

Duodenal variceal bleeding successfully treated with transjugular intrahepatic portosystemic shunt: A case report and review of the literature

Duodenal varis kanamasının transjugüler intrahepatik portosistemik şant ile başarılı tedavisi: Olgu sunumu ve literatür taraması

Tan ATTİLA¹, Kenneth J. KOLBECK², Zachary M. BLAND², Amy WANG¹, Sarah A. RODRIGUEZ¹

Division of ¹Gastroenterology and Hepatology, Dotter Interventional Institute, Department of ²Radiology, Oregon Health and Science University, Oregon, USA

Duodenal varices are uncommon causes of gastrointestinal bleeding. In the literature, treatment of duodenal variceal bleeding is limited to case reports and case series. Treatment options are endoscopic, interventional radiologic and surgical modalities. In this article, we presented a case of a successful duodenal variceal bleeding treatment with transjugular intrahepatic portosystemic shunt. The other treatment modalities are also reviewed.

Duodenal varisler nadir gastrointestinal sistem kanama sebeplerinden biridir. Literatürde duodenal varis kanama tedavisi vaka taktimleri veya vaka serileriyle sınırlıdır. Tedaviler endoskopik, girişimsel radyolojik ve cerrahi tedaviler olarak üçe ayrılır. Bu makalede duodenal varis kanama vakasının transjugüler intrahepatik portosistemik şant ile başarılı tedavisi takdir edilmektedir. Diğer tedavi yöntemleri hakkında da bilgi sunulmaktadır.

Key words: Duodenal varices, TIPS

Anahtar kelimeler: Duodenal varis, TIPS

INTRODUCTION

Duodenal varices are ectopic portosystemic shunts and are uncommon causes of gastrointestinal bleeding. Initially reported in 1931 by Alberti (1), the medical literature regarding the complications of duodenal varices and subsequent therapy has been mostly limited to case reports and small case series. Duodenal variceal bleeding in the absence of esophageal varices is an even rarer manifestation of portal hypertension. We report a case of isolated duodenal variceal bleeding that was successfully treated with transjugular intrahepatic portosystemic shunt (TIPS) placement.

CASE REPORT

A 78-year-old Caucasian female with a history of hypertension, hyperlipidemia, peptic ulcer disease and cirrhosis secondary to non-alcoholic steatohepatitis presented to a local hospital with a large maroon-colored bowel movement without any as-

sociated nausea, vomiting or abdominal pain. She previously had no complications due to cirrhosis other than mild ascites. There was no history of gastrointestinal bleeding. She denied any history of alcohol use. Her outpatient medications included metoprolol 50 mg QD, aspirin 162 mg QD, lasix 10 mg QD, and spironolactone 25 mg QD. She did not have any family history of liver disease or cirrhosis. At presentation, her complete blood count and blood chemistries were as follows: hematocrit 26.7% (normal: 37-47%), platelet count 213 K/mm³ (normal: 150-400 K/mm³), total bilirubin 1.6 mg/dl (normal: 0.2-1.2 mg/dl), alkaline phosphatase 39 IU/L (normal: 42-121 IU/L), alanine aminotransferase 14 IU/L (normal: 10-60 IU/L), aspartate aminotransferase 18 IU/L (normal: 12-45 IU/L), total protein 4.6 g/dl (normal: 6.2-8.2 g/dl), albumin 1.9 g/dl (normal: 3.5-5 g/dl), blood urea nitrogen 27 mg/dl (normal: 7-22 mg/dl), cre-

atinine 0.7 mg/dl (normal: 0.5-1.2 mg/dl), and international normalized ratio (INR) of 1.3. Despite receiving two units of packed red blood cells, her repeat hematocrit dropped to 22.2% then increased to 31.3% after three additional units. She was started on pantoprazole and octreotide drip as well as the antibiotic ceftriaxone. An emergent esophagogastroduodenoscopy (EGD) at a local hospital revealed a large bleeding duodenal lesion of unclear etiology. There was some initial concern that this lesion could represent the major duodenal papilla. Three ml of 1/10,000 epinephrine was injected around the lesion as a temporizing measure. However, given the appearance of the lesion, degree of recent blood loss, and high risk for recurrent bleeding, the patient was transferred to our institution for further management. Upon arrival, the patient had maroon colored stools and hematemesis. Her heart rate ranged from 80-110 beats/min and systolic blood pressure averaged 80-90 mmHg. Physical examination was significant for tachycardia, hyperactive bowel sounds with epigastric tenderness and no stigmata of chronic liver disease. An emergent EGD revealed an isolated dome-shaped lesion at the second portion of the duodenum with normal pink overlying mucosa. However, at the apex of the dome, there was a small dimple (similar in appearance to an ulcer crater) (Figure 1). No esophageal or gastric varices were seen, and there was no evidence of

