Submit a Manuscript: https://www.f6publishing.com

World J Gastrointest Endosc 2025 September 16; 17(9): 108787

ISSN 1948-5190 (online) DOI: 10.4253/wjge.v17.i9.108787

REVIEW

Role of endoscopy in the diagnosis and treatment of gastric mucosal lesions in liver cirrhosis patients

Elena R Olevskaya, Anastasia I Dolgushina, Dmitry V Garbuzenko, Alina O Khikhlova, Anna A Saenko, Guzel M Khusainova, Alla S Kuznetsova

Specialty type: Gastroenterology and hepatology

Provenance and peer review:

Invited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's classification

Scientific Quality: Grade A, Grade

Novelty: Grade B, Grade C Creativity or Innovation: Grade A,

Scientific Significance: Grade B,

Grade C

P-Reviewer: Al-Abachi KT, Professor, Iraq; Teherán AA, MD, Professor, Senior Researcher,

Colombia

Received: April 23, 2025 Revised: May 22, 2025 Accepted: August 19, 2025 Published online: September 16,

Processing time: 142 Days and 13

Hours



Elena R Olevskaya, Alina O Khikhlova, Department of Hospital Surgery, South Ural State Medical University, Chelyabinsk 454092, Russia

Anastasia I Dolgushina, Anna A Saenko, Guzel M Khusainova, Alla S Kuznetsova, Department of Hospital Therapy, South Ural State Medical University, Chelyabinsk 454092, Russia

Dmitry V Garbuzenko, Department of Faculty Surgery, South Ural State Medical University, Chelyabinsk 454092, Russia

Corresponding author: Dmitry V Garbuzenko, MD, PhD, Professor, Department of Faculty Surgery, South Ural State Medical University, 64 Vorovskogo Street, Chelyabinsk 454092, Russia. garb@inbox.ru

Abstract

Liver cirrhosis (LC) can cause a wide spectrum of gastric mucosal lesions, the main diagnostic method of which is esophagogastroduodenoscopy. In addition to gastric varices, portal hypertensive gastropathy, gastric antral vascular ectasia, portal hypertensive polyps, peptic ulcer disease can be detected. All of these leave LC patients susceptible to bleeding. If visual differential diagnosis is difficult, endoscopic ultrasound and new digital and optical endoscopic technologies such as magnifying endoscopy with narrow-band imaging may be useful. In many cases, endoscopic technologies are also the methods of choice for the treatment of gastric mucosal lesions in LC patients. In particular, argon plasma coagulation can be used for local treatment of portal hypertensive gastropathy bleeding, and argon plasma coagulation, radiofrequency ablation or endoscopic band ligation is recommended as the main method for local treatment of gastric antral vascular ectasia bleeding. Endoscopic therapy for peptic ulcer bleeding is carried out according to the current guidelines. Besides, endoscopic mucosal resection and endoscopic submucosal dissection may be used in LC patients for the treatment of early gastric cancer. The purpose of this review is to provide up-to-date information on the role of endoscopy in the diagnosis and treatment of gastric mucosal lesions in LC patients.

Key Words: Liver cirrhosis; Gastric mucosal lesions; Endoscopy; Diagnosis; Treatment; Portal hypertensive gastropathy; Gastric antral vascular ectasia; Portal hypertensive polyps; Peptic ulcer disease; Early gastric cancer

©The Author(s) 2025. Published by Baishideng Publishing Group Inc. All rights reserved.

Core Tip: Liver cirrhosis (LC) can cause a wide spectrum of gastric mucosal lesions, the main diagnostic method of which is esophagogastroduodenoscopy. If visual differential diagnosis is difficult, endoscopic ultrasound and new digital and optical endoscopic technologies such as magnifying endoscopy with narrow-band imaging may be useful. In many cases, endoscopic technologies are also the methods of choice for the treatment of gastric mucosal lesions in LC patients. The purpose of this review is to provide up-to-date information on the role of endoscopy in the diagnosis and treatment of gastric mucosal lesions in LC patients.

Citation: Olevskaya ER, Dolgushina AI, Garbuzenko DV, Khikhlova AO, Saenko AA, Khusainova GM, Kuznetsova AS. Role of endoscopy in the diagnosis and treatment of gastric mucosal lesions in liver cirrhosis patients. World J Gastrointest Endosc 2025; 17(9): 108787

URL: https://www.wjgnet.com/1948-5190/full/v17/i9/108787.htm

DOI: https://dx.doi.org/10.4253/wjge.v17.i9.108787

INTRODUCTION

Liver cirrhosis (LC) is one of the most severe chronic somatic diseases. In 2019, it accounted for 2.4% of global deaths[1]. According to the Global Burden of Disease Study 2017, the estimated number of people with compensated LC was 112 million worldwide, which corresponds to the age-standardized global prevalence of compensated LC of 1395 cases per 100000 population[2]. The natural history of LC is accompanied by complex metabolic disorders and the development of portal hypertension. This has a pathological effect on various organs and systems including the stomach. Esophagogastroduodenoscopy (EGD) is a common and generally safe endoscopic procedure for the diagnosis and treatment of upper gastrointestinal tract lesions[3]. LC patients have a wide spectrum of gastric mucosal lesions, the main diagnostic method of which is EGD. Gastric varices are less common than esophageal varices, occurring in about 17%-25% of patients with portal hypertension. The incidence of gastric varices varies from 16% at 1 year to 44% at 5 years[4]. In addition to gastric varices, portal hypertensive gastropathy (PHG), gastric antral vascular ectasia (GAVE), portal hypertensive polyps (PHPs), peptic ulcer disease (PUD) can be detected. All of these leave LC patients susceptible to bleeding[5]. If visual differential diagnosis is difficult, endoscopic ultrasound (EUS) and new digital and optical endoscopic technologies such as magnifying endoscopy with narrow-band imaging (NBI) may be useful [6,7]. Various endoscopic technologies have been proposed for their prevention and treatment[8]. For example, according to the decisions of the Baveno VII consensus workshop, endoscopic therapy, in particular, argon plasma coagulation (APC) can be used for local treatment of PHG bleeding, and APC, radiofrequency ablation (RFA) or endoscopic band ligation (EBL) is recommended as the main method for local treatment of GAVE bleeding[9]. Endoscopic therapy for peptic ulcer bleeding is carried out according to the current guidelines[10-12]. Besides, endoscopic mucosal resection (EMR) and endoscopic submucosal dissection (ESD) may be used in LC patients for the treatment of early gastric cancer[13].

The purpose of this review is to provide up-to-date information on the role of endoscopy in the diagnosis and treatment of gastric mucosal lesions in LC patients. The Web of Science platform, the EMBASE and PubMed databases, the Cochrane Database of Systematic Reviews, the Google Scholar retrieval system, Reference Citation Analysis (https:// www.referencecitationanalysis.com/), and the reference lists from related articles were used to search for suitable publications. Articles evaluating the role of endoscopy in the diagnosis and treatment of gastric mucosal lesions in LC patients were selected for 1995-2025. To limit the scope of this review, articles dealing with gastric varices were not included.

PHG

PHG occurs as a complication of cirrhotic or non-cirrhotic portal hypertension and is of great clinical importance, since it can be complicated by both acute and chronic upper gastrointestinal bleeding [14]. The prevalence of PHG ranges from 7% to 98%. The wide disparity in these results is due to differences in the criteria used for patient selection and endoscopic interpretations [15]. The development of PHG usually requires the presence of clinically significant portal hypertension. The prevalence of PHG among patients with compensated LC ranges from 49% to 80%, with lower prevalence in patients without gastroesophageal varices (GEVs) (11%) or small GEVs (35%) relative to those with medium or large GEVs (80%-97%)[16]. With long-term follow-up of LC patients, progression of PHG is frequently, and regression more rarely, observed. Factors contributing to the progression of PHG may be a high hepatic venous pressure gradient [17], severity of LC, as measured by Child-Turcotte-Pugh (CTP) score and/or model for end-stage liver disease (MELD) score[18], and endoscopic eradication of GEVs[19].

EGD is currently the gold-standard for the diagnosis of PHG. It is characterized by gastric mucosal lesions that is classically described as a mosaic pattern that resembles the skin of a snake, with or without red marks. Based on this, PHG is classified into mild (Figure 1A), when mosaic pattern alone is observed, and severe (Figure 1B), in the presence of red marks of any kind. With severe PHG, an erythema foci merge and hemorrhages appear [20]. The most common

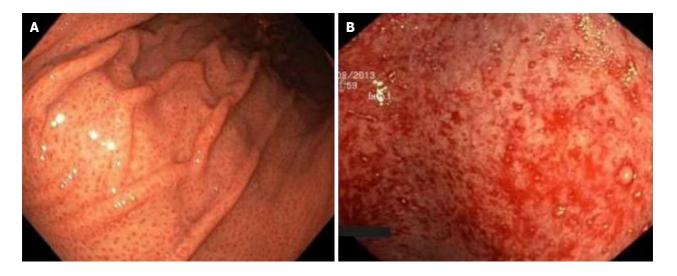


Figure 1 Portal hypertensive gastropathy. A: Mild portal hypertensive gastropathy. A mosaic mucosal pattern of the body and fundus of the stomach; B: Severe portal hypertensive gastropathy. Hemorrhages on the background of erythema foci.

locations for PHG are the body and fundus of the stomach[21]. The endoscopic findings are not specific for PHG. In some cases, it is difficult to distinguish from other gastric mucosal lesions, such as GAVE, Helicobacter pylori (H. pylori)associated gastritis, etc. Therefore, microscopic examination may be useful. Histologically, PHG is characterized by dilation and ectasia of the capillaries and venules of the gastric mucosa and submucosa, as well as edema and thinning of the arteriole and venule wall in the submucosa without signs of inflammation, erosion or fibrin thrombi[22].

In a study by Macedo et al[23] involving 20 patients with compensated LC, the role of contrast-enhanced EUS in the assessment of PHG was studied. Intense gastric mucosa and submucosa blood flow was identified in all patients with endoscopically confirmed PHG and only in one patient without endoscopic signs of PHG, presenting sensibility, sensitivity, positive predictive value and negative predictive value, respectively of 100.0%, 90.0%, 90.9%, 100.0% (P < 0.001). It can be assumed that with the help of contrast-enhanced EUS, it is possible to detect PHG earlier and with greater sensitivity than with conventional EGD.

The gastric mucosa in portal hypertension differs morphologically and functionally from normal. Its increased susceptibility to damaging agents, such as nonsteroidal anti-inflammatory drugs, alcohol, bile acids, acid-peptic factor, contributes to the development of bleeding [24]. PHG-related bleeding is usually mild to moderate in severity, and in over half of cases, hemostasis can occur on its own. Clinically significant bleeding is rare, but difficult to stop[25].

According to the European Association for the Study of the Liver clinical practice guidelines, acute PHG bleeding may be treated with somatostatin analogues or terlipressin but substantiating data are limited [26]. According to the decisions of the Baveno VII consensus workshop, non-selective β-blockers (NSBBs) are the first-line therapy for preventing recurrent bleeding from PHG. Endoscopic therapy, in particular, APC, may be used for local treatment of acute PHG bleeding, and APC or hemospray - in case of its recurrence. Transjugular intrahepatic portosystemic shunt should be considered for preventing recurrent transfusion-dependent bleeding from PHG despite traditional NSBBs or carvedilol and endoscopic therapy[9].

APC is an electrosurgical modality for the treatment of bleeding and devitalization of tissue. This is achieved by noncontact thermal coagulation, in which a high frequency, monopolar alternating current is applied to the target tissue through an argon plasma jet, creating effective hemostasis and homogeneous surface coagulation with limited penetration depth. The depth of coagulation depends on the setting of the generator power, the duration of application, and the distance from the tip of the probe to the target tissue (the optimal distance is 2-8 mm)[10]. In a study by Hanafy and El Hawary [27] involving 200 LC patients, APC alone and in combination with NSBBs was highly effective and safe in controlling bleeding from PHG. In a study by El Shahawy et al [28] involving 120 LC patients, APC significantly improved iron deficiency anemia and reduced transfusion requirements in patients with severe PHG as compared to carvedilol with a low risk of adverse events. In addition, the combination of APC and carvedilol had a synergistic beneficial effect in severe PHG.

