Chapter 18
Gastric Antral Vascular Ectasia (GAVE)

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Clinical Vignette

A 65-year-old woman with limited cutaneous systemic sclerosis (lcSSc) was admitted for the evaluation of worsening fatigue. Routine blood tests showed a hemoglobin level of 9.4 g/dL, with a ferritin level of 4 ng/mL. The fecal occult blood test was positive. Esophagogastroduodenoscopy revealed diffuse gastric vascular ectasias involving the antrum and converging towards the pylorus (Fig. 18.1). How should this case be managed?

Introduction

Gastric antral vascular ectasia (GAVE) is a rare acquired vascular disease involving the antral mucosa of the stomach [1]. The disease is also known as *watermelon stomach* because of its striking endoscopic appearance, characterized by multiple longitudinal stripes of red vessels originating

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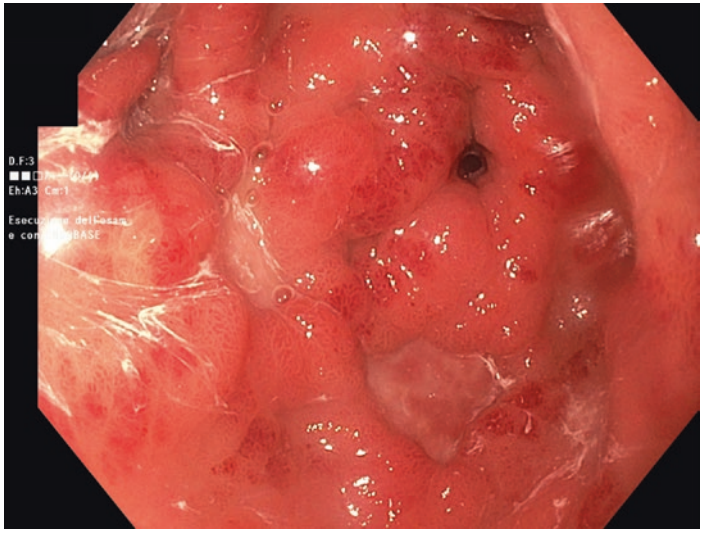

Figure 18.1 Endoscopic appearance of gastric antral vascular ectasia (GAVE): multiple longitudinal stripes of red vessels involving the gastric antrum and converging to the pylorus

from the pylorus and involving the antrum. The macroscopic appearance of GAVE is related to the underlying pathogenic mechanisms and microscopic features, characterized by capillary dilatation, small vessel fibrin deposition and microthrombosis [2].

Although GAVE is a known complication of cirrhosis, only a third of patients with GAVE actually have a chronic liver disease [3]. Among non-cirrhotic patients, GAVE can be mostly observed in patients with autoimmune connective tissue disorders, bone marrow transplantation and chronic renal failure [3]. Despite the rarity of both diseases, the association of GAVE and systemic sclerosis (SSc) has been clearly established in recent years, and several studies have shed light upon the prevalence, significance and prognosis of such an association.

Epidemiology

After *Jabbari et al* accurately described it in 1984 [1], GAVE has increasingly been associated with scleroderma and SSc-related features in the subsequent years [4–6].

To date, the prevalence of GAVE in patients with SSc is estimated between 1% and 22.3%, varying largely according to the different studies [7, 8]. The differences may be mostly accounted to the design of the study (i.e. retrospective or prospective) and the selection of the patients (i.e. whether patients were systematically screened or not regardless of the presence of symptoms).

Overall, retrospective studies report a lower prevalence (1–5.7%) [7, 8] than the prospective Scleroderma Cyclophosphamide Or Transplant (SCOT) trial that systematically assessed all patients by endoscopy regardless of clinical symptoms (22.3%) [9].

In most of the studies, GAVE appears to be an early complication of SSc, since on average it occurs within the first 18–36 months after the diagnosis of SSc [8, 10]. In 15–20% of the cases, the diagnosis of GAVE may precede or be concurrent to that of SSc [7, 11]. Finally, in up to a third of SSc patients GAVE may represent the first non-Raynaud symptom [8].