portal hypertensive gastropathy or other potential source of bleeding. During the initial evaluation, the lesion was not bleeding. The endoscopic examination was completed to the distal duodenum, which appeared unremarkable. Upon withdrawal of the endoscope, non-pulsatile steady stream bleeding was observed originating from the lesion (Figure 2). The endoscopic appearance of the lesion was consistent with a duodenal varix. Given its large size, the lesion was not felt to be amenable to endoscopic therapy. The patient underwent an emergent interventional radiological evaluation. Celiac and superior mesenteric arteriograms failed to demonstrate arterial extravasation or any other arterial abnormality. With the delayed images from the superior mesenteric artery injection (evaluating the superior mesenteric vein and portal venous system), a large portal varix was identified extending over the region of the duodenum and flowing towards the inferior vena cava through collaterals. Given this finding in the setting of a normal arteriogram, the procedure was converted to a TIPS placement. Ultrasound-guided right internal jugular venous access was obtained. The right hepatic vein was selected with a 5 Fr catheter. An occlusion balloon was advanced into the right hepatic vein and inflated. A CO₂ digital subtraction portal venogram was obtained, identifying a suitable target. Access to the portal vein was obtained with a Rosch-Uchida TIPS set (Cook,

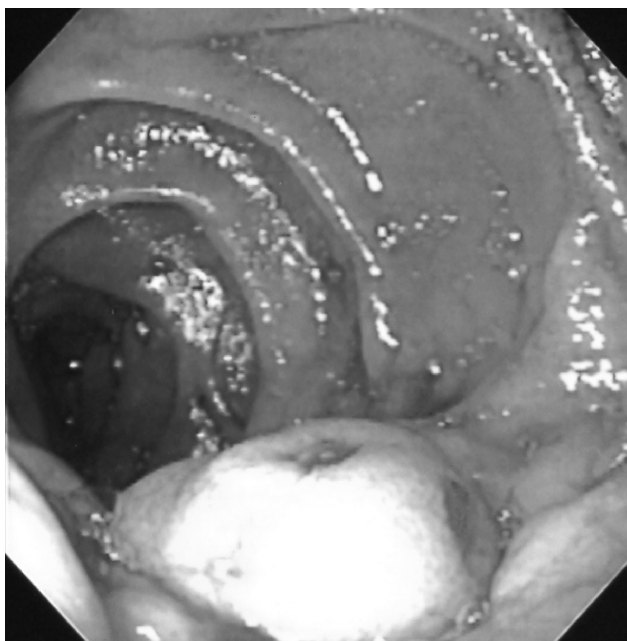


Figure 1. Isolated dome-shaped lesion in the second portion of the duodenum with an apical dimple.

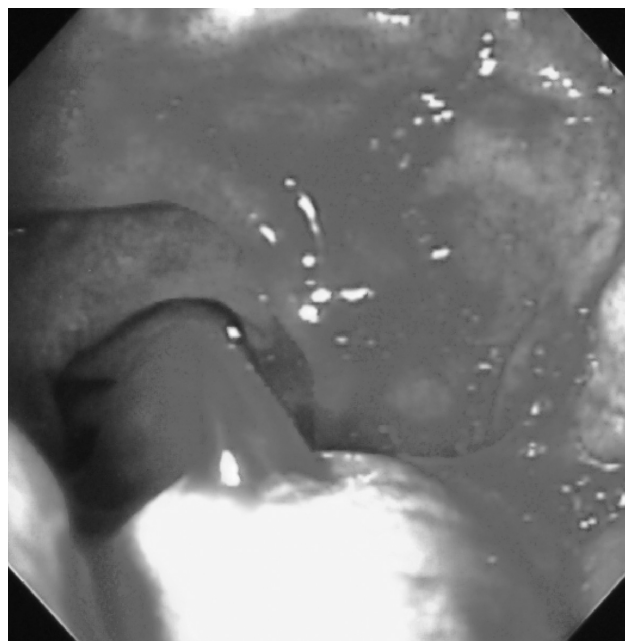


Figure 2. Active high-flow, non-pulsatile bleeding from the duodenal varix.