TC-325 (Hemospray®; Cook Medical, Winston-Salem, NC, United States) is a hygroscopic mineral-based powder that is applied using a pressurized carbon dioxide cylinder. When it comes into contact with blood, a clotting cascade is triggered, which leads to the formation of coagulate that ensures hemostasis. The powder peels off the mucosa over the next 24-72 hours. In a prospective international multicenter study involving 190 patients, TC-325 monotherapy was effective and safe, in particular, for bleeding after endoscopic interventions, when immediate hemostasis was achieved in 100% of cases[29]. Currently, the efficacy of TC-325 in PHG has been shown only in descriptions of clinical cases. For example, Smith et al[30] presented data on the successful use of TC-325 in four patients with acute PHG bleeding. Thus, the use of endoscopic technologies in PHG-related bleeding remains problematic and requires further research.

GAVE

GAVE is a disease characterized by dilation of the capillaries and venules of the gastric mucosa and submucosa, visually resembling blotchy erythematous or bloody streaks. The most common locations for GAVE is the gastric antrum. These lesions can cause about 4% of non-variceal upper gastrointestinal bleeding. As a rule, GAVE is clinically manifested by melena and iron deficiency anemia secondary to chronic blood loss. GAVE is usually associated with LC, autoimmune diseases, heart failure, chronic renal failure, bone marrow transplantation, as well as metabolic syndrome [31,32].

GAVE is mostly diagnosed by EGD. In some cases, capsule endoscopy may be useful [33]. The endoscopic patterns of gastric mucosa in GAVE may vary. Among these variations, linear striped ("watermelon stomach") phenotype is a term used to describe the classic GAVE phenotype, which is manifested by red vascular spots spiraling away from the pylorus (Figure 2). The "honeycomb stomach" is characterized by similar red gastric mucosal lesions spread out in a diffuse pattern. The third endoscopic GAVE phenotype is represented by nodules, which are often indistinguishable from other benign formations of the gastric antrum, and may require biopsy for diagnosis[34]. Diffuse (honeycomb, punctate) GAVE phenotype is most characteristic for LC patients (Figure 3)[35].

In some cases, GAVE is difficult to distinguish from other gastric mucosal lesions including PHG, erythema, ulceration, H. pylori-associated gastritis, or polyps. Therefore, microscopic examination may be useful. Histologically, all three variations have characteristic reactive epithelial hyperplasia and vascular ectasia, meanwhile, more specific manifestations include spindle cell proliferation of smooth muscle cells, microvascular thrombi, and fibrohyalinosis, the frequency of which depends on the GAVE phenotype[36].

The use of modern endoscopic technologies has significantly improved the diagnosis of GAVE. The characteristic EUS features of GAVE are wall thickening of the gastric antrum with preserved stratification, hypertrophy of the mucosa and submucosa with the presence of anechoic structures in them, and a well-defined muscularis propria[37-39].

NBI is an optical digital method of image-enhanced endoscopy. NBI is a light technology that uses special filters to separate the wavelengths of white light and highlight the red, green, and blue ranges. Additional filtering technology enhances the blue range. Due to the selective effect of these bands of light, the microvasculature is more clearly visible (Figure 3C)[7]. In a study by Chang et al[40] involving 27 LC patients, the high efficiency of magnifying endoscopy with NBI for the diagnosis of diffuse GAVE phenotype was demonstrated.

Versatile Intelligent Staining Technology (VIST) is based on the NBI concept, but instead of an optical filter uses digital image processing to refine the appearance of both vascular and mucosal patterns without the use of dye. It is easy to perform and allows for inspection of the whole endoscopic field with more accuracy by simply switching the button from the conventional view to the VIST view. In a study by Abdelmoneim et al [41] involving 50 LC patients, it was shown that VIST can be used as an alternative tool for the histopathological diagnosis of GAVE. There was a statistical significance between VIST and histopathology in the diagnosis of GAVE (P < 0.035). In addition, VIST had superior sensitivity than conventional white light endoscopy in the detection of GAVE (82.1% vs 7.1%), while conventional white light endoscopy had higher specificity (95.5% vs 59.1%) by VIST.

I-scan virtual chromoendoscopy with indigo carmine or crystal violet is a modern endoscopic technology that allows to evaluate, in particular, the gastric mucosa microvasculature. In a study by Al-Taee et al[42] involving 23 LC patients, Iscan virtual chromoendoscopy had higher specificity, sensitivity, and accuracy in the diagnosis of GAVE compared to high-definition white light endoscopy alone (82% vs 70%). However, the efficiency of these two methods in detecting PHG was similar.

The main methods of treatment for GAVE are pharmacotherapy, endoscopy, and surgery. The efficacy and safety of pharmacotherapy have not been sufficiently confirmed, and surgical intervention is considered only in cases where other treatment is ineffective [43]. According to the decisions of the Baveno VII consensus workshop, endoscopic therapy, in particular, APC, RFA or EBL is recommended as the main method for local treatment of GAVE bleeding[9].

APC

In recent years, APC has been the most commonly used endoscopic approach for GAVE with a good level of efficiency and safety. Endoscopic success, which is defined as persistent hemostasis and/or elimination of most of the gastric mucosal lesions visible during endoscopy, can reach 40%-100%. According to the literature, recurrences occurred in 25%-68.2% of cases, 5.9%-40% of patients required post-treatment blood transfusions. Adverse events and complications were rare. Fever, abdominal distension and epigastric pain, antrum stenosis, gastric hyperplastic polyps, Mallory-Weiss syndrome and gastric ulceration resulting in scarring were described (Table 1)[44-57].

In a retrospective single-center study by Tamari et al [58] involving 15 patients with GAVE, the combination therapy with polidocanol injection and APC was effective with a low recurrence rate. Endoscopic hemostasis has been achieved in all cases. The average number of attempts to stop bleeding with polidocanol injections was 1.5 ± 0.8 (1-4), and the average number of APC attempts was 2.1 ± 1.2 (1-5). Treatment-related complications occurred in two patients: Ulceration in one patient and hematoma in the other patient. Two patients had relapse of the disease during the follow-up period (the average period was 42 months).

RFA

RFA allows for a short period of time to transfer powerful energy for ablation of surface lesions, ensuring a uniform depth of impact with a low risk of wall perforation and no need for gas insufflation. A variety of ablative catheters ensures adequate tissue contact. The HALO® RFA system uses two different types of probes with a closely spaced electrode that thermally ablate tissue. The depth (0.5-1 mm) is dependent on the power, density and duration of contact. The generator is connected to either a HALO 360 catheter or a HELO 90 catheter to provide circular or more focused

Table 1 Results of argon plasma coagulation for the treatment of gastric antral vascular ectasia

Ref.	Total number of patients /with LC	Other gastric mucosa lesions	Number of APC sessions	Follow-up (month)	Efficacy	Recurrence, treatment failure, post- treatment blood transfusions, adverse events, complications
Probst et al [44]	17/6	N/A	2.2 (range 1-4)	30.4 (range 1-65)	Endoscopic success was 100%	Recurrence was 29.4%; one patient (5.9%) needed post-treatment blood transfusions; one patient (5.9%) developed antrum stenosis
Yusoff et al [45]	5/3	N/A	2.6 (range 1-4)	20 (range 14-27)	Endoscopic success was 100%	Recurrence was 40%; two patients (40%) needed post-treatment blood transfusions
Sebastian et al[46]	12/2	N/A	Median 2 (range 1-5)	Median 3 (range 1-4)	Endoscopic success was 100%	Two patients (16.7%) needed post- treatment blood transfusions; one of them had ulcer bleeding, second patient had no cause for blood loss identified at repeat endoscopies
Sato et al[47]	8/8	One patient (12.5%) had EV, one patient (12.5%) had PHG	1.8 (range 1-3)	28 (range 12-47)	Endoscopic success was 100%	Recurrence was 25%
Lecleire <i>et al</i> [48]	30/17	Eleven LC patients (64.7%) had EV, four LC patients (23.5%) had PHG	2.18 ± 1.74 in LC patients	Median 20 in LC patients	Endoscopic success in LC patients was 88.2%	Nine LC patients (53%) died during the follow-up; two LC patients (11.8%) who were not successfully treated died from intractable overt bleeding
Herrera et al [49]	8/ N/A	N/A	Median 3 (range 1-10)	28	Endoscopic success was 87.5%	One patient had a recurrence acute upper gastrointestinal bleeding 15 months after treatment completion
Fuccio et al [50]	20/20	All patients had EV, thirteen of them (65%) underwent EBL	Median 4.6 ± 4.6	N/A	Endoscopic success was 100%	Recurrence was 30%; two patients (10%) needed post-treatment blood transfusions; gastric hyperplastic polyps developed in 3 patients (15%) causing active bleeding in 2 cases
Naga et al [51]	29/22	Thirteen patients (44.8%) had PHG	Median 4.6 ± 4.6 (range 1-17)	N/A	N/A	Three patients (10.4%) required post-treatment blood transfusions
Sato et al[52]	22/20	Twelve LC patients (60%) had EV	2.3 (range 1-3)	16.6	There was no need for blood transfusion in most cases	Recurrence was 68.2%; seven patients (31.8%) died during follow-up period, including two cases with bleeding-related deaths
Chiu <i>et al</i> [53]	19/12	Five patients (26.3%) had PHG	2.4 ± 1.4	N/A	Endoscopic success was about 80%	Rebleeding occurred in 15 patients (78.9%); the median duration of rebleeding after treatment was 23 days; three LC patients (25%) died of rebleeding
Keohane et al[54]	15/N/A	Three patients (20%) had PHG	4.1 (range 1-11)	26 (range 1-60)	Endoscopic success was 46.7%	There was a non-significant increase from 7.7 to 9.3 post-treatment blood transfusions
Elhendawy et al[55]	44/44	N/A	3.48 ± 0.902	6	N/A	Nine patients (20.5%) had adverse events, in the form of fever in two patients, abdominal distension in four patients and epigastric pain in three patients
Garg <i>et al</i> [56]	20/16	Four LC patients (25%) had EV	Median 2 (range 1-7) in LC patients	Median 20 (range 2- 65)	Endoscopic success was 40%	Recurrence was 25%
Fábián <i>et al</i> [57]	45/17	N/A	4.43 ± 0.76 (for endoscopic remission)	13.49 ± 2.46	There was a significant increase in hemoglobin levels and a decrease in the need for blood transfusion	Complications occurred in four patients (8.8%): Two patients (4.4%) had Mallory-Weiss syndrome and two patients (4.4%) had gastric ulceration without active bleeding resulting in scarring

LC: Liver cirrhosis; GAVE: Gastric antral vascular ectasia; APC: Argon plasma coagulation; EV: Esophageal varices; PHG: Portal hypertensive gastropathy; EBL: Endoscopic band ligation; N/A: Not available.

ablation[59].

RFA is an alternative to APC for the treatment of GAVE with a good level of efficiency and safety. Technical success, which is defined as complete ablation of the gastric mucosal lesions visible during endoscopy, can reach 90%-100%. Clinical success, which is defined as complete independence from the need for blood transfusions for the 6-month followup period (after the last RFA) and negative endoscopy 6 months after completion of treatment, can reach 71%-87%.

