American College of Radiology
ACR Appropriateness Criteria®

Clinical Condition: **Radiologic Management of Upper Gastrointestinal Bleeding**

Variant 1: **Endoscopy reveals arterial bleeding source.**

Radiologic Procedure	Rating	Comments	RRL*
Arteriography visceral	8	Only for bleeding refractory to endoscopic management.	☢☢☢
CTA abdomen with contrast	3		☢☢☢
Tc-99m-labeled RBC scan abdomen and pelvis	3		☢☢☢
Rating Scale: 1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate			***Relative Radiation Level**

Variant 2: **Endoscopy reveals variceal bleeding source.**

Radiologic Procedure	Rating	Comments	RRL*
Wedge venography with pressures liver with TIPS	8	Primarily for bleeding refractory to medical and endoscopic management.	NS
US liver with Doppler	6		O
Wedge venography with pressures liver without TIPS	5	For determination of sinusoidal vs presinusoidal portal hypertension in select cases.	NS
CTA abdomen with contrast	5	May be useful for TIPS planning.	☢☢☢
Tc-99m-labeled RBC scan abdomen and pelvis	2		☢☢☢
Arteriography visceral	2		☢☢☢
Rating Scale: 1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate			***Relative Radiation Level**

Variant 3: **Endoscopy confirms UGIB without a clear source in a patient with a history of aortic reconstruction or pancreaticobiliary procedure.**

Radiologic Procedure	Rating	Comments	RRL*
CTA abdomen with contrast	8		☢☢☢
Arteriography visceral	7		☢☢☢
Tc-99m-labeled RBC scan abdomen and pelvis	4		☢☢☢
Rating Scale: 1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate			***Relative Radiation Level**

Clinical Condition: **Radiologic Management of Upper Gastrointestinal Bleeding**

Variant 4: **Negative endoscopy.**

Radiologic Procedure	Rating	Comments	RRL*
CTA abdomen with contrast	8		☢☢☢
Tc-99m-labeled RBC scan abdomen and pelvis	7	For slower bleeding.	☢☢☢
Arteriography visceral	7	For brisk, active bleeding.	☢☢☢
US liver with Doppler	3		O
Wedge venography with pressures liver without TIPS	3		NS
Wedge venography with pressures liver with TIPS	2		NS
Rating Scale: 1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate			*Relative Radiation Level

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Summary of Literature Review

Upper gastrointestinal bleeding (UGIB) by definition occurs proximal to the ligament of Treitz, originating from the esophagus, stomach, or duodenum. Typically, UGIB will present with hematemesis of varying shade depending on the extent of iron oxidation by gastric acid or with melena following digestion. If brisk, UGIB can also result in hematochezia, which is the presenting sign in 15% of cases of UGIB [1].

The incidence of UGIB ranges between 36 and 48 per 100,000 persons annually. Despite advances in medical care, the overall mortality has remained relatively constant over the past several decades due to the increasing proportion of elderly patients presenting with UGIB and additional comorbidities. According to the American Society for Gastrointestinal Endoscopy (ASGE) survey on UGIB, the most common etiologies are duodenal ulcer (24.3%), gastric erosions (23.4%), gastric ulcer (21.3%), varices (10.3%), Mallory-Weiss tears (7.2%), esophagitis (6.3%), duodenitis (5.8%), neoplasm (2.9%), stomal marginal ulcer (1.8%), esophageal ulcer (1.7%), and other/miscellaneous, including angiodyplasia or vascular malformations (6.8%), with some patients having multiple sources of bleeding. Other recent surveys have reported similar findings with a relatively higher proportion of variceal hemorrhage and erosive gastritis in inner-city populations [2-5]. Frequently, a directed history will reveal the underlying etiology.

In patients presenting with UGIB, aggressive volume resuscitation and maintenance of hemodynamic stability are the first priorities. Only then should an attempt be made to identify and treat the source of hemorrhage. It should be noted that UGIB will cease spontaneously in 70%-80% of cases. A nasogastric aspirate is often obtained to help establish the etiology, though 3%-16% of patients with UGIB bleeding may have a negative aspirate. The three most important diagnostic techniques in the investigation of UGIB are upper endoscopy, angiography, and computed tomography (CT).

Diagnosis and Management of Nonvariceal Upper Gastrointestinal Bleeding

Upper Endoscopy

Patients with presumed UGIB should first be examined by upper endoscopy (esophagogastroduodenoscopy or EGD) as it successfully identifies the source of hemorrhage in 95% of cases and provides prognostic information regarding rebleeding, the need for surgery, the level of hospital care required, and mortality. Emergent endoscopy is indicated in patients with persistent hemorrhage resulting in deviations of vitals signs or requiring repeated transfusions [6-7]. Endoscopy within the emergency room can result in safe discharge in nearly half of all stable patients with subsequent outpatient follow-up. When not performed in the emergency room, endoscopy within 24 hours of admission still effectively reduces resource utilization and shortens hospital stays [8-9]. Further, in patients with high-risk ulcer stigmata at the time of initial endoscopy, a second-look endoscopy may help reduce bleeding rates, surgery, and cost [10].

Endoscopic hemostatic therapy can be grouped into three categories: injection of sclerosants and/or vasoconstrictors, thermal coagulation techniques, and mechanical methods such as band ligation and clips. A meta-analysis of trials of therapeutic endoscopy demonstrated similar efficacy across all hemostatic modalities in reducing risk of rebleeding and the need for emergency surgery. Moreover, using combined methods may be advantageous [11-14]. With acute hemorrhagic gastritis, endoscopic therapy may be more difficult because of the potential for diffuse mucosal bleeding [15].

Angiography and Embolotherapy

When upper endoscopy is unable to control or localize the source of UGIB, angiography is indicated. The accuracy of diagnostic arteriography is increased in active hemorrhage, but it can also reveal structural lesions that bleed intermittently [16-17]. Visceral arteriography can detect bleeding in the UGI tract at rates as low as 0.5 mL/min. Only arterial or capillary bleeding can be detected by selective visceral arteriography; venous bleeding is rarely detected on the venous phase of an arteriogram.