Pathogenesis

The etiology of GAVE is still largely unknown [3]. However, several mechanisms have been suspected to be involved in the pathogenesis of watermelon stomach, including altered motility of the gastric antrum and mucosal trauma secondary to mechanical stress [3]. The role of vasoactive substances is uncertain, but locally released neurotransmitters may be responsible for vasodilatation and hence the propensity to bleed [12].

The microscopic features of GAVE further suggest that vascular ectasia is acquired rather than congenital. Several histopathological aspects have been described, including dilatation of mucosal capillaries with focal fibrin thromboses and fibromuscular hyperplasia of the lamina propria [13].

In SSc, GAVE may represent a complication belonging to the spectrum of vascular alterations, along with Raynaud's phenomenon, telangiectasias, pulmonary and renal vascular involvements [8].

Clinical Aspects

The clinical presentation of GAVE is related to its pathological aspects. Submucosal ectasic vessels can erode through the gastric mucosa, leading to chronic blood loss and substantial iron deficiency anemia. GAVE is responsible for around 4% of non-variceal upper gastrointestinal bleedings [14], with a substantial proportion of patients (60–70%) requiring blood transfusions due to recurrent anemia despite iron supplementation.

In SSc-associated GAVE, clinically meaningful anemia may develop in about 80–90% of patients, with half of them requiring blood transfusions [8]. Acute hemorrhage in the form of melena or hematemesis may be the first presentation of GAVE in few cases [7].

Several studies reported significant clinical associations between GAVE and SSc features. SSc patients with GAVE appear to have a low prevalence of anti-topoisomerase I antibodies [8–10], and a lower proportion of them exhibit pulmonary fibrosis on chest CT scan [8].

Conversely, anti-RNA polymerase III (RNAP III) positivity appears to be significantly associated to GAVE, as confirmed by several studies [8, 10, 15]. Patients that are positive to anti-RNAP III antibodies carry a significant higher risk of developing GAVE (OR 4.6, 95%CI 1.2–21.1) and

other vascular events such as scleroderma renal crisis and pulmonary hypertension [8], and are more likely to have a shorter disease duration, a more rapid disease onset (defined as the interval from appearance of Raynaud's phenomenon to first symptom other than Raynaud's) and faster skin thickening in the first months after SSc onset, compared to anti-topoisomerase I positive patients [10, 15]. Furthermore, patients with early diffuse cutaneous subset (dcSSc) seems to have a higher risk of developing GAVE [9, 10], although this has not been confirmed in all studies [8].

Finally, SSc patients with GAVE have a higher prevalence of telangiectasias and systemic hypertension [7, 8, 10].

At the functional level, patients with GAVE more frequently exhibit a diminished diffusing capacity (DLCO) with a low DLCO/alveolar volume (DLCO/AV) ratio at pulmonary function tests, suggesting an underlying vascular pulmonary involvement in these patients [8, 9]. Taken together, these data indicate that GAVE may be a significant manifestation of the "vascular phenotype" of SSc, along with renal, pulmonary and cutaneous vascular complications [8, 9].

Diagnosis

The diagnosis of GAVE is usually supported by the endoscopic appearance [14]. In long-standing diseases, gastroscopy may disclose the typical watermelon stomach characterized by prominent, erythematous stripes, radiating in a spoke-like fashion from the antrum to the pylorus, but in early stages the findings may be limited to multiple red spots or linear red stripes in the antrum (Fig. 18.1) [11].

Routine histopathological examination is not required for a definite diagnosis, but it may be useful for cases in which the diagnosis is uncertain, or when other diseases (i.e. antral gastritis) need to be investigated.

Management

Although in the last two decades several therapeutic options have become available for GAVE-related gastrointestinal bleeding, the management of watermelon stomach remains a challenging issue. Therapeutic options include medical, endoscopic and surgical procedures [14].

Medical Management

Different medical treatments have been proposed for the management of GAVE-related bleeding, though evidence is weak. Corticosteroids [16], thalidomide [17], tranexamic acid [18], octreotide [19] and hormone replacement [20] have been occasionally reported to be successful, mostly in individual case reports, but their efficacy may be limited because of the development of adverse events.