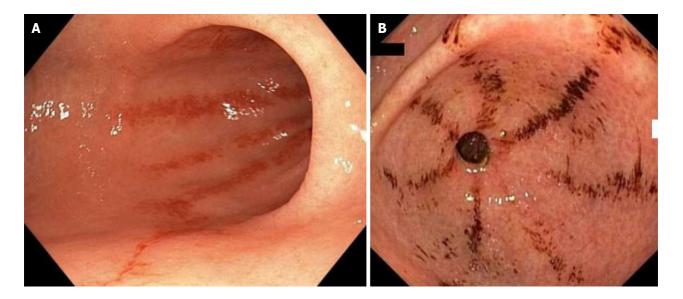


Figure 2 Gastric antral vascular ectasia. A: Linear striped phenotype. Red vascular spots spiraling away from the pylorus; B: Linear striped phenotype. Hemorrhages on the background of red vascular spots spiraling away from the pylorus.

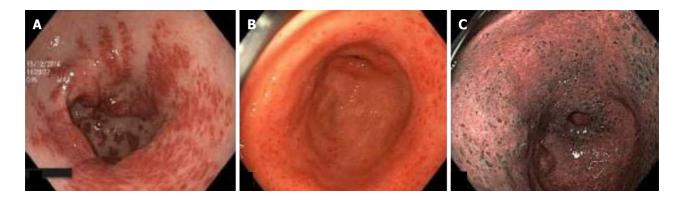


Figure 3 Gastric antral vascular ectasia. A: Diffuse phenotype. Multiple red spots spread out in a diffuse pattern along the gastric antrum; B: Diffuse phenotype in white light endoscopy; C: Diffuse phenotype in endoscopy with narrow-band imaging.

According to the literature, treatment failure, which is defined as any need for blood transfusions after completion of RFA therapy, occurred in 15%-33.3% of cases. Adverse events and complications were rare. Epigastric pain, superficial gastric ulceration and ulcer bleeding were described (Table 2)[60-68].

In a systematic review and meta-analysis of 24 studies (n = 508) using APC and 9 studies (n = 104) using RFA, the comparative efficacy and safety of these two endoscopic technologies for the treatment of GAVE was evaluated. A total of 47% of patients with RFA were refractory to previous APC treatment. RFA required fewer treatment sessions and was accompanied by fewer and less severe complications compared to APC (P < 0.001). Although endoscopic success was better for RFA compared with ARF (P < 0.001), an increase in total hemoglobin after treatment and a decrease in transfusion dependence were higher for APC compared with RFA (P < 0.001)[69].

Magee et al [70] conducted a study comparing the cost-efficacy of RFA and APC for the treatment of patients with APCresistant GAVE. According to their calculations, the introduction of RFA would result in reductions in the need for intravenous iron, endoscopic intervention and requirement for blood transfusions by 27.1%, 32.3% and 36.5%respectively. As a result, RFA will require 36.7% fewer procedures than APC.

EBL

EBL in patients with GAVE allows to reliably obliterate vascular structures in the deep gastric mucosa and submucosa, which reduces the need for further treatment. Ligation bands are applied to the antrum mucosa at the sites of pathological changes. The distal antrum is first treated, then the ligation bands are applied more proximally until most of the gastric mucosal lesions is treated [8]. Many studies have demonstrated the advantages of EBL over other endoscopic technologies for the treatment of GAVE.

In a systematic review and meta-analysis of 10 studies involving 194 patients with GAVE, EBL demonstrated excellent clinical outcomes with minimal adverse events. The pooled rate of treatment responders was 81%, and GAVE recurrence was 15.4%. The pooled mean number of treatment sessions required was 2.4, and the number of bands used to achieve eradication per patient was 15.1. The pooled mean difference of pre- to post-treatment hemoglobin was 1.5 (P = 0.001),

Table 2 Desults a	f vadiation	for the treatment of	monthis autual vasaular actoria
Table 2 Results o	or radiofrequency ablation	for the treatment of	gastric antral vascular ectasia

Ref.	Total number of patients /with LC	Other gastric mucosa lesions	Number of RFA sessions	Follow- up (month)	Efficacy	Recurrence, treatment failure, post- treatment blood transfusions, adverse events, complications
Gross et al [60]	6/N/A	One patient (16.7%) had superficial gastric ulcer	1.7 (range 1- 3)	N/A	There was a significant increase in hemoglobin levels	One patient (16.7%) remained dependent on blood transfusions, but the overall need for blood transfusion decreased
McGorisk et al[61]	21/18	Two patients (9.5%) had EV, five patients (23.8%) had PHG	1.9 ± 0.6 (range 1-3)	6	Technical and clinical successes were 90% and 86%, respectively	Three patients (14.3%) required post- treatment blood transfusions; one patient (4.8%) had superficial gastric ulceration, one patient (4.8%) had ulcer bleeding and one patient (4.8%) continued to have GAVE associated bleeding
Dray et al [62]	24/11	N/A	1.8 ± 0.8 (range 1-3)	6	There was a significant increase in hemoglobin levels and a decrease in the need for blood transfusion	There were no reports of any perioperative or inpatient postoperative complications
Jana <i>et al</i> [63]	7/2	N/A	Median 2 (range 1-3)	Median 6 (range 8- 15)	Technical and clinical successes were 100% and 71%, respectively	Two patients (29%) required post-treatment blood transfusions; there were no reports of any perioperative or inpatient postoperative complications
Raza et al [64]	9/4	One LC patient (25%) had PHG	Median 3 (range 2-6)	Median 11 (range 6- 21)	Technical success was 100%	Three patients (33.3%) had a recurrence of GAVE, and required blood transfusions and retreatment
Dunn <i>et al</i> [65]	15/3	N/A	Median 2	Median 2 (range 1-6)	Clinical and endoscopic responses were 87% and 20%, respectively. There was a significant increase in hemoglobin levels and a decrease in the need for blood transfusion	Four patients (26.7%) required post- treatment blood transfusions
Markos et al[66]	15/0	N/A	Median 2 (range 1-3)	Median 11 (range 5- 18)	Technical and clinical successes were 100% and 80%, respectively. There was a significant increase in hemoglobin levels	Five patients (33.3%) required post-treatment blood transfusions
Senzolo et al[67]	40/40	N/A	N/A	6	There was a significant increase in hemoglobin levels and a decrease in the need for blood transfusion	No major complication occurred and liver function remained stable in all patients
Magee et al[68]	20/4	N/A	Median 2 (range 1-2)	6	There was a significant increase in hemoglobin levels	Three patients (15%) required post- treatment blood transfusions; three patients (15%) experienced epigastric pain which was managed with oral pain medications

LC: Liver cirrhosis; GAVE: Gastric antral vascular ectasia; RFA: Radiofrequency ablation; EV: Esophageal varices; PHG: Portal hypertensive gastropathy; N/A: Not available.

pre- to post-treatment units of packed red blood cells transfused was 1.1 (P = 0.002), and pre- to post-treatment hospital length of stay was 0.5 days (P = 0.01). The overall incidence of adverse events was 15.9%[71].

In a systematic review and meta-analysis of 11 studies involving 393 patients with GAVE, EBL was safe and effective with improved outcomes when compared to APC. Endoscopic success was achieved in 87.84% with average number of treatment sessions of 2.50 ± 0.49 and average of 12.40 ± 3.82 bands applied. For eight studies comparing EBL (n = 143) vs APC (n = 174), there were no differences in baseline patient characteristics. However, endoscopic success was significantly higher for EBL (P = 0.002), which required fewer treatment sessions (P < 0.001). EBL was also associated with a greater increase in post-procedure hemoglobin (P = 0.0140), greater reduction in transfusions required (P = 0.033) and fewer rebleeding events (P < 0.001). There was no difference in adverse events or bleeding-associated mortality (P > 0.001). 0.050)[72].

In a systematic review and meta-analysis of four randomized controlled trials involving 204 patients with GAVE, it was shown that EBL was associated with a higher incidence of endoscopic eradication and fewer recurrence of bleeding than APC. Patients treated with EBL required fewer blood transfusions and hospitalizations. The number of sessions required to repair of gastric mucosal lesions was higher with APC. There were no differences in the frequency of adverse events[73].

In a systematic review and meta-analysis of five studies involving 207 patients with GAVE, over a follow-up period of at least 6 months, patients who underwent EBL had significantly lower post-procedural blood transfusion requirements and significantly higher changes in average hemoglobin levels compared to those who received endoscopic thermal therapy, including APC and electrocautery with heater probe. EBL has led to a reduction in the number of required sessions and a more pronounced change in blood transfusion requirements. There were no differences in the number of adverse events [74].

In a meta-analysis of 12 studies involving 571 patients with GAVE, the efficacy and safety of EBL, endoscopic thermal therapy using APC together with a non-contact device to destroy mucosal microvasculature, and RFA was compared. When compared with endoscopic thermal therapy, EBL allowed for better bleeding control (P = 0.01), higher hemoglobin improvement (P < 0.01), and fewer number of sessions (P = 0.01). In addition, EBL outperformed endoscopic thermal therapy in terms of improved endoscopic outcomes (P < 0.01), hospitalization (P < 0.01), and blood transfusion requirements (P = 0.01) with statistical significance, excluding mortality (P = 0.34) and complication rate (P = 0.14), while RFA did not show differences in any of these outcomes [75].

At the moment, the optimal endoscopic technologies for the treatment of GAVE has not yet been determined. This is due to the lack of sufficient number of randomized controlled trials involving a large cohort of patients necessary to evaluate them.

PHPs

Gastric hyperplastic polyps account for 30% to 93% of all gastric epithelial polyps. Their specific histological features are dilated, elongated, tortuous foveolae (which give them the appearance of a corkscrew) lined by hyperplastic epithelium containing mucin[76]. Gastric hyperplastic polyps are usually associated with H. pylori-associated gastritis, chronic atrophic gastritis, autoimmune gastritis, portal hypertension and gastric surgery [77]. Cases of their development after APC and EBL for GAVE are described [78,79].

Gastric hyperplastic polyps are a common finding during conventional EGD in LC patients. In a study by Topal et al [80] involving 200 LC patients, polypoid lesions were detected in 33.5% of cases, and their rate was statistically higher in the advanced chronic liver disease. PHPs have been described in LC patients with portal hypertension, portal venous obstruction or GAVE, have a low risk of malignant transformation, and can cause pyloric obstruction, acute and chronic upper gastrointestinal bleeding leading to anemia[81]. The prevalence of PHPs in LC patients ranges from 1% to 8%[82]. These polyploid lesions are associated with portal hypertension and therefore require its correction [83]. PHPs are similar to gastric hyperplastic polyps, but with presence of larger and more numerous vascular capillaries in the lamina propria. PHPs can be single or numerous, located in the antrum or the gastric corpus (Figure 4). Their size may range from a few millimeters to several centimeters [84].

Independent risk factors for the development of PHPs are thrombocytopenia (platelet count $< 130 \times 10^{\circ}/L$), CTP score > 6, and MELD score > 16. An unfavorable event is the preliminary EBL of GEVs, which may be due to the increased formation of portosystemic shunts in the gastric wall[85].

The management of gastric hyperplastic polyps is determined by their size and the presence of dysplasia. Endoscopic resection is recommended for gastric hyperplastic polyps that biopsy-proven dysplasia (usually > 10 mm), have pedunculated morphology and are symptomatic [86]. At the same time, there are no specific recommendations for the management of PHPs. Due to the benign nature of PHPs, frequent recurrence, and the risk of significant bleeding from the feeding vessels located deep within the gastric submucosa, it is most advisable to remove them only in case of complications[87]. In a single-center retrospective study by Nelson et al [88] involving 63 patients of whom 20 (31%) had LC, the efficacy of endoscopic resection for the treatment of transfusion-dependent iron-deficiency anemia or upper gastrointestinal bleeding due to gastric hyperplastic polyps was investigated. The majority of LC patients had upper gastrointestinal bleeding (n = 13.65%, P = 0.52), whereas the majority of non-cirrhotics presented with transfusion-dependent irondeficiency anemia (n = 30.70%, P = 0.01). Technical success was 100% with no adverse events. The clinical success rate was 94% (95% in LC patients and 93% in non-cirrhotics, P = 0.46). The recurrence rate was 32% (40% in LC patients and 28% in non-cirrhotics, P = 0.35) with mean time to recurrence of 17.3 ± 13.9 months (P = 0.22). Of those with recurrence, 75% had no further transfusion-dependent iron-deficiency anemia or upper gastrointestinal bleeding after repeat endoscopic resection (mean follow-up 20 ± 11 months). At the same time, in a study by Cui et al[89], the rate of post-endoscopic polypectomy bleeding in LC patients was 16.5%, while the rate of post-endoscopic polypectomy bleeding non-cirrhotic group was 2.4% (P < 0.001). CTP class B and C, severe GEVs, and polyps located in the stomach were independent risk factors for bleeding (P < 0.05).