Treatment of UGIB via transcatheter arterial embolization (TAE) has a high technical success rate and is associated with lower complication rates than transcatheter vasopressin infusion [18-20]. While originally relegated to only poor surgical candidates, TAE has been shown to

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be equally effective at controlling bleeding with lower overall complication rates and trends toward lower 30-day mortality rates [21-25]. Long-term clinical success is demonstrated in >60% of patients undergoing TAE for UGIB, irrespective of whether active contrast extravasation is observed at the time of angiography [18,26]. The clinical outcome after technically successful embolotherapy is largely dependent on patient comorbidities [27-28].

Computed Tomography Angiography

Several recent studies have documented the high sensitivity, specificity, and predictive value of multiphasic, multidetector CT (MDCT) in assessing UGIB [29-33]. MDCT was first applied in the evaluation of obscure origin and lower GI bleeding, and in both instances it compares favorably with endoscopic techniques [34-39]. Faster acquisition and thinner collimation, together with multiplanar and three-dimensional image rendering, have improved the sensitivity of MDCT for detecting active hemorrhage to as low as 0.5 mL/min in a swine model, while in vitro models have compared well to digital subtraction angiography (DSA) in first-order aortic branches, with a detection threshold of 0.35 mL/min [40-41].

Nuclear Medicine

Tc-99m-labeled erythrocyte scans ("tagged RBC scans") can detect bleeding rates as low as 0.05-0.1 mL/min. Tc-99m-labeled erythrocyte scans are favored over Tc-99m sulfur colloid scans for diagnosing GI bleeding because of the longer potential imaging interval and corresponding increased sensitivity in the detection and localization of bleeding [42-43]. Errors in localization are most likely to occur when hemorrhage arises from a gastric or duodenal source [44-46]. Moreover, most scintigraphy series included a substantial proportion of patients for whom upper endoscopy would be expected to identify the bleeding site, leaving only a small percentage of patients with UGIB for whom nuclear medicine studies would be of value [47-49].

Barium

Barium studies have no role in the evaluation of acute UGIB. Technically adequate studies may be difficult to obtain in critically ill patients. Barium in the gastrointestinal (GI) tract obscures active hemorrhage and may interfere with subsequent endoscopy or angiography. Barium studies may have a limited role in identifying lesions as potential sources of obscure GI bleeding [50], but they have been largely supplanted by endoscopic techniques [51-57]. Because endoscopy is more accurate than barium studies and potentially therapeutic, it should always precede barium studies in the evaluation of chronic UGIB as well.

Bleeding Due to Portal Hypertension

Most UGIB in the setting of portal hypertension results from ruptured distal esophageal varices, but bleeding from gastric varices, portal hypertensive gastropathy, ectopic varices, and arterial sources must also be considered. While esophageal variceal hemorrhage typically requires a portosystemic pressure gradient in excess of 12 mm Hg, gastric varices may bleed at lower pressure gradients due to the presence of spontaneous gastrorenal shunts. As with nonvariceal bleeding, prompt resuscitation of the patient is imperative. Various supportive measures are often employed to help achieve hemostasis, including the use of vasopressin, somatostatin analogues, and balloon tamponade. The aforementioned therapies can provide temporary stabilization, but emergent endoscopy remains the first line of treatment for esophageal varices as it reduces mortality by 25% and identifies the 30%-50% of cirrhotic patients who have nonvariceal hemorrhage [58-59]. Conversely, no definitive treatment algorithm exists for gastric varices, as their classification varies depending on size, location, and relationship to esophageal varices [60]. Therapeutic endoscopy remains the primary treatment modality for bleeding gastric varices in the United States and Europe, but the evolving standard in Japan is to perform balloon-occluded retrograde transvenous obliteration and, to a lesser degree, percutaneous transhepatic obliteration [61-66].

Endoscopic treatment for variceal hemorrhage consists of sclerotherapy or band ligation, which demonstrate similar efficacy. Of the two, band ligation is favored, given its lower rate of perforation and stenosis [58,67-70]. Endoscopic treatment may be unsuccessful in 10%-30% of patients, with recurrent rebleeding rates between 30%-50% [8,71].

If a second endoscopy is unsuccessful, an alternative treatment should be pursued. Surgical shunts are effective for managing variceal hemorrhage; however, emergent surgery in such patients carries with it a 50% mortality risk, and the paucity of donor organs precludes emergent liver transplant as an option in those with end-stage liver disease [71-73]. Percutaneous transcatheter embolization of the coronary vein and esophageal varices has been shown to control variceal bleeding in 83% of patients; however, since bleeding recurs in 55% of surviving patients at 6 months and in 66% at 1 year, it is no longer widely used [20]. A more comprehensive transcatheter evaluation including free hepatic venography and wedge hepatic venography with manometry can provide useful information in elucidating the cause of portal hypertension and periportal fibrosis [74-76]. Moreover, it can be performed in conjunction with transjugular liver biopsy or transjugular intrahepatic portosystemic shunt (TIPS) insertion.

TIPS has been shown to effectively stop variceal bleeding unresponsive to endoscopic therapy, with a further reduction in rebleeding rates if performed in combination with variceal embolization [77-80]. Numerous studies have demonstrated the benefits of TIPS in the management of poor surgical candidates with esophageal, gastric, or ectopic varices who fail medical and endoscopic therapy [57,81-82]. TIPS performed for variceal hemorrhage is equally efficacious when compared to distal splenorenal shunts, and it has potential cost advantages. New or worsened hepatic encephalopathy can be a potential complication of TIPS. In most cases, hepatic encephalopathy can be managed

effectively through medical means, including protein restriction and bowel catharsis and/or sterilization. Techniques for TIPS reduction have been described to correct refractory hepatic encephalopathy. Fulminant hepatic failure after TIPS is rare and is managed by TIPS reduction or emergent liver transplant [83-90].

TIPS can also relieve bleeding related to portal hypertensive gastropathy, but it has not been shown to be effective in gastric antral ectasia, which can have a similar appearance endoscopically [91]. Recurrent bleeding after TIPS can occur in 16%-30% of patients due to neointimal hyperplasia and stenosis within the stent or in the unstented portion of the hepatic vein, resulting in recurrent portal hypertension [92-94]. However, with the development and use of stent grafts as opposed to bare metal stents, primary and secondary TIPS patency rates have improved dramatically. Secondary patency rates with stent grafts are >90% at 6 months, and survival rates in treated populations are correspondingly higher [95-102]. Shunt patency can be documented through regular Doppler ultrasound (US) or venographic surveillance and maintained by angioplasty or additional stent placement as needed [20].