Bloomington, Indiana). The initial portal systemic gradient was 17 mmHg (normal: 6-8 mmHg). The TIPS tract was created in the standard fashion and a 10 Fr sheath was advanced through the intrahepatic tract into the main portal vein. A 10 mm diameter x 80 mm long (60 mm covered) stent graft was placed between the right portal vein and right hepatic vein. The stent graft was angioplastied to 8 mm in diameter. Post-TIPS, the portosystemic gradient dropped to 4 mmHg with brisk antegrade flow through the TIPS into the inferior vena cava. There was significantly reduced flow through the paraduodenal varix with routine injection of contrast into the portal vein (Figure 3). To further evaluate the source of bleeding, the paraduodenal portal varix was catheterized and digital subtraction angiographic evaluation was performed (Figure 4), without an occlusion balloon in place. Given the post TIPS gradient and significantly altered flow through the portal system, the decision was made not to embolize the varix at this time. The patient was observed over the next several days and monitored for repeat bleeding. The patient had an uneventful hospital course and was discharged home in stable condition. Direct embolization of the varix was not required. The patient did not have any recurrent bleeding four months following TIPS placement, and her liver function tests remained stable following the procedure.

DISCUSSION

Cirrhosis is the most common cause of portal hypertension. Varices are dilated portosystemic

venous collateral vessels that form as a direct consequence of portal hypertension. Varices most commonly develop in the gastroesophageal region. Approximately one-third of patients with varices experience hemorrhage (2), which is regarded as the most severe and morbid complication of end-stage liver disease. Six-week post-variceal hemorrhage mortality is approximately 30% (3).

Ectopic varices are large portosystemic venous collaterals occurring anywhere in the abdomen except in the cardia and esophagus (4). They are reported to occur in various locations, such as the gastric fundus, body and antrum of the stomach, duodenum, intra- and extrahepatic biliary tree, adjacent to surgically created enteric stoma, small bowel, colon, anorectum, intra-peritoneum, vesicle and vagina (5-11). Bleeding ectopic varices account for less than 5% of all variceal hemorrhages (12).

The prevalence of duodenal varices is not well known. Although duodenal varices were present in 40% of patients with portal hypertension who had angiographic evaluation (13), endoscopic incidence of duodenal varices was found to be 0.4% in a nine-year follow-up of patients with portal hypertension (14). This discrepancy is due to the serosal and submucosal location of duodenal varices (15), which limits visualization during endoscopy. The duodenal varices are reported to occur mostly in portal hypertension caused by cirrhosis and portal vein thrombosis (16). Less commonly, duodenal varices were noted with superior mesenteric vein stenosis (17), splenic vein obstruction

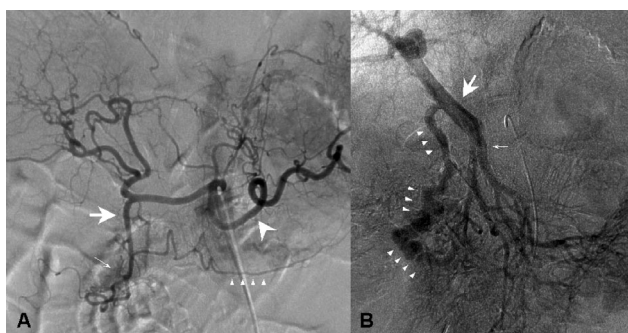


Figure 3. (A) Digital subtraction arteriogram of the celiac axis demonstrates a normal gastroduodenal artery (large arrow), gastropiploic artery (arrowheads), and branches of the superior pancreaticoduodenal vessels (small arrow). The splenic artery (white arrowhead) is also identified. (B) Venous phase of the superior mesenteric arteriogram demonstrates the superior mesenteric vein (small arrow) and duodenal variceal pathway (white arrowheads).



Figure 4. Opening portal venogram demonstrates a patent portal vein (large arrow), small gastroesophageal varices (small arrow), and the pathway of the duodenal varix. After TIPS placement, there is preferential flow through the shunt and no filling of the duodenal varix. (B) The pathway is clearly demonstrated with an occlusion balloon (small arrow) obstructing the outflow of the portal vein (large arrow), redirecting flow through the duodenal variceal pathway (white arrowheads) and back of the inferior vena cava.

and liver fibrosis due to schistosomiasis (18). Duodenal varices were also reported after successful treatment of esophageal varices (19,20). Vascular anatomical studies of the duodenal varices revealed that they originate from the superior or inferior pancreaticoduodenal vein and drain into the inferior vena cava (21). Duodenal variceal hemorrhage can present as profuse melena, hematochezia, or hematemesis. Hemorrhage from the ruptured varices may potentially be fatal (22). Currently, there are no data available for primary prophylaxis of duodenal varices, but beta-blockers have been tried to prevent rebleeding, despite the lack of supporting evidence.

