

World Journal of *Clinical Cases*

World J Clin Cases 2022 June 26; 10(18): 5934-6340



MINIREVIEWS

- 5934** Development of clustered regularly interspaced short palindromic repeats/CRISPR-associated technology for potential clinical applications
Huang YY, Zhang XY, Zhu P, Ji L
- 5946** Strategies and challenges in treatment of varicose veins and venous insufficiency
Gao RD, Qian SY, Wang HH, Liu YS, Ren SY
- 5957** Diabetes mellitus susceptibility with varied diseased phenotypes and its comparison with phenome interactome networks
Rout M, Kour B, Vuree S, Lulu SS, Medicherla KM, Suravajhala P

ORIGINAL ARTICLE**Clinical and Translational Research**

- 5965** Identification of potential key molecules and signaling pathways for psoriasis based on weighted gene co-expression network analysis
Shu X, Chen XX, Kang XD, Ran M, Wang YL, Zhao ZK, Li CX
- 5984** Construction and validation of a novel prediction system for detection of overall survival in lung cancer patients
Zhong C, Liang Y, Wang Q, Tan HW, Liang Y

Case Control Study

- 6001** Effectiveness and postoperative rehabilitation of one-stage combined anterior-posterior surgery for severe thoracolumbar fractures with spinal cord injury
Zhang B, Wang JC, Jiang YZ, Song QP, An Y

Retrospective Study

- 6009** Prostate sclerosing adenopathy: A clinicopathological and immunohistochemical study of twelve patients
Feng RL, Tao YP, Tan ZY, Fu S, Wang HF
- 6021** Value of magnetic resonance diffusion combined with perfusion imaging techniques for diagnosing potentially malignant breast lesions
Zhang H, Zhang XY, Wang Y
- 6032** Scar-centered dilation in the treatment of large keloids
Wu M, Gu JY, Duan R, Wei BX, Xie F
- 6039** Application of a novel computer-assisted surgery system in percutaneous nephrolithotomy: A controlled study
Qin F, Sun YF, Wang XN, Li B, Zhang ZL, Zhang MX, Xie F, Liu SH, Wang ZJ, Cao YC, Jiao W

- 6050** Influences of etiology and endoscopic appearance on the long-term outcomes of gastric antral vascular ectasia

Kwon HJ, Lee SH, Cho JH

Randomized Controlled Trial

- 6060** Evaluation of the clinical efficacy and safety of TST33 mega hemorrhoidectomy for severe prolapsed hemorrhoids

Tao L, Wei J, Ding XF, Ji LJ

- 6069** Sequential chemotherapy and icotinib as first-line treatment for advanced epidermal growth factor receptor-mutated non-small cell lung cancer

Sun SJ, Han JD, Liu W, Wu ZY, Zhao X, Yan X, Jiao SC, Fang J

Randomized Clinical Trial

- 6082** Impact of preoperative carbohydrate loading on gastric volume in patients with type 2 diabetes

Lin XQ, Chen YR, Chen X, Cai YP, Lin JX, Xu DM, Zheng XC

META-ANALYSIS

- 6091** Efficacy and safety of adalimumab in comparison to infliximab for Crohn's disease: A systematic review and meta-analysis

Yang HH, Huang Y, Zhou XC, Wang RN

CASE REPORT

- 6105** Successful treatment of acute relapse of chronic eosinophilic pneumonia with benralizumab and without corticosteroids: A case report

Izhakian S, Pertzov B, Rosengarten D, Kramer MR

- 6110** Pembrolizumab-induced Stevens-Johnson syndrome in advanced squamous cell carcinoma of the lung: A case report and review of literature

Wu JY, Kang K, Yi J, Yang B

- 6119** Hepatic epithelioid hemangioendothelioma after thirteen years' follow-up: A case report and review of literature

Mo WF, Tong YL

- 6128** Effectiveness and safety of ultrasound-guided intramuscular lauromacrogol injection combined with hysteroscopy in cervical pregnancy treatment: A case report

Ye JP, Gao Y, Lu LW, Ye YJ

- 6136** Carcinoma located in a right-sided sigmoid colon: A case report

Lyu LJ, Yao WW

- 6141** Subcutaneous infection caused by *Mycobacterium abscessus* following cosmetic injections of botulinum toxin: A case report

Deng L, Luo YZ, Liu F, Yu XH

- 6148** Overlapping syndrome of recurrent anti-N-methyl-D-aspartate receptor encephalitis and anti-myelin oligodendrocyte glycoprotein demyelinating diseases: A case report
Yin XJ, Zhang LF, Bao LH, Feng ZC, Chen JH, Li BX, Zhang J
- 6156** Liver transplantation for late-onset ornithine transcarbamylase deficiency: A case report
Fu XH, Hu YH, Liao JX, Chen L, Hu ZQ, Wen JL, Chen SL
- 6163** Disseminated strongyloidiasis in a patient with rheumatoid arthritis: A case report
Zheng JH, Xue LY
- 6168** CYP27A1 mutation in a case of cerebrotendinous xanthomatosis: A case report
Li ZR, Zhou YL, Jin Q, Xie YY, Meng HM
- 6175** Postoperative multiple metastasis of clear cell sarcoma-like tumor of the gastrointestinal tract in adolescent: A case report
Huang WP, Li LM, Gao JB
- 6184** Toripalimab combined with targeted therapy and chemotherapy achieves pathologic complete response in gastric carcinoma: A case report
Liu R, Wang X, Ji Z, Deng T, Li HL, Zhang YH, Yang YC, Ge SH, Zhang L, Bai M, Ning T, Ba Y
- 6192** Presentation of Boerhaave's syndrome as an upper-esophageal perforation associated with a right-sided pleural effusion: A case report
Tan N, Luo YH, Li GC, Chen YL, Tan W, Xiang YH, Ge L, Yao D, Zhang MH
- 6198** Camrelizumab-induced anaphylactic shock in an esophageal squamous cell carcinoma patient: A case report and review of literature
Liu K, Bao JF, Wang T, Yang H, Xu BP
- 6205** Nontraumatic convexal subarachnoid hemorrhage: A case report
Chen HL, Li B, Chen C, Fan XX, Ma WB
- 6211** Growth hormone ameliorates hepatopulmonary syndrome and nonalcoholic steatohepatitis secondary to hypopituitarism in a child: A case report
Zhang XY, Yuan K, Fang YL, Wang CL
- 6218** Vancomycin dosing in an obese patient with acute renal failure: A case report and review of literature
Xu KY, Li D, Hu ZJ, Zhao CC, Bai J, Du WL
- 6227** Insulinoma after sleeve gastrectomy: A case report
Lobaton-Ginsberg M, Sotelo-González P, Ramirez-Renteria C, Juárez-Aguilar FG, Ferreira-Hermosillo A
- 6234** Primary intestinal lymphangiectasia presenting as limb convulsions: A case report
Cao Y, Feng XH, Ni HX
- 6241** Esophagogastric junctional neuroendocrine tumor with adenocarcinoma: A case report
Kong ZZ, Zhang L