Special Considerations

Hemobilia and Hemosuccus Pancreaticus

The most common cause of hemobilia is iatrogenic, related to percutaneous transhepatic and endoscopic biliary procedures. Trauma, cholelithiasis, and hepatic artery aneurysms are also common etiologies [103]. In contrast, hemosuccus pancreaticus occurs when a peripancreatic artery communicates with a pancreatic duct directly or through a pseudocyst, afflicting 2%-10% of patients with chronic pancreatitis. While a rare entity, hemosuccus pancreaticus is estimated to be the responsible etiology in two of every 1,500 cases of UGIB [104-106].

In both cases, EGD may demonstrate ampullary blood, but it can not specify the site or cause of hemorrhage, and its role is limited to excluding more common causes of UGIB. Similarly, while endoscopic retrograde cholangiopancreatography (ERCP) may show clots within dilated biliary or pancreatic ducts, this is also a nonspecific finding [107-109]. Angiography can be diagnostic in both cases, and CT and magnetic resonance imaging (MRI) can also demonstrate a source aneurysm, pseudoaneurysm, or fistula [110-112]. Surgical ligation of the affected artery in conjunction with partial hepatectomy or pancreatectomy for hemobilia and hemosuccus pancreaticus, respectively, were once the treatments of choice, but these surgical procedures have largely been supplanted by selective arteriography with TAE or stenting of the culprit lesion [108,113-117]. Technical success rates for these catheter-based techniques are high, with lower morbidity and mortality rates than those for open surgery, but surgical intervention maintains a role in the 11%-37% with rebleeding [103,118] after TAE or stenting.

Aortoenteric Fistula

The proximity of the GI tract to the aorta allows for potential fistulization and resultant catastrophic GI hemorrhage. Primary fistulas are quite rare, estimated in an autopsy series at 0.04%-0.07% of the general population. An incidence of 0.1%-0.8% is reported in the presence of atherosclerotic aortic aneurysm, which occurs in 85% of these patients. Less commonly, primary fistulas can manifest as a sequela of foreign body ingestion or inflammatory, infectious, or neoplastic processes as well [119-120]. Secondary fistulas, in contrast, complicate up to 2% of reconstructed aortas [121]. While aortoesophageal and gastroaortic fistulas have been documented, they comprise <15% of all aortoenteric fistulas. Owing to its consistently close proximity, the third portion of the duodenum is involved in 80% of all aortoenteric fistulas [122].

Aortoenteric fistula will present with a herald hemorrhage in 85% of patients, which is typically self-limiting, followed by a latent period prior to exsanguination. The clinical triad of GI bleeding, abdominal pain, and a pulsatile mass is only present in 25% of patients. Bacteremia and sepsis can also occur, complicating the clinical presentation. EGD is useful in excluding other etiologies but is not typically diagnostic for this entity, having a reported sensitivity <50% [123]. Sensitive and suggestive findings on MDCT include focal bowel wall thickening, increased attenuation, loss of fat planes between the grafted aorta and adjacent bowel, perigraft hematoma, or ectopic gas; however, these signs are nonspecific given the overlap of these findings with perigraft infection. Active intravasation of contrast or displacement of graft material into the bowel lumen are more predictive signs, and generally MDCT remains very useful for diagnosis and therapeutic planning [124-131]. Untreated, mortality from aortoenteric fistula is 100%, and even with treatment long-term outcome is poor. Surgery remains the definitive treatment, but endovascular therapy using covered stents (ie, stent grafts) in conjunction with broad-spectrum antibiotics is proving increasingly successful as a temporizing measure in poor surgical candidates, decreasing perioperative morbidity and mortality [132-141].

Summary

- All patients with presumed UGIB should first be examined by upper endoscopy.
- Angiography and TAE should be considered for all patients with a known source of arterial UGIB refractory to endoscopic management for those with brisk, active bleeding and a negative endoscopy.
- CTA is particularly useful for the localizing of obscure UGIB and in the work-up of a patient with UGIB and a history of aortic reconstruction or pancreaticobiliary procedure.
- Tc-99m-labeled erythrocyte scans are of limited value in diagnosing UGIB but remain useful in certain cases of obscure UGIB.
- Variceal UGIB refractory to endoscopic management should be treated with TIPS insertion. Primary and

secondary TIPS patency rates have improved dramatically with the use of stent grafts rather than bare metal stents.

- Doppler US of the liver is useful for TIPS surveillance. Both Doppler US and CT may be used for TIPS planning.

Relative Radiation Level Information

Potential adverse health effects associated with radiation exposure are an important factor to consider when selecting the appropriate imaging procedure. Because there is a wide range of radiation exposures associated with different diagnostic procedures, a relative radiation level (RRL) indication has been included for each imaging examination. The RRLs are based on effective dose, which is a radiation dose quantity that is used to estimate population total radiation risk associated with an imaging procedure. Patients in the pediatric age group are at inherently higher risk from exposure, both because of organ sensitivity and longer life expectancy (relevant to the long latency that appears to accompany radiation exposure). For these reasons, the RRL dose estimate ranges for pediatric examinations are lower as compared to those specified for adults (see Table below). Additional information regarding radiation dose assessment for imaging examinations can be found in the ACR Appropriateness Criteria® Radiation Dose Assessment Introduction document.

Relative Radiation Level Designations

Relative Radiation Level*	Adult Effective Dose Estimate Range	Pediatric Effective Dose Estimate Range
O	0 mSv	0 mSv
☢	<0.1 mSv	<0.03 mSv
☢ ☢	0.1-1 mSv	0.03-0.3 mSv
☢ ☢ ☢	1-10 mSv	0.3-3 mSv
☢ ☢ ☢ ☢	10-30 mSv	3-10 mSv
☢ ☢ ☢ ☢ ☢	30-100 mSv	10-30 mSv

*RRL assignments for some of the examinations cannot be made, because the actual patient doses in these procedures vary as a function of a number of factors (eg, region of the body exposed to ionizing radiation, the imaging guidance that is used). The RRLs for these examinations are designated as NS (not specified).

