Duodenal ulcer hemorrhage treated by embolization : results in 28 patients

J.F. De Wispelaere[1], T. De Ronde[2], J.P. Trigaux[1], L. de Cannière[3], T. De Geeter[1]

(1) Department of Radiology ; (2) Department of Gastroenterology ; (3) Department of Surgery, Cliniques Universitaires UCL de Mont-Godinne, B-5530 Yvoir, Belgium.

Abstract

Background : **To assess the effectiveness and prospects of transcatheter gastroduodenal artery embolization in the control of massive duodenal bleeding and to relate our experience.**

Methods of study : **The study is based on the retrospective analysis of 165 patients with endoscopically detected bleeding duodenal ulcer who presented between 1991-1998. 28 patients were considered eligible for endovascular treatment either at initial presentation or following hemorrhage recurrence after endoscopic therapy.**

Results **: Technical failure was noted in 3 cases, thereafter treated by surgery. In the other 25 patients, embolization was performed : bleeding recurrence occurred in 7 cases. Four were treated only endoscopically. One was reembolized and the last two were treated by surgery. In 6 cases, a coaxial technique was used (guiding catheter in 2 and 3F microcatheter in 4). No complication related to the catheterization was observed.**

Conclusion : **Transcatheter embolization of the gastroduodenal artery appears to be an efficient procedure even in the absence of active bleeding at the time of the procedure. Failure and recurrence rates can be reduced by using a coaxial technique in the uneasy cases. Embolization seems to have a low recurrence rate and a very low complication rate.** (Acta gastroenterol. belg., **2002**, 65, **6-11**).

Key words : arteries, therapeutic blockades, duodenum, hemorrhage.

Introduction

Massive hemorrhage of the upper gastrointestinal tract accounts for approximately 1.5‰ of patients admitted to emergency medical care units in large US hospitals (1). In 80% of cases, bleeding ceases spontaneously. In the remaining 20% massive bleeding requires 4 to 6 units of red blood cells per 24 h and specific emergency therapeutic management (2). Mortality from bleeding peptic ulcer is still 11% in the 90's. Until the late 1970s, 35 to 55% of patients with massive upper gastrointestinal hemorrhage underwent emergency operation and mortality rate in the pre- and postoperative period was approximately 20% (1,3). With the advent of interventional endoscopy, the emergency surgical management rate is considered as 5% (4). However, despite considerable technical advance (laser, photo- and electrocoagulation, injection of sclerosing drugs), endoscopic therapy may prove to be inefficient or technically impossible, in particular in patients with massive hemorrhage.

Percutaneous transcatheter embolization (infusion of vasopressin and now embolization of the bleeding vessel) has progressively become the appropriate complementary treatment procedure in uneasy cases (2,5). We report our experience of transcatheter gastroduodenal artery embolization in the emergency management of massive duodenal bleeding and analyse the efficiency of the procedure and its complication rate.

Material and methods

Between 1991 and 1998, 289 patients with upper gastrointestinal bleeding and pressure instability were admitted to the emergency department. All underwent diagnostic endoscopy, which demonstrated an actively bleeding gastric or duodenal ulcer or signs of previous bleeding in 165 patients (57%). According to the Forrest's classification (6), endoscopic treatment was performed in 79 of the 165 patients (Forrest Ia, Ib, IIa and IIb) (Table I). The only endoscopic treatment consists of submucosal injection of 1:10,000 epinephrine followed by absolute alcohol.

Primary endoscopic failure (huge ulcers, active bleeding with uneasy access) or non-removed blood clot as we did in the first years of this study, was observed in 13 of the 79 patients (16%) : they were immediately referred for endovascular treatment. Bleeding recurrence occurred in 15 others of these 79 patients (19%) and were also referred for possible endovascular treatment.

The data are summarised in Table II (20 males / 8 females, mean age : 69, age range : 25-88). As the vascular anatomy and the following endovascular treatment are not the same for gastric hemorrhage, we did not include them in our study. In all cases, bleeding was due to a duodenal ulcer. Mean transfusion requirement was 7 units of packed red blood cells per 24 h. Most patients had one or more associated clinical conditions (cardiac disorder in 11, severe pulmonary disease in 8, cancer in 5, diabetes mellitus in 5).

Diagnostic angiography (global angiography first, and selective celiac angiography thereafter) documented a continuous bleeding with contrast extravasation in 39% of cases. Selective angiography of the superior mesenteric artery was made secondary in only twelve patients without proof of bleeding on celiac angiography : contrast extravasation was never documented by mesenteric angiography in these 12 patients. Transcatheter embolization was attempted in all patients,

Address for correspondence and reprint requests : Jean-François De Wispelaere, M.D., Department of Radiology, Cliniques Universitaires UCL de Mont-Godinne, B-5530 Yvoir, Belgium.

Table I. — **Forrest's classification**

FORREST's classification			Prevalence	Reccurrence without treatment	Mortality
Type I active bleeding	Ia	arterial spurting	18%	90%	40%
	Ib	oozing		10%	11%
Type II recent bleeding	IIa	visible vessel (elevated)	17%	50%	11%
	IIb	adherent blood clot	17%	33%	7%
	IIc	flat pigmented spot	20%	10%	3%
Type III no bleeding	III	clean based ulcer	42%	< 5%	2%

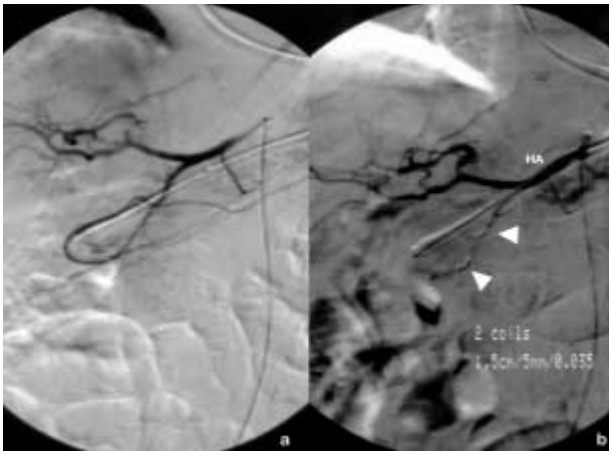

Fig. 1. — Typical embolization
a. Angiography before embolization.
b. Angiographic result immediately after proximal embolization along the gastroduodenal artery (GDA) with coils (arrowheads). The catheter stays in the common hepatic artery (HA).

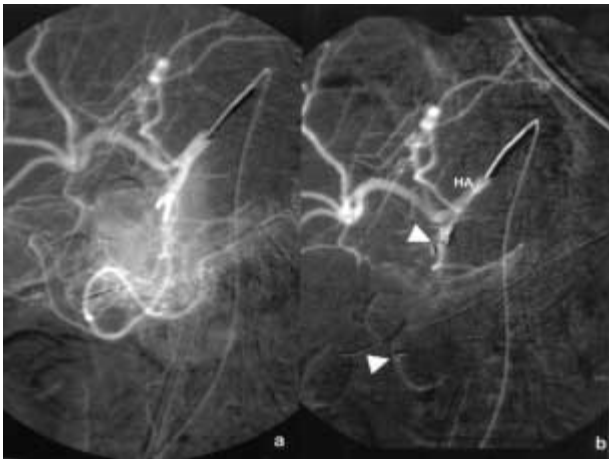

Fig. 2. — "Sandwich" embolization in another patient.
a. Angiography before embolization, with the catheter in the hepatic artery (HA).
b. Angiography immediately after. Arrowheads show the distal (in the right gastroepiploic artery) and proximal coils (in the GDA).

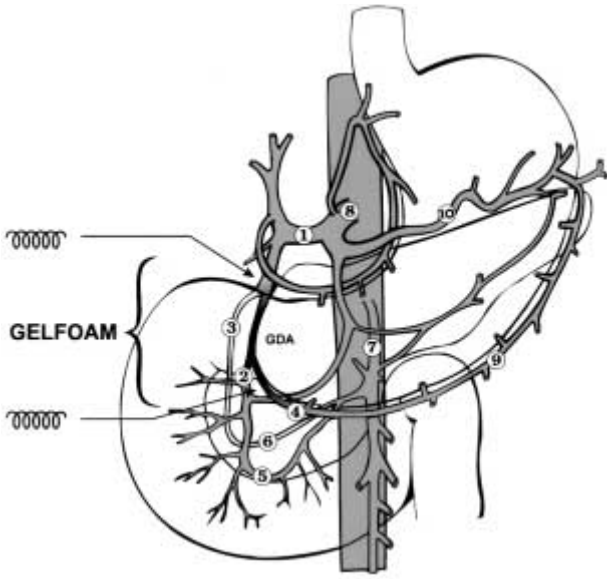

Fig. 3. — Classical anatomy and "sandwich" embolization.
GDA : gastroduodenal artery
① common hepatic artery
② pancreaticoduodenal antero-superior artery
③ pancreaticoduodenal postero-superior artery
④ right gastroepiploic artery
⑤ pancreaticoduodenal antero-inferior artery
⑥ pancreaticoduodenal postero-inferior artery
⑦ superior mesenteric artery
⑧ coeliac trunk
⑨ left gastroepiploic artery
⑩ splenic artery.
pancreaticoduodenal arcades

including those in whom active bleeding was not demonstrated at angiography. Technique selection depended on the practician's experience : (a) in three cases : superselective embolization of the proximal gastroduodenal artery (Fig. 1) first with handmade (± 1 mm diameter) gelatine sponge particles (GELFOAM®, Upjohn Company, Kalamazoo, MI, USA), and thereafter with coils (William COOK® Europe A/S Denmark) ; (b) in 7 cases coils only ; (c) in 15 other cases, we have first embolized coil(s) in the distal gastroduodenal artery or in a terminal branch (like a plug), then handmade GELFOAM® particles in the arterial trunk, and thereafter coil(s) in the proximal gastroduodenal artery (Fig. 2 and 3). If an extravasation was seen, the distal coil(s) were always placed beyond the blush. In one of these 15 cases, 350-500µ microemboli were used (polyvinyl-alcohol PVA Contours®, International Therapeutics Corp., Fremont CA, USA). We used usually flexible 4 or 5-F catheters (Surgimed MEADOX® Denmark or Glidex™ TERUMO® Japan). Four times

Table II. — **Clinical history and type of treatment of the 28 patients referred for endovascular management between 01.1991 and 12.1998**

PAT n°	Sex/Age	Underlying disease	Lowest Hb Level	Forrest	Endscopic Injection	Technique Classic	Technique Sandwich	Rebleeding (post-embolization)	Further Treatment	Final issue
1	M 52	Non steroid anti-inflam.	7.4	I b	**yes**	**failure**			surgery	OK
2	F 75	Chronic obstructive broncho-pneumopathy Arrhythmia Mitral stenosis	4.7	II b	**no** **?**	+		**yes**	surgery	OK
3	M 79	Chronic obstructive broncho-pneumopathy (corticoids) Diabetes mellitus II C/P insufficiency	8.6	II c	**no**		+	**no**	—	Death 1 month
4	F 68	Arterial hypertension	4.5	I a	**no**		+	**no**	—	OK
5	M 69	Pulmonary neoplasm Lymphatic leukemia Cirrhosis	8.5	I a	**yes**		+	**no**	—	Death 4 months
6	M 60	Mouth epidermoid esophagus cancer	6.6	I b	**no** **(huge ulcer)**	+		**no**	—	?
7	M 25	Hiatal hernia Gastric ulcer under Zantac® Aspirin for headaches	9.0	I a	yes		+	**yes**	endoscopic injection	OK
8	M 55	Transient stroke Chemotherapy for chronic myeloid leukemia 20 000 platelets	9.7	II c	no		+	**yes**	endoscopic injection	Death 6 months (CML)
9	M 77	AAA repaired	6.3	I b	yes	**failure**		**yes**	surgery	?
10	M 88	Chronic obstructive broncho-pneumopathy (corticoid) Stroke	7.2	II b	yes		+	**no**	—	OK Asymptotic pyloric stenosis
11	F 91	Cardiac infarct Atrial fibrillation Aortic stenosis Pneumonia	7.3	I b	no		+	**no**	—	Death 6 months
12	F 70	Angina post-infarct Coronary bypass	6.8	II b	yes	+		**yes**	surgery	?
13	M 85	Diabetes mellitus type II Hormonal treatment for prostatic neoplasm	5.6	I a	**no** **(uneasy access diverticulum)**	+		**no**	—	OK
14	M 72	Cardiac infarct Coronary bypass Diabetes type II Psoriasis	7.1	II b	**no** **(huge ulcer)**	+		**no**	—	?

PAT n°	Sex/Age	Underlying disease	Lowest Hb Level	Forrest	Endscopic Injection	Technique Classic	Technique Sandwich	Rebleeding (post-embolization)	Further Treatment	Final issue
15	F 60	Lymphoma 40 000 platelets Cachexia	7.8	I b	**yes**	+		**no**	—	Death d + 4
16	M 67	Bronchoemphysema Chronic hepatopathy 70 000 platelets Fraxiparine®	7.7	I b	**yes**	+		**no**	—	Death d + 12
17	M 71	Anthracosilicosis Alcoholism Carbonarcosis	5.2	I b	**yes**	+		**no**	—	Death d + 20
18	M 75	Chronic obstructive broncho-pneumopathy Right cardiac decompensation shock**o**	8.8	II a	**n** **(giant ulcer)**		+	**yes**	endoscopic injection	?
19	F 70	Polypathology Coronary bypass Corticoids for Certonciny syndrome	7.8	II a	**yes**		+	**no**	—	Death d + 30 cardiac failure
20	M 37	Psychopathology History of ulcers	6.9	II a	**yes**		+	**no**	—	OK
21	M 70	Chronic obstructive broncho-pneumopathy cortisoned	5.8	I b	**no**	+		**no**	—	Death d + 30
22	F 71	Osteosynthesis for hip fracture	6.4	I b	**yes** (twice)		+	**yes**	endoscopic injection	Death d + 10
23	M 74	Coronary bypass + valvular repair Cardiac failure	5.7	I a	**yes** (twice)		+ GDA + supraduodenal	**yes**	surgery (twice)	Death d + 16
24	M 76	15 years ago rectal carcinoma Pulmonary tumor Diabetes type II	5.9	II b	**no**		+	**no**	—	Death d + 16 Cachexia
25	M 79	Prostatectomy Cardiac failure after cardiac infarct Diabetes type II OK	6	II b	**no**		+	**no**	—	Endoscopic control : Death 3 months cardiac failure
26	F 87	Coronary bypass + aortic valve repair Renal failure Coumadine	5	I b	**yes** (twice)	+	+	**yes**	embolization	OK
27	M 56	Alcoholism	6.9	II b	**yes**		**failure**	**yes**	surgery	Fistula/MRSA OK
28	M 68	AAA repair ARDS – cardiac infarct	6.7	I b	**yes**		+	**no**	—	OK

we used a 3-F microcatheter (TRACKER® Target Therapeutic®, San Jose, CA, USA), coaxially with a 5F catheter. In two patients, when the 4 or 5-F catheter was not stable enough, we used a 8-F guiding catheter (Big-Max™ of Boston Scientific Corp.®, Watertown, MA, USA and Vistabritetip® of Cordis Corp.®, Miami, FL, USA) coaxially with the 4 or 5-F catheter. In all cases, care was taken to occlude the gastroduodenal artery as proximally as possible, in order to avoid residual patency of the posterior superior pancreaticoduodenal artery. In two patients, another vessel (a supraduodenal artery) was also selectively embolized, because it was recognised to be the site of bleeding.

Results

Technical failure was observed in 3 of the 28 patients, due to the particularly tortuous vessel course, preventing superselective catheterization and embolization of the GDA. These three patients were treated by surgery. After embolization, bleeding was controlled and the patient's condition stabilized in 18 cases. In the other 7 patients, bleeding recurred within 24 to 72 hours. Four of them, with moderate bleeding, were successfully managed with a new endoscopic procedure. Regarding the severity of the rebleeding, surgical correction had to be performed in two patients and the third one was reembolized. Thus, in five out of 28 patients (18%), surgical correction became necessary : in 3 cases due to catheterization failure and in 2 other cases because bleeding recurred massively. At the end of these various combined procedures, bleeding was controlled in all patients. In no case, death (n = 13) was due to rebleeding.

No complication related to catheterization was observed, in particular locoregional hematoma, thrombosis, or vascular embolism. However, due to the retrospective nature of our study, evolution of the renal function could not be recorded systematically in the acute phase. Middle term renal dialysis was never necessary.

Middle and long-term clinical follow-up (max. 24 months) is available in 23/28 patients ; one of these was examined with endoscopic and /or radiological procedures. In one clinically asymptomatic patient (n° 10), pyloric stenosis was detected at endoscopy (1/23 = 4%) : this patient had undergone 3-F catheterization and embolization with microemboli (PVA of 350-500µ). No other delayed complication was noted in our series.

Discussion

In the past, diagnostic arteriography for evaluation of acute upper gastrointestinal bleeding has commonly been considered as a useful method. Now, endoscopy has supplanted arteriography as the primary diagnostic modality (4,6,8). However, arteriography and embolization keep place as an alternate approach in the treatment of upper gastrointestinal bleeding, particularly after initial interventional endoscopy has failed (7).

Our series includes only patients with bleeding due to duodenal ulcer, which is the most common cause for upper gastrointestinal bleeding. Ulcers of the posterior wall are known to be more difficult to treat endoscopically. Other causes such as gastric ulcer, gastritis, peptic œsophagitis, or Mallory-Weiss tear have not been considered in the present study.

In most patients, localisation of the gastroduodenal artery was obtained by global arteriography and then by selective arteriography of the celiac trunk. It is essential to identify with great accuracy the mapping of digestive arteries in order to detect a possible anatomic variant and to prevent bleeding recurrence.

It is reported (12) that injection in the celiac trunk usually visualises active upper bleeding with a 60-80% sensitivity rate. In our series, celiac angiography demonstrated active extravasation in only 39% of cases (11/28). Superior mesenteric angiography was not systematically performed in our series, and only when the source of bleeding was not detected on celiac angiography : the aim of our procedure indeed was not to diagnose bleeding (it was achieved by endoscopy) but to treat as quickly as possible these patients in poor and haemodynamically instable conditions. If superior mesenteric angiography had been done systematically in our series, the number of documented extravasations would probably have increased. Moreover, it is now known that bleeding, even when massive, is usually intermittent probably due to variation of blood pressure (vasovagal reaction or blood loss) and/or vasospasm, and extravasation is detectable only if the arterial-bleeding rate is over 0.5 ml/min (12,13). Considering the associated clinical conditions and the presumed innocuity of the technique, embolization was attempted in the 17 patients in whom good angiographic documentation of arterial bleeding sites could not be obtained, on the basis of the results of endoscopy. In none of these cases, rebleeding or complication were observed. We therefore feel that in a patient with endoscopically documented duodenal bleeding, angiographic demonstration of active bleeding is in no way a prerequisite to embolization.

Vasopressin infusion has been widely used in the past years (14,15), but is presently being abandoned due to its rather disappointing results. Short acting occlusive agents such as e-aminocaproic acid-induced autologous clots have also been used successfully (16). The procedure seems to promise high effectiveness and may avoid ischemia but it is not performed currently in our institution.

Several teams (12,17) use Gelfoam® fragments that are absorbed within 1 to 3 week's time, often in association with steel coils (15,18). The use of excessively big Gelfoam® fragments results in a too proximal embolization that increases the risk of rebleeding due to reversal of blood flow in the right gastroepiploic artery and pancreaticoduodenal arches. In opposition, the use of Gelfoam® powder for embolization results in more

distal vascular occlusion but carries a higher risk of mucosal ischemia and secondary necrosis. Lang (19) verified this hypothesis in a series of 28 patients. He obtained control of bleeding in 27 cases with different types of embolic material (cyanoacrylates, Gelfoam® powder, and polyvinyl alcohol), but secondary duodenal stenosis occurred in 7 of 28 patients. More proximal embolization (with coils/gelatine sponge/blood clot) of the gastroduodenal artery was effective in only 25 of 29 (86%) other patients due to retrograde flow into the pancreaticoduodenal arches and gastroepiploic artery. With this technique, however, the complication rate is lower (2 out of 29), probably due to retrograde perfusion.

In our series, we used a mixture of coils and of Gelfoam fragments, at the discretion of the various angiographers, with definitive control of bleeding in 23/28 patients (82%) : by angiography alone in 19/28 patients (68%) and by angiography in conjunction with repeated endoscopy in 4/28 patients (14%). From a technical point of view, our aim was in all cases simply to occlude the gastroduodenal artery as quickly as possible to stop bleeding and to prevent reccurrence. And also as completely as possible to avoid reperfusion by collateral's (namely pancreaticoduodenal arches). From our experience, our preferred method is the "sandwich" technique (first to embolize the distal gastroduodenal artery with coils, then to occlude the trunk of the artery with Gelfoam® particles, and finally to lodge a last coil just beneath the ostium of the gastroduodenal artery).

Duodenal stenosis developed only in the patient treated with microemboli (clinically asymptomatic patient 10) ; however, it is not proved that embolization with this material was the cause of this complication : pyloric and duodenal stenosis are indeed well-recognised complications of peptic ulceration.

Our data from this retrospective study lack the statistical validity of a prospective trial, but it should be stressed that our series is rather homogeneous, as it is made of 165 consecutive patients admitted to the emergency unit with endoscopically-documented duodenal bleeding. The study is based on an emergency situation, which makes part of the everyday life of many hospitals. We therefore feel that population selection was in no way biased. It should also be stressed that our technique varied over time and consequently evolved as experience grew. As gastroenterologists accumulated more experience with radiological embolization, the number of surgical corrections in patients with postembolization rebleeding could be yet drastically reduced. According with the surgeons, in our institution, embolization is now considered as the first treatment after endoscopic failure, before surgery.

Conclusion

When the endoscopic treatment failed, we recommend that patients with bleeding duodenal ulcer be treated first with gastroduodenal embolization even when diagnostic angiography fails to demonstrate an active bleeding site, as far as it has been endoscopically documented. After reviewing our experience, this technique appears to be an efficient procedure both to stop bleeding in the acute phase and to prevent recurrence due to rich anastomotic circulation with very limited long-term ischemic complications. The use of a coaxial catheter system may possibly reduce the rate of technical failures.

References

1. GOLDMAN M.L., LAND W.C., BRADLEY E.L., ANDERSON J. Transcatheter therapeutic embolization in the management of massive upper gastrointestinal bleeding. *Radiology*, 1976, **120** : 513-521.
2. MALLORY A., SCHAEFER J.W., COHEN J.R., HOLT S.A., NORTON L.W. Selective intra-arterial vasopressin infusion for upper gastrointestinal tract hemorrage. *Arch. Surg*., 1980, **115** : 30-32.
3. VELLAKOT K.D., DRONFIELD M.W., ATKINSON M., LANGMAN M.J.S. Comparison of surgical and medical management of bleeding peptic ulcers. *Br. J. Med*., 1982, **284** : 548-550.
4. QVIST P., ARNESEN K.E., JACOBSEN C.D., ROSSELAND A.R. Endoscopic treatment and restrictive surgical policy in the management of peptic ulcer bleeding : five years'experience in a central hospital. *Scand. J. Gastroenterol*., 1994, **28** : 571-576.
5. TOYODA H., NAKANO S., KUMADA T., SUGIYAMA K., KIRIYAMA S, SUGA T. Transcatheter arterial embolization for massive bleeding from duodenal ulcers not controlled by endoscopic hemostasis. *Endoscopy*, 1995, **27** : 304-307.
6. FORREST J.A.H., FINLAYSON N.D.C., SCHEARMAN D.J.C. Endoscopy of upper gastrointestinal bleeding. *Lancet*, 1974, **2** : 394-397.
7. LIEBERMAN D.A., KELLER F.S., KATON R.M., RÖSCH J. Arterial embolization for massive upper gastrointestinal tract bleeding in poor surgical candidates. *Gastroenterology*, 1984, **86** : 876-885.
8. COOK D.J., GUYATT G.H., SALENA B.J., LAINE L.A. Endoscopic therapy for acute nonvariceal upper gastrointestinal hemorrage : a meta-analysis. *Gastroenterology*, 1992, **102** : 139-148.
9. KADIR S., LUNDELL C., SAEED M. Arterial and venous systems of the viscera. In : S Kadir, eds. Celiac, superior and inferior mesenteric arteries. Atlas of normal and variant angiographic anatomy. WB Saunders, Philadelphia, 1991 : section IV, chapter 13.
10. Welch J.P., Welch C.E. Peptic ulcer-stomach and duodenum. *In* : MORRIS P.J., MALT R.A. (eds). The stomach and duodenum. Oxford textbook of surgery. Oxford University Press, 1994 : section 15, chapter 1.
11. BELL S.D., LAU K.Y., SNIDERMAN K.W. Synchronous embolization of the gastroduodenal artery and the inferior pancreaticoduodenal artery in patients with massive duodenal hemorrage. *J. Vasc. Interv. Radiol*., 1995, **6** : 531-536.
12. VAN BEERS B., ROCHE A. L'artériographie dans les hémorragies digestives. *Acta Gastroenterol. Belg*., 1989, **LII** : 278-290.
13. SOS T.A., JACK G.L., WIXSON D., SNIDERMAN K.W. Intermittent bleeding from minute to minute in acute massive gastrointestinal hemorrage : arteriographic demonstration. *AJR*, 1978, **131** : 1015-1017.
14. CLARK R.A., COLLEY D.P., EGGERS F.M. Acute arterial gastrointestinal hemorrhage : efficacy of transcatheter control. *AJR*, 1981, **136** : 1185-1189.
15. GOMES A.S., LOIS J.F., MCCOY R.D. Angiographic treatment of gastrointestinal hemorrhage : comparison of vasopressin infusion and embolization. *AJR*, 1986, **146** : 1031-1337.
16. EISENBERG H., STEER M. The non operative management of massive pyloroduodenal hemorrhage by autologous clot embolization. *Surgery*, 1976, **79** : 414-420.
17. DEMPSEY D.T., BURKE D.R., REILLY R.S., MC LEAN G.K., ROSADO E.F. Angiography in poor-risk patients with massive nonvariceal upper gastrointestinal bleeding. *Am. J. Surg*., 1990, **159** : 282-286.
18. GRANMAYEH M., WALLACE S., SCHWARTEN D. Transcatheter occlusion of the gastroduodenal artery. *Radiology*, 1979, **131** : 59-64.
19. LANG E.K. Transcatheter embolization in management of hemorrhage from duodenal ulcer : long-term results and complications. *Radiology*, 1992, **182** : 703-707.