Due to the infrequency of duodenal variceal bleeding, there are no randomized studies on the management of this life-threatening condition, and medical literature on treatment options is limited to case reports or small series. Given the lack of data, no therapeutic modality has been favored over another. Therefore, management of acute duodenal variceal bleeding is best dictated by the local expertise and availability of resources.

Treatment options in duodenal variceal bleeding include endoscopic procedures (band ligation, sclerotherapy, clipping), interventional radiological procedures (TIPS), percutaneous transhepatic obliteration (PTO), trans-ileocolic vein obliteration (TIO), balloon-occluded retrograde transvenous obliteration (BRTO), and surgery (variceal ligation, duodenal resection, and extra-hepatic portosystemic shunt creation).

Endoscopic therapies include mechanical therapies (band ligation) and injection therapies (sclerotherapy with sclerosants or tissue adhesives). Mixed success with these therapies has been reported. Although the efficacy of endoscopic band ligation of esophageal varices is well established, its use in duodenal variceal bleeding is limited to only case reports. The first successful endoscopic duodenal variceal band ligation was reported more than a decade ago (24); however, this patient died of liver failure two days later. In another patient, initial hemostasis was achieved with variceal band ligation, followed by BRTO one month later (25). There are a few reports of successful treatment of duodenal variceal bleeding with endoscopic band ligation alone (26-30) or in combination with sclerotherapy (31).

Various sclerosants and tissue adhesives have been used in the management of duodenal variceal

bleeding. N-butyl-2-cyanoacrylate is a tissue adhesive that rapidly polymerizes upon contact with blood and embolizes the varix. It has been used to achieve hemostasis in patients with gastrointestinal variceal bleeding (33-37), and specifically has been successful in primary endoscopic hemostasis or secondary therapy of duodenal varices following failure of other endoscopic approaches (38-42). However, cyanoacrylate injection is not without risks. The reported complications are pulmonary emboli, portal and splenic vein thrombosis, cerebrovascular accidents, and recurrent bleeding following cast extrusion and impaction of the injector needle within the varix (43-48). This agent is currently not approved for the treatment of varices in the US. The success of various sclerosants such as ethanolamine oleate, sodium morrhuate, absolute alcohol and thrombin to achieve hemostasis of duodenal variceal hemorrhage has been mixed (49-54).

In addition to the lack of reported evidence, endoscopic intervention in duodenal varices can be more challenging than conventional treatment of esophageal varices. Inherent duodenal anatomy can make identifying the extent of the varix and maintaining full visualization of the lesion difficult. Furthermore, the relative thinness of the duodenal wall compared to the other parts of the upper gastrointestinal tract makes this region more at risk for perforation and complications (23). The failure to band the entire varix may potentially create a wide defect of the variceal wall after sloughing of the band. Given the usual proximity to the ampulla, it is also crucial to avoid papilla within the banded tissue to prevent biliary obstruction (32).

Interventional radiological therapy is the next step for patients with duodenal varices that are not amenable to or fail endoscopic therapies. TIPS placement reduces the pressure gradient between portal and systemic circulation. It is a less-invasive alternative to the surgical portacaval shunt surgeries, and is a hemodynamic equivalent of the side-to-side small-diameter portacaval shunt. Given the high morbidity and mortality of emergency surgical shunt placement in patients with decompensated cirrhosis, TIPS is a superior option in these patients, even in emergent situations (55). Furthermore, since the portal anatomy is not altered, TIPS placement does not preclude a patient from getting a liver transplantation. Rebleeding of duodenal varices secondary to the occlusi-

on of TIPS (62) and paradoxical cerebral emboli after TIPS placement for duodenal varices (63) have been noted in the literature.

There is robust evidence on the role of TIPS in the management of varices (56). However, as with the other treatment modalities, experience in the use of TIPS in duodenal variceal bleeding is limited only to case reports (57-61). Haskal et al. (57) retrospectively evaluated TIPS for the management of intestinal varices. Nine patients with small bowel varices, five of whom had active bleeding, underwent a TIPS procedure. Hemostasis was achieved in all patients with active bleeding. Only one patient had recurrence of bleeding 24 hours after the TIPS placement, which was controlled with coil embolization.