Supporting Document(s)

- ACR Appropriateness Criteria® Overview
- Procedure Information
- Evidence Table

References

1. Peter DJ, Dougherty JM. Evaluation of the patient with gastrointestinal bleeding: an evidence based approach. *Emerg Med Clin North Am* 1999; 17(1):239-261, x.
2. Gilbert DA, Silverstein FE, Tedesco FJ, Buenger NK, Persing J. The national ASGE survey on upper gastrointestinal bleeding. III. Endoscopy in upper gastrointestinal bleeding. *Gastrointest Endosc* 1981; 27(2):94-102.
3. Silverstein FE, Gilbert DA, Tedesco FJ, Buenger NK, Persing J. The national ASGE survey on upper gastrointestinal bleeding. I. Study design and baseline data. *Gastrointest Endosc* 1981; 27(2):73-79.
4. Sugawa C, Steffes CP, Nakamura R, et al. Upper GI bleeding in an urban hospital. Etiology, recurrence, and prognosis. *Ann Surg* 1990; 212(4):521-526; discussion 526-527.
5. van Leerdam ME. Epidemiology of acute upper gastrointestinal bleeding. *Best Pract Res Clin Gastroenterol* 2008; 22(2):209-224.
6. Chak A, Cooper GS, Lloyd LE, Kolz CS, Barnhart BA, Wong RC. Effectiveness of endoscopy in patients admitted to the intensive care unit with upper GI hemorrhage. *Gastrointest Endosc* 2001; 53(1):6-13.
7. Esrailian E, Gralnek IM. Nonvariceal upper gastrointestinal bleeding: epidemiology and diagnosis. *Gastroenterol Clin North Am* 2005; 34(4):589-605.
8. Adler DG, Leighton JA, Davila RE, et al. ASGE guideline: The role of endoscopy in acute non-variceal upper-GI hemorrhage. *Gastrointest Endosc* 2004; 60(4):497-504.
9. Van Dam J, Brugge WR. Endoscopy of the upper gastrointestinal tract. *N Engl J Med* 1999; 341(23):1738-1748.
10. Spiegel BM, Ofman JJ, Woods K, Vakil NB. Minimizing recurrent peptic ulcer hemorrhage after endoscopic hemostasis: the cost-effectiveness of competing strategies. *Am J Gastroenterol* 2003; 98(1):86-97.
11. Chung SS, Lau JY, Sung JJ, et al. Randomised comparison between adrenaline injection alone and adrenaline injection plus heat probe treatment for actively bleeding ulcers. *BMJ* 1997; 314(7090):1307-1311.
12. Cook DJ, Guyatt GH, Salena BJ, Laine LA. Endoscopic therapy for acute nonvariceal upper gastrointestinal hemorrhage: a meta-analysis. *Gastroenterology* 1992; 102(1):139-148.
13. Rollhauser C, Fleischer DE. Nonvariceal upper gastrointestinal bleeding. *Endoscopy* 2004; 36(1):52-58.
14. Zuccaro G, Jr. Bleeding peptic ulcer: pathogenesis and endoscopic therapy. *Gastroenterol Clin North Am* 1993; 22(4):737-750.
15. Chamberlain CE. Acute hemorrhagic gastritis. *Gastroenterol Clin North Am* 1993; 22(4):843-873.
16. Keller FS, Routh WD. Angiographic diagnosis and management. *Hepatogastroenterology* 1991; 38(3):207-215.
17. Rollins ES, Picus D, Hicks ME, Darcy MD, Bower BL, Kleinhoffer MA. Angiography is useful in detecting the source of chronic gastrointestinal bleeding of obscure origin. *AJR* 1991; 156(2):385-388.
18. Aina R, Oliva VL, Therasse E, et al. Arterial embolotherapy for upper gastrointestinal hemorrhage: outcome assessment. *J Vasc Interv Radiol* 2001; 12(2):195-200.
19. Miller M, Jr., Smith TP. Angiographic diagnosis and endovascular management of nonvariceal gastrointestinal hemorrhage. *Gastroenterol Clin North Am* 2005; 34(4):735-752.
20. Shapiro MJ. The role of the radiologist in the management of gastrointestinal bleeding. *Gastroenterol Clin North Am* 1994; 23(1):123-181.
21. Busch OR, van Delden OM, Gouma DJ. Therapeutic options for endoscopic haemostatic failures: the place of the surgeon and radiologist in gastrointestinal tract bleeding. *Best Pract Res Clin Gastroenterol* 2008; 22(2):341-354.
22. Defreyne L, De Schrijver I, Decruyenaere J, et al. Therapeutic decision-making in endoscopically unmanageable nonvariceal upper gastrointestinal hemorrhage. *Cardiovasc Intervent Radiol* 2008; 31(5):897-905.
23. Eriksson LG, Ljungdahl M, Sundbom M, Nyman R. Transcatheter arterial embolization versus surgery in the treatment of upper gastrointestinal bleeding after therapeutic endoscopy failure. *J Vasc Interv Radiol* 2008; 19(10):1413-1418.
24. Holme JB, Nielsen DT, Funch-Jensen P, Mortensen FV. Transcatheter arterial embolization in patients with bleeding duodenal ulcer: an alternative to surgery. *Acta Radiol* 2006; 47(3):244-247.
25. Ripoll C, Banares R, Beceiro I, et al. Comparison of transcatheter arterial embolization and surgery for treatment of bleeding peptic ulcer after endoscopic treatment failure. *J Vasc Interv Radiol* 2004; 15(5):447-450.
26. Padia SA, Geisinger MA, Newman JS, Pierce G, Obuchowski NA, Sands MJ. Effectiveness of coil embolization in angiographically