- 6247** Foreign body granuloma in the tongue differentiated from tongue cancer: A case report
Jiang ZH, Xu R, Xia L
- 6254** Modified endoscopic ultrasound-guided selective N-butyl-2-cyanoacrylate injections for gastric variceal hemorrhage in left-sided portal hypertension: A case report
Yang J, Zeng Y, Zhang JW
- 6261** Management of type IIb dens invaginatus using a combination of root canal treatment, intentional replantation, and surgical therapy: A case report
Zhang J, Li N, Li WL, Zheng XY, Li S
- 6269** Clivus-involved immunoglobulin G4 related hypertrophic pachymeningitis mimicking meningioma: A case report
Yu Y, Lv L, Yin SL, Chen C, Jiang S, Zhou PZ
- 6277** De novo brain arteriovenous malformation formation and development: A case report
Huang H, Wang X, Guo AN, Li W, Duan RH, Fang JH, Yin B, Li DD
- 6283** Coinfection of *Streptococcus suis* and *Nocardia asiatica* in the human central nervous system: A case report
Chen YY, Xue XH
- 6289** Dilated left ventricle with multiple outpouchings – a severe congenital ventricular diverticulum or left-dominant arrhythmogenic cardiomyopathy: A case report
Zhang X, Ye RY, Chen XP
- 6298** Spontaneous healing of complicated crown-root fractures in children: Two case reports
Zhou ZL, Gao L, Sun SK, Li HS, Zhang CD, Kou WW, Xu Z, Wu LA
- 6307** Thyroid follicular renal cell carcinoma excluding thyroid metastases: A case report
Wu SC, Li XY, Liao BJ, Xie K, Chen WM
- 6314** Appendiceal bleeding: A case report
Zhou SY, Guo MD, Ye XH
- 6319** Spontaneous healing after conservative treatment of isolated grade IV pancreatic duct disruption caused by trauma: A case report
Mei MZ, Ren YF, Mou YP, Wang YY, Jin WW, Lu C, Zhu QC
- 6325** Pneumonia and seizures due to hypereosinophilic syndrome – organ damage and eosinophilia without synchronisation: A case report
Ishida T, Murayama T, Kobayashi S
- 6333** Creutzfeldt-Jakob disease presenting with bilateral hearing loss: A case report
Na S, Lee SA, Lee JD, Lee ES, Lee TK

LETTER TO THE EDITOR

- 6338** Stem cells as an option for the treatment of COVID-19
Cuevas-González MV, Cuevas-González JC

ABOUT COVER

Editorial Board Member of *World Journal of Clinical Cases*, Cristina Tudoran, PhD, Assistant Professor, Department VII, Internal Medicine II, Discipline of Cardiology, "Victor Babes" University of Medicine and Pharmacy Timisoara, Timisoara 300041, Timis, Romania. cristina13.tudoran@gmail.com

AIMS AND SCOPE

The primary aim of *World Journal of Clinical Cases* (WJCC, *World J Clin Cases*) is to provide scholars and readers from various fields of clinical medicine with a platform to publish high-quality clinical research articles and communicate their research findings online.

WJCC mainly publishes articles reporting research results and findings obtained in the field of clinical medicine and covering a wide range of topics, including case control studies, retrospective cohort studies, retrospective studies, clinical trials studies, observational studies, prospective studies, randomized controlled trials, randomized clinical trials, systematic reviews, meta-analysis, and case reports.

INDEXING/ABSTRACTING

The WJCC is now indexed in Science Citation Index Expanded (also known as SciSearch®), Journal Citation Reports/Science Edition, Scopus, PubMed, and PubMed Central. The 2021 Edition of Journal Citation Reports® cites the 2020 impact factor (IF) for WJCC as 1.337; IF without journal self cites: 1.301; 5-year IF: 1.742; Journal Citation Indicator: 0.33; Ranking: 119 among 169 journals in medicine, general and internal; and Quartile category: Q3. The WJCC's CiteScore for 2020 is 0.8 and Scopus CiteScore rank 2020: General Medicine is 493/793.

RESPONSIBLE EDITORS FOR THIS ISSUE

Production Editor: *Ying-Yi Yuan*, Production Department Director: *Xu Guo*, Editorial Office Director: *Jin-Lei Wang*.

NAME OF JOURNAL

World Journal of Clinical Cases

ISSN

ISSN 2307-8960 (online)

LAUNCH DATE

April 16, 2013

FREQUENCY

Thrice Monthly

EDITORS-IN-CHIEF

Bao-Gan Peng, Jerzy Tadeusz Chudek, George Kontogeorgos, Maurizio Serati, Ja Hyeon Ku

EDITORIAL BOARD MEMBERS

<https://www.wjgnet.com/2307-8960/editorialboard.htm>

PUBLICATION DATE

June 26, 2022

COPYRIGHT

© 2022 Baishideng Publishing Group Inc

INSTRUCTIONS TO AUTHORS

<https://www.wjgnet.com/bpg/gerinfo/204>

GUIDELINES FOR ETHICS DOCUMENTS

<https://www.wjgnet.com/bpg/GerInfo/287>

GUIDELINES FOR NON-NATIVE SPEAKERS OF ENGLISH

<https://www.wjgnet.com/bpg/gerinfo/240>

PUBLICATION ETHICS

<https://www.wjgnet.com/bpg/GerInfo/288>

PUBLICATION MISCONDUCT

<https://www.wjgnet.com/bpg/gerinfo/208>

ARTICLE PROCESSING CHARGE

<https://www.wjgnet.com/bpg/gerinfo/242>

STEPS FOR SUBMITTING MANUSCRIPTS

<https://www.wjgnet.com/bpg/GerInfo/239>

ONLINE SUBMISSION

<https://www.f6publishing.com>



Retrospective Study

Influences of etiology and endoscopic appearance on the long-term outcomes of gastric antral vascular ectasia

Hyo Jin Kwon, Si Hyung Lee, Joon Hyun Cho

Specialty type: Gastroenterology and hepatology

Provenance and peer review:

Unsolicited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's scientific quality classification

Grade A (Excellent): 0
Grade B (Very good): B, B
Grade C (Good): C
Grade D (Fair): 0
Grade E (Poor): 0

P-Reviewer: El-Nakeep S, Egypt; Govindarajan KK, India

Received: December 18, 2021

Peer-review started: December 18, 2021

First decision: January 23, 2022

Revised: January 31, 2022

Accepted: April 24, 2022

Article in press: April 24, 2022

Published online: June 26, 2022



Hyo Jin Kwon, Si Hyung Lee, Joon Hyun Cho, Division of Gastroenterology and Hepatology, Department of Internal Medicine, Yeungnam University College of Medicine, Daegu 42415, South Korea

Corresponding author: Joon Hyun Cho, MD, PhD, Associate Professor, Division of Gastroenterology and Hepatology, Department of Internal Medicine, Yeungnam University College of Medicine, 170 Hyeonchung-ro, Nam-gu, Daegu 42415, South Korea.
ygowgo96@hanmail.net

Abstract

BACKGROUND

Gastric antral vascular ectasia (GAVE) has diverse associations and presumed causes, which include liver cirrhosis, chronic kidney disease, and autoimmune disease. This heterogeneity of underlying disorders suggests that the pathogenesis of GAVE may be variable.

AIM

To compare the clinical features and long-term outcomes of GAVE according to endoscopic patterns and etiologies.

