosa thins as a result of the focal pressure from these large "caliber-persistent" vessels, resulting in vascular wall erosion and bleeding. Massive hematemesis or melena usually occurs without any gastrointestinal symptoms and is followed by intermittent and severe bleeding over several days. Dieulafoy lesions have been documented in extragastric regions, including the esophagus, small bowel, rectum, and even outside the GI tract in the bronchus. The most common site of bleeding is 6 cm distal to the cardioesophageal junction, where the arteries that supply the stomach are largest.

Endoscopically, a Dieulafoy lesion appears as an isolated protruding vessel surrounded by normal mucosa. Because the overlying mucosal defect may be small and hidden between the gastric rugae, and the caliber-persistent vessel may constrict and retract after the bleeding episode, it can be difficult to find in a patient with UGI bleeding. EUS has also been used to help identify whether endoscopic therapy was successful by enhancing the identification of these abnormal submucosal vessels. When endoscopy fails to locate a site of hemorrhage, mesenteric angiography may be useful, especially in patients with lesions in the colon or rectum, where the view may be masked by active bleeding or poor bowel preparation. Endoscopic approaches for locating and treating Dieulafoy lesions have significantly reduced the requirement for surgical intervention and reduced the mortality of bleeding from 80% to 8%. Endoscopic therapies for hemostasis are generally safe and successful, with success rates ranging from 75% to 100%. Injection therapy, heater probe, APC, band ligation, hemoclips, and the use of over-the-scope clips are the options for treating bleeding Dieulafoy lesions. Endoscopic therapy with injection followed by thermal or mechanical therapy is preferable to monotherapy, with 95 % of cases achieving hemostasis. Rebleeding from these lesions is reported to be between 9% and 40%, and it is more common following endoscopic monotherapy than after combination therapy.

Hemangioma

Hemangiomas are commonly assumed to be hamartomas and are considered true neoplasms by some. Hemangiomas are the second most common vascular lesion of the colon, and they can appear as single or numerous lesions localized to the colon, as well as part of diffuse gastrointestinal or multisystem angiomatoses. Hemangiomas are structurally complicated lesions characterized by an abundance of blood vessels, generally veins and capillaries, in a focused area of submucosal connective tissue.

Hemangiomas can be classified as cavernous (Figure 3), capillary (Figure 4), or mixed types; however, capillary hemangioma is the most common type found in the GI tract [6]. The majority of hemangiomas are small, ranging in size from a few millimeters to 2 centimeters, but larger lesions, particularly in the rectum, do occur. Colonic hemangiomas normally bleed slowly, resulting in occult blood loss in the form of anemia or melena. Except with large, cavernous hemangiomas of the rectum, which can cause significant hemorrhage, hematochezia is less common. Because radiologic investigations, including angiography, are frequently normal, endoscopy, including enteroscopy, is the best way to establish the diagnosis.

The presence of phleboliths and displacement or distortion of the rectal air column on plain films of the abdomen can often suggest the diagnosis of cavernous hemangioma of the rectum. The affected rectal lumen displays narrowing and rigidity, scalloping of the rectal wall, and widening of the presacral space on barium enema. Endoscopic examination reveals increased plum-red nodules or vascular congestion, as well as ulcers and proctitis. CT and MRI imaging are highly accurate in identifying cavernous hemangiomas, and EUS can also aid in determining the extent of invasion into the anal canal and adjacent structures. Angiography can reveal these lesions; however, it is rarely necessary to make a diagnosis. Cavernous hemangiomas are polypoid or mound-like reddish-purple mucosal lesions.

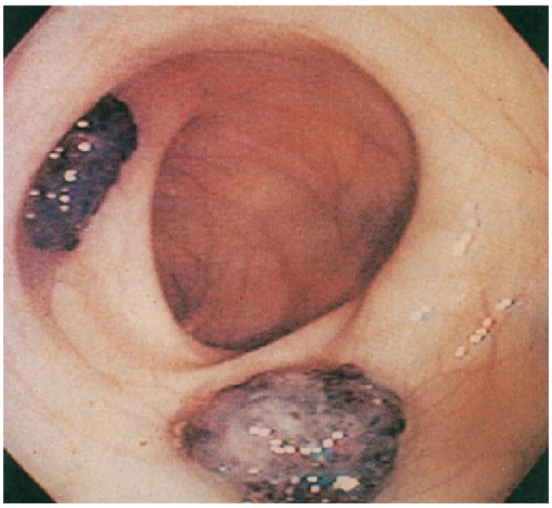

Figure 3: Endoscopic appearance of cavernous hemangioma

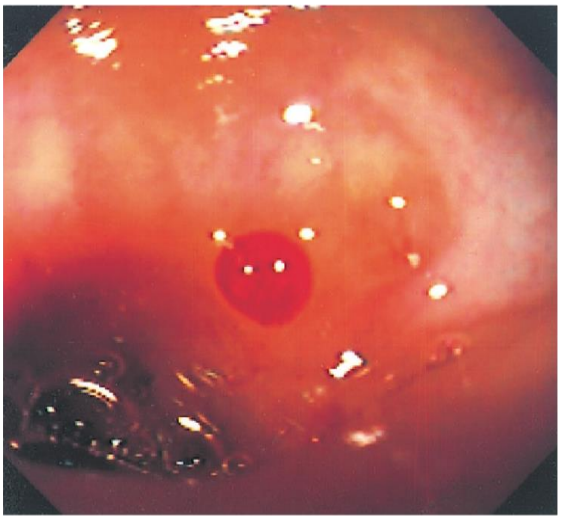

Figure 4: Endoscopic appearance of capillary hemangioma

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Numerous dilated, irregular blood-filled spaces are seen histologically within the mucosa and submucosa, and they occasionally extend through the muscular wall to the serosal surface. Flat endothelial cells with flat or plump nuclei line the vascular channels, depending on their age. Younger hemangiomas contain plump endothelial nuclei and frequently show mitotic activity, whereas older lesions have small and irregular vascular lumina. The endothelial cells flatten and decrease in number as the lesion matures. The fibrous septa thicken, endothelial cells are replaced by adipocytes, and vascular structures atrophy during involution. Despite the absence of a capsule, the capillary hemangioma is usually well-circumscribed, with a central feeding vessel and radiating, lobular extensions. Capillary hemangiomas are reddish-purple plaques or mounds made up of a proliferation of fine, closely packed, newly formed capillaries separated by stroma. Endothelial cells are large, hypertrophic, and can form solid cords or nodules with ill-defined capillary spaces. Ablation can be performed on small hemangiomas that are solitary or few in number and approachable endoscopically. Most large or numerous lesions necessitate excision of the hemangioma or the colon segment affected. Large lesions should not be ablated endoscopically unless they have been proven not to be transmural. Local treatments to reduce excessive bleeding from a rectum cavernous hemangioma are usually only effective for a short time. Although embolization and surgical ligation of major feeding vessels have been performed, rectum resection is frequently required.