detectable versus non-detectable sources of upper gastrointestinal hemorrhage. *J Vasc Interv Radiol* 2009; 20(4):461-466.
27. Loffroy R, Guiu B, Mezzetta L, et al. Short- and long-term results of transcatheter embolization for massive arterial hemorrhage from gastroduodenal ulcers not controlled by endoscopic hemostasis. *Can J Gastroenterol* 2009; 23(2):115-120.
28. Schenker MP, Duszak R, Jr., Soulen MC, et al. Upper gastrointestinal hemorrhage and transcatheter embolotherapy: clinical and technical factors impacting success and survival. *J Vasc Interv Radiol* 2001; 12(11):1263-1271.
29. Chua AE, Ridley LJ. Diagnostic accuracy of CT angiography in acute gastrointestinal bleeding. *J Med Imaging Radiat Oncol* 2008; 52(4):333-338.
30. Jaeckle T, Stuber G, Hoffmann MH, Freund W, Schmitz BL, Aschoff AJ. Acute gastrointestinal bleeding: value of MDCT. *Abdom Imaging* 2008; 33(3):285-293.
31. Jaeckle T, Stuber G, Hoffmann MH, Jeltsch M, Schmitz BL, Aschoff AJ. Detection and localization of acute upper and lower gastrointestinal (GI) bleeding with arterial phase multi-detector row helical CT. *Eur Radiol* 2008; 18(7):1406-1413.
32. Scheffel H, Pfammatter T, Wildi S, Bauerfeind P, Marincek B, Alkadhi H. Acute gastrointestinal bleeding: detection of source and etiology with multi-detector-row CT. *Eur Radiol* 2007; 17(6):1555-1565.
33. Yoon W, Jeong YY, Shin SS, et al. Acute massive gastrointestinal bleeding: detection and localization with arterial phase multi-detector row helical CT. *Radiology* 2006; 239(1):160-167.
34. Ettorre GC, Francioso G, Garribba AP, Fracella MR, Greco A, Farchi G. Helical CT angiography in gastrointestinal bleeding of obscure origin. *AJR* 1997; 168(3):727-731.
35. Hara AK, Leighton JA, Sharma VK, Heigh RI, Fleischer DE. Imaging of small bowel disease: comparison of capsule endoscopy, standard endoscopy, barium examination, and CT. *Radiographics* 2005; 25(3):697-711; discussion 711-698.
36. Huprich JE, Fletcher JG, Alexander JA, Fidler JL, Burton SS, McCullough CH. Obscure gastrointestinal bleeding: evaluation with 64-section multiphase CT enterography--initial experience. *Radiology* 2008; 246(2):562-571.
37. Laing CJ, Tobias T, Rosenblum DI, Banker WL, Tseng L, Tamarkin SW. Acute gastrointestinal bleeding: emerging role of multidetector CT angiography and review of current imaging techniques. *Radiographics* 2007; 27(4):1055-1070.
38. Stunell H, Buckley O, Lyburn ID, McGann G, Farrell M, Torreggiani WC. The role of computerized tomography in the evaluation of gastrointestinal bleeding following negative or failed endoscopy: a review of current status. *J Postgrad Med* 2008; 54(2):126-134.
39. Tew K, Davies RP, Jadun CK, Kew J. MDCT of acute lower gastrointestinal bleeding. *AJR* 2004; 182(2):427-430.
40. Kuhle WG, Sheiman RG. Detection of active colonic hemorrhage with use of helical CT: findings in a swine model. *Radiology* 2003; 228(3):743-752.
41. Roy-Choudhury SH, Gallacher DJ, Pilmer J, et al. Relative threshold of detection of active arterial bleeding: in vitro comparison of MDCT and digital subtraction angiography. *AJR* 2007; 189(5):W238-246.
42. Alavi A, Dann RW, Baum S, Biery DN. Scintigraphic detection of acute gastrointestinal bleeding. *Radiology* 1977; 124(3):753-756.
43. Bunker SR, Lull RJ, Tanasescu DE, et al. Scintigraphy of gastrointestinal hemorrhage: superiority of 99mTc red blood cells over 99mTc sulfur colloid. *AJR* 1984; 143(3):543-548.
44. Bentley DE, Richardson JD. The role of tagged red blood cell imaging in the localization of gastrointestinal bleeding. *Arch Surg* 1991; 126(7):821-824.
45. McKusick KA, Froelich J, Callahan RJ, Winzelberg GG, Strauss HW. 99mTc red blood cells for detection of gastrointestinal bleeding: experience with 80 patients. *AJR* 1981; 137(6):1113-1118.
46. Winzelberg GG, McKusick KA, Froelich JW, Callahan RJ, Strauss HW. Detection of gastrointestinal bleeding with 99mTc-labeled red blood cells. *Semin Nucl Med* 1982; 12(2):139-146.
47. Howarth DM. The role of nuclear medicine in the detection of acute gastrointestinal bleeding. *Semin Nucl Med* 2006; 36(2):133-146.
48. Robinson P. The role of nuclear medicine in acute gastrointestinal bleeding. *Nucl Med Commun* 1993; 14(10):849-855.