METHODS

The medical records and endoscopic images of 23 consecutive patients diagnosed with GAVE by endoscopy at Yeungnam University Hospital from January 2006 to December 2020 were retrospectively reviewed. Patients were allocated to cirrhosis (16 patients) and non-cirrhosis groups (7 patients). GAVE subtypes, as determined by endoscopy, were categorized as punctate (a diffuse, honeycomb-like appearance, 17 patients) or striped (a linear, watermelon-like appearance, 6 patients).

RESULTS

All GAVE patients with cirrhosis (16/16, 100%) had a punctate pattern by endoscopy, whereas the majority of patients (6/7, 85.7%) without cirrhosis had a striped pattern ($P < 0.001$). Overt GAVE bleeding (10/23, 43%) was significantly more common in the non-cirrhosis group than in the cirrhosis group (6/7, 85.7% vs 4/16, 25.0%; $P = 0.019$), and more common in the striped group than in the punctate group (5/6, 83.3% vs 5/17, 29.4%; $P = 0.052$). However, mean numbers of admissions due to GAVE bleeding and argon plasma coagulation (APC) sessions to address overt bleeding were similar in the cirrhosis and non-cirrhosis groups and in the punctate and striped groups. All patients with GAVE bleeding were

successfully treated by APC, and no patient died from GAVE-related blood loss during a median follow-up of 24 mo.

CONCLUSION

Punctate-type GAVE is strongly associated with liver cirrhosis, and GAVE patients without cirrhosis tend to be more prone to overt bleeding. However, the presence of cirrhosis and endoscopic patterns did not influence long-term clinical courses or outcomes in cases of overt bleeding.

Key Words: Gastric antral vascular ectasia; Cirrhosis; Endoscopy; Gastrointestinal bleeding; Argon plasma coagulation

©The Author(s) 2022. Published by Baishideng Publishing Group Inc. All rights reserved.

Core Tip: The study shows that punctate (diffuse, honeycomb)-type gastric antral vascular ectasia (GAVE) is strongly associated with liver cirrhosis, whereas striped (linear, watermelon)-type GAVE is strongly associated with non-cirrhotic underlying disease. Additionally, GAVE patients without cirrhosis tended to be more prone to overt bleeding. However, the presence of cirrhosis and endoscopic GAVE patterns did not influence clinical courses or the outcomes of overt bleeding after endoscopic APC treatment. It appears that clinical manifestations are dependent on etiologies, but that etiologies do not influence clinical courses in cases of GAVE bleeding.

Citation: Kwon HJ, Lee SH, Cho JH. Influences of etiology and endoscopic appearance on the long-term outcomes of gastric antral vascular ectasia. *World J Clin Cases* 2022; 10(18): 6050-6059

URL: <https://www.wjgnet.com/2307-8960/full/v10/i18/6050.htm>

DOI: <https://dx.doi.org/10.12998/wjcc.v10.i18.6050>

INTRODUCTION

Gastric antral vascular ectasia (GAVE), also known as “watermelon stomach”, is a rare clinical condition related to upper gastrointestinal (GI) bleeding, which varies from chronic occult blood loss requiring serial transfusions to acute hemorrhage[1]. GAVE was first described by Rider *et al*[2] in 1953 and fully defined by Jabbari *et al*[3] in 1984, who described patients with hyperemic striae in gastric antrum. GAVE is characterized by a unique endoscopic appearance, which takes two forms, punctate (a diffuse, honeycomb-like appearance) and striped (a linear, watermelon-like appearance). Striped GAVE has the endoscopic appearance of red stripes radiating from antrum and converging at pylorus, whereas the punctate form is characterized by diffuse red spots indicating dilated blood vessels in antrum.

Liver cirrhosis is the most frequent underlying condition associated with GAVE and portal hypertension is seen in approximately 40% of reported cases[4,5], though GAVE may not respond to therapies directed at reducing portal pressure. In addition, GAVE has been associated with other diverse medical conditions including chronic renal failure, diabetes, and autoimmune and connective tissue disorders such as systemic lupus erythematosus, rheumatoid arthritis, and systemic sclerosis[6-10]. Thus, the heterogeneity of underlying diseases observed in GAVE patients suggests its pathogenesis may be variable[11].

Epidemiologic evidence indicating that the clinical outcomes of GAVE patients with or without cirrhosis differ is lacking. Although numerous studies have investigated GAVE and many case reports and series have been reported, little information is available on the effects of its etiologies and gross endoscopic appearances on long-term clinical courses or outcomes, especially in Asians. In this study, we aimed to determine whether the presence of cirrhosis and endoscopic patterns are related to the clinical features and course of GAVE.

MATERIALS AND METHODS

Study design and patients

This retrospective cohort study was approved by the Institutional Review Board of Yeungnam University Hospital (IRB No. 2021-10-044), which waived the requirement for written informed consent due to the retrospective nature of the study. We reviewed the medical records of 23 consecutive patients diagnosed with GAVE by endoscopy at our institution from January 2006 to December 2020. Baseline

demographic data such as gender, etiology of liver disease, and severity of liver disease (as determined using Child-Pugh scores) were collected, as were details of medical comorbidities (liver cirrhosis, chronic renal failure, autoimmune or connective tissue disease, diabetes, and others), medications [proton pump inhibitors (PPIs), non-steroidal anti-inflammatory drugs (NSAIDs), and anticoagulant and antiplatelet use], and follow-up durations. Laboratory findings such as hemoglobin levels, platelet counts, serum urea nitrogen and creatinine, serum albumin, and prothrombin activity were also reviewed. Patients were categorized into two groups based on the presence (16 patients) or absence (7 patients) of cirrhosis. Cirrhosis was diagnosed based on a combination of clinical findings, liver function tests, and computed tomographic or ultrasonographic appearances of liver and spleen. Only patients aged 18 or older were included in the analysis. Median follow-up duration was 24 mo (range 6-84 mo) in the cirrhosis group and 26 mo (range 4-96 mo) in the non-cirrhosis group.

Diagnosis and endoscopic classification

GAVE was diagnosed during upper GI endoscopy by skilled gastroenterologists. Histologic specimens were not required to differentiate GAVE from portal hypertensive gastropathy. GAVE was diagnosed primarily based on endoscopic patterns and in uncertain cases by histology[12]. Endoscopy reports and photographs were reviewed independently by two gastroenterologists (Cho JH and Kwon HJ) unaware of clinical statuses. When these two gastroenterologists disagreed regarding diagnoses, a third independent gastroenterologist (Lee SH) reviewed the information available and made final decisions. Patients were classified into one of two groups according to the two typical endoscopic appearances of GAVE, that is, to a “punctate” (diffuse, honeycomb) group (17 patients) with sharply demarcated red spots diffusely spread within antrum (Figure 1A) or a “striped” (linear, watermelon) type group (6 patients) with prominent flat or raised red stripes radiating longitudinally from pylorus to antrum (Figure 1B)[6]. During upper GI endoscopy, bleeding from GAVE lesions and the presence of other nonbleeding lesions were noted (these included lesions due to portal hypertension in patients with cirrhosis). Overt GI bleeding was defined based on the presence of symptoms, such as hematemesis, melena, or hematochezia, a change in hemodynamics, or gross bleeding by upper GI endoscopy. Median follow-up duration was 23 mo (range 6-84 mo) in the punctate group and 27 mo (range 4-96 mo) in the striped group.