Another interventional radiologic option is BRTO. First reported in 1996 (64), BRTO is now accepted as a novel therapeutic modality for the treatment of gastric varices (65, 66). In this procedure, a balloon catheter is inserted into the shunt, occluding the outflow. After the blockage of blood flow with balloon inflation, a sclerosant agent is injected in a retrograde manner to fill the ectopic varix, resulting in the disappearance of the dilated vessel. Recently, BRTO has been successfully used in the management of duodenal varices (67-74); however, the procedure can also result in the emergence of esophageal varices.

Similar to other therapeutic modalities, the experience in the surgical treatment of duodenal variceal bleeding is also limited to case reports and case series (75-77). In a literature review, the un-

derlying cause of the majority of 16 surgically treated patients with duodenal varices was portal hypertension due to alcoholic cirrhosis (76). Four of the six patients who had surgical variceal ligation rebled and required surgical shunt placement. Three of five patients who had duodenal resection rebled and underwent surgical shunt placement. Since most of the patients who had surgical shunt placement did not rebleed, the authors concluded that end-to-side portacaval shunt was the most effective procedure in stopping acute and subsequent bleeding in patients with duodenal varices (76). Simple oversewing of duodenal varices through a duodenotomy was recently described as a novel treatment (77). However, effective endoscopic and interventional radiological methods have essentially replaced surgical shunt placement for variceal hemorrhage. The surgical procedure has become a third option and "last resort" for patients with variceal bleeding refractory to other therapies.

In conclusion, there is no consensus as to the best modality to treat bleeding duodenal varices. The treatment method depends on the local expertise. In our case, given the size of the duodenal varix and unavailability of cyanoacrylate in the US, TIPS placement was the preferred method to acutely control duodenal variceal hemorrhage. The patient did not have any recurrent bleeding as of the four-month follow-up after TIPS placement. TIPS placement is an effective modality for acute and long-term treatment of duodenal variceal bleeding.

REFERENCES

1. Alberti W. Über den roentgenologixhen nachweis von varizen in buolbus duodeni. *Forsch Geb Rontgenstr* 1931;43:60-5
2. Grace ND. Prevention of initial variceal hemorrhage. *Gastroenterol Clin North Am* 1992;21:149-61.
3. de Franchis R, Primignani M. Natural history of portal hypertension in patients with cirrhosis. *Clin Liver Dis* 2001;5:645-63.
4. Norton ID, Andrews JC, Kamath PS. Management of ectopic varices. *Hepatology* 1998;28:1154-8.
5. Chen WC, Hou MC, Lin HC, et al. An endoscopic injection with N-butyl-2-cyanoacrylate used for colonic variceal bleeding: a case report and review of the literature. *Am J Gastroenterol* 2000;95:540-2.
6. Freed JS, Schuchmacher PH, Bluestone L, Fano A. Massive colonic variceal bleeding secondary to abnormal splenic collaterals: report of a case. *Dis Colon Rectum* 1978; 21:126-7.
7. Kotfila R, Trudeau W. Extraesophageal varices. *Dig Dis* 1998;16:232-41.
8. Cutler CS, Rex DK, Lehman GA. Enteroscopic identification of ectopic small bowel varices. *Gastrointest Endosc* 1995;41:605-8.
9. Paquet KJ, Lazar A, Bickhart J. Massive and recurrent gastrointestinal hemorrhage due to jejunal varices in an afferent loop--diagnosis and management. *Hepatogastroenterology* 1994;41:276-7.
10. Getzlaff S, Benz CA, Schilling D, Riemann JF. Enteroscopic cyanoacrylate sclerotherapy of jejunal and gallbladder varices in a patient with portal hypertension. *Endoscopy* 2001;33:462-4.
11. Bruet A, Fingerhut A, Lopez Y, et al. Ileal varices revealed by recurrent hematuria in a patient with portal hypertension and Mekong Schistosomiasis. *Am J Gastroenterol* 1983;78:346-50.
12. Stiegmann GV. Diagnosis and management of ectopic varices. *J Gastroenterol Hepatol* 2004;19:168-73.
13. Stephen G, Miething R. Roentgendagnostik varicoser duodenal-veränderungen bei portaler hypertension. *Radiologie* 1968;8:90.