49. Zuckier LS, Freeman LM. Selective role of nuclear medicine in evaluating the acute abdomen. *Radiol Clin North Am* 2003; 41(6):1275-1288.
50. Cello JP, Thoeni RF. Gastrointestinal hemorrhage. Comparative values of double-contrast upper gastrointestinal radiology and endoscopy. *JAMA* 1980; 243(7):685-688.
51. Appleyard M, Glukhovsky A, Swain P. Wireless-capsule diagnostic endoscopy for recurrent small-bowel bleeding. *N Engl J Med* 2001; 344(3):232-233.
52. Cellier C, Tkoub M, Gaudric M, et al. [Comparison of push-type endoscopy and barium transit study of the small intestine in digestive bleeding and unexplained iron-deficiency anemia]. *Gastroenterol Clin Biol* 1998; 22(5):491-494.
53. Descamps C, Schmit A, Van Gossum A. "Missed" upper gastrointestinal tract lesions may explain "occult" bleeding. *Endoscopy* 1999; 31(6):452-455.
54. Iddan G, Meron G, Glukhovsky A, Swain P. Wireless capsule endoscopy. *Nature* 2000; 405(6785):417.
55. Waye JD. Enteroscopy. *Gastrointest Endosc* 1997; 46(3):247-256.
56. Yamamoto H, Sekine Y, Sato Y, et al. Total enteroscopy with a nonsurgical steerable double-balloon method. *Gastrointest Endosc* 2001; 53(2):216-220.
57. Zaman A, Chalasani N. Bleeding caused by portal hypertension. *Gastroenterol Clin North Am* 2005; 34(4):623-642.
58. Sclerotherapy after first variceal hemorrhage in cirrhosis. A randomized multicenter trial. The Copenhagen Esophageal Varices Sclerotherapy Project. *N Engl J Med* 1984; 311(25):1594-1600.
59. Goff JS. Gastroesophageal varices: pathogenesis and therapy of acute bleeding. *Gastroenterol Clin North Am* 1993; 22(4):779-800.
60. Ryan BM, Stockbrugger RW, Ryan JM. A pathophysiologic, gastroenterologic, and radiologic approach to the management of gastric varices. *Gastroenterology* 2004; 126(4):1175-1189.
61. Chikamori F, Kuniyoshi N, Kawashima T, Shibuya S, Takase Y. Percutaneous transhepatic obliteration for isolated gastric varices with gastropericardiac shunt: case report. *Abdom Imaging* 2006; 31(2):249-252.
62. Fukuda T, Hirota S, Sugimura K. Long-term results of balloon-occluded retrograde transvenous obliteration for the treatment of gastric varices and hepatic encephalopathy. *J Vasc Interv Radiol* 2001; 12(3):327-336.
63. Kitamoto M, Imamura M, Kamada K, et al. Balloon-occluded retrograde transvenous obliteration of gastric fundal varices with hemorrhage. *AJR* 2002; 178(5):1167-1174.
64. Lunderquist A, Vang J. Transhepatic catheterization and obliteration of the coronary vein in patients with portal hypertension and esophageal varices. *N Engl J Med* 1974; 291(13):646-649.
65. Ninoi T, Nakamura K, Kaminou T, et al. TIPS versus transcatheter sclerotherapy for gastric varices. *AJR* 2004; 183(2):369-376.
66. Ninoi T, Nishida N, Kaminou T, et al. Balloon-occluded retrograde transvenous obliteration of gastric varices with gastrorenal shunt: long-term follow-up in 78 patients. *AJR* 2005; 184(4):1340-1346.
67. Karsan HA, Morton SC, Shekelle PG, et al. Combination endoscopic band ligation and sclerotherapy compared with endoscopic band ligation alone for the secondary prophylaxis of esophageal variceal hemorrhage: a meta-analysis. *Dig Dis Sci* 2005; 50(2):399-406.
68. Kravetz D. Prevention of recurrent esophageal variceal hemorrhage: review and current recommendations. *J Clin Gastroenterol* 2007; 41 Suppl 3:S318-322.
69. Stiegmann GV. Evolution of endoscopic therapy for esophageal varices. *Surg Endosc* 2006; 20 Suppl 2:S467-470.
70. Stiegmann GV, Goff JS, Michaletz-Onody PA, et al. Endoscopic sclerotherapy as compared with endoscopic ligation for bleeding esophageal varices. *N Engl J Med* 1992; 326(23):1527-1532.
71. Cello JP, Grendell JH, Crass RA, Trunkey DD, Cobb EE, Heilbron DC. Endoscopic sclerotherapy versus portacaval shunt in patients with severe cirrhosis and variceal hemorrhage. *N Engl J Med* 1984; 311(25):1589-1594.
72. Henderson JM. Surgery versus transjugular intrahepatic portal systemic shunt in the treatment of severe variceal bleeding. *Clin Liver Dis* 2006; 10(3):599-612, ix.
73. Luketic VA, Sanyal AJ. Esophageal varices. II. TIPS (transjugular intrahepatic portosystemic shunt) and surgical therapy. *Gastroenterol Clin North Am* 2000; 29(2):387-421, vi.