Endoscopic procedure

Argon plasma coagulation (APC) treatment for GAVE consisted of electrocoagulation of all angiectatic lesions during an endoscopic session with an APC probe introduced through the working channel of an endoscope. Argon gas flow ranged from 0.8 L/min to 1 L/min, and electric power output from 50 W to 70 W. Angiectasia was managed using a combination of focal pulse and “spray-painting” techniques. During endoscopic sessions, argon and coagulation smoke were regularly aspirated, and coagulum on probes was removed when required. Treatment time per session varied from 20 min to 40 min. All patients received PPI therapy for 3 wk to 4 wk to promote mucosal healing after the procedure. The endpoint of treatment was defined as complete or near complete disappearance of vascular ectasia. In cases of anemia recurrence requiring transfusion of packed red blood cells or cases of overt GI bleeding recurrence, another APC session was performed. The criteria for successful APC treatment were cessation of GI bleeding and the need for transfusion. Numbers of APC sessions per patient were documented and APC treatment success rates were evaluated.

Statistical analysis

Data were analyzed using SPSS version 20.0 for Windows (SPSS Inc, Chicago, IL). GAVE patients with or without cirrhosis were compared with respect to multiple factors, which included demographics, associated underlying diseases, laboratory values, and clinical outcomes of endoscopic APC treatment. Patients with punctate or striped GAVE were also compared. Continuous variables are expressed as mean \pm SD and were compared using the Student's *t* test or the Mann-Whitney test. Categorical variables are expressed as absolute numbers and proportions and were compared using Fisher's exact test. All statistical tests were two-sided, and statistical significance was accepted for $P < 0.05$.

RESULTS

Patient characteristics

Over the fifteen-year study period (January 2006 to December 2020), 23 patients were diagnosed with GAVE at our institution. Mean patient age was 59.8 ± 12.6 years (range 41-79) and 14 (60.9%) were male. Indications for initial endoscopy were screening for varices (8 patients), iron deficiency anemia (5), melena (8), or hematemesis (2). The etiology of cirrhosis was viral in 6 patients, alcohol in 6, autoimmune hepatitis in 1, nonalcoholic steatohepatitis in 1, and others in 2 (Table 1). The Child-Pugh classification was used to grade liver disease severity in cirrhotic patients. Eight patients with cirrhosis were classified as Child-Pugh B (50%), five as A (31.3%), and three as C (18.7%). Ten patients (62.5%) with

Table 1 Comparison of endoscopic patterns and baseline characteristics in patients with or without cirrhosis

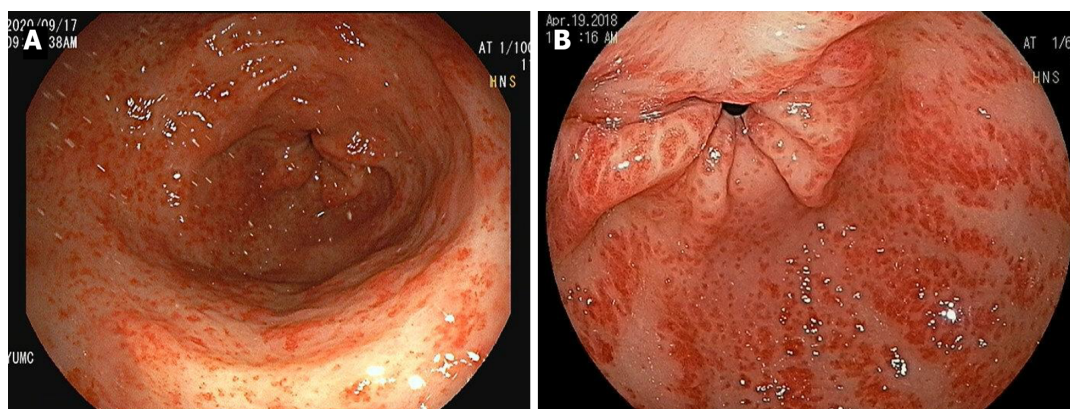
	Cirrhosis (n = 16)	No cirrhosis (n = 7)	P value
Endoscopic pattern of GAVE, n (%)			< 0.001
Punctate (Diffuse)	16 (100)	1 (14.3)	
Stripe (Linear)	0	6 (85.7)	
Age, yr, mean \pm SD	55.4 \pm 12.2	69.8 \pm 6.1	0.015
Gender (males/females)	12/4	2/5	0.066
BMI, kg/m ² , mean \pm SD	23.4 \pm 4.0	25.0 \pm 4.6	0.397
Etiology of cirrhosis, n (%)			
Viral	6 (37.5)		
Alcohol	6 (37.5)		
Autoimmune hepatitis	1 (6.3)	NA	NA
NASH	1 (6.3)		
Cryptogenic	2 (12.5)		
Child Pugh Score, n (%)			
Class A	5 (31.3)		
Class B	8 (50.0)	NA	NA
Class C	3 (18.7)		
Other associated diseases, n (%)			
Chronic kidney disease	2 (12.5)	5 (71.4)	0.011
SLE	0	1 (14.3)	0.304
Hypertension	3 (18.8)	2 (28.6)	0.621
Diabetes mellitus	5 (31.3)	3 (42.9)	0.657
Medication, n (%)			
PPI use	6 (37.5)	3 (42.9)	1.000
Antiplatelet use	0	1 (14.3)	0.304
Anticoagulant use	0	1 (14.3)	0.304
Initial laboratory findings, mean \pm SD			
Hemoglobin level, g/dL	7.8 \pm 3.4	6.2 \pm 1.6	0.494
Platelet count, k/ μ L	103.9 \pm 73.0	176.8 \pm 62.2	0.041
Total bilirubin, mg/dL	1.74 \pm 1.35	1.15 \pm 1.30	0.188
Albumin, g/dL	2.52 \pm 0.36	3.03 \pm 0.60	0.048
INR	1.23 \pm 0.27	1.09 \pm 0.25	0.179
BUN, mg/dL	23.3 \pm 14.0	44.9 \pm 39.1	0.416
Creatinine, mg/dL	1.12 \pm 0.44	3.65 \pm 2.93	0.244

BMI: Body mass index; NA: Not applicable; NASH: Nonalcoholic steatohepatitis; NSAID: Nonsteroidal anti-inflammatory inhibitor drug; PPI: Proton pump inhibitor; SLE: Systemic lupus erythematosus; INR: International normalized ratio; BUN: Blood urea nitrogen.

cirrhosis had esophageal varices.

Comparison between patients with or without cirrhosis

The characteristics of patients in the cirrhosis and non-cirrhosis groups at GAVE diagnosis are summarized in Table 1. Mean age was significantly greater in the cirrhosis group (69.8 \pm 6.1 *vs* 55.4 \pm 12.2, *P* = 0.015). Men predominated in the cirrhosis group (12/16, 75.0%) and women in the non-cirrhosis group (5/7, 71.4%). Mean group body mass indices (BMIs) were not significantly different. In the non-cirrhosis group, the most common associated disease was chronic kidney disease (5/7, 71.4%),



DOI: 10.12998/wjcc.v10.i18.6050 Copyright ©The Author(s) 2022.

