Identification of Perforators in Patients with Duodenal Varices by Endoscopic Ultrasound—A Case Series [with video]

Malay Sharma*, Pazhanivel Mohan**, Chittapuram S. Rameshbabu†, Venkataraman Jayanthi‡

*Jaswant Rai Speciality Hospital, Saket, Meerut 250 001, Uttar Pradesh, **Department of Gastroenterology, PSG Institute of Medical Sciences and Research, Coimbatore, Tamil Nadu, †L.L.R.M., Medical College, Meerut, Uttar Pradesh, and ‡Stanley Medical College Hospital, Chennai, Tamil Nadu, India

Background/Aims: Duodenal varices (DV) are ectopic varices which can cause massive gastrointestinal bleeding. The diagnosis of DV may be difficult; sometimes they can be hidden behind duodenal folds. The aim of the study was to evaluate DV by endoscopic ultrasound. **Methods:** Endoscopic ultrasound was done in patients detected or suspected to be having DV. The para duodenal varices were identified and subsequently hemodynamic evaluation of DV was done. **Results:** Endoscopic ultrasound identified perforators in seven cases of DV. **Conclusion:** The endoscopic ultrasound can help in detection of DV underlying thickened folds. It can also help in hemodynamic evaluation of DV. (J CLIN EXP HEPATOL 2012;2:229–237)

Duodenal varices (DV) account for 1–3% of all varices in patients with portal hypertension.¹ Angiography has revealed DV in up to 43% of patients with portal hypertension; however, in contrast with esophageal varices, bleeding complications are relatively rare. The rarity of bleeding from DV may be related to their smaller diameter, shorter length and deeper location on the outer wall of the duodenum (paraduodenal varices).^{2–4} Paraduodenal varices (PDV) can communicate through perforators with vascular channels in the submucosa of the duodenum which gradually enlarge over a period of time to form DV. In the submucosa, DV may be endoscopically inevident initially, appearing as thickened duodenal folds, but generally become endoscopically evident before they bleed. Bleeding from DV is generally severe *per se*, with a mortality rate of up to 40%.

Afferents to DV are related to pancreaticoduodenal venous arcades which are in communication with the portal venous system (Figure 1).⁵ Efferents from DV reveal two different patterns. In cirrhosis, the DV efferents are formed in the descending or transverse parts of the duodenum and

flow hepatofugally via retroperitoneal shunts, also called veins of Retzius, into the inferior vena cava (IVC) (Figure 2a). In extra hepatic portal vein obstruction (EHPVO) DV efferents are formed in the duodenal bulb and flow hepatopetally via portoportal collaterals into the liver.^{6–11} Portoportal collaterals develop from either preformed venous plexuses near the common bile duct (CBD) or from patent tributaries of the portal vein (PV) above the occluded part of the portal venous system (Figure 2b).¹² DV are formed when the dilated veins of Retzius or portoportal collaterals cannot accommodate the increased flow of blood in portal hypertension.^{13,14}

Confirmation of DV as the source of bleeding requires a high index of suspicion, careful inspection of the duodenal bulb, second, third and fourth parts of the duodenum and repeated endoscopies.¹ Endoscopic ultrasound (EUS) has been shown to be more sensitive than conventional endoscopy at detecting DV and portoportal collaterals related to pancreaticoduodenal venous arcades.^{15–17} Once a diagnosis is made, endoscopic management is often an initial therapeutic option but re-bleeding may occur, which may result in mortality.^{18,19} In such cases surgical management or interventional radiological procedures may be required.²⁰ Endoscopic and interventional radiological procedures may be combined to control bleeding and to reduce the probability of recurrence.^{21–27} Surgery may be preferred in selected cases.^{5,7}

The effective management of DV depends on confirmation of the diagnosis (thickened duodenal fold or DV), evaluation of site (bulb, descending or horizontal duodenum), size (assessed by endoscopy and EUS) and hemodynamic assessment (HDA) of pathways by CT scan, MRI and, occasionally, by EUS.^{28,29} Noninvasive HDA of DV is important

Keywords: duodenal varices, endoscopic ultrasound, portal hypertension, endoscopic variceal ligation

Received: 24.4.2012; Accepted: 8.8.2012; Available online: 13.8.2012

Address for Correspondence: Dr. Malay Sharma, Gastroenterologist, Jaswant Rai Speciality Hospital, Saket, Meerut 250 001, Uttar Pradesh, India. Tel.: +91 9837031148 (mobile)