Cyclophosphamide (CYC), with or without intravenous methylprednisolone, may be effective for the resolution of GAVE-related bleeding in SSc patients as reported by several case reports and case series [21–25]. These patients were mostly refractory to conventional endoscopic treatments, and CYC was able to achieve stable hemoglobin levels. Due to its unfavorable safety profile, CYC should be reserved for patients with refractory disease.

Autologous hematopoietic stem cell transplantation (HSCT), that recently became a treatment option for SSc patients [26], may also be effective for SSc-related GAVE. In a series of three patients with persistent bleeding despite multiple endoscopic treatments, HSCT was successful to achieve transfusion independence and maintain stable hemoglobin at 1 year. Interestingly, the surveillance endoscopy showed significant improvement of vascular ectasia [27].

Endoscopic Management

Endoscopy is the treatment of choice for the management of GAVE-related gastrointestinal bleeding [14]. Endoscopic techniques have also been successfully applied in patients with SSc [7, 8].

Treatments modalities that have been proven to be useful in GAVE include thermal and mechanical methods, with meaningful success rates and acceptable safety profiles (Table 18.1).

Table 18.1 Endoscopic treatment for gastric antral vascular ectasia

Method	Bleeding cessation rate[a]	Mean number of sessions	Complications[b]
Cryotherapy	50–71%	2–6	Major: ~0%; minor 0–8%
Nd: YAG laser	60–100%	1–5	Major 0–13%; minor ~0%
Argon plasma coagulation	30–100%	2–6	Major: ~0%; minor ~0%
Endoscopic band ligation	65–95%	2–3	Major: ~0%; minor 8–12%
Radiofrequency ablation	67–86%	2–3	Major ~0%; minor 1–2%

Reproduced from Hsu WH, Wang YK, Hsieh MS, Kuo FC, Wu MC, Shih HY, et al. Insights into the management of gastric antral vascular ectasia (watermelon stomach). Therap Adv Gastroenterol. 2018;11:1–9

Nd: YAG, neodymium-yttrium-aluminium garnet

[a]Definition of bleeding cessation: rising hemoglobin level and no requirement for blood transfusion

[b]Major complication: death, perforation, stenosis; minor complication: gastrointestinal upset

Endoscopic treatments are usually considered effective if they are able to improve gastrointestinal bleeding, achieving bleeding cessation, a condition in which hemoglobin levels rise and blood transfusions are no longer required [14].

Cryotherapy is an endoscopic technique using nitrous oxide to apply an extremely cold temperature on affected tissue and achieve hemostasis by thermal destruction or necrosis of the mucosa [14].

Two small studies demonstrated limited efficacy of cryotherapy on GAVE-related bleeding, with a cessation rate of 50–71% after a median of 2–6 sessions of treatment [28, 29].

Neodymium-yttrium-aluminum garnet laser coagulation (Nd: YAG) laser is a thermal device that causes tissue destruction by absorption of laser light without direct contact [14]. The efficacy of Nd: YAG laser in stopping bleeding and decreasing the requirement for blood transfusions ranges between 60% and 100% after a median of 1–4 sessions [30–32]. However, Nd: YAG lasers have been largely replaced by other treatment modalities because of higher cost and serious complications, such as perforation, antral narrowing, and mortality, compared to other treatment modalities [14].

Argon plasma coagulation (APC) is another thermal method that has gradually become the treatment of choice for GAVE. APC produces high frequency electrical current flows to achieve tissue coagulation by using ionized argon gas (plasma) as a medium. Compared with Nd: YAG lasers, APC is easier to apply and safer due to its favorable side-effect profile, and less expensive [14].