Figure 1 Two main typical endoscopic views of gastric antral vascular ectasia. A: Punctate (diffuse, honeycomb)-type gastric antral vascular ectasia (GAVE) showing sharply demarcated, punctate red spots diffusely scattered in antrum; B: Striped (linear, watermelon)-type GAVE showing bright red bands radiating longitudinally from pylorus.

and this was followed by diabetes mellitus (3/7, 42.9%), and the prevalence of chronic kidney disease was significantly greater in the non-cirrhosis group (5/7, 71.4% *vs* 2/16, 12.5%; $P = 0.011$). The frequencies of other associated diseases, except chronic kidney disease, were similar in the two groups. Although no patient received corticosteroids or NSAIDs, 2 patients in the non-cirrhosis group were on antiplatelet or anticoagulant medication, respectively, which did not amount to a significant difference versus the cirrhotic group. As regards initial laboratory findings, platelet count and serum albumin level were significantly lower in the cirrhosis group (103.9 ± 73.0 k/ μ L *vs* 176.8 ± 62.2 k/ μ L, $P = 0.041$; and 2.52 ± 0.36 g/dL *vs* 3.03 ± 0.60 g/dL, $P = 0.048$, respectively). Mean hemoglobin levels were not significantly different in the cirrhosis and non-cirrhosis groups (7.8 ± 3.4 *vs* 6.2 ± 1.6 , respectively, $P = 0.494$). Total bilirubin and international normalized ratio (INR) were non-significantly greater in the cirrhosis group (1.74 ± 1.35 *vs* 1.15 ± 1.30 , and 1.23 ± 0.27 *vs* 1.09 ± 0.25 ; $P = 0.188$ and 0.179 , respectively). Blood urea nitrogen (BUN) and serum creatinine were higher in the non-cirrhosis group (44.9 ± 39.1 *vs* 23.3 ± 14.0 , and 3.65 ± 2.93 *vs* 1.12 ± 0.44 , respectively), but again differences were not significant (NS, $P = 0.416$ and 0.244 , respectively). However, group endoscopic patterns were significantly different; all patients in the cirrhosis group had punctate GAVE, whereas 6/7 patients (85.7%) in the non-cirrhosis group had striped GAVE ($P < 0.001$).

Comparison between endoscopic types

The characteristics of patients in the punctate and striped groups at diagnosis are summarized in Table 2. Mean age was significantly greater in the striped group (70.8 ± 6.3 *vs* 56.1 ± 12.0 , $P = 0.011$). Men predominated in the punctate group (13/17, 76.5%), and women predominated in the striped group (5/6, 83.3%). Group BMIs were not significantly different. Sixteen of the 17 patients in the punctate group (16/17, 94.1%) had concurrent cirrhosis, whereas no patient in the striped group (0/6, 0%) had cirrhosis ($P < 0.001$). In the striped group, the most common associated disease was chronic kidney disease (5/6, 83.3%), which was followed by diabetes mellitus (3/6, 50%), and patients in this group had an increased prevalence of chronic kidney disease (5/6, 83.3% *vs* 2/17, 11.8%; $P = 0.014$). The frequencies of other associated diseases were not significantly different in the two groups. As regards initial laboratory findings, serum bilirubin was significantly higher in the punctate group (1.88 ± 1.39 *vs* 0.64 ± 0.41 , $P = 0.044$), but creatinine was significantly higher in the striped group (4.27 ± 2.79 *vs* 1.07 ± 0.45 , $P = 0.034$), and BUN was non-significantly higher in the striped group (51.9 ± 39.9 *vs* 22.5 ± 13.8 , $P = 0.130$). Mean hemoglobin levels were similar in the punctate and striped groups (7.5 ± 3.4 *vs* 6.6 ± 1.4 , $P = 0.528$), but platelet counts and serum albumin levels were non-significantly lower in the punctate group (114.5 ± 81.5 *vs* 159.6 ± 51.1 , and 2.51 ± 0.39 *vs* 3.00 ± 0.59 ; $P = 0.168$ and 0.070 , respectively). INR was non-significantly higher in the punctate group (1.23 ± 0.26 *vs* 1.08 ± 0.28 , respectively, $P = 0.130$).

Clinical outcomes

Ten of the 23 patients (43.5%) were diagnosed with overt GAVE bleeding, and this was significantly more common in the non-cirrhosis group than in the cirrhosis group (6/7, 85.7% *vs* 4/16, 25.0%; $P = 0.019$, Table 3), and more common in the striped group than in the punctate group (5/6, 83.3% *vs* 5/17, 29.4%; $P = 0.052$, Table 4). Endoscopic APC treatment was required significantly more frequently in the non-cirrhosis group than in the cirrhosis group (6/7, 85.7% *vs* 4/16, 25.0%; $P = 0.019$), and more frequently required in the striped group than in the punctate group (5/6, 83.3% *vs* 5/17, 29.4%; $P = 0.052$). However, numbers of admissions due to GAVE bleeding were no different in the cirrhosis and non-cirrhosis groups (3.00 ± 2.45 *vs* 3.00 ± 2.83 , NS) and in the punctate and striped groups (3.00 ± 2.83

Table 2 Comparison of the baseline characteristics of patients with punctate or striped patterns

	Punctate (n = 17)	Stripe (n = 6)	P value
Age, yr, mean \pm SD	56.1 \pm 12.0	70.8 \pm 6.3	0.011
Gender (males/females)	13/4	1/5	0.018
BMI, kg/m ² , mean \pm SD	23.5 \pm 3.9	25.2 \pm 2.6	0.612
Liver cirrhosis, n (%)	16 (94.1)	0	< 0.001
Other associated diseases, n (%)			
Chronic kidney disease	2 (11.8)	5 (83.3)	0.003
SLE	0	1 (16.7)	0.261
Hypertension	3 (17.6)	2 (33.3)	0.576
Diabetes mellitus	5 (29.4)	3 (50.0)	0.621
Initial laboratory findings, mean \pm SD			
Hemoglobin level, g/dL	7.5 \pm 3.4	6.6 \pm 1.4	0.528
Platelet count, k/ μ L	114.5 \pm 81.5	159.6 \pm 51.1	0.168
Total bilirubin, mg/dL	1.88 \pm 1.39	0.64 \pm 0.41	0.044
Albumin, g/dL	2.51 \pm 0.39	3.00 \pm 0.59	0.070
INR	1.23 \pm 0.26	1.08 \pm 0.28	0.130
BUN, mg/dL	22.5 \pm 13.8	51.9 \pm 39.9	0.130
Creatinine, mg/dL	1.07 \pm 0.45	4.27 \pm 2.79	0.034

BMI: Body mass index; SLE: Systemic lupus erythematosus; INR: International normalized ratio; BUN: Blood urea nitrogen.