14. Hashizume M, Tanoue K, Ohta M, et al. Vascular anatomy of duodenal varices: angiographic and histopathological assessments. *Am J Gastroenterol* 1993;88:1942-5.
15. Sukigara M, Koyama I, Komazaki T, et al. Bleeding varices located in the second portion of the duodenum. *Jpn J Surg* 1987;17:130-5.
16. Lebrech D, Benhamou JP. Ectopic varices in portal hypertension. *Clin Gastroenterol* 1985;14:105-21.
17. Yen HH, Chen YY, Wu HK. Clinical challenges and images in GI. Image 2. *Gastroenterology* 2007;133:747-1058.
18. Al-Mofarreh M, Al-Moagel-Alfarag M, Ashoor T, Shadochy F. Duodenal varices. Report of 13 cases. *Z Gastroenterol* 1986;24:673-80.
19. Eleftheriadis E. Duodenal varices after sclerotherapy for esophageal varices. *Am J Gastroenterol* 1988;83:439-41.
20. Wu WC, Wang LY, Yu FJ, et al. Bleeding duodenal varices after gastroesophageal varices ligation: a case report. *Kaohsiung J Med Sci* 2002;18:578-81.
21. Hashizume M, Tanoue K, Ohta M, et al. Vascular anatomy of duodenal varices: angiographic and histopathological assessments. *Am J Gastroenterol* 1993;88:1942-5.
22. Amin R, Alexis R, Korzis J. Fatal ruptured duodenal varix: a case report and review of literature. *Am J Gastroenterol* 1985;80:13-8.
23. Desai RK, Tagliabue JR, Wegryn SA, Einstein DM. CT evaluation of wall thickening in the alimentary tract. *Radiographics* 1991;11:771-83; discussion 784.
24. Tazawa J, Sakai Y, Koizumi K, et al. Endoscopic ligation for ruptured duodenal varices. *Am J Gastroenterol* 1995;90:677-8.
25. Haruta I, Isobe Y, Ueno E, et al. Balloon-occluded retrograde transvenous obliteration (BRTO), a promising nonsurgical therapy for ectopic varices: a case report of successful treatment of duodenal varices by BRTO. *Am J Gastroenterol* 1996;91:2594-7.
26. Tan NC, Ibrahim S, Tay KH. Successful management of a bleeding duodenal varix by endoscopic banding. *Singapore Med J* 2005;46:723-5.
27. Bosch A, Marsano L, Varilek GW. Successful obliteration of duodenal varices after endoscopic ligation. *Dig Dis Sci* 2003; 48: 1809-12.
28. Shiraishi M, Hiroyasu S, Higa T, et al. Successful management of ruptured duodenal varices by means of endoscopic variceal ligation: report of a case. *Gastrointest Endosc* 1999;49:255-7.
29. Yeh YY, Hou MC, Lin HC, et al. Case report: successful obliteration of a bleeding duodenal varix using endoscopic ligation. *J Gastroenterol Hepatol* 1998;13:591-3.
30. Fayad N, Nammour F, Elfant A. Endoscopic variceal ligation for bleeding duodenal varices. *J Clin Gastroenterol* 2004;38:467.
31. Omata F, Itoh T, Shibayama Y, et al. Duodenal variceal bleeding treated with a combination of endoscopic ligation and sclerotherapy. *Endoscopy* 1998;30:62-3.
32. Silberzweig JE, Atillasoy EO, Sheiner PA, Mitty HA. Biliary obstruction caused by endoscopic band ligation of a duodenal varix. *Am J Gastroenterol* 1997;92:1060-2.
33. D'Imperio N, Piemontese A, Baroncini D, et al. Evaluation of undiluted N-butyl-2-cyanoacrylate in the endoscopic treatment of upper gastrointestinal tract varices. *Endoscopy* 1996;28:239-43.
34. Soehendra N, Grimm H, Nam VC, Berger B. N-butyl-2-cyanoacrylate: a supplement to endoscopic sclerotherapy. *Endoscopy* 1987;19: 221-4.
35. Marques P, Maluf-Filho F, Kumar A, et al. Long-term outcomes of acute gastric variceal bleeding in 48 patients following treatment with cyanoacrylate. *Dig Dis Sci* 2008;53:544-50.