Several studies demonstrated that APC is effective in GAVE, with variable bleeding cessation rates (30–100%), but also reported that usually repeated endoscopic sessions are warranted [33–35]. APC may be unsatisfactory in long-term follow-up and yield high bleeding recurrence rates [36–38]. The recurrent bleeding following APC may be related to its limited depth of mucosal coagulation [14]. Although APC presumably has a favorable side effect profile due to its non-contact method and limited depth of mucosal injury, some major complications are still a concern. Gastric outlet obstruc-

tion and hyperplastic polyps were reported as serious adverse events after APC treatment [39, 40].

Radiofrequency ablation (RFA) is another thermal technique that has been recently proposed as treatment modality for GAVE. The principle of RFA is to apply high-energy coaptive coagulation to destroy the superficial mucosal capillary ectasia with the subsequent regeneration of epithelium composed of a normal capillary structure [14]. RFA has a success rate of 67–86% in achieving bleeding cessation after a median of 2–3 sessions, with a low rate of minor complications [41–43]. The minimal time interval of 6 weeks between each treatment session is recommended and proton pump inhibitors should be prescribed. RFA appears to be a well-tolerated and feasible method for patients with GAVE with poor response to other treatment modalities [14].

Among mechanical methods, endoscopic band ligation (EBL), usually used for the treatment of esophageal varices, may be also effective for GAVE-related bleeding [14]. In retrospective studies, EBL showed higher rates of bleeding cessation and fewer treatment sessions compared to APC, whereas in prospective studies EBL reached a 95% success rate for bleeding cessation [44–47]. EBL appears to be safe and well tolerated and thus it may be an option for the treatment of patients refractory to thermoablative methods.

Surgical Treatments

With the advances of endoscopic techniques, surgery is now reserved for patients that fail to improve despite medical and endoscopic treatments [14]. Surgical approaches include antrectomies (with Billroth I, II, and Roux-en-Y reconstructions), partial gastrectomy, total gastrectomy and esophagogastrectomy [48]. The results of surgical hemostasis are usually satisfying but several complications may develop, such as late dumping syndrome, nutritional deficiencies and death [14].

Prognosis

The natural history of SSc-associated GAVE has not been fully elucidated given the paucity of data. Bleeding recurrence and transfusion dependence are common issues in patients with long-standing disease [7, 8, 11]. The median time of GAVE recurrence is 10 months after the initial diagnosis [7].

These issues may be overcome by planning re-treatment endoscopic sessions or integrating different endoscopic techniques and medical therapy [7].

Although frequently associated with the positivity of anti-RNAP III antibody, that is burdened with substantial morbidity, characterized by the rapid evolution of skin thickening and the occurrence of scleroderma renal crisis, the presence of GAVE does not appear to impact on mortality of SSc patients, at least in the study with the longer follow-up (30 months) [8].

Conclusion

GAVE is a rare but burdensome complication that appears to belong to the broad spectrum of vascular alterations of SSc. The presence of watermelon stomach should be considered when unexplained iron-deficiency anemia occurs in SSc patients, especially in patients with early diffuse skin involvement. Given the close association of GAVE and anti-RNA polymerase III antibodies, screening these high risk patients with endoscopy should be considered even in absence of anemia or gastric-related symptoms. Endoscopic treatment is effective, safe and well tolerated in patients with SSc-related GAVE with significant bleeding.