74. Cavaluzzi JA, Sheff R, Harrington DP, et al. Hepatic venography and wedge hepatic vein pressure measurements in diffuse liver disease. *AJR* 1977; 129(3):441-446.
75. Heeney DJ, Bookstein JJ, Bell RH, Orloff MJ, Miyai K. Correlation of hepatic and portal wedged venography and manometry with histology in alcoholic cirrhosis and periportal fibrosis. *Radiology* 1982; 142(3):591-597.
76. Smith GW, Westgaard T, Bjorn-Hansen R. Hepatic venous angiography in the evaluation of cirrhosis of the liver. *Ann Surg* 1971; 173(4):469-480.
77. Coldwell DM, Ring EJ, Rees CR, et al. Multicenter investigation of the role of transjugular intrahepatic portosystemic shunt in management of portal hypertension. *Radiology* 1995; 196(2):335-340.
78. LaBerge JM, Ring EJ, Gordon RL, et al. Creation of transjugular intrahepatic portosystemic shunts with the wallstent endoprosthesis: results in 100 patients. *Radiology* 1993; 187(2):413-420.
79. Rossle M, Haag K, Ochs A, et al. The transjugular intrahepatic portosystemic stent-shunt procedure for variceal bleeding. *N Engl J Med* 1994; 330(3):165-171.
80. Tesdal IK, Filser T, Weiss C, Holm E, Dueber C, Jaschke W. Transjugular intrahepatic portosystemic shunts: adjunctive embolotherapy of gastroesophageal collateral vessels in the prevention of variceal rebleeding. *Radiology* 2005; 236(1):360-367.
81. Boyer TD, Haskal ZJ. American Association for the Study of Liver Diseases Practice Guidelines: the role of transjugular intrahepatic portosystemic shunt creation in the management of portal hypertension. *J Vasc Interv Radiol* 2005; 16(5):615-629.
82. Vidal V, Joly L, Perreault P, Bouchard L, Lafortune M, Pomier-Layrargues G. Usefulness of transjugular intrahepatic portosystemic shunt in the management of bleeding ectopic varices in cirrhotic patients. *Cardiovasc Intervent Radiol* 2006; 29(2):216-219.
83. Boyer TD, Henderson JM, Heerey AM, et al. Cost of preventing variceal rebleeding with transjugular intrahepatic portal systemic shunt and distal splenorenal shunt. *J Hepatol* 2008; 48(3):407-414.
84. Henderson JM, Boyer TD, Kutner MH, et al. Distal splenorenal shunt versus transjugular intrahepatic portal systematic shunt for variceal bleeding: a randomized trial. *Gastroenterology* 2006; 130(6):1643-1651.
85. Khan S, Tudur Smith C, Williamson P, Sutton R. Portosystemic shunts versus endoscopic therapy for variceal rebleeding in patients with cirrhosis. *Cochrane Database Syst Rev* 2006; (4):CD000553.
86. Hauenstein KH, Haag K, Ochs A, Langer M, Rossle M. The reducing stent: treatment for transjugular intrahepatic portosystemic shunt-induced refractory hepatic encephalopathy and liver failure. *Radiology* 1995; 194(1):175-179.
87. Madoff DC, Wallace MJ, Ahrar K, Saxon RR. TIPS-related hepatic encephalopathy: management options with novel endovascular techniques. *Radiographics* 2004; 24(1):21-36; discussion 36-27.
88. Maleux G, Verslype C, Heye S, Wilms G, Marchal G, Nevens F. Endovascular shunt reduction in the management of transjugular portosystemic shunt-induced hepatic encephalopathy: preliminary experience with reduction stents and stent-grafts. *AJR* 2007; 188(3):659-664.
89. Saket RR, Sze DY, Razavi MK, et al. TIPS reduction with use of stents or stent-grafts. *J Vasc Interv Radiol* 2004; 15(7):745-751.
90. Sze DY, Hwang GL, Kao JS, et al. Bidirectionally adjustable TIPS reduction by parallel stent and stent-graft deployment. *J Vasc Interv Radiol* 2008; 19(11):1653-1658.
91. Kamath PS, Lacerda M, Ahlquist DA, McKusick MA, Andrews JC, Nagorney DA. Gastric mucosal responses to intrahepatic portosystemic shunting in patients with cirrhosis. *Gastroenterology* 2000; 118(5):905-911.
92. Cura M, Cura A, Suri R, El-Merhi F, Lopera J, Kroma G. Causes of TIPS dysfunction. *AJR* 2008; 191(6):1751-1757.
93. LaBerge JM, Somberg KA, Lake JR, et al. Two-year outcome following transjugular intrahepatic portosystemic shunt for variceal bleeding: results in 90 patients. *Gastroenterology* 1995; 108(4):1143-1151.
94. Rosemurgy AS, Serafini FM, Zweibel BR, et al. Transjugular intrahepatic portosystemic shunt vs. small-diameter prosthetic H-graft portacaval shunt: extended follow-up of an expanded randomized prospective trial. *J Gastrointest Surg* 2000; 4(6):589-597.
95. Angermayr B, Cejna M, Koenig F, et al. Survival in patients undergoing transjugular intrahepatic portosystemic shunt: ePTFE-covered stentgrafts versus bare stents. *Hepatology* 2003; 38(4):1043-1050.
96. Barrio J, Ripoll C, Banares R, et al. Comparison of transjugular intrahepatic portosystemic shunt dysfunction in PTFE-covered stent-grafts versus bare stents. *Eur J Radiol* 2005; 55(1):120-124.
97. Biecker E, Roth F, Heller J, Schild HH, Sauerbruch T, Schepke M. Prognostic role of the initial portal pressure gradient reduction after TIPS in patients with cirrhosis. *Eur J Gastroenterol Hepatol* 2007; 19(10):846-852.
98. Bureau C, Pagan JC, Layrargues GP, et al. Patency of stents covered with polytetrafluoroethylene in patients treated by transjugular intrahepatic portosystemic shunts: long-term results of a randomized multicentre study. *Liver Int* 2007; 27(6):742-747.
99. Charon JP, Alaeddin FH, Pimpalwar SA, et al. Results of a retrospective multicenter trial of the Viatorr expanded polytetrafluoroethylene-covered stent-graft for transjugular intrahepatic portosystemic shunt creation. *J Vasc Interv Radiol* 2004; 15(11):1219-1230.
100. Jung HS, Kalva SP, Greenfield AJ, et al. TIPS: comparison of shunt patency and clinical outcomes between bare stents and expanded polytetrafluoroethylene stent-grafts. *J Vasc Interv Radiol* 2009; 20(2):180-185.
101. Vignali C, Bargellini I, Grosso M, et al. TIPS with expanded polytetrafluoroethylene-covered stent: results of an Italian multicenter study. *AJR* 2005; 185(2):472-480.
102. Bureau C, Garcia-Pagan JC, Otal P, et al. Improved clinical outcome using polytetrafluoroethylene-coated stents for TIPS: results of a randomized study. *Gastroenterology* 2004; 126(2):469-475.
103. Green MH, Duell RM, Johnson CD, Jamieson NV. Haemobilia. *Br J Surg* 2001; 88(6):773-786.
104. Ammori BJ, Madan M, Alexander DJ. Haemorrhagic complications of pancreatitis: presentation, diagnosis and management. *Ann R Coll Surg Engl* 1998; 80(5):316-325.
105. Etienne S, Pessaux P, Tuech JJ, et al. Hemosuccus pancreaticus: a rare cause of gastrointestinal bleeding. *Gastroenterol Clin Biol* 2005; 29(3):237-242.
106. Suter M, Doenz F, Chapuis G, Gillet M, Sandblom P. Haemorrhage into the pancreatic duct (Hemosuccus pancreaticus): recognition and management. *Eur J Surg* 1995; 161(12):887-892.
107. Akpinar H, Dicle O, Ellidokuz E, et al. Hemosuccus pancreaticus treated by transvascular selective arterial embolization. *Endoscopy* 1999; 31(2):213-214.
108. Lygidakis NJ, Okazaki M, Damtsios G. Iatrogenic hemobilia: how to approach it. *Hepatogastroenterology* 1991; 38(5):454-457.
109. Sakorafas GH, Sarr MG, Farley DR, Que FG, Andrews JC, Farnell MB. Hemosuccus pancreaticus complicating chronic pancreatitis: an obscure cause of upper gastrointestinal bleeding. *Langenbecks Arch Surg* 2000; 385(2):124-128.
110. Balthazar EJ, Fisher LA. Hemorrhagic complications of pancreatitis: radiologic evaluation with emphasis on CT imaging. *Pancreatology* 2001; 1(4):306-313.
111. Koizumi J, Inoue S, Yonekawa H, Kunieda T. Hemosuccus pancreaticus: diagnosis with CT and MRI and treatment with transcatheter embolization. *Abdom Imaging* 2002; 27(1):77-81.
112. van Rooyen W, van Blankenstein M, Eeftinck Schattenkerk M, et al. Haemorrhage from the pancreatic duct: a rare form of upper gastrointestinal bleeding. *Br J Surg* 1984; 71(2):137-140.
113. Dasgupta R, Davies NJ, Williamson RC, Jackson JE. Haemosuccus pancreaticus: treatment by arterial embolization. *Clin Radiol* 2002; 57(11):1021-1027.
114. Moodley J, Singh B, Lalloo S, Pershad S, Robbs JV. Non-operative management of haemobilia. *Br J Surg* 2001; 88(8):1073-1076.
115. Koren M, Kinova S, Bedeova J, Javorka V, Kovacova E, Kekenak L. Hemosuccus pancreaticus. *Bratisl Lek Listy* 2008; 109(1):37-41.
116. Massani M, Bridda A, Caratozzolo E, Bonariol L, Antoniutti M, Bassi N. Hemosuccus pancreaticus due to primary splenic artery aneurysm: a diagnostic and therapeutic challenge. *Jop* 2009; 10(1):48-52.
117. Sugiki T, Hatori T, Imaizumi T, et al. Two cases of hemosuccus pancreaticus in which hemostasis was achieved by transcatheter