E-mail: sharmamalay@hotmail.com

Abbreviations: DV: duodenal varices; PDV: paraduodenal varices; IVC: inferior vena cava; EHPVO: extra hepatic portal vein obstruction; CBD: common bile duct; PV: portal vein; EUS: Endoscopic ultrasound; HDA: hemodynamic assessment; SMV: superior mesenteric vein; EVL: endoscopic variceal ligation; ECD: epicholedochal; PCD: para-choledochal

<http://dx.doi.org/10.1016/j.jceh.2012.07.004>

Figure 1 Venous drainage of duodenum and Afferent channels of duodenal varices. Four small pancreaticoduodenal veins drain the head of the pancreas. There are two superior and two inferior pancreaticoduodenal veins. The posterior superior pancreaticoduodenal vein joins the portal vein. The anterior superior pancreaticoduodenal vein is related to either superior mesenteric vein or one of two major tributaries of superior mesenteric vein. The two major tributaries of superior mesenteric vein are the gastroduodenal trunk and the first jejunal trunk which join the SMV roughly at the same level but on opposite sides. IVC = inferior vena cava, CA = celiac artery, HA = hepatic artery, SA = splenic artery, GDA = gastroduodenal artery, SMA = superior mesenteric artery, ASPDV = anterior superior pancreaticoduodenal vein, PV = portal vein, PSPDV = posterior superior pancreaticoduodenal vein, SV = splenic vein, LRV = left renal vein, LRA = left renal artery, CBD = common bile duct, RGEV = right gastroepiploic vein, RCV = right colic vein, RRV = right renal vein, RRA = right renal artery, GT = gastroduodenal trunk, 1st JT = first Jejunal trunk, PIPDV = posterior inferior pancreaticoduodenal vein, AIPDV = anterior inferior pancreaticoduodenal vein, SMV = superior mesenteric vein, IPDV = inferior pancreaticoduodenal vein, Middle Colic vein, DV = duodenal Varix.

before definitive therapy is undertaken, to increase the efficacy of therapy and to avoid systemic complications. The utility of a single noninvasive imaging modality may be limited as it may not provide complete HDA and a combination of imaging modalities may be required before offering definitive therapy. Normal portal venous anatomy has been defined in detail in humans and evaluated by endoscopic ultrasound.³⁰⁻³² Pathways of portal collateral circulation in mammals with portal hypertension have been classified on CT scan.³³ EUS has shown perforators in esophageal, gastric and rectal walls.^{16,17,34} Only a few studies have utilized noninvasive imaging modalities to provide HDA of DV. In this series, we report demonstration of perforators and attempt to define the collateral pathways in patients with DV using EUS.

PATIENTS AND METHODS

One hundred twenty four consecutive patients with portal hypertension seen between January 2009 and March 2012

underwent repeated sessions of upper GI endoscopy. DV were suspected in 16 patients; endoscopic appearance was quite suggestive of DV in four. Definite DV on endoscopic observation were classified into three types: tortuous (F1), nodular (F2), and tumorous (F3). Endoscopic findings suspicious of DV included submucosal tumor like appearance and thickened duodenal folds. All patients with appearances suggestive or suspicious of DV underwent endosonography with a radial EUS scope (Pentax EG 3630 UR) in the same session to study the duodenal and periduodenal vascular system. Endoscopic and the corresponding endosonographic findings were documented and correlated. Color Doppler EUS of the course of superior mesenteric vein (SMV), PV and their tributaries was done from duodenum. PDV were identified either anterior or posterior to the wall of duodenum (Figure 3). DV in the submucosal layer of the duodenal wall were identified after filling the duodenum with 50 to 100 ml water. Perforators were identified as the communication between the DV and the PDV. Inflowing perforators were identified as flow signals toward the probe (red color) and outflowing perforators were identified as flow signals away from the probe (blue color).

Variceal banding was done in patients with clinical and endoscopic evidence of bleeding from the DV. Multiple bands were applied from below upwards. The first band was placed on the most distal and the most prominent point of DV and subsequent bands were applied cranially. Follow up endoscopy was done 3-7 days after banding and follow up endoscopy was done after 1 and 3 or 6 months for bleeders.

Proper informed consent was obtained from all patients prior to endosonographic evaluation. The study protocol had been approved by the Institutional Ethics Committee.