Table 3 Comparison of the clinical outcomes of patients with or without cirrhosis

	Cirrhosis (n = 16)	No cirrhosis (n = 7)	P value
Overt bleeding, n (%)	4 (25.0)	6 (85.7)	0.019
APC treatment, n (%)	4 (25.0)	6 (85.7)	0.019
No. of admissions d/t GAVE bleeding, mean \pm SD	3.00 \pm 2.45	3.00 \pm 2.83	NS
No. of APC sessions, mean \pm SD	4.50 \pm 4.95	4.80 \pm 5.08	NS
Success of APC	4/4	6/6	NS
Surgery	0	0	NS
Death due to GAVE bleeding	0	0	NS
Follow-up, month, median (range)	24 (6-84)	26 (4-96)	NS

APC: Argon plasma cauterization; GAVE: Gastric antral vascular ectasia; NS: Not significant.

vs 3.00 \pm 2.45, NS). In addition, no patient in either group needed therapy escalation to surgery. All 10 patients with GAVE bleeding underwent endoscopic APC treatment on a weekly or fortnightly basis, and all responded well to treatment. The mean numbers of APC sessions in patients with GAVE bleeding were similar in the cirrhosis and non-cirrhosis groups (4.50 \pm 4.95 *vs* 4.80 \pm 5.08, NS) and in the punctate and striped groups (4.00 \pm 4.34 *vs* 5.25 \pm 5.44, NS). Median follow-up durations were 24 mo and 26 mo in cirrhosis and non-cirrhosis groups, respectively, and 23 mo and 27 mo in punctate group and striped groups, respectively (neither intergroup difference was significant). During follow-up, 3 patients in the cirrhosis group and 1 patient in the non-cirrhosis group died of a cause other than GAVE bleeding (hepatocellular carcinoma in 1, hepatic failure in 1, and multiple organic failure in 2). No patient succumbed to GI hemorrhage.

Table 4 Comparison of the clinical outcomes of patients with punctate or striped patterns

	Punctate (n = 17)	Stripe (n = 6)	P value
Overt bleeding, n (%)	5 (29.4)	5 (83.3)	0.052
APC treatment, n (%)	5 (29.4)	5 (83.3)	0.052
No. of admissions d/t GAVE bleeding, mean \pm SD	3.00 \pm 2.83	3.00 \pm 2.45	NS
No. of APC sessions, mean \pm SD	4.00 \pm 4.34	5.25 \pm 5.44	NS
Success of APC	5/5	5/5	NS
Surgery	0	0	NS
Death due to GAVE bleeding	0	0	NS
Follow-up, month, median (range)	23 (6-84)	27 (4-96)	NS

APC: Argon plasma cauterization; GAVE: Gastric antral vascular ectasia; NS: Not significant.

DISCUSSION

The pathogenesis of GAVE is unknown but seems to be related to liver cirrhosis in most cases. Several authors have suggested that portal hypertension is the most influential contributor to the development of GAVE. Tobin *et al*[7] reported that patients with GAVE-induced gastric bleeding after bone marrow transplantation had hepatic veno-occlusive disease, which supports suggestions that portal hypertension might be a predisposing factor of GAVE. On the other hand, it has been demonstrated that GAVE lesions may develop in patients with cirrhosis independently of portal hypertension[8,11]. Indeed, Spahr *et al*[8] reported that portal decompression using a transjugular intrahepatic portosystemic shunt or a surgical end-to-side portacaval shunt was ineffective at treating GAVE bleeding. In addition to cirrhosis, several other disorders such as chronic kidney disease[9], chronic valvular, ischemic, or hypertensive heart disease, and a variety of autoimmune diseases[13], including Raynaud's phenomena, rheumatoid arthritis, primary biliary cirrhosis, and systemic sclerosis (diffuse and limited), have been also associated with the development of GAVE.

The remarkable heterogeneity of diseases associated with GAVE generates a wide array of factors that might cause angioectasia. Several factors have been suggested to play roles in the pathogenesis of GAVE. Charneau *et al*[14] reported that gastric antral motility was remarkably different in cirrhotic patients with or without GAVE. Others have suggested prostaglandin E2[15], gastrin[16], vasoactive intestinal polypeptide (VIP), and the proliferation of local neuroendocrine cells containing 5-hydroxy-tryptamine (5-HT)[17] are causative factors. Thus, it appears GAVE is probably caused by vasodilation and impaired motility[17] driven by these neurohumoral factors[15-18]. However, the pathophysiological changes leading to GAVE have not been fully explained[19,20]. It seems complex interplay between various factors underlies the development of vascular ectasias, and that differences in gross endoscopic appearances, *i.e.*, punctate-type or striped-type, depend on the relative influences of these factors. As described above, the assumed pathophysiology of GAVE is entirely different from that of portal hypertensive gastropathy, although at diagnosis, GAVE can be confused with this condition.

In our study, all 16 patients with cirrhosis had punctate-type GAVE and almost all patients (16/17) with punctate-type GAVE had concurrent cirrhosis. In contrast, 6 of 7 patients without cirrhosis had striped-type GAVE; the other patient had punctate-type GAVE. Thus, in this study punctate-type GAVE appeared to be associated with cirrhosis, and striped-type GAVE was not. Several reports have described linear lesions within antrum in non-cirrhotic patients and diffuse lesions in cirrhotic patients [1,6,21,22], and our results concur with these observations.

We also found that GAVE patients without cirrhosis more frequently exhibited overt bleeding and required APC treatment than those with cirrhosis. Unfortunately, we could not adjust our multivariate analysis for GI bleeding confounders due to the small number of overt GAVE bleeding cases included. Nonetheless, these observations do indicate GAVE with cirrhosis-associated nonvariceal GI bleeding is rare. However, in contrast to our results, in a retrospective study of 30 patients that underwent APC for GAVE bleeding (17 patients had cirrhosis), Lecleire *et al*[23] reported that patients with cirrhosis more frequently exhibited overt bleeding (65% *vs* 15%, $P = 0.01$), whereas patients without cirrhosis more frequently had occult bleeding as revealed by isolated iron deficiency anemia (35% *vs* 85%, $P = 0.01$). However, Wang *et al*[24] in a retrospective case-control study, reported that multivariate regression analysis adjusted for confounders showed the absence of cirrhosis best predicted active bleeding from GAVE with an odds ratio of 5.151 (95% confidence interval: 1.08-24.48, $P = 0.039$). In this previous study, of 84 GAVE patients with cirrhosis, 27 (32.1%) had overt bleeding, and of 26 patients without cirrhosis, 17 (63.4%) had overt bleeding. The reason for a significant difference between the incidences of overt bleeding according to the presence or absence of cirrhosis in GAVE remains unclear, although it has

been shown circulating neurohumoral factor levels, such as those of prostaglandin E, VIP, and 5-HT, differ in GAVE patients with or without cirrhosis[15-18]. Future studies are needed to clarify mechanistic differences between patients with or without cirrhosis in order to determine whether neurohumoral factors are responsible for clinically observed differences.