Hot snare polypectomy (HSP) is the most acceptable procedure for endoscopic resection of gastric hyperplastic polyps. In a study by Elsabaawy et al[90] involving 100 LC patients, the efficacy and safety of EBL and HSP in resecting gastric hyperplastic polyps were compared. It turned out that the average operational time with EBL was more than two times shorter than with HSP (P < 0.001). There were no complications in 94% of EBL cases, compared with 78% for HSP. Bleeding occurred only with HSP (20%). The cost was significantly lower with EBL than with HSP (P < 0.001). The recurrence rate of hyperplastic gastric polyps and the number of required sessions did not differ significantly.

Indeed, post-polypectomy bleeding is one of the most common and serious complications, especially in LC patients. To increase the efficacy of conventional methods of endoscopic hemostasis, the "metallic tulip-bundle" technique, combining through the scope and over-the-scope clips, can be applied[91].

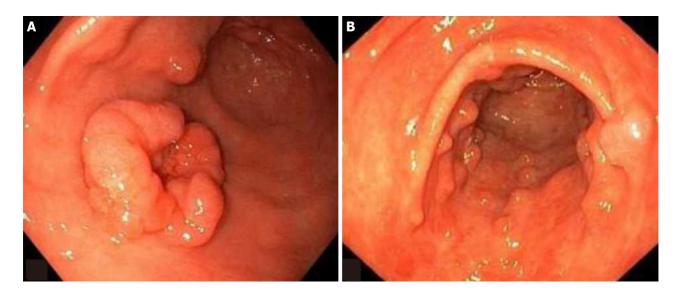


Figure 4 Portal hypertensive polyps. A: Polypoid lesion in the gastric antrum up to 1 cm in diameter with an indentation in the middle, and of a soft-elastic consistency; B: Multiple polypoid lesions in the gastric antrum on a wide base from 3 mm to 7 mm in diameter.

PUD IN LC PATIENTS

LC patients have a high risk of developing PUD, which is associated with a significant prevalence of *H. pylori* infection [92]. In a study by Voulgaris et al[93] involving 100 LC patients, PUD was found in 19 (19%) (14 gastric, 5 duodenal). H. pylori infection was diagnosed in 54% LC patients. PUD was unrelated to the etiology and the severity of liver disease or to other endoscopic manifestations of portal hypertension (GEVs and PHG), was asymptomatic and had no known risk factors of ulcerogenicity.

It was also noted that LC patients have a high risk of peptic ulcer bleeding, which significantly worsens their prognosis. For example, in a study by Gado et al [94], among 103 LC patients who were admitted with non-variceal upper gastrointestinal bleeding, 62 (60%) had peptic ulcer bleeding. A systematic review including 23 studies involving a total of 1288 LC patients showed that peptic ulcer bleeding was the most common cause of acute non-variceal bleeding, followed by PHG, GAVE, Mallory-Weiss syndrome, Dieaulafoy lesions, portal hypertensive colopathy, and hemorrhoids [95].

In a study by Pariente et al [96] showed that in-hospital mortality was significantly greater in LC patients with peptic ulcer bleeding as compared to the general population (17.4% vs 4.7%). In a study by Ardevol et al [97] involving 790 LC patients, 646 (81.8%) were admitted with acute variceal bleeding and 144 (18.2%) with peptic ulcer bleeding. The CTP and MELD scores were the same. Further bleeding was more frequent in the acute variceal bleeding group than those in the peptic ulcer bleeding group. However, the risk of mortality at 45 days was the same in both groups (P = 0.48). Various parameters such as CTP score, acute kidney injury, acute on chronic liver failure, or presence of shock or bacterial infection, but not the cause of bleeding, were associated with the risk of death. Only 2% of peptic ulcer bleeding group vs 3% of acute variceal bleeding group died with uncontrolled bleeding (P = 0.39), while the majority of patients in both groups died from liver failure or attributed to other comorbidities.

In a study by Lu et al[98] involving 650 LC patients, 402 (61.9%) had acute variceal bleeding and 248 (38.1%) had peptic ulcer bleeding. Univariate and multivariate analysis identified prothrombin time [odds ratio (OR) = 0.884, 95% confidence interval (CI): 0.786-0.995; P = 0.041], MELD score (OR = 1.153, 95%CI: 1.073-1.240; P = 0.000), emergency intervention (OR = 8.656, 95%CI: 2.219-33.764; P = 0.002), hepatic encephalopathy before bleeding (OR = 8.119, 95%CI: 2.084-31.637; P = 0.003) and hepatorenal syndrome before bleeding (OR = 3.877, 95%CI: 1.152-13.045; P = 0.029) as the independent predictors for 42-day mortality.

Endoscopic therapy for peptic ulcer bleeding is carried out according to the current guidelines, in particular, the European Society of Gastrointestinal Endoscopy (ESGE). ESGE recommends the Forrest (F) classification be used in all patients with peptic ulcer bleeding to differentiate low risk and high risk endoscopic stigmata. The basic principles of endoscopic therapy for peptic ulcer bleeding are as follows: (1) For actively bleeding ulcers (FIa, FIb), combination therapy is performed using epinephrine injection plus a second hemostasis modality (contact thermal or mechanical therapy); (2) In selected actively bleeding ulcers (FIa, FIb), specifically those > 2 cm in size, with a large visible vessel > 2 mm, or located in a high-risk vascular area (e.g., gastroduodenal, left gastric arteries), or in excavated/fibrotic ulcers, endoscopic hemostasis using a cap-mounted clip should be considered as first-line therapy; (3) Patients with an ulcer with a nonbleeding visible vessel (FIIa) are recommended contact or noncontact thermal therapy, mechanical therapy, or injection of a sclerosing agent, each as monotherapy or in combination with epinephrine injection; (4) Patients with persistent bleeding refractory to standard hemostasis modalities, the use of a topical hemospray/powder or cap-mounted clip should be considered; and (5) The use of hemostatic forceps as an alternative endoscopic hemostasis option in peptic ulcer bleeding can be considered[10].

For patients with acid-related causes of non-variceal upper gastrointestinal bleeding different from PUD (e.g., erosive gastritis), ESGE recommends treatment with high dose proton pump inhibitors. Endoscopic hemostasis is usually not required[99].

EARLY GASTRIC CANCER IN LC PATIENTS

A higher incidence of gastric cancer in LC patients has been reported [100]. This is due to the existence of such unfavorable factors as a high incidence of gastric ulcer and PHG, zinc deficiency, alcohol drinking and tobacco use and gut dysbiosis[101], as well as a significant prevalence of *H. pylori* infection[102]. The presence of LC creates significant difficulties for the treatment of gastric cancer patients [103]. Early gastric cancer is an invasive carcinoma that affects only the gastric mucosa or submucosa, regardless of lymph node status [104]. EMR and ESD are usually used to treat it [105]. It has been shown that these endoscopic technologies can be successfully applied in LC patients with early gastric cancer [106].

Endoscopic resection of low-grade dysplasia is safe (perforation and bleeding rates are 1% and 7%, respectively) and is associated with reduced rates of progression to high-grade dysplasia or carcinoma. Lesions greater than 10 mm with high-grade dysplasia or depressed lesions are more likely to harbor carcinoma and should be resected with ESD. Compared with EMR, ESD has significantly higher rates of "en bloc" resection, higher rates of complete resection (negative histologic margins), and lower recurrence rates but requires longer procedure times and results in significantly higher perforation rates. There are no significant differences in post-procedure bleeding between EMR and ESD[13].

In a cross-sectional retrospective cohort study by Horiki et al [107], the four LC patients with early gastric cancer received EMR. No patients died of the cancer and no postprocedural complications occurred in the 10-year follow-up

In a case-control multicenter retrospective study involving 69 LC patients (53 of whom had CTP class A) with early gastric cancer, the short- and long-term outcomes of ESD was examined. It turned out that although LC patients had significantly worse long-term outcomes than noncirrhotic patients (the 5-year overall survival rates were 60% vs 91%, respectively), the LC patients CTP class A had an overall survival almost equivalent to that of outcomes than noncirrhotic patients. The short-term outcomes of LC patients CTP class B or C were no worse, but they benefited less from ESD[108].

In a multicenter retrospective study involving 43 patients with HBV-related decompensated LC undergoing ESD for the treatment of early gastric cancer, the procedural outcomes and post-procedural complications (bleeding or perforation) did not differ significantly between LC patients and noncirrhotic patients, as well as between LC patients CTP class A and LC patients CTP class B. None of the patients showed a deterioration of the CTP score 1 month after ESD compared to baseline values. During the average follow-up period of 66 months, the recurrence rate of gastric cancer in LC patients and noncirrhotic patients was the same (P = 0.925), and all recurrent gastric cancers were completely resected by additional ESD. The overall mortality rate was higher in LC patients compared with noncirrhotic patients (P = 0.034), and 8 of 10 deaths were associated with liver-related diseases (such as hepatocellular carcinoma, complications of portal hypertension, hepatic failure)[109].

Barakat et al [110] performed a systematic review of MEDLINE and Cochrane database followed by a pooled dataanalysis, to evaluate the safety and efficacy of ESD in LC patients with early gastric cancer. "En bloc" resection was achieved in 94% of lesions (P = 0.92) and R0 margins were achieved in 90% of lesions (P = 0.65). There were 9% of bleeding events (P = 0.51), all of which occurred in LC patients CTP class B (P < 0.0001), with platelets count less than 105000/mL and international normalized ratio of more than 1.3. There was one (0.9%) reported perforation (P = 0.57) and no mortality related to the ESD procedure.

In a retrospective study by Kim et al [111], the long-term efficacy and safety of ESD for the treatment of early gastric cancer in LC patients (n = 17) and noncirrhotic patients (n = 671) was analyzed. No significant differences were found in the type of procedure, complications, "en bloc" resection rate, and complete resection rate between the two groups. However, cancer recurrence was more common in LC patients (P = 0.038).

In a retrospective cohort study by Pecha et al[112], the outcomes of ESD for the treatment of early gastric cancer in LC patients (n = 16) and noncirrhotic patients (n = 43) was evaluated. "En bloc" and curative resection was achieved in 84.21%, 78.94% of LC patients and 88.89%, 68.89% of noncirrhotic patients, respectively, with no significant differences. LC patients had significantly higher rates of intraprocedural coagulation grasper use for control of bleeding (47.37% vs 20%; P = 0.02). There were otherwise no significant differences in adverse event rates. A higher rates of recurrence was found in LC patients compared to noncirrhotic patients (40% vs 5.26%; P = 0.019).