Numerous lesions, usually of the cavernous type, affect the stomach, small bowel, and colon in diffuse intestinal hemangiomatosis; hemangiomas of the skin or soft tissues of the head and neck are commonly present. Bleeding or anemia in childhood typically leads to the diagnosis, and surgical intervention may be required for continuous, slow bleeding or intussusception. Intraoperative endoscopy had been useful in detecting small lesions, but SBE and DBE would likely be tried first today.

Congenital Arteriovenous Malformation

AVMs are developmental anomalies that are caused by embryonic growth defects. AVMs are most commonly found in the extremities, but they can appear elsewhere in the vascular system. They can be small and mimic AEs in the colon, or they can include a long segment of the bowel. The rectum and sigmoid are the most common sites for AVMs. Histologically, AVMs are persistent congenital communications between arteries and veins, particularly in the submucosa. The veins are “arterialized,” which is a characteristic feature (i.e., tortuosity, dilatation, and thick walls with smooth muscle hypertrophy and intimal thickening or sclerosis). The arteries in long-standing AVMs are dilated and show atrophic and sclerotic degeneration. The most common method of diagnosis is angiography. In small lesions, early-filling veins are common, as is extensive dilatation of arteries or veins in large lesions. Patients with substantial bleeding from large AVMs should have the affected segment resected; for smaller lesions, transendoscopic treatment may be effective.

Aneurysms

Abdominal Aortic Aneurysm

Atherosclerosis is the most common cause of abdominal aortic aneurysms (AAA) (Figure 5); however, genetic susceptibility is also a factor, less common causes include trauma, vasculitis, infection, and congenital anomalies. Age greater than 60 years, male gender, white race, smoking, and hypertension are all risk factors for the development of a AAA. AAA is observed to be present in 3.9 to 7.2%o f men and 1.0 to 1.3% of women in population-based studies of adults over 50 years old. In 15% to 20% of cases, familial clustering of AAA has been observed, and an abnormality in chromosome 16 has been identified in some families; defects in procollagen III in patients with Ehlers-Danlos syndrome type IV and altered gene expression causing abnormalities in the elastin and collagen content of AAA have been observed in other families. The CDKN2BAS gene [7], also known as ANRIL, encodes an antisense RNA that regulates expression of the cyclin-dependent kinase inhibitors CDKN2A and CDKN2B, and DAB2IP, which encodes a cell growth and survival inhibitor, has the strongest supporting evidence of contributing to the genetic risk for AAA to date. Because most AAAs are asymptomatic until they rupture and are detected incidentally on abdominal ultrasound, CT scan, or MRI done for another reason, current guidelines recommend screening with a “one-time” abdominal ultrasound in males aged 65 to 74 who have a history of smoking. Epigastric pain, which often radiates to the back, is the most common symptom of AAA; severe pain may indicate rupture. A pulsatile epigastric mass may be palpable on physical examination.

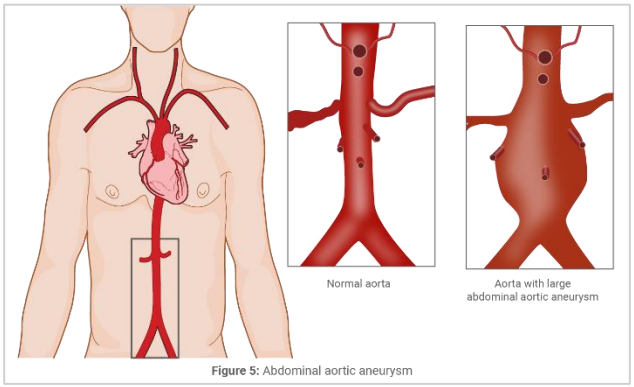

Figure 5: Abdominal aortic aneurysm

On physical examination, distinguishing an aneurysm from an overlying abdominal mass with transmitted pulsations can be difficult and is best done by imaging investigations. A bruit may be present, although it is usually of minimal diagnostic value unless it is recent in onset. In the region of the abdominal aorta, plain abdominal films may reveal a soft tissue mass with peripheral calcification. Large aneurysms can result in erosion of the lumbar vertebrae or displacement of surrounding viscera, such as the bowel, kidneys, and ureters. Because plain film investigations are insufficiently sensitive to determine the presence or size of an aneurysm, ultrasonography, computed tomography, and magnetic resonance imaging (MRI) have become the standard for diagnosis. Because it is highly sensitive (95 to 100%) and specific (100%), and relatively inexpensive, US is the imaging screening technique of choice for AAA. Additionally, for serial monitoring of a AAA to detect changes in its size, US is the preferred test. Preoperatively, CT and MRI are utilized to demonstrate aortic and vascular architecture and to help customize stent-grafts. Because intraluminal laminated

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thrombus limits delineation of the entire lumen and, more importantly, simpler, less invasive, and less expensive imaging modalities are now commonly available, angiography is not employed to determine the size of the aneurysm. The most serious complication of AAA is rupture, which is marked by the sudden onset or worsening of abdominal, flank, or back pain; when “leakage” occurs before overt rupture, an insidious presentation characterized by weeks of pain can develop. Lying in a recumbent position might aggravate pain, whereas sitting or leaning forward can relieve it. Aortic dissection can cause severe abdominal pain as the splanchnic vessels become weakened and acute intestinal ischemia develops.

The size of the aneurysm is the most important predictor of AAA rupture, according to vascular surgeons. AAAs between 3.0 and 3.9 cm in diameter have an almost 0% annual risk of rupture, 1% for those between 4.0 and 4.9 cm in diameter, and 11% for those between 5.00 and 5.99 cm in diameter. Hypertension and the presence of chronic obstructive pulmonary disease are the risk factors associated with rupture. AAA most usually ruptures into the tissues that surround the aorta in the retroperitoneum. Aneurysms that communicate with the peritoneal cavity are less common, in which case hemorrhagic shock develops rapidly. Aneurysmal rupture into the small intestine usually occurs in the third or fourth part of the duodenum and is characterized by massive gastrointestinal bleeding; intermittent bleeding can with clot formation and subsequent dislodgement from the eroded bowel or fistulous opening.