APC has been the endoscopic treatment of choice for GAVE since its introduction. In general, patients exhibit good initial response and low complication rates, but recurrent bleeding rates are usually high, and rates of 35.0% to 78.9% have been reported[25-27]. In the present study, endoscopic APC was performed in 10 patients with GAVE bleeding (4 patients had cirrhosis) and no complication was recorded after APC treatment. However, as has also been reported, 8 of the 10 patients required additional hospitalization due to GAVE bleeding after APC treatment and only two patients did not (one in the non-cirrhosis group and one in the cirrhosis group). In order to compare the clinical courses of GAVE bleeding cases with or without cirrhosis or with punctate or striped GAVE, we focused on the number of admissions due to GAVE bleeding. Although the proportion of patients with overt GAVE bleeding that received APC treatment was significantly greater among those without cirrhosis than those with cirrhosis, mean numbers of admissions due to GAVE bleeding and of APC sessions in overt bleeding patients were similar in the cirrhosis and non-cirrhosis groups and in the striped and punctate groups. These results suggest vulnerability to bleeding depends on GAVE etiology, but that clinical course after overt bleeding does not.

All patients with GAVE bleeding were successfully treated by APC. No patient needed surgery or died of GAVE-related blood loss, and mortalities during follow-up were similar in the cirrhosis and non-cirrhosis groups and in the punctate and striped groups. These findings suggest that APC is effective and safe for treating GAVE bleeding. Furthermore, the outcomes of endoscopic APC were similar regardless of endoscopic appearance or the presence of cirrhosis, which concurs with the results of a previous study[1]. Based on the above findings, we conclude that the presence of cirrhosis and endoscopic type have no influence on response to endoscopic APC treatment. We cannot explain why regardless of etiology, APC is an effective hemostatic treatment for GAVE. Indeed, the pathophysiologic features of GAVE are undetermined, and thus, open to debate.

Some limitations of the present study warrant consideration. First, some clinical data such as radiologically determined portal vein diameters and liver fibroscan data were missing due to the retrospective design of the study and lack of availability. Second, the sample size was small, especially numbers of bleedings and APC cases, and thus, it was not possible to compare study groups with respect to some variables or perform further multivariate analysis to identify risk factors of GAVE bleeding. Nevertheless, this study is one of the few to compare long-term clinical courses according to the presence or absence of cirrhosis and endoscopic type in a non-Western region.

CONCLUSION

GAVE patients without cirrhosis tended to be more prone to overt bleeding; however, the long-term clinical courses of GAVE bleeding after endoscopic APC treatment were similar irrespective of the presence of cirrhosis or endoscopic appearance. These findings suggest that the etiologies of GAVE may result in different clinical manifestations, especially bleeding, but do not influence the clinical course of GAVE bleeding. Additional studies are needed to identify the factors that play key roles in the development and clinical course of GAVE and to clarify its pathophysiologic mechanism. Furthermore, although these issues remain to be clarified, we recommend that when GAVE is diagnosed endoscopically, a careful investigation should be undertaken to determine whether liver cirrhosis and other non-cirrhotic disease processes associated with GAVE are present.

ARTICLE HIGHLIGHTS

Research background

Gastric antral vascular ectasia (GAVE) is associated with diverse medical conditions such as liver cirrhosis, chronic kidney disease, and autoimmune disease. This heterogeneity of underlying disorders suggests that the pathogenesis of GAVE may not be uniform.

Research motivation

Many studies have sought to determine whether clinical features differ in GAVE with or without cirrhosis. However, few have examined the effects of its etiologies and endoscopic patterns on long-term clinical courses or outcomes, especially in Asians.

Research objectives

To determine whether etiologies and endoscopic patterns are related to the clinical features and course of GAVE.

Research methods

A retrospective analysis of 23 consecutive patients diagnosed with GAVE from January 2006 to December 2020 was conducted. Patients were allocated to cirrhosis (16 patients) and non-cirrhosis groups (7 patients), and GAVE subtypes, as determined by endoscopy, were categorized as punctate (a diffuse, honeycomb-like appearance, 17 patients) or striped (a linear, watermelon-like appearance, 6 patients).

Research results

Punctate-type GAVE was strongly associated with liver cirrhosis, whereas striped-type GAVE was strongly associated with non-cirrhotic underlying disease. Additionally, GAVE patients without cirrhosis experienced overt bleeding more often and required APC treatment more frequently than those with cirrhosis. However, mean numbers of admissions due to GAVE bleeding and of APC sessions for overt bleeding were similar in the cirrhosis and non-cirrhosis groups and in the striped and punctate groups.

Research conclusions

GAVE etiologies may result in different clinical manifestations, especially as regards bleeding. However, etiologies and endoscopic patterns were not found to influence long-term clinical courses or treatment outcomes in cases of overt bleeding.

Research perspectives

This study is one of the few to analyze the effects of GAVE etiologies and endoscopic patterns on long-term clinical courses and outcomes. Additional studies are needed to identify those factors that play key roles in the development and clinical course of GAVE and to clarify its pathophysiologic mechanism.

FOOTNOTES

Author contributions: Cho JH and Lee SH designed the study; Kwon HJ and Cho JH performed the research; Kwon HJ and Cho JH analyzed the data; Cho JH wrote the paper; Lee SH and Cho JH revised the manuscript.

Supported by the 2020 Yeungnam University Research Grant.

Institutional review board statement: The study was reviewed and approved by the Institutional Review Board of Yeungnam University Hospital (IRB No. 2021-10-044).

Conflict-of-interest statement: There are no conflicts of interest to report.

Data sharing statement: No additional data are available.

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/Territory of origin: South Korea

ORCID number: Hyo Jin Kwon 0000-0003-4214-6369; Si Hyung Lee 0000-0001-7221-7506; Joon Hyun Cho 0000-0002-3584-6300.

Corresponding Author's Membership in Professional Societies: The Korean Society of Gastroenterology.

S-Editor: Chen YL

L-Editor: A

P-Editor: Chen YL

REFERENCES

- 1 **Dulai GS**, Jensen DM, Kovacs TO, Gralnek IM, Jutabha R. Endoscopic treatment outcomes in watermelon stomach patients with and without portal hypertension. *Endoscopy* 2004; **36**: 68-72 [PMID: 14722858 DOI: 10.1055/s-2004-814112]
- 2 **RIDER JA**, KLOTZ AP, KIRSNER JB. Gastritis with veno-capillary ectasia as a source of massive gastric hemorrhage. *Gastroenterology* 1953; **24**: 118-123 [PMID: 13052170]