RESULTS

A total of seven cases with DV were identified. The clinical, endoscopic and EUS features of the patients are given in Table 1. Endoscopic appearances were classical in three and suspicious of DV in four patients. One had tortuous DV (Figure 4a & b, Video 1), another had nodular DV along with antral varices (Figure 5a & b, Video 2) and the third had tumorous DV in the bulb (Figure 6a & b, Video 3). Of the remaining four cases one had an appearance of submucosal tumor in the bulb (Figure 7a & b, Video 4) and three had thickened duodenal folds (Figures 8 & 9, Videos 5-7).

On EUS, DV, ranging from 3 to 6 mm diameter, and PDV were identified in all the seven cases. PDV related to inflowing perforators were identified in five cases near the posterior wall of duodenum and in two cases near the anterior wall of duodenum while outflowing perforators were seen only in five cases. One case had two inflowing perforators coming from anterior and posterior

Figure 2 Efferent channels of duodenal varices. a: Hepatofugal channels of duodenal varices. The efferents from DV can be hepatopetal or hepatofugal. The figure shows hepatofugal pathways from the duodenal varices which can go to following tributaries. 1. Right renal vein*, 2. right gonadal vein*, 3. lumbar veins, 4. iliac vein*, 5. IVC = inferior vena cava, 6. right supra renal vein, 7. right inferior phrenic vein, 8. right ascending lumbar vein, 9. right subcostal vein, 10. azygos vein, 11. tributary of right renal vein–right inferior adrenal vein*. The veins marked by stars (*) have been demonstrated to act as hepatofugal collaterals of duodenal varices. b: Hepatopetal channels from duodenal varices. Hepatopetal pathways from the duodenal varices going toward liver can include tributaries or branches of portal vein above the blocked part of portal venous system or pericholedochal veins. Pericholedochal veins consist of two groups of epicholedochal (ECD) venous plexus of Saint and para-choledochal (PCD) venous plexus of Petren which provide bidirectional flow and venous drainage of bile duct and can also act as efferent channels of the DV with predominant hepatopetal flow. 1. Left gastric vein*, 2. right gastric vein, 3. posterior superior pancreaticoduodenal vein, 4. cystic vein, 5. paraumbilical vein, 6. paracholedochal venous plexus, 7. epicholedochal venous plexus, 8. right portal vein branch, 9. left portal vein branch, 10. gastroduodenal trunk, 11. first jejunal vein, 12. gallbladder wall varix, 13. splenic vein, 14. superior mesenteric vein. The veins marked by stars (*) have been demonstrated to act as efferent channels of duodenal varices that drain into portal system and establish a hepatopetal flow of DV. The places marked by arrows point out to the possible places of communication via the venous plexus of Petren.

paraduodenal varices. One case had inflowing and outflowing perforator in the same location. Three patients had EHPVO with cavernous transformation of portal vein and predominant flow was noted toward the liver hilum. In two cases the predominant flow could be identified toward IVC and in two cases no assessment of direction of flow was possible. The exact venous tributary into which the efferent vessel drained could not be identified in any case.

Variceal banding was done in four patients with history of GI bleeding while beta-blockers were used in three cases with no history of bleeding (Figure 10). Three bands were applied in three cases and two bands were applied in one case. During banding, the first band was applied on the most distal part of the DV while subsequent bands were applied more cranially, if needed. In one patient with varices extended into the antrum requiring additional banding in the antrum where four bands were placed. (Figure 11) In one patient with thickened duodenal folds, exact banding

site was difficult to find because of narrow view offered by ligator and two bands were applied in anterior wall of duodenum. In this case, follow up EUS after 4 days showed absence of signal in duodenal wall. Follow up endoscopy done after 3 or 6 months showed absence of varices in three patients with endoscopically evident DV at the index procedure.

DISCUSSION

DV is a submucosal lesion which can be accurately diagnosed by endoscopy in only 44% of cases since they lack the red color signs usually seen in EV.^{35,36} In some cases CT scan has highlighted retroperitoneal varices.^{19,29} EUS has been found to be more sensitive than conventional endoscopy for detecting submucosal varices in stomach and duodenum.^{9,15,37} In the present series, endoscopic findings were classical of DV in 3 cases and suspicious in four (submucosal lesion in one and thickened duodenal folds

Figure 3 Endoscopic ultrasound probe lies close to the medial part of the head of pancreas in duodenum. From this position the SMV and SMA lie anterior to the uncinate process of head of pancreas and aorta and IVC lie posterior to the head of pancreas. In this position the aorta and IVC lie in a plane posterior to pancreas and the SMV and SMA lie anterior to pancreas. An inverted V shape position is commonly achieved where the right limb of v is formed by aorta or IVC and the left limb is formed by SMV or SMA. The uncinate process of pancreas and the pancreatic duct lie between the two limbs. In this position the paraduodenal collaterals can be identified near the anterior or posterior surface of pancreas close to the respective pancreaticoduodenal venous plexuses. An imaginary blue line divides the approximate anterior and posterior location of the paraduodenal varices when imaging is done from duodenum. On EUS an attempt can be made to individually trace the smaller tributary which can act as the afferent vessel to the DV. The figure shows the arrangement of tributaries of SMV as they contribute to the formation of venous plexuses. As the superior mesenteric vein crosses the uncinate process, it is joined by the gastrocolic trunk, first jejunal trunk and the middle colic vein. Gastrocolic trunk joins the right antero-lateral surface and first jejunal trunk joins the left posterolateral surface of the superior mesenteric vein roughly at the same level but on opposite sides. Middle colic vein may drain either into the anterior surface of SMV or drain into the gastrocolic trunk before joining the superior mesenteric vein. The surface of SMV lying close to the scope is the posterior surface on which no tributary joins. The surface lying away from the scope is the anterior surface on which middle colic vein may join. The first jejunal vein lies in between the aorta and SMA and goes anteriorly toward the SMV to join its left border whereas the gastrocolic trunk joins the right border. 1. Superior mesenteric vein, 2. Gastrocolic trunk, 3. Anterior superior pancreaticoduodenal vein, 4. Right colic vein, 5. Right gastroepiploic vein, 6. Middle colic vein, 7. 1st jejunal trunk, 8. Inferior pancreaticoduodenal vein, Pan = pancreas, CBD = common bile duct, IVC = inferior vena cava.

in three). EUS confirmed the presence of DV in all the 7 cases, suggesting that EUS is superior to conventional endoscopy in detection of DV and that frequency of DV may be higher than reported in literature.^{38,39}

EUS is useful for noninvasive HDA of the portal venous system.⁴⁰ EUS has demonstrated perforators in esophageal, gastric, rectal and CBD wall.^{16,17,34,41} Although angiography has demonstrated paraduodenal varices in

portal hypertension, duodenal perforators have not been demonstrated so far. Their demonstration is important as their obliteration is associated with decreased rebleeding rates in patients with esophageal varices.⁴² Unlike arterial embolization for gastrointestinal hemorrhage, the goal of therapy in patients with bleeding ectopic varices is to occlude the feeding vein on the portal venous side rather than only to occlude the bleeding site.^{24,43} Obliteration of duodenal perforators should be a suitable goal and end point of endotherapy for successful treatment of DV and can be achieved easily in the thin walled duodenum. After the obliteration of duodenal perforators, collateral flow in PDV, which are generally not obliterated by endotherapy, can continue to transmit some of the pressure away from the DV toward the systemic veins.⁴⁴ Thus, obliterating DV close to the inflowing duodenal perforators may be more effective in control of acute bleeding and in prevention of rebleeding in patients with DV. In our series inflow reduction was achieved as was seen by disappearance of DV on the endoscopic aspect in four cases. A repeat EUS was not considered necessary to establish inflow disappearance.

Endoscopic procedures are less invasive, quicker and easier when compared to surgery or interventional radiological procedures.^{44,45} Endoscopic sclerotherapy can cause severe liver damage if the flow of DV is hepatopetal.⁴⁶ Glue injection carries a high risk of thromboembolic complications and endoscopic variceal ligation (EVL) is considered unsafe for DV larger than 15 mm.⁴⁵ Extensive DV have been shown to be difficult to control with EVL alone and can often result in mortality.⁴⁷ During this evaluation, the maximum size of bleeding DV measured on EUS was 6 mm in transverse axis and 16 mm. in tangential axis. On real time evaluation of the videos (Videos 1–7) it appeared that in these cases a single tortuous varix gave a false impression of large diameter. Banding was safely done in four cases. As the HDA in these cases had shown inflowing duodenal perforators more distally in the duodenum multiple bands could be applied from below upwards on the tortuous course of varix. In one case the inflowing and outflowing perforator was seen on the same place where blood was demonstrated during endoscopy under a thickened duodenal fold. Banding was done at two places around this thickened fold of duodenum (Figure 10). Successful application of multiple bands for DV has been reported by others also.⁴⁸

In the current case series three patients had EHPVO and DV in bulb and D1 whereas four patients had cirrhosis and DV mainly in D2, an observation similar to other studies.⁴⁹ One patient had unusual varices extending from antrum to D2. One of the three patients with EHPVO had outgoing perforators going toward liver hilum. Outgoing perforators were seen in three of four patients with cirrhosis. Two had hepatofugal flow toward the IVC and one had flow toward gallbladder varices. In two cases the direction

Table 1 Clinical, endoscopic and endoscopic ultrasound features of seven cases of duodenal varices.