The results of the conducted studies show that EMR and ESD are suitable for the treatment of early gastric cancer, and the main procedure-associated complications are bleeding and perforation. The risk of bleeding during endoscopic procedures in LC patients is increasing with the rise of portal pressure and worsening of hemostasis disorders, and is statistically significantly higher in CTP class C or MELD score > 11.5. The risk factors for bleeding can be divided into three groups: (1) Risk of vascular origin; (2) Risk related to disorders of platelet count and function; and (3) Risk related to deficiency of coagulation factors and disorders of fibrinolysis [113]. Currently, there are no specific recommendations for the use of ESD for the treatment of early gastric cancer accompanied by GEVs. There are reports that this may be safe after their prior eradication[114]. Disturbances of coagulation and hemostasis are common in LC patients. A study by Repici et al[115] showed that LC patients with either international normalized ratio > 1.33 and/or platelets count < 105000/mm³ should be regarded at increased risk of bleeding following ESD. In a study by Drolz et al[116], the platelet count $< 30 \times 10^{9}$ /L, fibrinogen level < 60 mg/dL, and activated partial thromboplastin time values > 100 seconds were the strongest independent predictors for severe bleeding and served as contraindications for performing invasive procedures in LC patients.

CONCLUSION

LC can cause a wide spectrum of gastric mucosal lesions, the main diagnostic method of which is EGD. This is a common and generally safe endoscopic procedure for the diagnosis and treatment of upper gastrointestinal tract lesions. If visual differential diagnosis is difficult, EUS and new digital and optical endoscopic technologies such as magnifying endoscopy with NBI may be useful. In this aspect, the development of software based on artificial intelligence as a decision-making assistance system is promising. In many cases, endoscopic technologies are also the methods of choice for the treatment of gastric mucosal lesions in LC patients and their use is recommended by current guidelines. New endoscopic therapeutic options are currently being proposed to replace outdated and not always harmless ones. Further clinical trials involving a large cohort of LC patients are required to evaluate their efficacy and safety.

FOOTNOTES

Author contributions: Olevskaya ER contributed to conception; Olevskaya ER and Dolgushina AI contributed to design; Dolgushina AI and Garbuzenko DV approved the final version; Olevskaya ER, Garbuzenko DV, and Khikhlova AO wrote the manuscript; Dolgushina AI, Olevskaya ER, Garbuzenko DV, Khikhlova AO, Saenko AA, Khusainova GM, and Kuznetsova AS contributed to analysis; Olevskaya ER, Garbuzenko DV, Khikhlova AO, Saenko AA, Khusainova GM, and Kuznetsova AS contributed to acquisition, and interpretation of

Conflict-of-interest statement: All the authors report no relevant conflicts of interest for this article.

Open Access: This article is an open-access article that was selected by an in-house editor and fully peer-reviewed by external reviewers. It is distributed in accordance with the Creative Commons Attribution NonCommercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited and the use is non-commercial. See: https://creativecommons.org/Licenses/by-nc/4.0/

Country of origin: Russia

ORCID number: Elena R Olevskaya 0000-0002-7385-8505; Anastasia I Dolgushina 0000-0003-2569-1699; Dmitry V Garbuzenko 0000-0001-9809-8015; Alina O Khikhlova 0000-0001-6444-4573; Anna A Saenko 0000-0002-3481-8092; Guzel M Khusainova 0000-0001-5954-3112; Alla S Kuznetsova 0000-0002-0357-5702.

S-Editor: Bai Y L-Editor: A P-Editor: Lei YY

REFERENCES

- Huang DQ, Terrault NA, Tacke F, Gluud LL, Arrese M, Bugianesi E, Loomba R. Global epidemiology of cirrhosis aetiology, trends and predictions. Nat Rev Gastroenterol Hepatol 2023; 20: 388-398 [RCA] [PMID: 36977794 DOI: 10.1038/s41575-023-00759-2] [FullText] [Full
- 2 GBD 2017 Cirrhosis Collaborators. The global, regional, and national burden of cirrhosis by cause in 195 countries and territories, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet Gastroenterol Hepatol 2020; 5: 245-266 [RCA] [PMID: 31981519 DOI: 10.1016/S2468-1253(19)30349-8] [FullText] [Full Text(PDF)]
- Nagula S, Parasa S, Laine L, Shah SC. AGA Clinical Practice Update on High-Quality Upper Endoscopy: Expert Review. Clin Gastroenterol 3 Hepatol 2024; 22: 933-943 [RCA] [PMID: 38385942 DOI: 10.1016/j.cgh.2023.10.034] [FullText]
- Maydeo A, Patil G. How to Approach a Patient With Gastric Varices. Gastroenterology 2022; 162: 689-695 [RCA] [PMID: 34979100 DOI: 4 10.1053/j.gastro.2021.12.277] [FullText]
- 5 Ma C, Chen CH, Liu TC. The spectrum of gastric pathology in portal hypertension-An endoscopic and pathologic study of 550 cases. Pathol Res Pract 2016; 212: 704-709 [RCA] [PMID: 27461830 DOI: 10.1016/j.prp.2016.05.005] [FullText]
- 6 Laleman W, Vanderschueren E, Mehdi ZS, Wiest R, Cardenas A, Trebicka J. Endoscopic procedures in hepatology: Current trends and new developments. J Hepatol 2024; 80: 124-139 [RCA] [PMID: 37730125 DOI: 10.1016/j.jhep.2023.08.032] [FullText]
- Gono K. Narrow Band Imaging: Technology Basis and Research and Development History. Clin Endosc 2015; 48: 476-480 [RCA] [PMID: 26668792 DOI: 10.5946/ce.2015.48.6.476] [FullText] [Full Text(PDF)]
- Luigiano C, Iabichino G, Judica A, Virgilio C, Peta V, Abenavoli L. Role of endoscopy in management of gastrointestinal complications of portal hypertension. World J Gastrointest Endosc 2015; 7: 1-12 [RCA] [PMID: 25610530 DOI: 10.4253/wjge.v7.i1.1] [Full Text] [Full Text (PDF)]
- de Franchis R, Bosch J, Garcia-Tsao G, Reiberger T, Ripoll C; Baveno VII Faculty. Baveno VII Renewing consensus in portal hypertension. J Hepatol 2022; 76: 959-974 [RCA] [PMID: 35120736 DOI: 10.1016/j.jhep.2021.12.022] [FullText]
- Gralnek IM, Stanley AJ, Morris AJ, Camus M, Lau J, Lanas A, Laursen SB, Radaelli F, Papanikolaou IS, Cúrdia Gonçalves T, Dinis-Ribeiro M, Awadie H, Braun G, de Groot N, Udd M, Sanchez-Yague A, Neeman Z, van Hooft JE. Endoscopic diagnosis and management of

11



- nonvariceal upper gastrointestinal hemorrhage (NVUGIH): European Society of Gastrointestinal Endoscopy (ESGE) Guideline Update 2021. Endoscopy 2021; **53**: 300-332 [RCA] [PMID: 33567467 DOI: 10.1055/a-1369-5274] [FullText]
- Laine L, Barkun AN, Saltzman JR, Martel M, Leontiadis GI. ACG Clinical Guideline: Upper Gastrointestinal and Ulcer Bleeding. Am J Gastroenterol 2021; 116: 899-917 [RC4] [PMID: 33929377 DOI: 10.14309/ajg.000000000001245] [FullText]
- Fujishiro M, Iguchi M, Ono S, Funasaka K, Sakata Y, Mikami T, Kataoka M, Shimaoka S, Michida T, Igarashi Y, Tanaka S. Guidelines for 12 endoscopic management of nonvariceal upper gastrointestinal bleeding (second edition). Dig Endosc 2025; 37: 447-469 [RCA] [PMID: 40114631 DOI: 10.1111/den.15019] [FullText]
- Morgan DR, Corral JE, Li D, Montgomery EA, Riquelme A, Kim JJ, Sauer B, Shah SC. ACG Clinical Guideline: Diagnosis and Management 13 of Gastric Premalignant Conditions. Am J Gastroenterol 2025; 120: 709-737 [RCA] [PMID: 40072510 DOI: 10.14309/ajg.00000000000003350]
- Gjeorgjievski M, Cappell MS. Portal hypertensive gastropathy: A systematic review of the pathophysiology, clinical presentation, natural history and therapy. World J Hepatol 2016; 8: 231-262 [RCA] [PMID: 26855694 DOI: 10.4254/wjh.v8.i4.231] [FullText] [Full Text(PDF)]
- Casas M, Vergara M, Brullet E, Junquera F, Martínez-Bauer E, Miquel M, Sánchez-Delgado J, Dalmau B, Campo R, Calvet X. Inter and intra-15 observer concordance for the diagnosis of portal hypertension gastropathy. Rev Esp Enferm Dig 2018; 110: 166-171 [RCA] [PMID: 29320862 DOI: 10.17235/reed.2018.5169/2017] [FullText]
- 16 Kaplan DE, Ripoll C, Thiele M, Fortune BE, Simonetto DA, Garcia-Tsao G, Bosch J. AASLD Practice Guidance on risk stratification and management of portal hypertension and varices in cirrhosis. Hepatology 2024; 79: 1180-1211 [RCA] [PMID: 37870298 DOI: 10.1097/HEP.0000000000000647] [FullText]
- Marrache MK, Bou Daher H, Rockey DC. The relationship between portal hypertension and portal hypertensive gastropathy. Scand J 17 Gastroenterol 2022; 57: 340-344 [RCA] [PMID: 34904513 DOI: 10.1080/00365521.2021.2012591] [FullText]
- Kim MY, Choi H, Baik SK, Yea CJ, Won CS, Byun JW, Park SY, Kwon YH, Kim JW, Kim HS, Kwon SO, Kim YJ, Cha SH, Chang SJ. 18 Portal hypertensive gastropathy: correlation with portal hypertension and prognosis in cirrhosis. Dig Dis Sci 2010; 55: 3561-3567 [RCA] [PMID: 20407828 DOI: 10.1007/s10620-010-1221-6] [FullText]
- Elnaser SS, El-Ebiary S, Bastawi MB, Shafei AL, Abd-Elhafee A. Effect of endoscopic injection sclerotherapy and variceal ligation on development of portal hypertensive gastropathy and duodenopathy. J Egypt Soc Parasitol 2005; 35: 253-264 [RCA] [PMID: 15881011] [Full
- 20 Carpinelli L, Primignani M, Preatoni P, Angeli P, Battaglia G, Beretta L, Bortoli A, Capria A, Cestari R, Cosentino F, Crotta S, Gerunda G, Lorenzini I, Maiolo P, Merighi A, Rossi A, Sangiovanni A, de Franchis R. Portal hypertensive gastropathy: reproducibility of a classification, prevalence of elementary lesions, sensitivity and specificity in the diagnosis of cirrhosis of the liver. A NIEC multicentre study. New Italian Endoscopic Club. Ital J Gastroenterol Hepatol 1997; 29: 533-540 [RCA] [PMID: 9513828] [FullText]
- Rockey DC. An Update: Portal Hypertensive Gastropathy and Colopathy. Clin Liver Dis 2019; 23: 643-658 [RCA] [PMID: 31563216 DOI: 21 10.1016/j.cld.2019.07.002] [FullText]
- 22 Bella MR, Casas M, Vergara M, Brullet E, Junquera F, Martínez-Bauer E, Miquel M, Sánchez-Delgado J, Dalmau B, Campo R, Calvet X. Utility of histology for the diagnosis of portal hypertensive gastroenteropathy. Concordance between the endoscopic image and gastrointestinal biopsies. Role of the CD34 marker. Gastroenterol Hepatol 2019; 42: 150-156 [RCA] [PMID: 30459058 DOI: 10.1016/j.gastrohep.2018.09.008] [FullText]
- Macedo G, Aranzana E, Ribeiro M, Szutan L, Ferreira F. Endoscopic contrast enhanced ultrasound in the study of portal hypertensive 23 gastropathy. HPB 2016; 18: e529-e530 [DOI: 10.1016/j.hpb.2016.03.411] [FullText]
- 24 Yi Z, Zhang M, Ma Z, Tuo B, Liu A, Deng Z, Zhao Y, Li T, Liu X. Role of the posterior mucosal defense barrier in portal hypertensive gastropathy. Biomed Pharmacother 2021; 144: 112258 [RCA] [PMID: 34614465 DOI: 10.1016/j.biopha.2021.112258] [FullText]
- Fontana RJ, Sanyal AJ, Ghany MG, Bonkovsky HL, Morgan TR, Litman HJ, Reid AE, Lee WM, Naishadham D; HALT-C Trial Study 25 Group. Development and progression of portal hypertensive gastropathy in patients with chronic hepatitis C. Am J Gastroenterol 2011; 106: 884-893 [RCA] [PMID: 21139575 DOI: 10.1038/ajg.2010.456] [FullText]
- 26 European Association for the Study of the Liver. EASL Clinical Practice Guidelines for the management of patients with decompensated cirrhosis. J Hepatol 2018; 69: 406-460 [RCA] [PMID: 29653741 DOI: 10.1016/j.jhep.2018.03.024] [FullText]
- Hanafy AS, El Hawary AT. Efficacy of argon plasma coagulation in the management of portal hypertensive gastropathy. Endosc Int Open 27 2016; 4: E1057-E1062 [RCA] [PMID: 27747278 DOI: 10.1055/s-0042-114979] [Full Text(PDF)]
- El Shahawy MS, Shady ZM, Gaafar A. The Efficacy of Argon Plasma Coagulation versus Carvedilol for Treatment of Portal Hypertensive 28 Gastropathy. Digestion 2020; 101: 651-658 [RCA] [PMID: 31563912 DOI: 10.1159/000502814] [FullText]
- Papaefthymiou A, Aslam N, Hussein M, Alzoubaidi D, Gross SA, Serna A, Varbobitis I, Hengehold TA, López MF, Fernández-Sordo JO, Rey JW, Hayee B, Despott EJ, Murino A, Moreea S, Boger P, Dunn JM, Mainie I, Mullady D, Early D, Latorre M, Ragunath K, Anderson JT, Bhandari P, Goetz M, Kiesslich R, Coron E, Santiago ER, Gonda TA, O'Donnell M, Norton B, Telese A, Simons-Linares R, Haidry R. Hemospray® (hemostatic powder TC-325) as monotherapy for acute gastrointestinal bleeding: a multicenter prospective study. Ann Gastroenterol 2024; 37: 418-426 [RCA] [PMID: 38974074 DOI: 10.20524/aog.2024.0897] [FullText] [Full Text(PDF)]
- Smith LA, Morris AJ, Stanley AJ. The use of hemospray in portal hypertensive bleeding; a case series. J Hepatol 2014; 60: 457-460 [RCA] [PMID: 24140803 DOI: 10.1016/j.jhep.2013.10.008] [FullText]
- Fuccio L, Mussetto A, Laterza L, Eusebi LH, Bazzoli F. Diagnosis and management of gastric antral vascular ectasia. World J Gastrointest 31 Endosc 2013; 5: 6-13 [RCA] [PMID: 23330048 DOI: 10.4253/wjge.v5.i1.6] [FullText] [Full Text(PDF)]
- Smith E, Davis J, Caldwell S. Gastric Antral Vascular Ectasia Pathogenesis and the Link to the Metabolic Syndrome. Curr Gastroenterol Rep 32 2018; **20**: 36 [*RCA*] [PMID: 29961911 DOI: 10.1007/s11894-018-0644-7] [FullText]
- 33 Alkhormi AM, Memon MY, Algarawi A. Gastric Antral Vascular Ectasia: A Case Report and Literature Review. J Transl Int Med 2018; 6: 47-51 [RCA] [PMID: 29607305 DOI: 10.2478/jtim-2018-0010] [FullText]
- Aryan M, Jariwala R, Alkurdi B, Peter S, Shoreibah M. The misclassification of gastric antral vascular ectasia. J Clin Transl Res 2022; 8: 218-34 223 [*RCA*] [PMID: 35813901] [FullText] [Full Text(PDF)]
- Kwon HJ, Lee SH, Cho JH. Influences of etiology and endoscopic appearance on the long-term outcomes of gastric antral vascular ectasia. 35 World J Clin Cases 2022; 10: 6050-6059 [RCA] [PMID: 35949841 DOI: 10.12998/wjcc.v10.i18.6050] [FullText] [Full Text(PDF)]
- Thomas A, Koch D, Marsteller W, Lewin D, Reuben A. An Analysis of the Clinical, Laboratory, and Histological Features of Striped, 36 Punctate, and Nodular Gastric Antral Vascular Ectasia. Dig Dis Sci 2018; 63: 966-973 [RCA] [PMID: 29450749 DOI:



- 10.1007/s10620-018-4965-z] [FullText]
- Parente F, Petrillo M, Vago L, Bianchi Porro G. The watermelon stomach: clinical, endoscopic, endosonographic, and therapeutic aspects in 37 three cases. Endoscopy 1995; 27: 203-206 [RCA] [PMID: 7601056 DOI: 10.1055/s-2007-1005663] [FullText]
- Shudo R, Yazaki Y, Sakurai S, Uenishi H, Yamada H, Sugawara K. Diffuse antral vascular ectasia: EUS after argon plasma coagulation. 38 Gastrointest Endosc 2001; 54: 623 [RCA] [PMID: 11677481 DOI: 10.1067/gien.2001.0001] [FullText]
- 39 Angelov T, Churchev S, Vladimirov B, Golemanov B. Endoscopic ultrasound of gastric antral vascular ectasia. Br J Hosp Med (Lond) 2021; 82: 1 [RCA] [PMID: 33792386 DOI: 10.12968/hmed.2020.0547] [FullText]
- Chang CY, Chen PH, Hou MC, Chang WC, Yang TC, Hsin IF, Liao WC, Lee FY. Magnifying endoscopy with narrow-band image for diagnosing diffuse type of gastric antral vascular ectasia in cirrhotic patients. Eur J Gastroenterol Hepatol 2021; 33: 495-500 [RCA] [PMID: 32433425 DOI: 10.1097/MEG.000000000001757] [FullText]
- Abdelmoneim RSE, Abdelmoety AA, Baddour N, Salem P, Metawea M. The classification of gastric antral vascular ectasia in cirrhotic 41 patients by Versatile Intelligent Staining Technology. Egypt Liver J 2022; 12: 34 [RCA] [PMID: 35702616 DOI: 10.1186/s43066-022-00198-9] [FullText] [Full Text(PDF)]
- Al-Taee AM, Cubillan MP, Hinton A, Sobotka LA, Befeler AS, Hachem CY, Hussan H. Accuracy of virtual chromoendoscopy in differentiating gastric antral vascular ectasia from portal hypertensive gastropathy: A proof of concept study. World J Hepatol 2021; 13: 2168-2178 [RCA] [PMID: 35070017 DOI: 10.4254/wjh.v13.i12.2168] [FullText] [Full Text(PDF)]
- Peng M, Guo X, Yi F, Shao X, Wang L, Wu Y, Wang C, Zhu M, Bian O, Ibrahim M, Chawla S, Qi X. Endoscopic treatment for gastric antral 43 vascular ectasia. Ther Adv Chronic Dis 2021; 12: 20406223211039696 [RCA] [PMID: 34408826 DOI: 10.1177/20406223211039696] [Full Text] [Full Text(PDF)]
- Probst A, Scheubel R, Wienbeck M. Treatment of watermelon stomach (GAVE syndrome) by means of endoscopic argon plasma coagulation (APC): long-term outcome. Z Gastroenterol 2001; 39: 447-452 [RCA] [PMID: 11474999 DOI: 10.1055/s-2001-15722] [FullText]
- 45 Yusoff I, Brennan F, Ormonde D, Laurence B. Argon plasma coagulation for treatment of watermelon stomach. *Endoscopy* 2002; 34: 407-410 [RCA] [PMID: 11972274 DOI: 10.1055/s-2002-25287] [FullText]
- 46 Sebastian S, McLoughlin R, Qasim A, O'Morain CA, Buckley MJ. Endoscopic argon plasma coagulation for the treatment of gastric antral vascular ectasia (watermelon stomach): long-term results. Dig Liver Dis 2004; 36: 212-217 [RCA] [PMID: 15046192 DOI: 10.1016/j.dld.2003.11.028] [FullText]
- Sato T, Yamazaki K, Toyota J, Karino Y, Ohmura T, Akaike J, Kuwata Y, Suga T. Efficacy of argon plasma coagulation for gastric antral 47 vascular ectasia associated with chronic liver disease. Hepatol Res 2005; 32: 121-126 [RCA] [PMID: 15967712 DOI: 10.1016/j.hepres.2005.04.004] [FullText]
- Lecleire S, Ben-Soussan E, Antonietti M, Goria O, Riachi G, Lerebours E, Ducrotté P. Bleeding gastric vascular ectasia treated by argon plasma coagulation: a comparison between patients with and without cirrhosis. Gastrointest Endosc 2008; 67: 219-225 [RCA] [PMID: 18226684 DOI: 10.1016/j.gie.2007.10.016] [FullText]
- Herrera S, Bordas JM, Llach J, Ginès A, Pellisé M, Fernández-Esparrach G, Mondelo F, Mata A, Cárdenas A, Castells A. The beneficial effects of argon plasma coagulation in the management of different types of gastric vascular ectasia lesions in patients admitted for GI hemorrhage. Gastrointest Endosc 2008; 68: 440-446 [RCA] [PMID: 18423466 DOI: 10.1016/j.gie.2008.02.009] [FullText]
- Fuccio L, Zagari RM, Serrani M, Eusebi LH, Grilli D, Cennamo V, Laterza L, Asioli S, Ceroni L, Bazzoli F. Endoscopic argon plasma 50 coagulation for the treatment of gastric antral vascular ectasia-related bleeding in patients with liver cirrhosis. *Digestion* 2009; 79: 143-150 [RCA] [PMID: 19329853 DOI: 10.1159/000210087] [FullText]
- Naga M, Esmat S, Naguib M, Sedrak H. Long-term effect of argon plasma coagulation (APC) in the treatment of gastric antral vascular ectasia 51 (GAVE). Arab J Gastroenterol 2011; 12: 40-43 [RCA] [PMID: 21429455 DOI: 10.1016/j.ajg.2011.01.012] [FullText]
- 52 Sato T, Yamazaki K, Akaike J. Endoscopic band ligation versus argon plasma coagulation for gastric antral vascular ectasia associated with liver diseases. Dig Endosc 2012; 24: 237-242 [RCA] [PMID: 22725108 DOI: 10.1111/j.1443-1661.2011.01221.x] [FullText]
- Chiu YC, Lu LS, Wu KL, Tam W, Hu ML, Tai WC, Chiu KW, Chuah SK. Comparison of argon plasma coagulation in management of upper 53 gastrointestinal angiodysplasia and gastric antral vascular ectasia hemorrhage. BMC Gastroenterol 2012; 12: 67 [RCA] [PMID: 22681987 DOI: 10.1186/1471-230X-12-67] [FullText] [Full Text(PDF)]
- Keohane J, Berro W, Harewood GC, Murray FE, Patchett SE. Band ligation of gastric antral vascular ectasia is a safe and effective endoscopic 54 treatment. Dig Endosc 2013; 25: 392-396 [RCA] [PMID: 23808945 DOI: 10.1111/j.1443-1661.2012.01410.x] [FullText]
- Elhendawy M, Mosaad S, Alkhalawany W, Abo-Ali L, Enaba M, Elsaka A, Elfert AA. Randomized controlled study of endoscopic band 55 ligation and argon plasma coagulation in the treatment of gastric antral and fundal vascular ectasia. United European Gastroenterol J 2016; 4: 423-428 [RCA] [PMID: 27403309 DOI: 10.1177/2050640615619837] [FullText]
- 56 Garg S, Aslam B, Nickl N. Endoscopic resolution and recurrence of gastric antral vascular ectasia after serial treatment with argon plasma coagulation. World J Gastrointest Endosc 2017; 9: 263-266 [RCA] [PMID: 28690769 DOI: 10.4253/wjge.v9.i6.263] [FullText] [Full Text
- Fábián A, Bor R, Szabó E, Kardos V, Bálint A, Farkas K, Milassin Á, Rutka M, Szántó K, Molnár T, Szűcs M, Lőrinczy K, Orbán-Szilágyi Á, Gyökeres T, Gyimesi G, Szepes A, Kovács V, Rácz I, Szepes Z. Endoscopic treatment of gastric antral vascular ectasia in real-life settings: Argon plasma coagulation or endoscopic band ligation? J Dig Dis 2021; 22: 23-30 [RCA] [PMID: 33128340 DOI: 10.1111/1751-2980.12958]
- Tamari H, Oka S, Tanaka S, Hiyama Y, Ninomiya Y, Kotachi T, Boda T, Yuge R, Urabe Y, Kitadai Y, Chayama K. Clinical usefulness of 58 combination therapy with polidocanol injection and argon plasma coagulation for gastric antral vascular ectasia. JGH Open 2021; 5: 465-469 [RCA] [PMID: 33860097 DOI: 10.1002/jgh3.12517] [FullText] [Full Text(PDF)]
- 59 Patel A, Patel S, Wickremesinghe PC, Vadada D. Radiofrequency ablation using Barrx(®) for the endoscopic treatment of gastric antral vascular ectasia: a series of three cases and a review of the literature on treatment options. Clin Exp Gastroenterol 2017; 10: 113-120 [RCA] [PMID: 28744150 DOI: 10.2147/CEG.S80241] [FullText] [Full Text(PDF)]
- Gross SA, Al-Haddad M, Gill KR, Schore AN, Wallace MB. Endoscopic mucosal ablation for the treatment of gastric antral vascular ectasia with the HALO90 system: a pilot study. Gastrointest Endosc 2008; 67: 324-327 [RCA] [PMID: 18226696 DOI: 10.1016/j.gie.2007.09.020]