- 3 **Jabbari M**, Cherry R, Lough JO, Daly DS, Kinnear DG, Goresky CA. Gastric antral vascular ectasia: the watermelon stomach. *Gastroenterology* 1984; **87**: 1165-1170 [PMID: [6332757](#)]
- 4 **Egger C**, Kreczy A, Kirchmair R, Waldenberger P, Jaschke W, Vogel W. Gastric antral vascular ectasia with portal hypertension: treatment with TIPSS. *Am J Gastroenterol* 1997; **92**: 2292-2294 [PMID: [9399773](#)]
- 5 **Fisher NC**. Gastric antral vascular ectasia and its relation with portal hypertension. *Gut* 2000; **46**: 441-442 [PMID: [10733318](#) DOI: [10.1136/gut.46.3.440b](#)]
- 6 **Sebastian S**, O'Morain CA, Buckley MJ. Review article: current therapeutic options for gastric antral vascular ectasia. *Aliment Pharmacol Ther* 2003; **18**: 157-165 [PMID: [12869075](#) DOI: [10.1046/j.1365-2036.2003.01617.x](#)]
- 7 **Tobin RW**, Hackman RC, Kimmey MB, Durtschi MB, Hayashi A, Malik R, McDonald MF, McDonald GB. Bleeding from gastric antral vascular ectasia in marrow transplant patients. *Gastrointest Endosc* 1996; **44**: 223-229 [PMID: [8885337](#) DOI: [10.1016/s0016-5107\(96\)70155-4](#)]
- 8 **Spahr L**, Villeneuve JP, Dufresne MP, Tassé D, Bui B, Willems B, Fenyves D, Pomier-Layrargues G. Gastric antral vascular ectasia in cirrhotic patients: absence of relation with portal hypertension. *Gut* 1999; **44**: 739-742 [PMID: [10205216](#) DOI: [10.1136/gut.44.5.739](#)]
- 9 **Stefanidis I**, Liakopoulos V, Kapsoritakis AN, Ioannidis I, Eleftheriadis T, Mertens PR, Winograd R, Mavvaka E, Psychos AK, Potamianos SP. Gastric antral vascular ectasia (watermelon stomach) in patients with ESRD. *Am J Kidney Dis* 2006; **47**: e77-e82 [PMID: [16731286](#) DOI: [10.1053/j.ajkd.2006.02.185](#)]
- 10 **Elkayam O**, Oumanski M, Yaron M, Caspi D. Watermelon stomach following and preceding systemic sclerosis. *Semin Arthritis Rheum* 2000; **30**: 127-131 [PMID: [11071584](#) DOI: [10.1053/sarh.2000.9623](#)]
- 11 **Payen JL**, Calès P, Voigt JJ, Barbe S, Pilette C, Dubuisson L, Desmorat H, Vinel JP, Kervran A, Chayvialle JA. Severe portal hypertensive gastropathy and antral vascular ectasia are distinct entities in patients with cirrhosis. *Gastroenterology* 1995; **108**: 138-144 [PMID: [7806035](#) DOI: [10.1016/0016-5085\(95\)90018-7](#)]
- 12 **Vesoulis Z**, Naik N, Maseelall P. Histopathologic changes are not specific for diagnosis of gastric antral vascular ectasia (GAVE) syndrome: a review of the pathogenesis and a comparative image analysis morphometric study of GAVE syndrome and gastric hyperplastic polyps. *Am J Clin Pathol* 1998; **109**: 558-564 [PMID: [9576573](#) DOI: [10.1093/ajcp/109.5.558](#)]
- 13 **Sargeant IR**, Loizou LA, Rampton D, Tulloch M, Bown SG. Laser ablation of upper gastrointestinal vascular ectasias: long term results. *Gut* 1993; **34**: 470-475 [PMID: [8491392](#) DOI: [10.1136/gut.34.4.470](#)]
- 14 **Charneau J**, Petit R, Calès P, Dauver A, Boyer J. Antral motility in patients with cirrhosis with or without gastric antral vascular ectasia. *Gut* 1995; **37**: 488-492 [PMID: [7489933](#) DOI: [10.1136/gut.37.4.488](#)]
- 15 **Saperas E**, Pigué JM, Perez-Ayuso R, Bombi JA, Bordas JM, Sentis J, Rodés J. Comparison of snare and large forceps biopsies in the histologic diagnosis of gastric vascular ectasia in cirrhosis. *Endoscopy* 1989; **21**: 165-167 [PMID: [2776701](#) DOI: [10.1055/s-2007-1012933](#)]
- 16 **Quintero E**, Pique JM, Bombi JA, Bordas JM, Sentis J, Elena M, Bosch J, Rodés J. Gastric mucosal vascular ectasias causing bleeding in cirrhosis. A distinct entity associated with hypergastrinemia and low serum levels of pepsinogen I. *Gastroenterology* 1987; **93**: 1054-1061 [PMID: [3498659](#) DOI: [10.1016/0016-5085\(87\)90569-5](#)]
- 17 **Lowes JR**, Rode J. Neuroendocrine cell proliferations in gastric antral vascular ectasia. *Gastroenterology* 1989; **97**: 207-212 [PMID: [2785944](#) DOI: [10.1016/0016-5085\(89\)91437-6](#)]
- 18 **Qureshi K**, Al-Osaimi AM. Approach to the management of portal hypertensive gastropathy and gastric antral vascular ectasia. *Gastroenterol Clin North Am* 2014; **43**: 835-847 [PMID: [25440929](#) DOI: [10.1016/j.gtc.2014.08.012](#)]
- 19 **Selinger CP**, Ang YS. Gastric antral vascular ectasia (GAVE): an update on clinical presentation, pathophysiology and treatment. *Digestion* 2008; **77**: 131-137 [PMID: [18391491](#) DOI: [10.1159/000124339](#)]
- 20 **Beales IL**. Watermelon stomach in the CREST syndrome. *Postgrad Med J* 1994; **70**: 766-767 [PMID: [7831185](#) DOI: [10.1136/pgmj.70.828.766-a](#)]
- 21 **Ito M**, Uchida Y, Kamano S, Kawabata H, Nishioka M. Clinical comparisons between two subsets of gastric antral vascular ectasia. *Gastrointest Endosc* 2001; **53**: 764-770 [PMID: [11375585](#) DOI: [10.1067/mge.2001.113922](#)]
- 22 **Fuccio L**, Mussetto A, Laterza L, Eusebi LH, Bazzoli F. Diagnosis and management of gastric antral vascular ectasia. *World J Gastrointest Endosc* 2013; **5**: 6-13 [PMID: [23330048](#) DOI: [10.4253/wjge.v5.i1.6](#)]
- 23 **Lecleire S**, Ben-Soussan E, Antonietti M, Gorla 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 [PMID: [18226684](#) DOI: [10.1016/j.gie.2007.10.016](#)]
- 24 **Wang J**, Stine JG, Cornella SL, Argo CK, Cohn SM. Patients with Gastric Antral Vascular Ectasia (GAVE) Are at a Higher Risk of Gastrointestinal Bleeding in the Absence of Cirrhosis. *J Clin Transl Hepatol* 2015; **3**: 254-259 [PMID: [26807380](#) DOI: [10.14218/JCTH.2015.00031](#)]
- 25 **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 [PMID: [11474999](#) DOI: [10.1055/s-2001-15722](#)]
- 26 **Yusoff I**, Brennan F, Ormonde D, Laurence B. Argon plasma coagulation for treatment of watermelon stomach. *Endoscopy* 2002; **34**: 407-410 [PMID: [11972274](#) DOI: [10.1055/s-2002-25287](#)]
- 27 **Roman S**, Saurin JC, Dumortier J, Perreira A, Bernard G, Ponchon T. Tolerance and efficacy of argon plasma coagulation for controlling bleeding in patients with typical and atypical manifestations of watermelon stomach. *Endoscopy* 2003; **35**: 1024-1028 [PMID: [14648415](#) DOI: [10.1055/s-2003-44594](#)]



Published by **Baishideng Publishing Group Inc**
7041 Koll Center Parkway, Suite 160, Pleasanton, CA 94566, USA

Telephone: +1-925-3991568

E-mail: bpgoffice@wjgnet.com

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

<https://www.wjgnet.com>