Details	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7
Age/sex	35/m	40/m	50/m	38/F	23/M	55/m	35/m
Cause of portal hypertension	HCV	HBV	EHPVO	EHPVO	ALD	ALD	ALD + EHPVO
Presenting symptom	Recurrent melena	UGI bleed	UGI bleed	Follow up case on EVL	Follow up case on EVL	Follow up case on EVL	Follow up case on EVL
Therapy offered number of bands	Multiple EVL (3)	Multiple EVL + antral banding (3 in duodenum + 4 in antrum)	EVL (3)	Beta blocker	Beta blocker	Beta blocker	EVL (2)
Endoscopy finding and form	Serpiginous varices F1	Tortuous varices F2	Tumorous varix F3	Appearance of submucosal tumor	Thickened duodenal folds	Thickened duodenal folds	Thickened duodenal folds
Associated Vx	Obliterated EV	Obliterated EV + Antral Vx	EV Gr 1	EV Gr 2	EV Gr 3	EV Gr 1	Obliterated EV & rectal varices
Past H/o treatment	Underwent EVL for EV	Underwent EVL for EV	Undergoing EVL for OV	Undergoing EVL + EIS	Undergoing EVL	Undergoing EVL	Undergoing EVL
Paraduodenal vessel	Present	Present	Present	Present	Present	Present	Present
Site of DV	D2	D1, D2	D1	Bulb	D2	D2	D1
Tangential diameter of DV (mm)	5	3	4	3	6	6	6
Transverse diameter of DV (mm)	10	12	16	12	15	15	15
Number of perforators	2	1 + 1	1	1	1 + 1	1 + 1	1 + 1
Type of perforator inflowing/outflowing	Both inflowing	1 Inflowing and 1 outflowing	Inflowing	Outflowing	1 Inflowing and 1 outflowing	1 Inflowing and 1 outflowing	1 Inflowing and 1 outflowing
Position of afferent vein in duodenal wall	1 Anterior and 1 posterior	1 Posterior	1 Posterior	Not identified	1 Posterior	1 Anterior	Same location of inflow and outflow perforator
Position of Efferent veins hepatopetal/hepatofugal	Not identified	Hepatopetal	Not identified	Hepatopetal	Hepatofugal	Hepatofugal	Not identified

HCV = Hepatitis C virus, HBV = Hepatitis B virus, ALD = Alcoholic liver disease, EHPVO = Extra hepatic portal vein obstruction, EVL = Endoscopic variceal ligation, EIS = Injection sclerotherapy, D1 = First part of duodenum, D2 = Second part of duodenum, EV = Esophageal varices, Gr = Grade.

Figure 4 The 35-year-old man having chronic liver diseases due to hepatitis C virus had undergone successful obliteration of esophageal varices by EVL. He came with complain of recurrent melena. Upper GI endoscopy showed obliterated esophageal varices and bluish serpiginous multiple varices in duodenum. (a) He was transfused with 4 units of packed cells and had massive upper GI bleed during hospitalization. Repeat endoscopy showed fresh blood in duodenum. An endoscopic ultrasound showed DV and inflowing perforators. (b) He underwent banding of duodenal varices.

of flow could not be identified. Partial HDA was thus possible in five cases. The information about HDA can be of importance to the radiologist who requires a roadmap of draining tributaries before offering interventional radiological procedures and may not be able to offer any therapy if DV have a predominant hepatopetal flow without availability of veins of Retzius or without patency of portal vein.

Endotherapy was done for four cases of bleeding DV and three cases were given beta blockers. Although guidelines exist for treatment of bleeding esophageal and gastric varices, there is no unanimity of opinions with regard to treatment of DV.⁵⁰ While a case may be made for prophylactic endoscopic therapy for endoscopically evident DV, further data from follow up studies are required to establish the natural history of endoscopically in evident DV which have been demonstrated by EUS in this series and the need for definitive intervention in them. HDA done in this series can identify the

course of shunt and be helpful in evaluating the role of pharmacotherapy for inflow reduction to the DV. Inflow reduction has been achieved temporarily by vaso-pressin or somatostatin in DV and more effectively by the use of nitroglycerin induced hypotension in management of bleeding from intracholedochal varices related to pancreaticoduodenal veins.^{7,46} Its efficacy in DV remains to be determined.

In conclusion role of EUS in the evaluation of ectopic varices is still emerging and bleeding from ectopic varices continue to pose a challenge to clinicians.⁵⁰ DV are ectopic varices which may be endoscopically in evident in a significant number of cases. While EUS with use of color Doppler detects the presence of DV, their presence may not be an indication for treatment in all cases. The presence of DV below thickened folds mandates caution during duodenal biopsies in portal hypertension. Identification of perforators provides insight into the formation of DV and may allow a choice of

Figure 5 A 40-year-old man having cirrhosis due to hepatitis B virus undergoing banding for esophageal varices came with complain of upper GI bleed. Upper GI endoscopy showed obliterated esophageal varices, prominent antral varices (a) and tortuous duodenal varices in bulb and duodenum with presence of fresh blood. An endoscopic ultrasound showed DV and perforators. (b) The patient underwent multiple banding of antral and DV.

Figure 6 A 50-year-old man a known case of portal hypertension due to EHPVO for the last 15 years. He has received multiple blood transfusions and recently acquired hepatitis B infection also. Upper GI endoscopy showed a tumorous varix in duodenal bulb. (a) Initially it appeared to be an infiltrative lesion. An endoscopic ultrasound showed DV almost occupying the entire wall of duodenum and inflowing perforator (b). Banding of the varix was done.

Figure 7 A 38-year-old lady who has been undergoing EVL for esophageal varices came for routine follow up. Upper GI endoscopy revealed obliterated varices in esophagus and an elevated submucosal area in bulb (a). An endoscopic ultrasound showed DV and perforators (b). The patient was kept on beta blockers.

Figure 8 A 23-year-old man (case no 5) with alcoholic cirrhosis was undergoing EVL. An endoscopy 6 months back had shown obliterated varices in esophagus. Upper GI endoscopy as a part of follow up showed grade three esophageal varices and significantly thickened duodenal folds which did not disappear on distension of duodenum with air.

Figure 9 A 35-year-old man a known case of EHPVO with obliterated varices presented with GI bleed. Endoscopy showed thickened duodenal fold and presence of blood in duodenum. Endoscopic ultrasound shows an inflowing and outflowing perforator. This figure shows bidirectional perforator in the same location in duodenum.

Figure 10 The same case as in Figure 9 endoscopic appearance after 3 days of banding.

Figure 11 The appearance in antrum in the case shown in Figure 5a and b with duodenal and antral varices 4 days after banding.

therapeutic intervention suited to obliterate DV close to inflowing perforators. HDA of DV is not done as a routine practice in management before offering definitive therapy but may be important for targeted management. The true utility of EUS for the diagnosis of DV would be in detecting DV in patients with no or little suspicion of DV and in assessing the effect of duodenal variceal ligation on DV and PDV, on obliteration of entry and exit (feeding and draining) perforators and in predicting risk of recurrence of DV.

DISCLOSURES

The authors report that there are no disclosures relevant to this publication.

CONFLICTS OF INTEREST

All authors have none to declare.

ACKNOWLEDGMENTS

We would like to thank Mr. Pran Prakash for preparing the illustrations and videos.

SUPPLEMENTARY MATERIAL

Supplementary data related to this article can be found online at <http://dx.doi.org/10.1016/j.jceh.2012.07.004>.