13

McGorisk T, Krishnan K, Keefer L, Komanduri S. Radiofrequency ablation for refractory gastric antral vascular ectasia (with video). Gastrointest Endosc 2013; 78: 584-588 [RCA] [PMID: 23660565 DOI: 10.1016/j.gie.2013.04.173] [FullText]



- Dray X, Repici A, Gonzalez P, Fristrup C, Lecleire S, Kantsevoy S, Wengrower D, Elbe P, Camus M, Carlino A, Pérez-Roldán F, Adar T, Marteau P. Radiofrequency ablation for the treatment of gastric antral vascular ectasia. Endoscopy 2014; 46: 963-969 [RCA] [PMID: 25111135 DOI: 10.1055/s-0034-1377695] [FullText]
- Jana T, Thosani N, Fallon MB, Dupont AW, Ertan A. Radiofrequency ablation for treatment of refractory gastric antral vascular ectasia (with 63 video). Endosc Int Open 2015; 3: E125-E127 [RCA] [PMID: 26135652 DOI: 10.1055/s-0034-1391323] [FullText] [Full Text(PDF)]
- Raza N, Diehl DL. Radiofrequency ablation of treatment-refractory gastric antral vascular ectasia (GAVE). Surg Laparosc Endosc Percutan Tech 2015; 25: 79-82 [RCA] [PMID: 24743681 DOI: 10.1097/SLE.000000000000033] [FullText]
- Dunn J, Boger P, Cheong E, Fullard M, Leahy A, Murphy T, Smart H. PWE-010 Radiofrequency ablation (rfa) is safe and effective for the 65 treatment of gastric antral vascular ectasia refractory to apc - results of a national audit. Gut 2015; 64: A214.1-A214 [DOI: 10.1136/gutjnl-2015-309861.459] [FullText]
- Markos P, Bilic B, Ivekovic H, Rustemovic N. Radiofrequency ablation for gastric antral vascular ectasia and radiation proctitis. Indian J 66 Gastroenterol 2017; 36: 145-148 [RCA] [PMID: 28367587 DOI: 10.1007/s12664-017-0748-2] [FullText]
- 67 Senzolo M, Realdon S, Zanetto A, Simoncin B, Schepis F, Caronna S, Saracco GM, De Angelis CG, Debernardi Venon W. Endoscopic radiofrequency ablation for the treatment of severe gastric antral vascular ectasia in patients with cirrhosis. Eur J Gastroenterol Hepatol 2021; 33: 1414-1419 [RCA] [PMID: 32925507 DOI: 10.1097/MEG.000000000001889] [FullText]
- Magee C, Lipman G, Alzoubaidi D, Everson M, Sweis R, Banks M, Graham D, Gordon C, Lovat L, Murray C, Haidry R. Radiofrequency 68 ablation for patients with refractory symptomatic anaemia secondary to gastric antral vascular ectasia. United European Gastroenterol J 2019; 7: 217-224 [*RCA*] [PMID: 31080606 DOI: 10.1177/2050640618814659] [FullText]
- McCarty TR, Rustagi T. Comparative Effectiveness and Safety of Radiofrequency Ablation Versus Argon Plasma Coagulation for Treatment of Gastric Antral Vascular Ectasia: A Systematic Review and Meta-Analysis. J Clin Gastroenterol 2019; 53: 599-606 [RCA] [PMID: 29952856 DOI: 10.1097/MCG.000000000001088] [FullText]
- 70 Magee C, Graham D, Leonard C, McMaster J, Davies H, Skotchko M, Lovat L, Murray C, Mealing S, Smart H, Haidry R. The costeffectiveness of radiofrequency ablation for treating patients with gastric antral vascular ectasia refractory to first line endoscopic therapy. Curr Med Res Opin 2020; 36: 977-983 [RCA] [PMID: 32212980 DOI: 10.1080/03007995.2020.1747997] [FullText]
- Mohan BP, Toy G, Kassab LL, Ponnada S, Chandan S, Parbhu S, Chandna S, Adler DG. Endoscopic band ligation in the treatment of gastric 71 antral vascular ectasia: a systematic review and meta-analysis. Gastrointest Endosc 2021; 94: 1021-1029.e10 [RCA] [PMID: 34480922 DOI: 10.1016/j.gie.2021.08.017] [FullText]
- McCarty TR, Hathorn KE, Chan WW, Jajoo K. Endoscopic band ligation in the treatment of gastric antral vascular ectasia: a systematic review and meta-analysis. Endosc Int Open 2021; 9: E1145-E1157 [RCA] [PMID: 34222641 DOI: 10.1055/a-1401-9880] [Full Text] [Full Text
- Hirsch BS, Ribeiro IB, Funari MP, de Moura DTH, Matuguma SE, Sánchez-Luna SA, Mancini FC, de Oliveira GHP, Bernardo WM, de 73 Moura EGH. Endoscopic Band Ligation Versus Argon Plasma Coagulation in the Treatment of Gastric Antral Vascular Ectasia: A Systematic Review and Meta-Analysis of Randomized Controlled Trials. Clin Endosc 2021; 54: 669-677 [RCA] [PMID: 34053215 DOI: 10.5946/ce.2021.063] [FullText] [Full Text(PDF)]
- 74 Chalhoub JM, Umar J, Groudan K, Hamadeh N, Desilets DJ, Greeff Y. Endoscopic band ligation compared to thermal therapy for gastric antral vascular ectasia: A systematic review and meta-analysis. United European Gastroenterol J 2021; 9: 150-158 [RCA] [PMID: 33210983 DOI: 10.1177/2050640620975243] [FullText] [Full Text(PDF)]
- Che CC, Shiu SI, Ko CW, Tu YK, Chang CH. Comparisons Between Endoscopic Band Ligation, Radiofrequency Ablation and Endoscopic 75 Thermal Therapy for Gastric Antral Vascular Ectasia: A Meta-Analysis. Dig Dis Sci 2023; 68: 3534-3541 [RCA] [PMID: 37490152 DOI: 10.1007/s10620-023-08028-7] [FullText] [Full Text(PDF)]
- 76 Markowski AR, Markowska A, Guzinska-Ustymowicz K. Pathophysiological and clinical aspects of gastric hyperplastic polyps. World J Gastroenterol 2016; 22: 8883-8891 [RCA] [PMID: 27833379 DOI: 10.3748/wjg.v22.i40.8883] [FullText] [Full Text(PDF)]
- Jeong CY, Kim N, Lee HS, Yoon H, Shin CM, Park YS, Kim JW, Lee DH. Risk Factors of Multiple Gastric Polyps according to the 77 Histologic Classification: Prospective Observational Cohort Study. Korean J Gastroenterol 2019; 74: 17-29 [RCA] [PMID: 31344769 DOI: 10.4166/kjg.2019.74.1.17] [FullText]
- Nishino K, Kawanaka M, Suehiro M, Yoshioka N, Nakamura J, Urata N, Tanigawa T, Sasai T, Oka T, Monobe Y, Saji Y, Kawamoto H, Haruma K. Gastric Hyperplastic Polyps after Argon Plasma Coagulation for Gastric Antral Vascular Ectasia in Patients with Liver Cirrhosis: A Case Suggesting the "Gastrin Link Theory". Intern Med 2021; 60: 1019-1025 [RCA] [PMID: 33116013 DOI: 10.2169/internalmedicine.5837-20] [FullText] [Full Text(PDF)]
- Correia C, Almeida N, Cipriano A, Figueiredo P. Gastric antral vascular ectasia: when treatment prolongs bleeding. Rev Esp Enferm Dig 2022; **114**: 181 [*RCA*] [PMID: 34779216 DOI: 10.17235/reed.2021.8411/2021] [FullText]
- Topal F, Akbulut S, Karahanlı C, Günay S, Sarıtaş Yüksel E, Topal FE. Portal Hypertensive Polyps as Gastroscopic Finding in Liver Cirrhosis. Gastroenterol Res Pract 2020; 2020: 9058909 [RCA] [PMID: 32184817 DOI: 10.1155/2020/9058909] [FullText] [Full Text(PDF)]
- Martín Domínguez V, Díaz Méndez A, Santander C, García-Buey L. Portal hypertensive polyps, a new entity? Rev Esp Enferm Dig 2016; 81 **108**: 279-280 [*RCA*] [PMID: 27188590] [FullText]
- Héroin L, Saviano A, Fenouil T, Sosa-Valencia L, Baumert TF, Habersetzer F, Mayer P. Differences between sporadic hyperplastic gastric 82 polyps and portal hypertensive gastric polyps: a review. Eur J Gastroenterol Hepatol 2022; 34: 260-266 [RCA] [PMID: 34432677 DOI: 10.1097/MEG.0000000000002278] [FullText]
- Lemmers A, Evrard S, Demetter P, Verset G, Gossum AV, Adler M, Devière J, Moine OL. Gastrointestinal polypoid lesions: a poorly known 83 endoscopic feature of portal hypertension. *United European Gastroenterol J* 2014; 2: 189-196 [RCA] [PMID: 25360302 DOI: 10.1177/2050640614529108] [FullText]
- Polese L, La Raja C, Fasolato S, Frigo AC, Angeli P, Merigliano S. Endoscopic diode laser therapy for gastric hyperplastic polyps in cirrhotic 84 patients. Lasers Med Sci 2021; 36: 975-979 [RCA] [PMID: 32815064 DOI: 10.1007/s10103-020-03127-7] [FullText]
- Kara D, Hüsing-Kabar A, Schmidt H, Grünewald I, Chandhok G, Maschmeier M, Kabar I. Portal Hypertensive Polyposis in Advanced Liver 85 Cirrhosis: The Unknown Entity? Can J Gastroenterol Hepatol 2018; 2018: 2182784 [RCA] [PMID: 30155451 DOI: 10.1155/2018/2182784] [FullText] [Full Text(PDF)]
- Kővári B, Kim BH, Lauwers GY. The pathology of gastric and duodenal polyps: current concepts. Histopathology 2021; 78: 106-124 [RCA] 86 [PMID: 33382489 DOI: 10.1111/his.14275] [FullText]