Many of these patients may experience a "herald bleed," which will be followed by massive hemorrhage hours or days later. The most sensitive way for diagnosing this complication is endoscopy. AAA can occasionally rupture into the inferior vena cava, causing a loud bruit. Asymptomatic patients with AAAs greater than 5.5 cm, as well as symptomatic patients with aneurysms of any size, should undergo surgical repair to avoid rupture. Those with an AAA of 3.0 to 5.4 cm should be surveyed every 3 to 12 months by US or CT. AAAs have a varied growth rate, which has been lower in current research than in older ones. An AAA grows at a rate of 0.35 cm per year on average. An open technique, either retroperitoneally or transabdominally or an endovascular approach, which involves the insertion of an endograft into the vascular lumen to exclude the aneurysm from blood flow, reducing the risk of rupture, are the options for AAA repair. Endovascular aneurysm repair (EVAR) is increasingly being used as an alternative to open AAA surgery. EVAR may offer a number of short-term benefits over open surgery, including the avoidance of general anesthesia, shorter operative time, reduced blood loss, and less postoperative pain. Open repair of an AAA is recommended in patients who have a low or moderate risk of operative complications, while EVAR is recommended in patients who have a high risk of complications.

Splanchnic Artery Aneurysms

Splanchnic artery aneurysms (SAAs) are frequently asymptomatic and detected on imaging investigations incidentally. Because SAAs grow slowly and the smallest aneurysm rupture was seen at 2.3 cm, it is recommended that asymptomatic patients with SAAs less than 2.5 cm be monitored. When SAAs are symptomatic, they can cause abdominal pain or gastrointestinal bleeding. A bruit may be heard on auscultation during a physical examination, although an abdominal mass is rarely palpable due to the small size of the aneurysms. Up to 25% of SAAs can be complicated by rupture, which has a mortality rate of 25 to 70% [8]. If an SAA erodes into the GI lumen, it might cause sporadic GI bleeding, known as the "herald bleed," similar to aneurysms of the aorta. An AV fistula can form when a mesenteric vein ruptures, resulting in portal hypertension and variceal hemorrhage. Plain films of the abdomen may show an SAA if it is sufficiently calcified, but the diagnosis is usually made by CT, MRI, or splanchnic angiography, which has the added benefit of potential therapeutic intervention. Treatment is determined by the aneurysm's presentation, location, and size. Even if the aneurysm is asymptomatic, treatment should be considered if the diameter is higher than 2 cm. Treatment options are embolization, surgical repair, or endovascular stenting. Embolization may be preferred for aneurysms that are difficult to treat surgically and for patients who are at high risk.

Splenic Artery Aneurysms

Splenic artery aneurysms are generally saccular (Figure 6), and 20% of patients have multiple aneurysms. Symptoms if present include left upper quadrant or epigastric pain that may radiate to the left shoulder. Atherosclerosis and portal hypertension are the most common causes, while splenic arterial dissection, septic emboli, hypertension, polyarteritis nodosa, SLE, Ehlers-Danlos syndrome, fibromuscular dysplasia, and neurofibromatosis are the less common causes. There is a 3 to 4:1 female to male predominance, which is linked with pregnancy. The increased prevalence in pregnant women may be linked to an increase in splenic blood flow and estrogen's effects on the tunica media's elastic tissue, which might cause splenic artery dilation and predispose to aneurysm formation. Except in pregnant women, where the risk of rupture is substantially higher, aneurysmal rupture occurs in less than 2% of patients. Aneurysms in pregnant women are diagnosed after they rupture in more than 95% of cases, and they are linked to a 75% maternal and 95% fetal mortality rate.

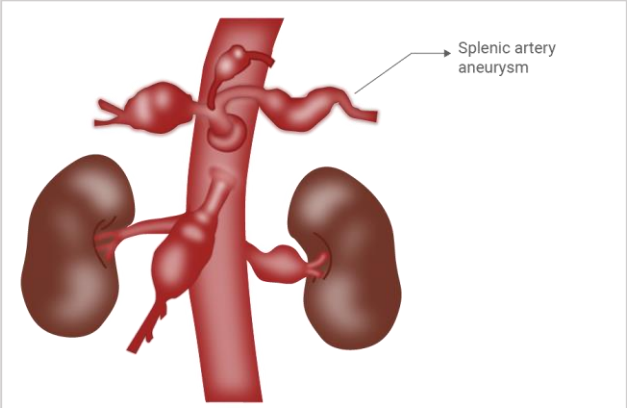

Figure 6: Splenic artery aneurysm

The patient can remain hemodynamically stable if the aneurysm ruptures into the lesser sac, but if the blood overflows into the greater intraperitoneal sac through the foramen of Winslow, diffuse abdominal pain and hypovolemic shock will develop; this is known as the "double-rupture" phenomenon. According to

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reports, in about 25% of patients, the time when the bleeding is localized in the smaller sac allows for surgical intervention. Treatment options for splenic artery aneurysms are determined by the location and presentation of the aneurysm. Splenectomy is the most common treatment for ruptured aneurysms. Emergency surgery has a mortality rate of up to 40%, compared to a very low mortality rate after elective repair.

A pregnant woman or a woman planning to become pregnant who has a symptomatic aneurysm or an aneurysm of any size should undergo repair before pregnancy. If the aneurysm is proximal and larger than 2 cm, surgical therapy, which includes resection and end-to-end vascular repair, should be considered; if the aneurysm is distal or involves the hilum, splenectomy is indicated. Imaging should be used to monitor aneurysms that are between 1 and 2 cm in diameter. Embolization may be performed if surgery is not a possibility. Because the extensive collateral circulation makes surgery more difficult in patients with portal hypertension, embolization is preferred. Splenic infarction and aneurysm reperfusion are the complications of embolization that might occur in 5 to 20% of patients. The use of CT or MRI imaging once a year to check for leaks and subsequent growth is recommended.

Celiac Artery Aneurysms

Historically, the infection caused by syphilis or tuberculosis was the most common cause of celiac artery aneurysms (CAAs), and most cases were identified at autopsy after an aneurysm ruptured. Atherosclerosis, trauma, dissection, and Takayasu arteritis are the most common causes of CA aneurysms now that syphilis and tuberculosis are more readily detected and treated. Aneurysms of the carotid artery are frequently asymptomatic [6,7]. Due to the location, the symptoms can mimic pancreatitis. Esophageal compression can cause dysphagia. The risk of rupture is about 6%. CA aneurysms larger than 2.5 cm should be considered for repair, however asymptomatic lesions smaller than that can be monitored with imaging. A transabdominal or thoracolumbar technique can be used to do a traditional open repair. Aortohepatic bypass or direct aortic reimplantation can be performed after ligation. Prosthetic grafts have a reduced risk of occlusion than saphenous vein grafts in patients undergoing revascularization, but they are more challenging to place due to its location. If the aneurysm ruptures, ligation or percutaneous transcatheter embolization may be done.