REFERENCES

1. Hashizume M, Tanoue K, Ohta M, et al. Vascular anatomy of duodenal varices: angiographic and histopathological assessments. *Am J Gastroenterol*. 1993;88:1942–1945.
2. Nardone G, Budillon G. Treatment of duodenal varices by endoscopic sclerotherapy. *Gastrointest Endosc*. 1991;37:407–408.
3. Stephan G, Miething R. Roentgendagnostik varicoser duodenalveränderungen bei. *Radiology*. 1968;8:90–95.
4. Khan MQ, Al-Momen S, Alghssab A. Duodenal varices causing massive lower gastrointestinal hemorrhage. *Ann Saudi Med*. 1999;19(5):440–443.
5. Cottam D, Clark R, Hayn E, Shaftan G. Duodenal varices: a novel treatment and literature review. *Am Surg*. 2002;68(5):407–409.
6. Ibukuro K, Tsukiyama T, Mori K, et al. Veins of Retzius at CT during arterial portography: anatomy and clinical importance. *Radiology*. 1998;209:793–800.
7. McAlister VC, Al-Saleh NA. Duodenal dearterialization and stapling for severe hemorrhage from duodenal varices with portal vein thrombosis. *Am J Surg*. 2005;189(1):49–52.
8. Perchik L, Max TC. Massive hemorrhage from varices of the duodenal loop in cirrhotic patient. *Radiology*. 1963;80:641–644.
9. 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(6):591–593.
10. Watanabe N, Toyonaga A, Kojima S, et al. Current status of ectopic varices in Japan: results of a survey by the Japan Society for Portal Hypertension. *Hepatol Res*. 2010;40(8):763–776.
11. Kang HK, Jeong YY, Choi JH, et al. Three-dimensional multi-detector row CT portal venography in the evaluation of portosystemic collateral vessels in liver cirrhosis. *Radiographics*. 2002;22:1053–1061.
12. Vellar ID. Preliminary study of the anatomy of the venous drainage of the intrahepatic and extrahepatic bile ducts and its relevance to the practice of hepatobiliary surgery. *ANZ J Surg*. 2001;71(7):418–422.
13. Sonomura T, Horiata K, Yamahara K, et al. Ruptured duodenal varices successfully treated with balloon-occluded retrograde transvenous obliteration: usefulness of microcatheters. *AJR*. 2003;181:725–727.
14. Tominaga K, Montani A, Kuga T, et al. Combined balloon-occluded embolization for treatment of concurrent duodenal, gastric, and esophageal varices: a case report. *Gastrointest Endosc*. 2001;53(6):665–668.
15. Irani S, Kowdley KV, Kozarek RA. Gastric varices: an updated review of management. *J Clin Gastroenterol*. 2011;45:133–148.
16. Sharma M, Pathak A. Perforators of common bile duct wall in portal hypertensive biliopathy (with videos). *Gastrointest Endosc*. 2009;70(5):1041–1043.
17. Sharma M, Pathak A. Intrahepatocholel varices in portal hypertensive biliopathy. *European J Radiol Extra*. 2009;72(3):119–123.

18. Lee MS, Cho JY, Cheon YK, et al. Use of detachable snares and elastic bands for endoscopic control of bleeding from large gastric varices. *Gastrointest Endosc.* 2002;56(1):83–88.
19. 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(5):794–797.
20. Hotta M, Yoshida H, Mamada Y. Successful management of duodenal varices by balloon-occluded retrograde transvenous obliteration. *J Nippon Med Sch.* 2008;75(1):36–40.
21. Soga K, Tomikashi K, Fukumoto K, et al. Successful endoscopic hemostasis for ruptured duodenal varices after balloon-occluded retrograde transvenous obliteration. *Dig Endosc.* 2010;22(4):329–333.
22. Nishida N, Sakai G, Morimoto A, et al. Gadolinium enhanced three-dimensional magnetic resonance portography with subtraction. *Br J Radiol.* 2001;74(878):147–152.
23. Al-Mofarreh M, Al-Moagel-Alfarag M, Ashoor T, et al. Duodenal varices: report of 13 cases. *Z Gastroenterol.* 1986;24(11):673–680.
24. Norton ID, Andrews JC, Kamath PS. Management of ectopic varices. *Hepatology.* 1998;28:1154–1158.
25. Ota K, Okazaki M, Higashihara H. Combination of transileocolic vein obliteration and balloon-occluded retrograde transvenous obliteration is effective for ruptured duodenal varices. *J Gastroenterol.* 1999;34(6):694–699.
26. Sans M, Llach J, Bordas JM, et al. Thrombin and ethanolamine injection therapy in arresting uncontrolled bleeding from duodenal varices. *Endoscopy.* 1996;28(4):403.
27. Hirochika Makino, Chikara Kunisaki, Hidenori Masui. A case of duodenal varices successfully treated by intraoperative ligation and injection sclerotherapy. *Jpn J Gastroenterol Surg.* 1999;2664–2668.
28. Almeida JR, Trevisan L, Guerrazzi F, Mesquita MA. Bleeding duodenal varices successfully treated with TIPS. *Dig Dis Sci.* 2006;51(10):1738–1741.
29. Weishaupt D, Pfammatter T, Marincek B, et al. Detecting bleeding duodenal varices with multislice helical CT. *AJR Am J Roentgenol.* 2002;178(2):399–401.
30. Sharma M, Rameshbabu CS, Garg S, et al. Portal venous system and its tributaries: a radial endosonographic assessment. *Endoscopic Ultrasound.* (in press).
31. Wataru K. Surgical anatomy of the pancreas for limited resection. *J Hepatobiliary Pancreat Surg.* 2000;7:473–479.
32. Katz MH, Fleming JB, Pisters PW, et al. Anatomy of the Superior mesenteric vein with special reference to the surgical management of first-order branch involvement at pancreaticoduodenectomy. *Ann Surg.* 2008;248(6):1098–1102.
33. Bertolini G. Acquired portal collateral circulation in the dog and cat. *Vet Radiol Ultrasound.* 2010;51(1):25–33.
34. Sharma M, Somasundaram A. Massive lower GI bleed from an endoscopically in evident rectal varices: diagnosis and management by EUS (with videos). *GIE.* 2010;72(5):1106–1108.
35. Khouqeer F, Morrow C, Jordan P. Duodenal varices as a cause of massive upper gastrointestinal bleeding. *Surgery.* 1987;102:548–552.
36. Matsui S, Kudo M, Ichikawa T, et al. The clinical characteristics, endoscopic treatment, and prognosis for patients presenting with duodenal varices. *Hepatogastroenterology.* 2008;55(84):959–962.
37. Rana SS, Bhasin DK, Singh K. Duodenal varix diagnosed by endoscopic ultrasound. *Clin Gastroenterol Hepatol.* 2010;8(12):A24.
38. Itzchak Y, Glickman MG. Duodenal varices in extrahepatic portal obstruction. *Radiology.* 1977;124:619–624.
39. Olson RW, Hodgson JR, Adson MA. The significance of duodenal deformity in patients with extrahepatic portal obstruction. *Mayo Clin Proc.* 1963;38:289.
40. El-Saadany M, Jalil S, Irisawa A, et al. EUS for portal hypertension: a comprehensive and critical appraisal of clinical and experimental indications. *Endoscopy.* 2008;40(8):690–696.
41. Sharma M, Rameshbabu CS, Dhiman RK, et al. Induced hypotension in management of acute hemobilia during therapeutic ERCP in a patient with portal biliopathy (with videos). *Gastrointest Endosc.* 2010;72(6):1317–1319.
42. McCormack TT, Rose JD, Smith PM, et al. Perforating veins and blood flow in oesophageal varices. *Lancet.* 1983;2(8365–8366):1442–1444.
43. Toubia N, Sanyal AJ. Portal hypertension and variceal hemorrhage. *Med Clin North Am.* 2008;92(3):551–574.
44. Seo YS, Kwon YD, Park S, et al. Complete eradication of duodenal varices after endoscopic injection sclerotherapy with ethanolamine oleate: a case report. *Gastrointest Endosc.* 2008;67(4):759–762.
45. Fayad N, Nammour F, Elfant A. Endoscopic variceal ligation for bleeding duodenal varices. *J Clin Gastroenterol.* 2004;5(38):467.
46. Suzuki R, Irisawa A, Hikichi T, et al. Hemorrhagic duodenal varices treated successfully with endoscopic injection sclerotherapy using cyanoacrylate and ethanolamine-oleate: a case report. *Surg Laparosc Endosc Percutan Tech.* 2009;19(6):233–236.
47. Machida T, Sato K, Kojima A, et al. Ruptured duodenal varices after endoscopic ligation of esophageal varices: an autopsy case. *Gastrointest Endosc.* 2006;63(2):352–354.
48. Trikudanathan G, Myers M, Karasik M, et al. Successful obliteration of bleeding duodenal varices using band ligation. *J Gastroenterol Hepatol.* 2011;26(1):209.
49. Kakizaki S, Toyoda M, Ichikawa T, et al. Clinical characteristics and treatment for patients presenting with bleeding duodenal varices. *Dig Endosc.* 2010;22(4):275–281.
50. Garcia-Tsao G, Sanyal AJ, Grace ND, Carey W. Practice Guidelines Committee of the American Association for the Study of Liver Diseases; Practice Parameters Committee of the American College of Gastroenterology. Prevention and management of gastroesophageal varices and variceal hemorrhage in cirrhosis. *Hepatology.* 2007;46(3):922–938.