36. Rengstorff DS, Binmoeller KF. A pilot study of 2-octyl cyanoacrylate injection for treatment of gastric fundal varices in humans. *Gastrointest Endosc* 2004;59:553-8.
37. Greenwald BD, Caldwell SH, Hespenheide EE, et al. N-2-butyl-cyanoacrylate for bleeding gastric varices: a United States pilot study and cost analysis. *Am J Gastroenterol* 2003;98:1982-8.
38. Ota K, Shirai Z, Masuzaki T, et al. Endoscopic injection sclerotherapy with n-butyl-2-cyanoacrylate for ruptured duodenal varices. *J Gastroenterol* 1998;33:550-5.
39. Yoshida Y, Imai Y, Nishikawa M, et al. Successful endoscopic injection sclerotherapy with N-butyl-2-cyanoacrylate following the recurrence of bleeding soon after endoscopic ligation for ruptured duodenal varices. *Am J Gastroenterol* 1997;92:1227-9.
40. Onozato Y, Kakizaki S, Iizuka H, et al. Ectopic varices rupture in the gastroduodenal anastomosis successfully treated with N-butyl-2-cyanoacrylate injection. *Acta Med Okayama* 2007; 61: 361-5.
41. Paterlini A, Rolfi F, Buffoli F, et al. Endoscopic treatment of a bleeding duodenal varix using N-butyl-2-cyanoacrylate. *Endoscopy* 1993; 25: 434.
42. Sato H, Fujishiro H, Rumi MA, et al. Successful endoscopic injection sclerotherapy for duodenal varices. *J Gastroenterol* 2002; 37: 143-4.
43. Alexander S, Korman MG, Sievert W. Cyanoacrylate in the treatment of gastric varices complicated by multiple pulmonary emboli. *Intern Med J* 2006; 36: 462-5.
44. Al Hamad A, Kabbani A, Al Kadhi Y. N-butyl-2-cyanoacrylate (Histoacryl) complication: a case report. *Ann Saudi Med* 2006; 26: 71-2.
45. Shim CS, Cho YD, Kim JO, et al. A case of portal and splenic vein thrombosis after Histoacryl injection therapy in gastric varices. *Endoscopy* 1996; 28: 461.
46. See A, Florent C, Lamy P, et al. Cerebrovascular accidents after endoscopic obturation of esophageal varices with isobutyl-2-cyanoacrylate in 2 patients. *Gastroenterol Clin Biol* 1986; 10: 604-7.
47. Huang YH, Yeh HZ, Chen GH, et al. Endoscopic treatment of bleeding gastric varices by N-butyl-2-cyanoacrylate (Histoacryl) injection: long-term efficacy and safety. *Gastrointest Endosc* 2000; 52: 160-7.
48. Wu WC, Wang LY, Yu FJ, et al. Bleeding duodenal varices after gastroesophageal varices ligation: a case report. *Kaohsiung J Med Sci* 2002; 18: 578-81.
49. Barbish AW, Ehrinpreis MN. Successful endoscopic injection sclerotherapy of a bleeding duodenal varix. *Am J Gastroenterol* 1993; 88: 90-2.
50. Sauerbruch T, Weinzierl M, Dietrich HP, et al. Sclerotherapy of a bleeding duodenal varix. *Endoscopy* 1982; 14: 187-9.
51. Rai R, Panzer SW, Miskovsky E, Thuluvath PJ. Thrombin injection for bleeding duodenal varices. *Am J Gastroenterol* 1994; 89: 1871-3.
52. Sans M, Llach J, Bordas JM, et al. Thrombin and ethanolamine injection therapy in arresting uncontrolled bleeding from duodenal varices. *Endoscopy* 1996; 28: 403.
53. Wang CS, Jeng LB, Chen MF. Duodenal variceal bleeding-successfully treated by mesocaval shunt after failure of sclerotherapy. *Hepatogastroenterology* 1995; 42: 59-61.
54. Jonnalagadda SS, Quiason S, Smith OJ. Successful therapy of bleeding duodenal varices by TIPS after failure of sclerotherapy. *Am J Gastroenterol* 1998; 93: 272-4.
55. Boyer TD. Transjugular intrahepatic portosystemic shunt: current status. *Gastroenterology* 2003; 124: 1700-10.
56. Boyer TD, Haskal ZJ. American Association for the Study of Liver Diseases. The role of transjugular intrahepatic portosystemic shunt in the management of portal hypertension. *Hepatology* 2005; 41: 386-400.