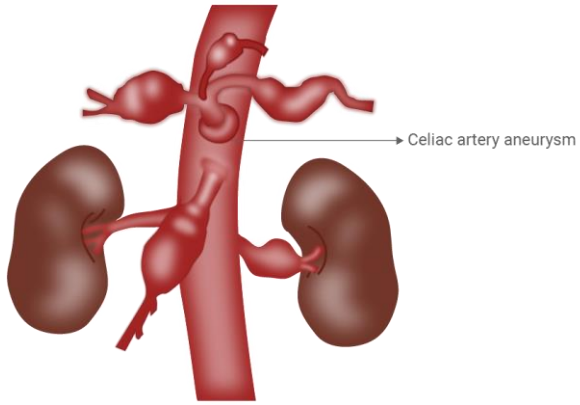

Figure 7: Celiac artery aneurysm

Superior Mesenteric Artery Aneurysms

Aneurysms of the superior mesenteric artery (SMA) are uncommon. They mainly affect the SMA's proximal 5 cm (Figure 8). Aneurysm-related thrombus or dissection can occur, resulting in intestinal ischemia symptoms. In the past, infectious causes were the most common, with septic emboli accounting for a third of all SMA aneurysms. Atherosclerosis, polyarteritis nodosa, pancreatitis, biliary tract disease, neurofibromatosis, and trauma are common causes, according to recent studies. More than 90% of SMA aneurysms are symptomatic, causing abdominal pain and gastrointestinal bleeding. Patients can present with rupture in up to 50% of cases, with a 30% mortality rate. β-Adrenergic blockers have been shown to protect against rupture. Because of the high probability of complications, intervention is suggested for all symptomatic patients and all patients at low surgical risk; for asymptomatic aneurysms smaller than 2.5 cm, surveillance is recommended. Aneurysmectomy and either interposition vein grafting or ligation of a branch of a mesenteric artery are used to treat the aneurysm surgically.

In addition, any ischemic part of the bowel is resected. Endovascular stenting and transcatheter embolization are also options; however, the latter may raise the risk of mesenteric ischemia if the SMA branches are blocked by the stent. Individuals who are reluctant to undergo interventional procedures should take β-adrenergic blockers [8].

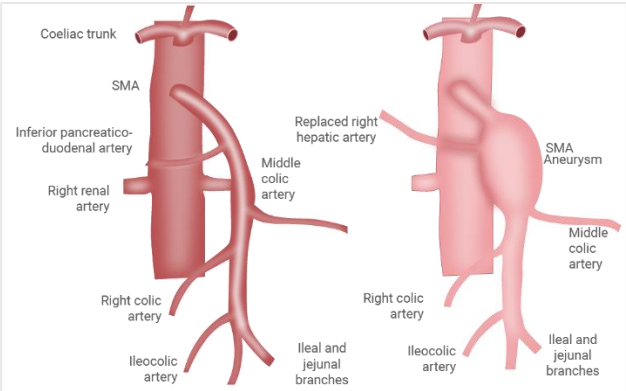

Figure 8: Superior mesenteric artery aneurysm

Mycotic Aneurysm

Aortic and splanchnic vessels mycotic aneurysms are uncommon. Sir William Osler named them so because of their fungus-like appearance. Septic emboli from bacterial endocarditis used to be the most common cause of mycotic aneurysms. The usage of IV drugs is the main risk factor today. Contiguous spread from adjacent infectious processes, arterial manipulation, and immunocompromised (e.g., alcoholism, diabetes mellitus, chemotherapy, glucocorticoid therapy) are also other important risk factors. The most common infectious organisms are Salmonella (particularly Salmonella choleraesuis) and Staphylococcus. The CA, followed by SMA, and IMA are the most commonly affected. Symptoms of mycotic aneurysms are nonspecific early in the course; fever, chills, and abdominal pain

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usually appears later. The vasculature is imaged to diagnose mycotic aneurysms, which are often lobulated and saccular. The destructive process can accelerate, resulting in rapid expansion and rupture. Management is surgical excision of the aneurysm and vascular reconstruction followed by IV antibiotics for 6 weeks. The long-term suppressive oral antibiotic medication also has been used to prevent prosthetic graft infection.

Paraprosthetic-Enteric and Aortoenteric Fistulas

The formation of a fistula between the graft and the adjacent bowel, usually the third or fourth portion of the duodenum because of its proximity to the infrarenal abdominal aorta, is an uncommon but potentially catastrophic complication of aortic aneurysmectomy and other procedures in which vascular prostheses are placed in the retroperitoneum or abdomen. This complication is called as a secondary aortoenteric fistula, and its frequency ranges between 0.36-2%. The average time between aortic surgery and the development of a secondary aortoenteric fistula is 44 months, but fistulas have been reported as early as 21 days and as late as 14 years after surgery; fistulas develop sooner, at a mean interval of 22 months when concurrent intra-abdominal infections are present.

Secondary aortoenteric fistulas are thought to develop as a result of local circumstances present at the time of graft placement or afterward, such as infection, injury to the duodenum or its blood supply during dissection, and graft-induced erosion of the duodenal wall. Endovascular grafts, non-absorbable sutures, antibiotics, strict hemostasis, and suture line coverage with retroperitoneal tissue and peritoneum are some of the newer surgical procedures that may lower the frequency of fistula formation. Atherosclerosis, infection (most usually *Salmonella spp.* and *Klebsiella spp*.) are all linked to primary aortoenteric fistulas. Other organisms that are less common include *Staphylococcus* spp., *Streptococcus* spp., *Clostridium septicum*, *Escherichia coli*, *Enterococcus faecalis*, and *Lactobacillus*), trauma, malignancy, radiotherapy, and foreign body. With an incidence of 0.04 to 0.07%, primary aortoenteric fistulas are less common than secondary aortoenteric fistulas [9,10]. Patients with aortoenteric fistulas have upper or lower gastrointestinal bleeding that, if left untreated, can become massive and rapidly fatal. To rule out other diagnoses and plan treatment, EGD is paired with CT imaging. A high index of suspicion is required to make the diagnosis, typically in a patient who has had aorotoiliac graft surgery and presents with GI bleeding, and quick diagnosis and surgical correction are crucial for survival.