References

1. Jabbari M, Cherry R, Lough JO, Daly DS, Kinnear DG, Goresky CA. Gastric Antral vascular Ectasia: the watermelon stomach. Gastroenterology. 1984;87(5):1165–70.
2. Ruhl GH, Schnabel R, Peiseler M, Seidel D. Gastric antral vascular ectasia: a case report of a 10 year follow-up with special consideration of histopathological aspects. Z Gastroenterol. 1994;32(3):160–4.
3. Burak KW, Lee SS, Beck PL. Portal hypertensive gastropathy and gastric antral vascular ectasia (GAVE) syndrome. Gut. 2001;49(6):866 LP-872.
4. Jouanolle H, Bretagne JF, Ramée MP, Lancien G, Le Jean-Colin I, Heresbach D, et al. Antral vascular ectasia and scleroderma. Endoscopic, radiologic and anatomopathologic aspects of an uncommon association. Gastroenterol Clin Biol. 1989;13(2):217–21.
5. Murphy FT, Enzenauer RJ, Cheney CP. Watermelon stomach. Arthritis Rheum. 1999;42(3):573.
6. Watson M, Hally RJ, McCue PA, Varga J, Jiménez SA. Gastric antral vascular ectasia (watermelon stomach) in patients with systemic sclerosis. Arthritis Rheum. 1996;39(2):341–6.
7. Marie I, Ducrotte P, Antonietti M, Herve S, Levesque H. Watermelon stomach in systemic sclerosis: its incidence and management. Aliment Pharmacol Ther. 2008;28(4):412–21.
8. Ghrénassia E, Avouac J, Khanna D, Derk CT, Distler O, Suliman YA, et al. Prevalence, correlates and outcomes of gastric antral vascular ectasia in systemic sclerosis: a eustar case-control study. J Rheumatol. 2014;41(1):99–105.
9. Hung EW, Mayes MD, Sharif R, Assassi S, Machicao VI, Hosing C, et al. Gastric Antral vascular Ectasia and its clinical correlates in patients with early diffuse systemic sclerosis in the SCOT trial. J Rheumatol. 2013;40(4):455 LP-460.
10. Ingraham KM, O'Brien MS, Shenin MAX, Derk CT, Steen VD. Gastric Antral vascular Ectasia in systemic sclerosis: demographics and disease predictors. J Rheumatol. 2010;37(3):603–7.
11. El-Gendy H, Shohdy KS, Maghraby GG, Abadeer K, Mahmoud M. Gastric antral vascular ectasia in systemic sclerosis: where do we stand? Int J Rheum Dis. 2017;20(12):2133–9.

12. Lowes JR, Rode J. Neuroendocrine cell proliferations in gastric antral vascular ectasia. Gastroenterology. 1989;97(1):207–12.
13. Suit PF, Petras RE, Bauer TW, Petrini JL. Gastric antral vascular ectasia. A histologic and morphometric study of "the watermelon stomach". Am J Surg Pathol. 1987;11(10):750–7.
14. Hsu WH, Wang YK, Hsieh MS, Kuo FC, Wu MC, Shih HY, et al. Insights into the management of gastric antral vascular ectasia (watermelon stomach). Ther Adv Gastroenterol. 2018;11:1–9.
15. Ceribelli A, Cavazzana I, Airò P, Franceschini F. Anti-RNA polymerase III antibodies as a risk marker for early gastric Antral vascular Ectasia (GAVE) in systemic sclerosis. J Rheumatol. 2010;37(7):1544.
16. Calès P, Voigt JJ, Payen JL, Bloom E, Berg P, Vinel JP, et al. Diffuse vascular ectasia of the antrum, duodenum, and jejunum in a patient with nodular regenerative hyperplasia. Lack of response to portosystemic shunt or gastrectomy. Gut. 1993;34(4):558–61.
17. Dunne KA, Hill J, Dillon JF. Treatment of chronic transfusion-dependent gastric antral vascular ectasia (watermelon stomach) with thalidomide. Eur J Gastroenterol Hepatol. 2006;18(4):455–6.
18. McCormick PA, Ooi H, Crosbie O. Tranexamic acid for severe bleeding gastric antral vascular ectasia in cirrhosis. Gut. 1998;42(5):750 LP-752.
19. Nardone R, Balzano B. The efficacy of octreotide therapy in chronic bleeding due to vascular abnormalities of the gastrointestinal tract. Aliment Pharmacol Ther. 1999;13(11):1429–36.
20. Tran A, Villeneuve J-P, Bilodeau M, Willems B, Marleau D, Fenyves D, et al. Treatment of chronic bleeding from gastric Antral vascular Ectasia (Gave) with estrogen-progesterone in cirrhotic patients: an open pilot study. Am J Gastroenterol. 1999;94(10):2909–11.
21. Lorenzi AR, Johnson AH, Davies G, Gough A. Gastric antral vascular ectasia in systemic sclerosis: complete resolution with methylprednisolone and cyclophosphamide. Ann Rheum Dis. 2001;60(8):796 LP-798.
22. Azam J, Caperon A, Lawson CA, Gough A. Medical versus laser therapy for gastric antral vascular ectasia: comment on the clinical images by Thonhofer et al. Arthritis Rheum. 2010;62(11):3517.
23. Schulz SW, O'Brien M, Maqsood M, Sandorfi N, Del Galdo F, Jimenez SA. Improvement of severe systemic sclerosis-associated gastric antral vascular ectasia following immunosuppressive treatment with intravenous cyclophosphamide. J Rheumatol. 2009;36(8):1653–6.

24. Papachristos DA, Nikpour M, Hair C, Stevens WM. Intravenous cyclophosphamide as a therapeutic option for severe refractory gastric antral vascular ectasia in systemic sclerosis. Intern Med J. 2015;45(10):1077–81.
25. Sawada T, Mizuuchi T, Hayashi H, Tsubouchi S, Yasuda T, Tahara K, et al. Intravenous cyclophosphamide for gastric Antral vascular Ectasia associated with systemic sclerosis refractory to endoscopic treatment: a case report and review of the pertinent literature. Intern Med. 2018;58(1):135–9.
26. Del Papa N, Pignataro F, Zaccara E, Maglione W, Minniti A. Autologous hematopoietic stem cell transplantation for treatment of systemic sclerosis. Front Immunol. 2018;9:2390.
27. Bhattacharyya A, Sahhar J, Milliken SAM, Ma D, Tymms K, Danta M, et al. The Journal of Rheumatology Autologous Hematopoietic Stem Cell Transplant for Systemic Sclerosis Improves Anemia from Gastric Antral Vascular Ectasia The Journal of Rheumatology is a monthly international serial edited by Earl D . in rheumatology and rela. 2015;42(3).
28. Kantsevoy SV, Cruz-Correa MR, Vaughn CA, Jagannath SB, Pasricha PJ, Kalloo AN. Endoscopic cryotherapy for the treatment of bleeding mucosal vascular lesions of the GI tract: a pilot study. Gastrointest Endosc. 2003;57(3):403–6.
29. Cho S, Zanati S, Yong E, Cirocco M, Kandel G, Kortan P, et al. Endoscopic cryotherapy for the management of gastric antral vascular ectasia. Gastrointest Endosc. 2008;68(5):895–902.
30. Potamiano S, Carter CR, Anderson JR. Endoscopic laser treatment of diffuse gastric antral vascular ectasia. Gut. 1994;35(4):461–3.
31. Calamia KT, Scolapio JS, Viggiano TR. Endoscopic YAG laser treatment of watermelon stomach (gastric antral vascular ectasia) in patients with systemic sclerosis. Clin Exp Rheumatol. 2000;18(5):605–8.
32. Mathou NG, Lovat LB, Thorpe SM, Bown SG. Nd:YAG laser induces long-term remission in transfusion-dependent patients with watermelon stomach. Lasers Med Sci. 2004;18(4):213–8.
33. Fuccio L, Zagari RM, Serrani M, Eusebi LH, Grilli D, Cennamo V, et al. Endoscopic argon plasma coagulation for the treatment of gastric Antral vascular Ectasia-related bleeding in patients with liver cirrhosis. Digestion. 2009;79(3):143–50.
34. Boltin D, Gingold-Belfer R, Lichtenstein L, Levi Z, Niv Y. Long-term treatment outcome of patients with gastric vascular ectasia

treated with argon plasma coagulation. Eur J Gastroenterol Hepatol. 2014;26(6):588–93.
35. Rosenfeld G, Enns R. Argon photocoagulation in the treatment of gastric antral vascular ectasia and radiation proctitis. Can J Gastroenterol. 2009;23(12):801–4.
36. Naga M, Esmat S, Naguib M, Sedrak H. Long-term effect of argon plasma coagulation (APC) in the treatment of gastric antral vascular ectasia (GAVE). Arab J Gastroenterol. 2011;12(1):40–3.
37. 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(3):212–7.
38. Kwan V, Bourke MJ, Williams SJ, