CASE Reports

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Duodenal Dieulafoy's Lesion : Case Series and Literature Review

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ABSTRACT

We present four cases of duodenal Dieulafoy's lesion from our practicing in gastrointestinal endoscopy since 1994. It also includes review published English literature of duodenal Dieulafoy's lesion by searching from PubMed and references list of relevant major articles. The diagnosis of the reported cases base on the endoscopic visual criteria, three cases under went successful endoscopic treatment. One underwent oversew ligation after failed therapeutic endoscopy . Forty nine cases of duodenal Dieulafoy's lesion have been reported in English literature since 1988. Endoscopy is one of the essential procedures for diagnosis but multiple endoscopic sessions are usually required. Scintigraphy and angiography are valuable after endoscopy failed to identify the bleeding lesion. Endoscopic treatment provided a high successful rate of permanent hemostasis with minimal procedure related morbidity and mortality. Causes of death mostly resulted from comorbidity of these eldery patients.

Key words : Duodenal, Dieulafoy's

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INTRODUCTION

Characteristic pathology of Dieulafoy's lesion (DL) consists of two components.: 1) A large tortuous submucosal artery or the other term called "caliber persistent artery" which usually 1-3 mm in diameter without histologic evidence of aneurysm or artherosclerosis or vasculitis. This is generally accepted that it is congenital in origin. 2)small mucosal defect which usually 2 - 5 mm. in diameter and solitary without evidence of ulceration[1,2]. When this exposed artery rupture, sudden onset of gastrointestinal bleeding (GIB) will occur which usually massive. Unrecognized lesion can be life threatening. The pathogenesis of the development of mucosal defect and bleeding is still unclear but may be multifactorial. DL was first reported in 1884 by Gallard as "Milliary aneurysm", thereafter it was reported in different names in subsequent literatures due to misunderstanding of its pathology. Dieulafoy has been named after a French surgeon who fully described and characerised this lesion in 1897. He called the superficial gastric mucosal lesion as "Exulceratio simplex".

Before the endoscopic era, diagnosis of DL could be made only by laparotomy or autopsy which had tissue for histologic confirmation but mortality rate was

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very high[3,4]. Currently, endoscopy is the best diagnostic procedure but repeat endoscopy is often necessary because initial endoscopy usually fails to identify the bleeding point. A minute lesion or obscure visualization by blood clot are some other explanations. Scintigraphy and selective mesenteric angiography are the alternative methods for demonstration or localization of DL. Therapeutic endoscopy is the treatment of choice while surgery and selective mesenteric embolisation are helpful if endoscopy is inaccessible or fail[5-11].

Endoscopic treatment modalities can be summarized in to three methods[12-14]. 1). Injection sclerotherapy. Generally epinephirne, polidoconal, hypertonic NaCl or ethanol are more favorable than other agents. They may be used as a single or combination therapy. These agents produce tissue inflammation, necrosis and vascular thrombosis. 2).Thermal therapy, tissue heating at 60˚C causes coagulation and contraction. Coaptation treatments are electrocoagulation (monopolar, multipolar, BICAP) , thermocoagulation (heater probe) and Laser photocoagulation (Nd : YAG). 3) Mechanical therapy by application of hemoclip or band ligation. No comparative study to confirm what is the most effective hemostatic procedure but perilesion epinephrine injection followed by coaqultation therapy are more preferable especially with active bleeding[15-19]. Permanent hemostasis can be achieved in 80-95% of cases[6,7,14]. Surgical treatments are oversew ligation ,wide wedge resection and segmental resection[5,20]. Intragastric surgery and lapraloscopic surgery with or without endoscopic assisted have also been reported[21-23]. Selective arterial embolization had a high recurrent bleeding rate due to extensive collateral circulation in gastroduodenal area[24-26]. Overall morality was about 10%. Causes of death mostly resulted from comorbidity especially elderly patient.

Acute GIB is the main clinical presentation. It accounts for 0.3-9% of cases,which depended on criteria for selection[8-10]. Some patients present with signs and symptoms of anemia from chronic blood loss. The extremely rare case DL was small bowel intussusception[28] and DL was found to be the leading point. DL can present in patient at the age range from 20 months to 93 years. Men at 60 years or above are the main group. Comorbidity of medical problem can be found in 45-90 % of cases[2-8,13]. More than 400 cases of gastrointestinal (GI) tract DL have been reported. Proximal stomach especially 6 cm from esophagogastric junction at lesser curvature is the most common site. Extragastrointestinal tract DL lesion can be found in the lip and bronchus[30,31]. Extragastric DL have also been reported in esophagus, duodenum, jejunum, ileum, caecum, colon, rectum and anus[29,32]. Forty nine cases of duodenal Dieulafoy's lesion (D-DL) have been reported since 1988[13,33-49].

CASE SERIES

The endoscopic criteria used for diagnosis of D-DL herein report were based on the endoscopic criteria those of Dy NM *et al.*[14] namely, 1). Active arterial spurting or micropulsatile streaming from a minute mucosal defect or through normal surrounding mucosa 2). Visualization of a protruding vessel with or without active bleeding within a minute mucosal defect or through normal surrounding mucosa. 3). Fresh, densely adherent clot with a narrow point of attachment to a minute mucosal defect or to normal appearing mucosa. The options for endoscopic hemostatsis at author's institution are injection sclerotherapy, heater probe thermocoagulation and rubber band ligation.

Case 1

A 35-year-old man presented with hematemesis and melena about two hours before admission. Initial evaluation revealed normal vital sign, physical examination was unremarkable, initial hematocrit (Hct) was 30%. He had a history of occasionally taken of NSAIDs to relief pain from acute attack of gouty arthritis. During the past ten years he developed five episodes of acute UGIB which required hospitalization. Three in five of gastroscopic records depicted a small ulcer size about 3-5 mm diameter with adherent clot located at the posterior wall of duodenal bulb, and evidence of duodenitis in another two gastroscopies. After provided basic resuscitation, esophagogastroduodenoscopy (EGD) was performed at 6 hours after admission. It showed densely adherent clot on normal appearing mucosal at the posterior wall of duodenal bulb which refractory to forceful water irrigation (Figure 1A). Hemostasis was performed by application of a heater probe. Since target lesion was smaller than heat probe tip (Olympus CD 120), then it was applied directly to lesion with firm pressure. (Figure1B) Unfortunately adherent clot was displaced away from mucosal defect due to duodenal movement during respi-

ration, torrential bleeding suddenly occured and rapidly pooling of blood precluded further endoscopic procedure. (Figure 1C) Surgical treatment by oversew ligation was done. One unit of packed red cell (PRC) was transfused postoperatively, he had no further UGIB during follow-up period of 43 months.

Case 2

A 45-year-old man experienced of melena for two days before admission. Initial evaluation revealed BP 84/50 mmHg, HR 120/min. Physical examination was unremarkable except pale conjuntiva. Initial Hct was 25%. After stabilized vital sign by basic resuscitations, EGD was performed at twenty hours after admission. It demonstrated a protruded visible vessel with sentinel clot inside and surrounded by normal mucosa at the posterolateral wall of duodenal bulb (Figure 2A, 2B). Endoscopic hemostasis was achieved by perilesion injection of 1:10,000 epinephrine (4 × 1.5cc). This was followed by heater probe application, perilesion and directly to lesion (8 × 15 joules). One unit of PRC was transfused. Gastroscopy at twelve weeks showed normal duodenal bulb. There was no further melena during 41 months of follow-up.

Case 3

A 66-year-old man with a history of alcoholic abuse developed an abrupt onset of hematemesis and melena about 3 hours before admission. The initial evaluation revealed BP 96/60 mmHg, HR 90/min , he looked pale, jaundice with minimal ascites. The rest of physical examination was unremarkable. Initial Hct was 17%, and LFT showed total protein 4.6 mg % (albumin 2.0/ globulin 2.6), AST 334U/L, ALT 123 U/L, total bilirubin 2.5 mg% (direct 10.9/ indirect 0.6), alkaline phosphatase 112 U/L. He was resuscitated with intravenous fluid, blood and fresh frozen plasma. EGD was performed with in 6 hours after admission. It revealed active arterial spurting through normal appearing mucosa of the duodenal bulb. (Figure 3A). Hemostasis was achieved by preliminary perilesion injection of 1:10,000 epinephrine (6 × 1.5cc),then spurting gradually ceased (Figure 3B, 3C). Heater probe application was appliced, perilesionly and directly to bleed-

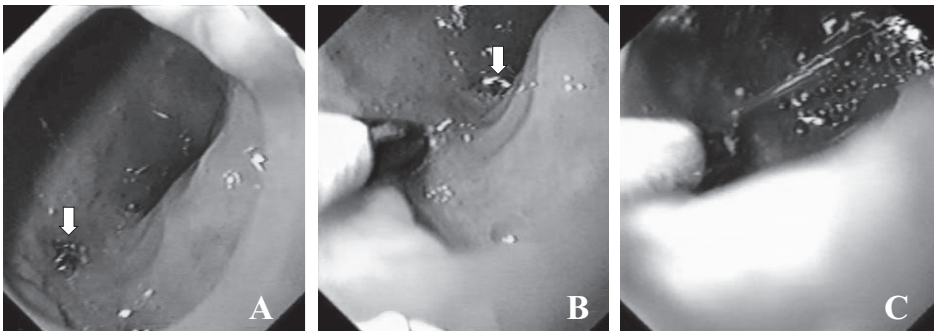

Figure 1 An adherent clot at the duodenal bulb (A-arrow) which was very small comparing to 2.5mm of HP tip (B), Torrential bleeding occurred after adherent clot had been removed by the heater probe (C)

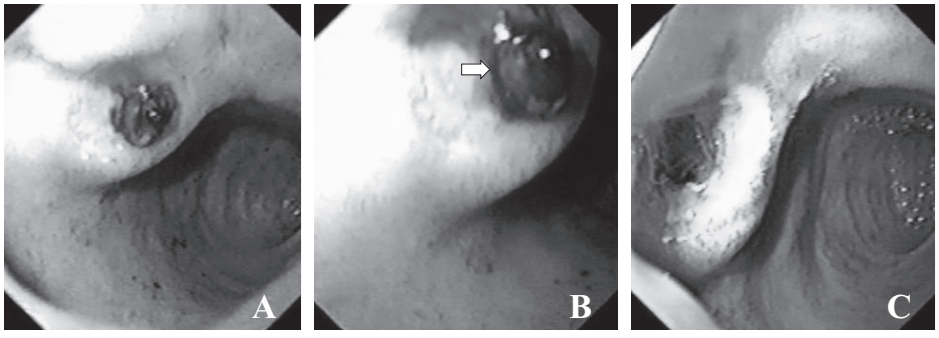

Figure 2 (A) An elevated normal appearing mucosa with a protruded visible vessel containing (B) sentinel clot inside.(C) Vascular thrombosis was seen after complete application of heater probe.

ing vessel (5 × 20 Joules). Patient had an unevenful recovery except developing alcohol withdrawal symptom (delirium tremens) during the first day. He received 6 units of blood and 4 units of fresh frozen plasma. Jaundice and ascites disappeared within two weeks and he had no recurrent UGIB during 37 months of follow-up period.

Case 4

A 79-year-old man was admitted because of dizziness, anorexia and fatique without a past history of significant medical problem. On the next morning he passed a large amount of melena. This accompanied with sign and symptom of hypovolemia, Hct dropped from 28% to 19%. He was resuscitated to stabilize vital signs by intravenous fluid, blood transfusion. Initial EGD performed at six hours after the onset was not able to identify the bleeding point due to blood clot in stomach and fresh blood in duodenum obscuring the veiw. Second EGD at one hour after vigorous nasogastric tube irrigation, revealed micropulsatile streaming through normal appearing mucosa of posterolateral wall of duodenal bulb (Figure 4A). Hemostasis was achieved by preliminary perilesion injection with 1:10,000 epinephrine (6 × 1.5 cc) to stop the bleeding (Figure 4B) then follow by heater probe application, perilesion then directly to bleeding vessel (6 × 15 Jouls) (Figure 4C). Seven units of PRC were transfused, he had no further bleeding during three months of follow-up period.

Results and Discussion

D-DL was first reported since 1988, up to December 2003, fifty three cases including this series have been reported[13,33-49]. (Table 1) Only half of these cases had detail data for evaluation. Patients age varied from 10 to 90 years (median age = 61 years, male:female = 4:1). Melena and hematochezia with or without hematemesis were the main clinical presentations in 87% of cases. Only 13% of cases presented with hematemesis alone. Significant number of cases had instability of hemodynamic status at initial presentation along with a history of significcant comorbidity.

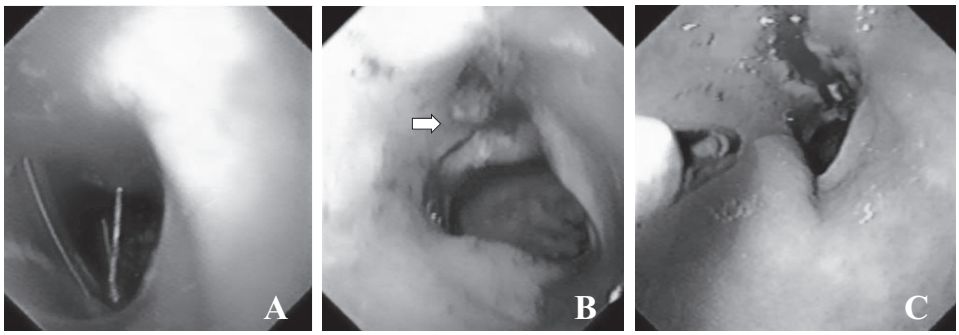

Figure 3 (A) Spurting through normal appearing mucosa (B) clot formation after perilesion epinephrine injection.(C) coagulated tissue after heater probe application.

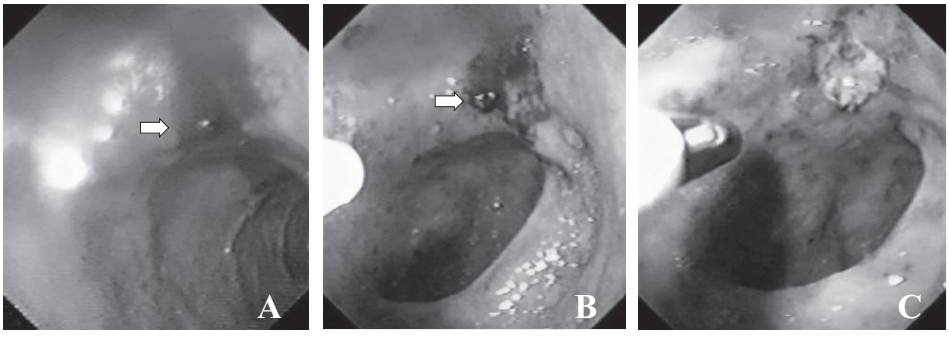

Figure 4 (A) Micropulsatile streaming through nomal appraring mucosa(B) red clot formation following perilesion epinephrine injection (C) coagulum formation after heater probe application.

Table 1 Summary of Duodenal Dieulafoy's lesion. (1988-2003)

Author/Year	Case	Age/Sex	Site	No.of SRH	Treatment	EGD (Retreatment)
McClave SA 1988	1	16/M	D1	2	NBVV	Suture ligation
	2	61/M	D1	2	Adherent clot	Suture ligation
	3	42/M	D2	7*	Spurting	Suture ligation
	4	62/M	D1	2*	NBVV	Suture ligation
Goldenberg SP 1990	5	68/F	D1	1	Spurting	Inj.Epi+BICAP
Golding MI 1990	6	81/F	D1	2	Active bleed	HP
Pollack R 1993	7	63/M	D2	4*	Adherent clot	Embolization (BICAP) (Inj. Epi, 100% Alcohol)
Choudari CP 1993	8	55/M	D1	1	Oozing	Inj. Epi + Nd: YAG Laser
Knudsen FB 1993	9	90/F	ns	1	ns	Inj. Polidocanol
Steinert D 1996	10	57/M	D1	2	Pulsatile	BICAP (Inj. Epi + BICAP)
Hokama A 1996	11	10/M	D1	1	Pulsatile	Hemoclip
Sueoka N 1997	12	50/M	D1	1	Pulsatile	Inj. Ethanol + Hemoclip
	13	28/M	D2	1	Pulsatile	Inj. Ethanol + Hemoclip
Hokama 1998	14	66/F	D2	ns	ns	Hemoclip
Gadenstatter M 1998	15	32/M	D1	2	Adherent clot	Inj. Polidocanol
Norton ID 1999	16-31	Detail information unavailable (Include Dy NM[14], Stark ME[12])				
Schmulewitz N 2001	32	75/	D1	2	ns	Inj. Epi + HP
	33	84/	D1/2	1	ns	Inj. Epi
	34	66/	D2	4	ns	HP
	35	44/	D1	2	ns	HP
	36	78/	D1	1	ns	Inj. Epi + HP
	37	77/	D1/2	2	ns	Inj. Epi + HP
	38	80/	D2	1	ns	Inj. Epi + HP
Nikolaidis N 2001	39-40	ns	D1, D2	ns	ns	EBL
Kasapidis P 2002	41	51/M	D1	ns	Active bleed	Inj. Epi + HP
	42	55/M	D1	ns	Active bleed	Inj. Epi + HP
	43	52/M	D1	ns	Active bleed	Inj. Mixed. Epi + EO
	44	44/M	D1	ns	NBVV	Inj. Epi + HP
Ibrarullah M 2003	45	31/M	D2	2	Active bleed	Inj. Epi
	46	25/F	D1	1	Oozing	Oozing
Hurlstone DP 2003	47	87/M	D2	ns	Spurting	Inj. Mixed Nacl + Epi (EBL)
Mumtaz R 2003	48	77/	ns	ns	Not active	HP
	49	76/	ns	ns	Not active	Inj. Epi + HP
Treesaranuwattana	50	46/M	D1	1	Adherent clot	HP (oversew ligation)
S, Khemtai C.2003	51	45/M	D1	1	Adherent clot	Inj. Epi + HP
	52	66/M	D1	1	Spurting	Inj. Epi + HP
	53	79/M	D1	2	Micropulsatile	Inj. Epi + HP

D1, D2 = *1st, 2nd part duodenum* — *D1/2* = *junction of 1st and 2nd part of duodenum,*
EGD = *esophagogastroduodenoscope* — *NBVV* = *non-bleeding visible vessel*
Inj = *injection* — *Epi* = *epinephrine*
EO = *Ethanolamide oleate* — *EBL* = *endoscopic band ligation*
HP = *heat probe* — *SRH* = *stigmata of recent hemorrhage*
ns = *not state* — * = *Coincidence of duodenal ulcer, M = male, F = female*

Although they had no report of blood transfusion in every cases but by estimation of initial Hct showed that most of D-DL required blood transfusion. First part of duodenum (bulb) is the most common site (68%) follow by second part (26%) and junction of first and second part (6%) respectively. No lesion was reported in the third or fourth part.

Endoscopy was performed within 24 hours in most of the cases. Initial endoscopy can identify D-DL in 50% of the cases othre[36,44]. Major causes of failure initial endoscopy were obscured visualization from pooling of fresh blood or blood clot. Prior vigorous nasograstric tube irrigation and changing position of patient during endoscopy are valuable[5,10]. New type of therapentic gastroscope with a working channel of 6 mm can rapidly removed blood. Water jet irrigation can also visualized the underneath lesion[50]. Lesion in the second part or superior duodenal angle can be better visualized and easily applied with side view doudenoscope[36,44,51]. Early endoscopy especially immediately after stabilized hemodynamic status can increase the sensivity of endoscopic diagnosis by early detection of stigmata for recent hemorrhage. Concurrent endoscopic findings such as peptic ulcer, gastroduodenitis, vascular ectasia without evidence of bleeding could be misinterpreted as the cause bleeding. Coincidence of duodenal ulcer in D-DL without identify the bleeding point can mislead the surgeon to perform ulcer operation and ending up with recurrent bleeding in early postoperative period[33,36].

Two international series demonstrated that, scintigraphy, bleeding scan and selective visceral angiography were performed in five cases (9%) but required multiple attempts, only in two cases[35,36] (40%). Scintigraphy and angiography yield the positive result only in active bleeding phase. Currently, more improvement in quality of endoscopic equipment can help diagnosing DL. After 1993 only one case of D-DL reported by Schmulewitz N[44] requirel angiographic study. It also be noted that D-DL in the second part always require multiple endoscopic sessions and multiple angiographic study or scintigraphy.

Endoscopic visualization of stigmata of recent hemorrhage (SRH) of these D-DL were active bleeding (63%) and non-active bleeding (37%). (Active bleeding defined as spurting, pulsatile bleeding, micropulsatile bleeding, or continuous oozing. Non-active bleeding defined as non-bleeding visible vessel or adherent clot.)

The first four cases of D-DL reported McClave SA in 1988[33] were successfully treated by surgery . Subsequently reports since 1990, therapeutic endoscopy has become the treatment of choice by using the same principle as gastric DL. Three methods of therapeutic endoscopy used for treatment of D-DL are injection sclerotherapy, thermal and mechanical method. One of these method can be used alone as monotherapy or combination of these two methods. Injecting sclerotherapy followed by thermocoagulation is the most favorable method. Thirty three cases of D-DL had initial endoscopic treatment, four cases were failed, three of these four case were successful on the second attempt. Another one was operated by oversew ligation. Initial hemostasis of D-DL was 88% and permanent hemostasis was 97%.

Sclerosing agents used in D-DL were epinephrine 1:10,000, 100% alcohol or ethanol, 1% polidocanol, hypertonic NaCl (3%), 5% ethanolamide oleate. Usually epinephrine was the only agent used for injections however some authors used two sclerosing agents injected on one lesion or mixed up of two sclerosing agent for each injection such as 8 cc epinephrine plus 2 cc of 5% ethanolamide oleate[46] or 9 cc of 3% NaCL plus 1 cc of epinephrine[48]. Injection of sclerosing agents cause tissue inflammation, mucosal ulceration, tissue necrosis and finally vascular thrombosis. Initial hemostasis attributed to volume temponade and permanent hemostasis resulting from vascular thrombosis[52,53]. Excessive volume of sclerosing agent may cause perforation[55]. In gastric DL it was recommend to use not over than 2 cc for ethanol and 10cc for polidocanol or epinephrine[8,18,19,35]. In D-DL, 0.3 cc, 0.4 cc of ethanol were injected without mucosal ulceration contined by repeat endoscopy at one week later[41]. Epinephrine injection produces mild tissue inflammation without vascular thrombosis.

Heater probe and BICAP are the thermal methods commonly used in D-DL lesion. Only one case had Md:YAG Laser as a treatment. Monotherapy with heater probe or BICAP yielded high a failure rate on initial hemostasis (50%). Combination therapy with injection sclerotherapy had a lower failure rate. Preliminary epinephrine injection can prevent deeper tissue destruction from heater probe or BICAP. In the presence of active bleeding, it can also slower the bleeding[13,55].

Hemoclipping and band ligation were used in D-DL both as monotherpy or combination therapy with

preliminary injection sclerotherapy. It provided permanent hemostasis in all of D-DL. Normal surrounding mocosa and lack of inflammation or fibrosis, contribute to easier technique and provide a good result[40-42].

Surgery was performed in five cases of D-DL, the first four cases reported in 1988 were in the period of early experience of therapeutic endoscopy in UGIB especially in DL, another case was surgically treated after failure from heater probe application without second endoscopic attempt. However at laparotomy, it may be overlooked because of its tiny lesion. Usnally after duodenotomy when all blood is removed, if bleeding is not seen, abrading duodenal mucosa with dry gauze can provoked active bleeding[20,33]. Many surgeons prefer oversew ligation over wide wedge resection or segmental resection. Since oversew ligation demand a higher skill and may increase complication rate for D-DL. In contract, wedge resection has a lower rebleeding rate.

Selective visceral aterial embolization was performed in one case of D-DL[36]. Unfortunately it failed to stop bleeding, this may be due to extensive collateral circulation in the gastroduodenal area[26,27]. In general embolization is indicated only in patient who is a poor candidate for surgery or endoscopy.

Recently endoscopic ultrasound with doppler examination is the novel procedure for confirmation and improving the treatment outcome by demonstrating vascular structure and also guiding for injection sclerotherapy. Absence of vascular signal is the indicator for therapeutic efficacy[56-60]. Some authors advocate the term "Dieulafoy's Like lesion" or "Endoscopic Dieulafoy's lesion" for patient who had no histologic confirmation[140].

CONCLUSION

Incidence of D-DL ranges from 9.7% to18% of all gastrointestinal DL[14,29]. It is the most common location of extragastric Dieulafoy's lesion. In general pathology, clinical presentation, principle of diagnosis and treatment are similar to gastric Dieulafoy's lesion. Melena or hematochezia with or without hematemesis is the main presenting symptom. Hematemesis alone was rarely presented. Initial EGD finds bleeding lesion in about half of cases. In case of massive acute UGIB with failure of initial endoscopic diagnosis, repeat endoscopy is mandatory with meticulous searching. All blood clot must be removed, hematin and adherent clot should be irrigate for good visualization of mucosa to confirm the clearance of the clot. Side view duodenoscope is valuable in providing a of better visualization of en face view for diagnosis and application of therapeutic procedure if the lesion locates in the 2nd part or at the duodenal angle. Currently endoscopic treatment is the standard approach which permanent hemostasis can be achieved in 97% of cases of D-DL with an acceptable rate of procedure related morbidity and mortality. Surgery is indicated if endoscopy is inaccessible or fail. It should be kept in mind that thin-walled duodenum predispose to perforation if excessive volume of sclcresing agent is injected or high power of thermal treatment is applied. Moreover, delayed perforation from peritoneal entrapment by the clip can occur[41].

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