- Boyd JT, Lee L. Portal hypertension-associated gastric polyps. BMJ Case Rep 2014; 2014: bcr2013202312 [RCA] [PMID: 24403387 DOI: 87 10.1136/bcr-2013-202312] [FullText]
- Nelson M, Ganger D, Keswani R, Grande D, Komanduri S. Endoscopic resection is effective for the treatment of bleeding gastric hyperplastic 88 polyps in patients with and without cirrhosis. Endosc Int Open 2016; 4: E874-E877 [RCA] [PMID: 27540576 DOI: 10.1055/s-0042-109773]
- Cui H, Han T, Xu BG, Wang HY, Zhao ZG, Li Y. [Risk factors of gastrointestinal polypectomy concurrent with bleeding in patients with liver cirrhosis]. Zhonghua Gan Zang Bing Za Zhi 2023; 31: 147-154 [RCA] [PMID: 37137829 DOI: 10.3760/cma.j.cn501113-20210410-00176]
- 90 Elsabaawy M, Elbahr O, Edrees A, Badr R, Kamal A, Afify S. Endoscopic band ligation versus hot snare resection for hyperplastic gastric polyps in cirrhotic patients. Clin Exp Hepatol 2024; 10: 14-19 [RCA] [PMID: 38765908 DOI: 10.5114/ceh.2024.136216] [Full Text] [Full Text
- Estevinho MM, Pinho R, Afecto E, Correia J, Freitas T. A full hemostatic repertoire in a complex cirrhotic patient. Rev Esp Enferm Dig 2023; 91 **115**: 145-146 [*RCA*] [PMID: 35791791 DOI: 10.17235/reed.2022.9039/2022] [FullText]
- 92 Feng H, Zhou X, Zhang G. Association between cirrhosis and Helicobacter pylori infection: a meta-analysis. Eur J Gastroenterol Hepatol 2014; **26**: 1309-1319 [*RCA*] [PMID: 25304251 DOI: 10.1097/MEG.0000000000000220] [FullText]
- Voulgaris T, Karagiannakis D, Siakavellas S, Kalogera D, Angelopoulos T, Chloupi E, Karamanolis G, Papatheodoridis G, Vlachogiannakos 93 J. High prevalence of asymptomatic peptic ulcers diagnosed during screening endoscopy in patients with cirrhosis. Ann Gastroenterol 2019; 32: 451-456 [RCA] [PMID: 31474790 DOI: 10.20524/aog.2019.0399] [FullText] [Full Text(PDF)]
- Gado A, Ebeid B, Axon A. Prevalence and outcome of peptic ulcer bleeding in patients with liver cirrhosis. Alex J Med 2014; 50: 143-148 94 [RCA] [DOI: 10.1016/j.ajme.2014.01.001] [FullText]
- Demetiou G, Augoustaki A, Kalaitzakis E. Endoscopic management and outcome of non-variceal bleeding in patients with liver cirrhosis: A 95 systematic review. World J Gastrointest Endosc 2022; 14: 163-175 [RCA] [PMID: 35432740 DOI: 10.4253/wjge.v14.i3.163] [FullText] [Full Text(PDF)]
- Pariente A, Nahon S; Groupe des hémorragies digestives hautes de l'ANGH. Letter: Peptic ulcer bleeding and liver cirrhosis--different 96 diseases and different prognosis, too? Aliment Pharmacol Ther 2013; 37: 368-369 [RCA] [PMID: 23281728 DOI: 10.1111/apt.12155] [Full
- 97 Ardevol A, Ibañez-Sanz G, Profitos J, Aracil C, Castellvi JM, Alvarado E, Cachero A, Horta D, Miñana J, Gomez-Pastrana B, Pavel O, Dueñas E, Casas M, Planella M, Castellote J, Villanueva C. Survival of patients with cirrhosis and acute peptic ulcer bleeding compared with variceal bleeding using current first-line therapies. Hepatology 2018; 67: 1458-1471 [RCA] [PMID: 28714072 DOI: 10.1002/hep.29370] [Full
- Lu Z, Sun X, Han J, Jin B, Zhang W, Han J, Ma X, Liu B, Yu X, Wu Q, Wang Y, Li H. Characteristics of peptic ulcer bleeding in cirrhotic patients with esophageal and gastric varices. Sci Rep 2020; 10: 20068 [RCA] [PMID: 33208832 DOI: 10.1038/s41598-020-76530-3] [FullText] [Full Text(PDF)]
- Gralnek IM, Dumonceau JM, Kuipers EJ, Lanas A, Sanders DS, Kurien M, Rotondano G, Hucl T, Dinis-Ribeiro M, Marmo R, Racz I, Arezzo A, Hoffmann RT, Lesur G, de Franchis R, Aabakken L, Veitch A, Radaelli F, Salgueiro P, Cardoso R, Maia L, Zullo A, Cipolletta L, Hassan C. Diagnosis and management of nonvariceal upper gastrointestinal hemorrhage: European Society of Gastrointestinal Endoscopy (ESGE) Guideline. Endoscopy 2015; 47: a1-46 [RCA] [PMID: 26417980 DOI: 10.1055/s-0034-1393172] [FullText]
- Morais R, Moreira J, Gaspar R, Santos-Antunes J, Marques M, Coelho R, Alves R, Ferreira-Silva J, Dias E, Pereira P, Lopes S, Cardoso H, Sousa-Pinto B, Faria-Ramos I, Gullo I, Carneiro F, Liberal R, Macedo G. Higher frequency of gastric neoplasia in advanced chronic liver disease patients: Impact of screening endoscopy in an intermediate-high risk country. Dig Liver Dis 2024; 56: 2133-2142 [RCA] [PMID: 38811247 DOI: 10.1016/j.dld.2024.04.035] [FullText]
- Jeng KS, Chang CF, Sheen IS, Jeng CJ, Wang CH. Upper Gastrointestinal Cancer and Liver Cirrhosis. Cancers (Basel) 2022; 14: 2269 [RCA] [PMID: 35565397 DOI: 10.3390/cancers14092269] [FullText] [Full Text(PDF)]
- Moss SF. The Clinical Evidence Linking Helicobacter pylori to Gastric Cancer. Cell Mol Gastroenterol Hepatol 2017; 3: 183-191 [RCA] [PMID: 28275685 DOI: 10.1016/j.jcmgh.2016.12.001] [FullText] [Full Text(PDF)]
- Xiang Z, Li Y, Zhu C, Hong T, He X, Zhu H, Jiang D. Gastrointestinal Cancers and Liver Cirrhosis: Implications on Treatments and Prognosis. Front Oncol 2021; 11: 766069 [RCA] [PMID: 34746008 DOI: 10.3389/fonc.2021.766069] [Full Text(PDF)]
- Chiarello MM, Fico V, Pepe G, Tropeano G, Adams NJ, Altieri G, Brisinda G. Early gastric cancer: A challenge in Western countries. World 104 J Gastroenterol 2022; 28: 693-703 [RCA] [PMID: 35317273 DOI: 10.3748/wjg.v28.i7.693] [FullText] [Full Text(PDF)]
- Ono H, Yao K, Fujishiro M, Oda I, Uedo N, Nimura S, Yahagi N, Iishi H, Oka M, Ajioka Y, Fujimoto K. Guidelines for endoscopic submucosal dissection and endoscopic mucosal resection for early gastric cancer (second edition). Dig Endosc 2021; 33: 4-20 [RCA] [PMID: 33107115 DOI: 10.1111/den.13883] [FullText]
- 106 Yang H, Mou Y, Hu B. Safety and efficacy of common endoscopic treatments in patients with decompensated liver cirrhosis. Ann Hepatol 2022; **27**: 100689 [*RCA*] [PMID: 35192965 DOI: 10.1016/j.aohep.2022.100689] [FullText]
- 107 Horiki N, Omata F, Uemura M, Suzuki S, Ishii N, Fukuda K, Fujita Y, Ninomiya K, Tano S, Katurahara M, Tanaka K, Gabazza EC, Takei Y. Risk for local recurrence of early gastric cancer treated with piecemeal endoscopic mucosal resection during a 10-year follow-up period. Surg Endosc 2012; **26**: 72-78 [RCA] [PMID: 21792719 DOI: 10.1007/s00464-011-1830-y] [FullText]
- Kato M, Nishida T, Hamasaki T, Kawai N, Yoshio T, Egawa S, Yamamoto K, Ogiyama H, Komori M, Nakahara M, Yabuta T, Nishihara A, Hayashi Y, Yamada T, Takehara T. Outcomes of ESD for patients with early gastric cancer and comorbid liver cirrhosis: a propensity score analysis. Surg Endosc 2015; 29: 1560-1566 [RCA] [PMID: 25294528 DOI: 10.1007/s00464-014-3841-y] [FullText]
- Choe WH, Kim JH, Park JH, Kim HU, Cho DH, Lee SP, Lee TY, Lee SY, Sung IK, Park HS, Shim CS. Endoscopic Submucosal Dissection of Early Gastric Cancer in Patients with Liver Cirrhosis. Dig Dis Sci 2018; 63: 466-473 [RCA] [PMID: 29282635 DOI: 10.1007/s10620-017-4814-5] [FullText]
- Barakat M, Singh B, Salafia C, Eskaros S. The safety and efficacy of endoscopic submucosal dissection for early gastric cancer with concomitant liver cirrhosis. Eur J Gastroenterol Hepatol 2018; 30: 118 [RCA] [PMID: 29084075 DOI: 10.1097/MEG.0000000000000996]
- Kim SH, Joo MK, Yoo AY, Kim SM, Kim WS, Lee BJ, Park JJ, Chun HJ, Lee SW. Long-term outcome of the endoscopic submucosal dissection of early gastric cancer: A comparison between patients with and without liver cirrhosis. Oncol Lett 2022; 24: 404 [RCA] [PMID: 36276485 DOI: 10.3892/ol.2022.13524] [FullText]

15



- Pecha RL, Ayoub F, Patel A, Muftah A, Wright MW, Khalaf MA, Othman MO. Outcomes of endoscopic submucosal dissection in cirrhotic patients: First American cohort. World J Hepatol 2024; 16: 784-790 [RCA] [PMID: 38818291 DOI: 10.4254/wjh.v16.i5.784] [Full Text] [Full
- Simon K, Orłowska I, Pazgan-Simon M. The risk of complications of endoscopic procedures in patients with liver cirrhosis. Clin Exp Hepatol 2017; 3: 135-140 [RCA] [PMID: 29062903 DOI: 10.5114/ceh.2017.70284] [FullText] [Full Text(PDF)]
- Dias E, Marques M, Macedo G. Endoscopic management of esophageal and gastric lesions with underlying varices. Ann Gastroenterol 2022; **35**: 452-461 [*RCA*] [PMID: 36061164 DOI: 10.20524/aog.2022.0739] [FullText] [Full Text(PDF)]
- Repici A, Pagano N, Hassan C, Cavenati S, Rando G, Spaggiari P, Sharma P, Zullo A. Endoscopic submucosal dissection of gastric neoplastic lesions in patients with liver cirrhosis: a systematic review. J Gastrointestin Liver Dis 2012; 21: 303-307 [RCA] [PMID: 23012672] [FullText]
- Drolz A, Horvatits T, Roedl K, Rutter K, Staufer K, Kneidinger N, Holzinger U, Zauner C, Schellongowski P, Heinz G, Perkmann T, Kluge S, 116 Trauner M, Fuhrmann V. Coagulation parameters and major bleeding in critically ill patients with cirrhosis. Hepatology 2016; 64: 556-568 [RCA] [PMID: 27124745 DOI: 10.1002/hep.28628] [FullText]





Published by Baishideng Publishing Group Inc

7041 Koll Center Parkway, Suite 160, Pleasanton, CA 94566, USA

Telephone: +1-925-3991568

E-mail: office@baishideng.com

Help Desk: https://www.f6publishing.com/helpdesk

https://www.wjgnet.com