Vascular Lesions Associated with Systemic Disorders Or Manifestations

Hereditary Hemorrhagic Telangiectasia (HHT)

This autosomal dominant familial disorder is also called as Osler-Weber-Rendu disease, is characterized by telangiectasia of the skin and mucous membranes and recurrent GI bleeding. Mutations in the endoglin (ENG) and activin receptor-like kinase-1 (ALK-1) genes, which play a key role in determining the properties of endothelial cells during angiogenesis, are linked to the etiology in some patients. Recurrent epistaxis in childhood is characteristic, and lesions are usually seen in the first few years of life. By the age of ten, about half of the patients have experienced some GI bleeding. Before the fourth decade, severe bleeding is uncommon, and it peaks in the sixth decade. In most patients, hemorrhage presents as melena; BRBPR and hematemesis are less common. In a patient with HHT, hematochezia indicates bleeding from a source other than telangiectasia. Patients may receive more than 60 transfusions in their lifetime due to intermittent and chronic bleeding. 80% of HHT patients have a familial history of the condition; however, it is less common in individuals who bleed later in life. Lips, oral and nasopharyngeal membranes, tongue, and periungual areas are all common sites for telangiectasias; the absence of these sites raises doubts about the diagnosis.

At least three of four relevant clinical criteria must be present for HHT to be diagnosed. These so-called Curacao criteria include

- Epistaxis (spontaneous and recurring nosebleeds)
- Telangiectasias (many lesions at characteristic sites, e.g., lips, oral cavity, fingers, nose) **(Figure 9)**
- Visceral lesions (e.g., pulmonary, hepatic, cerebral, spinal, GI)
- Positive family history (a first-degree relative with HHT); molecular genetic screening can validate the clinical diagnosis

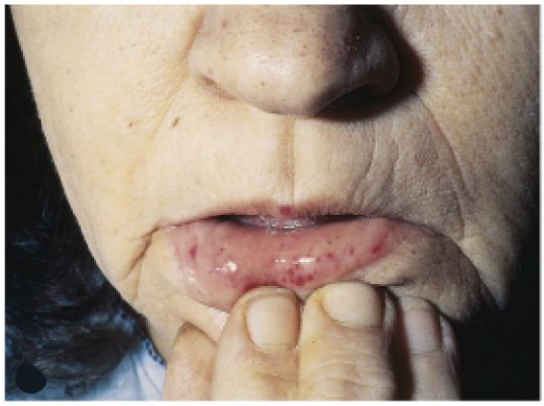

Figure 9: Multiple telangiectasias on the nose and lips

HHT is usually caused by mutations in one of the two HHT genes. Type 1 HHT is caused by mutations in the ENG gene. Endoglin, a type III transforming growth factor-β (TGF-β) receptor, is encoded by the ENG gene, which is found on chromosome 9. Type 2 HHT is caused by mutations in the activin gene. People with type 1 HHT develop symptoms earlier than those with type 2 HHT and are more likely to have blood vessel abnormalities in the lungs and brain; patients with type 2 HHT are more likely to have liver involvement with portal hypertension. The ACVRL1 protein, a type I TGF-β receptor, is encoded by the activin gene, which is found on chromosome 12. Both receptors are predominantly expressed on vascular endothelium and play critical functions in maintaining vascular integrity. Despite genotypic variation in HHT, clinical expression appears to be similar across genotypes. Increased VEGF production is a feature of both HHT and nonhereditary intestinal AE. Patients with HHT

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have high serum levels of VEGF, which correlates with the severity of bleeding.

In HHT, especially type 2, vascular involvement of the liver is common and often asymptomatic. Hepatic symptoms include high-output heart failure due to AV shunting, portal hypertension, and biliary tract disease, which are present in 8% to 31% of patients during the course of the disease. Complications of liver involvement include liver failure, which necessitates liver transplantation, and considerable morbidity and mortality. Patients with HHT may develop telangiectasias in the colon, but they are more common in the stomach and small intestine, where they are more likely to cause significant bleeding. Telangiectasias are apparent on endoscopy, although they may become less noticeable or even invisible in the presence of severe anemia, blood loss, or hypotension; after blood volume and blood pressure correction, they become prominent. Hepatic parenchyma enhancement dilated and tortuous intrahepatic arterial branches, conglomerate masses of abnormal vessels, aneurysms, AV communications, phlebectasia, and hepatic artery and portal vein enlargement may be unrevealing or demonstrate heterogeneous enhancement by conventional angiography or newer techniques such as helical CTA and MRA. When angiography reveals multiple vascular abnormalities, it is possible that some of these lesions are in the mesentery rather than the intestine and so are not potential sites of GI blood loss [8,9]. HHT telangiectasias are the size of millet seeds and appear as cherry red, smooth hillocks. The capillaries and venules are the most affected pathologically, but arterioles may also be affected. Lesions are made up of irregular, ectatic, tortuous blood spaces lined by a fine layer of fibrous connective tissue and supported by a delicate single layer of endothelial cells. Because these arteries lack an elastic lamina or muscular tissue, they are unable to contract, which may explain why telangiectasias bleed.

For telangiectasias, various treatments have been suggested, including estrogens, aminocaproic acid, endoscopic thermal ablation, and resection of the affected bowel. When lesions are within reach of the endoscope and are not widely diffuse, endoscopic ablation, including the use of the APC and thermal contact devices, is most promising. Endoscopic therapy can be done during active bleeding or in between episodes of bleeding and has lessened the need for emergency bowel resection. In a trial of 25 patients with HHT and associated severe hepatic vascular malformations with high cardiac output, bevacizumab reduced cardiac output and reduced epistaxis, but liver vascularity, liver volume, and liver tests did not alter substantially at the 6-month follow-up. This experience contrasted with that of a 47-year-old woman with HHT and severe liver involvement who was treated 4 years ago and experienced cholestasis reversal, resolution of cardiac failure and ascites, and improvement in nutritional status 3 months after starting treatment, as well as a significant reduction in liver vascularity and liver volume over a 6-month period. Although data on the use of bevacizumab for the treatment of GI bleeding in patients with HHT is still limited, case reports and small studies have demonstrated that it can reduce the need for transfusions while also increasing the quality of life.