57. Haskal ZJ, Scott M, Rubin RA, Cope C. Intestinal varices: treatment with the transjugular intrahepatic portosystemic shunt. *Radiology* 1994; 191: 183-7.
58. Sort P, Elizalde I, Llach I, et al. Duodenal variceal bleeding treated with a transjugular intrahepatic portosystemic shunt. *Endoscopy* 1995; 27: 626-7.
59. Jonnalagadda SS, Quiason S, Smith OJ. Successful therapy of bleeding duodenal varices by TIPS after failure of sclerotherapy. *Am J Gastroenterol* 1998; 93: 272-4.
60. Illuminati G, Smail A, Azoulay D, et al. Association of transjugular intrahepatic portosystemic shunt with embolization in the treatment of bleeding duodenal varix refractory to sclerotherapy. *Dig Surg* 2000; 17: 398-400.
61. Almeida JR, Trevisan L, Guerrazzi F, et al. Bleeding duodenal varices successfully treated with TIPS. *Dig Dis Sci* 2006; 51: 1738-41.
62. McChesney L, Jensen D, Matalon T, et al. Duodenal varices: a case report and review of the literature. *HPB Surgery* 1995; 9: 31-5.
63. Ponc RJ, Kowdley KV. Paradoxical cerebral emboli after transjugular intrahepatic portosystemic shunt and coil embolization for treatment of duodenal varices. *Am J Gastroenterol* 1997; 92: 1372-3.
64. Kanagawa H, Mima S, Kouyama H, et al. Treatment of gastric fundal varices by balloon-occluded retrograde transvenous obliteration. *J Gastroenterol Hepatol* 1996; 11: 51-8.
65. Cho SK, Shin SW, Lee IH, et al. Balloon-occluded retrograde transvenous obliteration of gastric varices: outcomes and complications in 49 patients. *AJR Am J Roentgenol* 2007; 189: W365-72.
66. Hiraga N, Aikata H, Takaki S, et al. The long-term outcome of patients with bleeding gastric varices after balloon-occluded retrograde transvenous obliteration. *J Gastroenterol* 2007; 42: 663-72.
67. Tanaka O, Ohno K, Ohno T, et al. Should balloon-occluded retrograde transvenous obliteration be the first-line interventional radiologic treatment for bleeding duodenal varices? A case report and review of the literature. *Acta Radiol* 2008; 49: 32-6.
68. Haruta I, Isobe Y, Ueno E, et al. Balloon-occluded retrograde transvenous obliteration (BRTO), a promising nonsurgical therapy for ectopic varices: a case report of successful treatment of duodenal varices by BRTO. *Am J Gastroenterol* 1996; 91: 2594-7.
69. Ohta M, Yasumori K, Saku M, et al. Successful treatment of bleeding duodenal varices by balloon-occluded retrograde transvenous obliteration: a transjugular venous approach. *Surgery* 1999; 126: 581-3.
70. Sonomura T, Horihata K, Yamahara K, et al. Ruptured duodenal varices successfully treated with balloon-occluded retrograde transvenous obliteration: usefulness of microcatheters. *AJR Am J Roentgenol* 2003; 181: 725-7.
71. Akazawa Y, Murata I, Yamao T, et al. Successful management of bleeding duodenal varices by endoscopic variceal ligation and balloon-occluded retrograde transvenous obliteration. *Gastrointest Endosc* 2003; 58: 794-7.
72. Takamura K, Miyake H, Mori H, et al. Balloon occluded retrograde transvenous obliteration and percutaneous transhepatic obliteration for ruptured duodenal varices after operation for rectal cancer with multiple liver metastasis: report of a case. *J Med Invest* 2005; 52: 212-7.
73. Tsurusaki M, Sugimoto K, Matsumoto S, et al. Bleeding duodenal varices successfully treated with balloon-occluded retrograde transvenous obliteration (B-RTO) assisted by CT during arterial portography. *Cardiovasc Intervent Radiol* 2006; 29: 1148-51.
74. Zamora CA, Sugimoto K, Tsurusaki M, et al. Endovascular obliteration of bleeding duodenal varices in patients with liver cirrhosis. *Eur Radiol* 2006; 16: 73-9.
75. Wang CS, Jeng LB, Chen MF. Duodenal variceal bleeding successfully treated by mesocaval shunt after failure of sclerotherapy. *Hepatogastroenterology* 1995; 42: 59-61.
76. Khouqeer F, Morrow C, Jordan P. Duodenal varices as a cause of massive upper gastrointestinal bleeding. *Surgery* 1987; 102: 548-52.
77. Cottam DR, Clark R, Hayn E, Shaftan G. Duodenal varices: a novel treatment and literature review. *Am Surg* 2002; 68: 407-9.