arterial embolization. *J Hepatobiliary Pancreat Surg* 2003; 10(6):450-454.
118. Boudghene F, L'Hermine C, Bigot JM. Arterial complications of pancreatitis: diagnostic and therapeutic aspects in 104 cases. *J Vasc Interv Radiol* 1993; 4(4):551-558.
119. Dossa CD, Pipinos, II, Shepard AD, Ernst CB. Primary aortoenteric fistula: Part II. Primary aortoesophageal fistula. *Ann Vasc Surg* 1994; 8(2):207-211.
120. Saers SJ, Scheltinga MR. Primary aortoenteric fistula. *Br J Surg* 2005; 92(2):143-152.
121. Pipinos, II, Carr JA, Haithcock BE, Anagnostopoulos PV, Dossa CD, Reddy DJ. Secondary aortoenteric fistula. *Ann Vasc Surg* 2000; 14(6):688-696.
122. Hollander JE, Quick G. Aortoesophageal fistula: a comprehensive review of the literature. *Am J Med* 1991; 91(3):279-287.
123. Ihama Y, Miyazaki T, Fuke C, et al. An autopsy case of a primary aortoenteric fistula: a pitfall of the endoscopic diagnosis. *World J Gastroenterol* 2008; 14(29):4701-4704.
124. Hagspiel KD, Turba UC, Bozlar U, et al. Diagnosis of aortoenteric fistulas with CT angiography. *J Vasc Interv Radiol* 2007; 18(4):497-504.
125. Hughes FM, Kavanagh D, Barry M, Owens A, MacErlaine DP, Malone DE. Aortoenteric fistula: a diagnostic dilemma. *Abdom Imaging* 2007; 32(3):398-402.
126. Perks FJ, Gillespie I, Patel D. Multidetector computed tomography imaging of aortoenteric fistula. *J Comput Assist Tomogr* 2004; 28(3):343-347.
127. Roos JE, Willmann JK, Hilfiker PR. Secondary aortoenteric fistula: active bleeding detected with multi-detector-row CT. *Eur Radiol* 2002; 12 Suppl 3:S196-200.
128. Vu QD, Menias CO, Bhalla S, Peterson C, Wang LL, Balfe DM. Aortoenteric fistulas: CT features and potential mimics. *Radiographics* 2009; 29(1):197-209.
129. Mylona S, Ntai S, Pomoni M, Kokkinaki A, Lepida N, Thanos L. Aorto-enteric fistula: CT findings. *Abdom Imaging* 2007; 32(3):393-397.
130. Low RN, Wall SD, Jeffrey RB, Jr., Sollitto RA, Reilly LM, Tierney LM, Jr. Aortoenteric fistula and perigraft infection: evaluation with CT. *Radiology* 1990; 175(1):157-162.
131. Orton DF, LeVeen RF, Saigh JA, et al. Aortic prosthetic graft infections: radiologic manifestations and implications for management. *Radiographics* 2000; 20(4):977-993.
132. Baril DT, Carroccio A, Ellozy SH, et al. Evolving strategies for the treatment of aortoenteric fistulas. *J Vasc Surg* 2006; 44(2):250-257.
133. Bergqvist D, Bjorck M, Nyman R. Secondary aortoenteric fistula after endovascular aortic interventions: a systematic literature review. *J Vasc Interv Radiol* 2008; 19(2 Pt 1):163-165.
134. Brountzos EN, Vasdekis S, Kostopanagiotou G, et al. Endovascular treatment of a bleeding secondary aorto-enteric fistula. A case report with 1-year follow-up. *Cardiovasc Intervent Radiol* 2007; 30(5):1037-1041.
135. Busuttil SJ, Goldstone J. Diagnosis and management of aortoenteric fistulas. *Semin Vasc Surg* 2001; 14(4):302-311.
136. Karkos CD, Vlachou PA, Hayes PD, Fishwick G, Bolia A, Naylor AR. Temporary endovascular control of a bleeding aortoenteric fistula by transcatheter coil embolization. *J Vasc Interv Radiol* 2005; 16(6):867-871.
137. Leonhardt H, Mellander S, Snygg J, Lonn L. Endovascular management of acute bleeding arterioenteric fistulas. *Cardiovasc Intervent Radiol* 2008; 31(3):542-549.
138. Papacharalambous G, Skourtis G, Saliveros A, et al. Endovascular treatment of a primary aortoduodenal fistula: 2-year follow-up of a case report. *Vasc Endovascular Surg* 2007; 41(3):265-270.
139. Suzuki S, Imoto K, Uchida K, Hashiyama N, Takanashi Y. Endovascular repair of a presumed aortoduodenal fistula. *Ann Thorac Cardiovasc Surg* 2005; 11(6):424-428.
140. Verhey P, Best A, Lakin P, Nachiondo J, Petersen B. Successful endovascular treatment of aortoenteric fistula secondary to eroding duodenal stent. *J Vasc Interv Radiol* 2006; 17(8):1345-1348.
141. Finch L, Heathcock RB, Quigley T, Jiranek G, Robinson D. Emergent treatment of a primary aortoenteric fistula with N-butyl 2-cyanoacrylate and endovascular stent. *J Vasc Interv Radiol* 2002; 13(8):841-843.