Blue Rubber Bleb Nevus Syndrome

A connection of cutaneous vascular nevi, intestinal lesions, and GI bleeding was described in 1860, and Bean called this constellation of symptoms blue rubber bleb nevus syndrome (BRBNS) to distinguish it from other cutaneous vascular lesions almost a century later. The eyes, nasopharynx, parotid glands, lungs, liver, spleen, heart, brain, skeletal muscles, urine bladder, and penis may be affected in addition to the GI tract. Orthopedic abnormalities may be present, and thrombosis, calcification, and consumptive coagulopathy may occur within the lesions. Although a few cases of autosomal dominant transmission have been described, and one investigation has identified a responsible locus on chromosome 9, family history is uncommon. BRBNS may be caused by somatic mutations in TEK, the gene encoding TIE2, the endothelial cell tyrosine kinase receptor for angiopoietins, according to a recent study. The presence of TEK mutations in 15 of 17 individuals with BRBNS was found in this study, supporting the theory that these genetic mutations are the cause of the cutaneomucosal venous malformations. The lesions are blue and raised, with diameters ranging from 0.1 to 5 cm (Figure 10) [11]. Direct pressure can be used to empty the contained blood, leaving a "wrinkled sac" until it fills again. Lesions on the trunk, extremities, and face might be solitary or multiple. They can affect any part of the GI tract, although they are most frequent in the small bowel. They are more common distally in the colon. Barium or angiographic investigations are infrequently used to detect them, while CT and MRI are more effective. The most important diagnostic test for this syndrome is endoscopy, but VCE has also been utilized. The lesions were initially thought to be hemangiomas, but they have recently been identified as venous malformations. For recurrent hemorrhage, resection of the affected intestinal segment is suggested. Because these lesions can include the full thickness of the bowel wall, APC can be dangerous; nonetheless, successful sclerotherapy and band ligation of GI tract lesions have been documented.

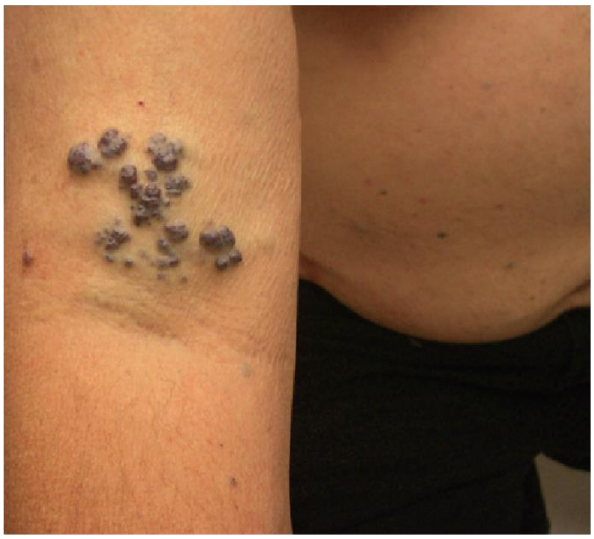

Figure 10: Cutaneous venous malformations in patient with blue rubber bleb nevus syndrome

Progressive Systemic Sclerosis (Scleroderma)

Calcinosis, Raynaud phenomenon, esophageal dysmotility, scleroderma, and telangiectasia (CREST) variants of progressive systemic sclerosis have telangiectasia (Figure 11) as a prominent feature. The hands, lips, tongue, and face are the most common sites for these lesions; however, gastric, intestinal, and colorectal

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lesions have also been documented. These microscopic lesions can cause occult or clinically significant bleeding and are treated with endoscopic thermal ablation if possible.

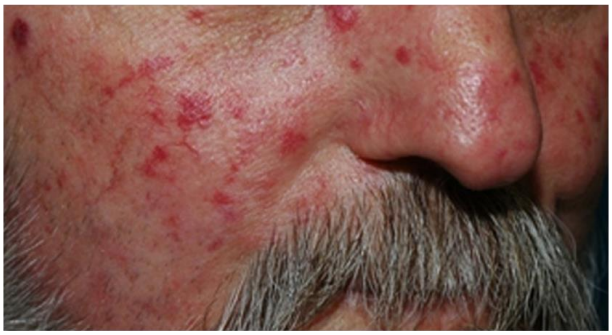

Figure 11: Telangectasias affecting face

Klippel-Trenaunay and Parkes Weber Syndromes

The Klippel-Trenaunay syndrome (KTS) was first described as consisting of (1) a vascular nevus involving the lower limb; (2) varicose veins limited to the affected side that occurred at birth or in childhood; and (3) Hypertrophy of the affected limb's tissues, particularly the bones. Following that, a variety of vascular lesions were linked to the hypertrophic limb, and some authors now separate the syndrome into two types: Klippel-Trenaunay and Parkes Weber; the former is defined by low-flow AV fistulas, while the latter is characterized by higher-flow AV fistulas. In patients with KTS, several genetic abnormalities in the regulation of the angiogenic factor VG5Q have been found. The etiology of bone elongation is unknown, but one theory suggests that it is caused by in utero venous hypertension and stasis. Edema of the affected leg is common, and if the thigh is involved, a variety of lymphatic abnormalities are typical (e.g., chylous mesenteric cysts, chyloperitoneum, protein-losing enteropathy) (Figure 12). Visceral lesions have been described involving the GI tract, kidney, lung, heart, liver, spleen, bladder, and genital organs. The GI tract involvement is more common than previously thought and may occur in as many as 20% of patients, some of whom may not be diagnosed to have visceral involvement because they are asymptomatic.

GI bleeding is the most common symptom of visceral GI involvement, and it usually starts in the first decade of life and is intermittent; after that, GI bleeding from KTS can range from the occult to massive. The distal colon and rectum are the most common bleeding sites in the GI tract, and involvement of the entire GI tract is uncommon. A rectal vascular lesion localized rectovaginal varices due to obstruction of the internal iliac system or portal hypertension with varices are the most common causes of GI bleeding [13]. Consumption coagulopathy, which can occur within the smaller sinusoids of the vascular lesion, can exacerbate bleeding. Although a physical examination is sufficient for diagnosis, different imaging modalities are employed to identify the anatomy and plan surgical correction. MRA is utilized to diagnose and identify AV shunting; angiography is still the gold standard and may allow therapeutic intervention. Endoscopic thermal ablation therapy can help in controlling hemorrhage and prevent or reduce recurrent GI bleeding, especially when the lesions are well-localized; however, patients with clinically significant hemorrhage may require surgical resection.

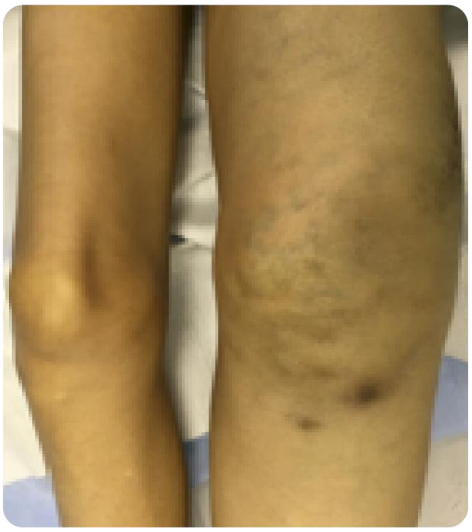

Figure 12: Edema of the involved leg and associated vascular lesions

Radiation-Induced Mucosal Injury

Radiation injury can cause obliterative endarteritis and endothelial proliferation, which can lead to neovascularization and telangiectasia, which may bleed. Radiation injury can occur anywhere in the gastrointestinal tract, although it is most typically seen in the rectosigmoid as a result of pelvic radiation for prostate and cervical malignancies.

Gastric antral vascular ectasia

Gastric antral vascular ectasia (GAVE) (watermelon stomach) is a vascular lesion of the gastric antrum that is characterized by tortuous, dilated vessels radiating outward from the pylorus-like spokes of a wheel and mimicking the dark stripes on the surface of a watermelon. Acute hemorrhage, chronic occult bleeding, or both may be caused by this lesion. Its etiology is unknown, though it has been suggested that gastric peristalsis causes the loose antral mucosa to prolapse, causing the mucosal vessels to elongate. GAVE has also been linked to hypergastrinemia, prostaglandin E2, 5-hydroxytryptamine (serotonin), and vasoactive intestinal polypeptide, as well as delayed gastric emptying. GAVE is more common in middle-aged and older women, and it is linked to achlorhydria/atrophic gastritis, cirrhosis and portal hypertension, cardiac disease, chronic kidney disease, autoimmune and connective tissue disorders, and bone marrow transplantation. Cirrhosis and portal hypertension are linked in around 40% of GAVE cases; consequently, GAVE could be caused by portal hypertension or hepatic veno-occlusive disease, despite the fact that GAVE may not react to therapy aimed at lowering portal pressure. However, given case reports of GAVE remission following liver transplantation, hepatic insufficiency may play a role in its pathogenesis. Although not pathognomonic, microscopic features of GAVE include mucosal capillary ectasia and congestion, spindle cell proliferation, focal thrombosis, and fibrohyalinosis that surrounds the ectatic capillaries of the lamina propria.

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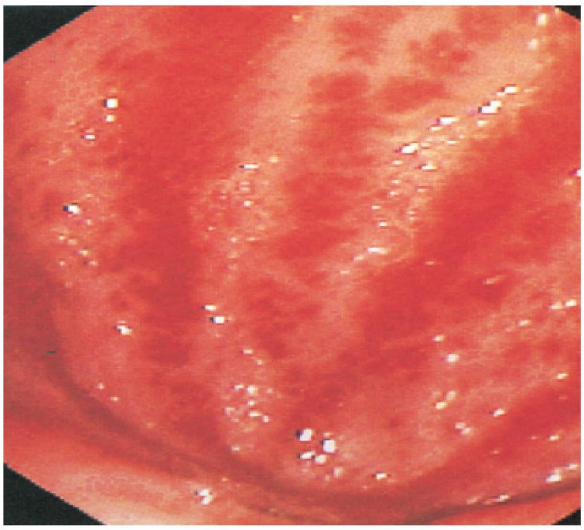

Figure 13: Endoscopic appearance of gastric antral vascular ectasia (GAVE)

Some researchers believe that GAVE and portal hypertensive gastropathy (PHG) are separate manifestations of the same pathogenetic process, although the literature demonstrates that they are independent entities that must be distinguished in order to apply effective therapeutic interventions. The combination of estrogen and progesterone has been attempted and appears to be effective. Tranexamic acid, an antifibrinolytic drug, and thalidomide have also been used successfully. In the absence of portal hypertension, TIPS does not appear to be beneficial for GAVE. Antrectomy has been used as a last resort for patients who have failed to respond to pharmacological and endoscopic treatments. The current mainstay of management for GAVE is transendoscopic treatment. APC has been demonstrated in several studies to improve anemia and reduce the need for transfusions, particularly in cirrhotic patients; side effects are low; however, multiple treatments are usually required. In retrospective studies, band ligation has also been proven to be beneficial. If APC is not available, alternative options include RFA, cryotherapy, and the use of cyanoacrylate spray [14]. In a patient with GAVE and GI bleeding, portal hypertension makes the bleeding more difficult to manage since the bleeding is frequently more severe and resistant to treatment. GAVE reversal after liver transplantation has been seen in several case reports. However, unless the patient is otherwise a liver transplant candidate, the results are insufficient to advocate this therapy. When GAVE is linked with portal hypertension or when bleeding from GAVE associated with PHG cannot be managed endoscopically, TIPS is an option.

Portal Hypertensive Gastropathy (PHG), Enteropathy and Colopathy

Due to a lack of uniform diagnostic criteria and classification, the prevalence of PHG in cirrhotic patients ranges from 20-98%. In general, there is a correlation with more severe portal hypertension. PHG patients can be asymptomatic or have symptoms like chronic GI bleeding, which has been reported in 3 to 60% of patients. Acute gastrointestinal bleeding is less common, with prevalence estimates ranging between 2 and 12%. PHG has 3 endoscopic patterns:

- Fine red speckling of the mucosa
- Superficial reddening, especially at the tips of the gastric rugae
- The presence of a mosaic pattern with red spots (snakeskin appearance) in the gastric fundus or body. (Figure 14)

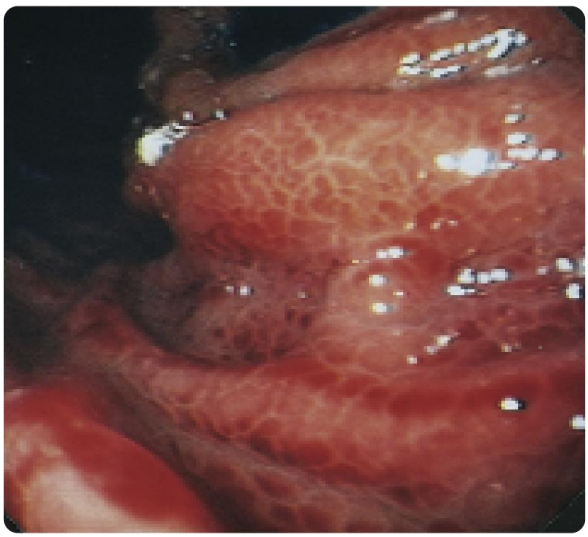

Figure 14: Portal hypertensive gastropathy: Severe gastropathy with diffuse subepithelial hemorrhage in a snakeskin pattern

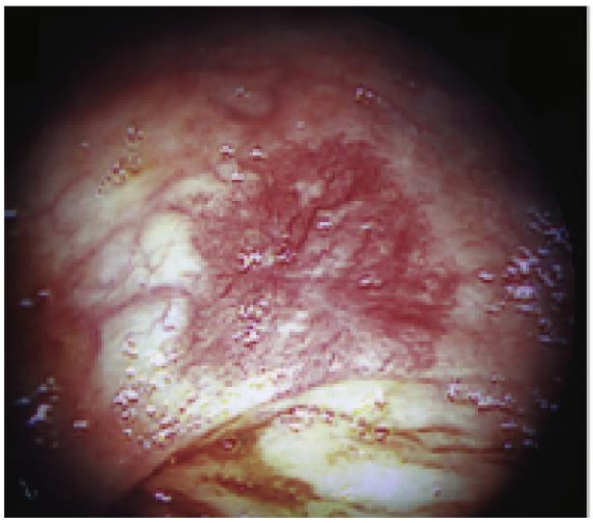

Figure 15: Portal colopathy: A solitary lesion resembling a spider like telangiectasia

Histologically, PHG arises in the oxyntic mucosa and is characterized by dilated, tortuous, irregular veins with no fibrin thrombi, as well as an intimal thickening in the absence of substantial inflammation. The goal of PHG management is to reduce portal pressures, mostly with pharmacotherapy. The first line of treatment is non-selective β-adrenergic blockers. TIPS is a treatment option for patients who have failed to respond to beta-blocker medication. In the majority of situations, it has been effective. Somatostatin analogs, such as octreotide, which have been demonstrated to be beneficial for acute variceal bleeding, have also been shown to be effective for acute PHG hemorrhage.

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Vasopressin and its analog, terlipressin, have also been attempted, with mixed results. Varices and spider-like telangiectasias also can be seen in the small intestine, warranting the term portal enteropathy. The phrase "portal colopathy" is used to describe the vascular presentation of portal hypertension in the colon, which includes varices, hemorrhoids, and spider-like telangiectasias (Figure 15). Portal colopathy mucosal lesions endoscopically mimic those found in PHG and may have a diffuse, colitis-like appearance, including erythema, telangiectasia, and friability. Changes in the histology of portal colopathy and enteropathy are the same as those seen in portal gastropathy. The same thermal therapies that are used to treat GAVE and PHG can be utilized to treat portal enteropathy and colopathy.

Anatomic Abnormalities of the Vasculature

Superior Mesenteric Artery (SMA) Syndrome

The root of the SMA and the wall of the aorta form a 45-degree angle around which the third section of the duodenum is cradled. The SMA impinges on the duodenum when the angle narrows to less than 25 degrees, causing gastric and intestinal obstruction, a condition known as Wilkie's syndrome or the SMA syndrome [11,13,14]. Epigastric pain, vomiting, and early satiety are common symptoms, which can be acute or chronic. Immobilization in a body cast, rapid growth in children, and significant, rapid weight loss in adults, particularly young women with eating disorders, have all been linked to the syndrome. Anatomic anomalies, such as a high ligament of Treitz or a low origin of the SMA, can predispose to the condition. When the patient is supine, barium studies may reveal an abrupt cutoff in the third segment of the duodenum with dilatation proximally. CTA and MRA can provide noninvasive, detailed anatomic information that can be used to diagnose the condition and determine a surgical strategy. After regaining lost weight or removing a body cast, symptoms usually improve. Surgery is only required in rare cases. This condition has been treated by a duodenojejunostomy, which has been performed laparoscopically.

Celiac Axis Compression (Median Arcuate Ligament) Syndrome

Since postprandial pain and an epigastric bruit were described in a patient with angiography showing narrowing of the CA caused by compression from a fibrotic celiac ganglion, whether celiac axis compression syndrome (CACS) is a cause of GI ischemia has been a subject of controversy. The murmur and postprandial pain disappeared after the artery was released. Compression of the CA by the diaphragm's median arcuate ligament and the celiac ganglion has been frequently identified since Harjola's report in 1963, but it is still poorly understood. Postprandial epigastric pain, diarrhea, weight loss, and an abdominal bruit that intensifies with deep expiration when the CA ascends more than the diaphragm and artery compression increases are the clinical features that should be present to diagnose CACS. Lateral aortography or selective CA investigations can demonstrate the compression of CA. Noninvasive methods of illustrating vascular anatomy and CA compression include EUS, CTA, and MRA. Compression of the superior aspect of the CA by the crural fibers of the diaphragm or the celiac ganglion causes a smooth, asymmetrical narrowing and displacement of the CA toward the SMA.

Because the implicated anatomic lesion in this condition is constriction of the major artery that perfuses the upper abdominal viscera, the pain that characterizes CACS is most usually attributed to ischemia. A frequent alternative to ischemia is that the pain originates in the celiac ganglion itself, potentially as a result of the compressed artery's pressure or throbbing. The association between pain and meals could be explained by the increased splanchnic blood flow and arterial dilation that occurs when food is consumed. Division of the median arcuate ligament, with or without ganglionectomy, arterial reconstruction, or bypass are all surgical options for CACS; a laparoscopic technique has been beneficial in alleviating the compression [12].

Summary

Vascular lesions are a common cause of GI bleeding. Colonic Angioectasia is the most common GI vascular anomaly and probably the most common cause of recurrent or chronic lower intestine bleeding in individuals over age 60 and is almost always limited to the cecum or ascending colon. Angiodysplasias are most typically identified in the stomach, and small intestine of patients with chronic kidney disease but are also seen in about 10% of patients with colonic AEs. The blue rubber bleb, hemangioma, angioma, Dieulafoy lesion, and portal hypertensive enteropathy are the other vascular lesions that can occur in the small intestine. AVMs are developmental anomalies that are caused by embryonic growth defects.

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Conflict of Interest
The authors of this manuscript do not have any conflict of interest to declare.

Author Contributions
J. Imai, H. Ichikawa, M. Kaneko, H. Ito, S. Takashimizu, T. Sirai, T. Tajiri, and N. Watanabe, were responsible for the study concept and design, data collection, discussion, and drafting of the manuscript. J. Imai and H. Ichikawa reassessed the contents and English grammar of the manuscript. H. Suzuki supervised the whole process of the study. All the authors read and approved the final manuscript.

Data Availability Statement: All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author.

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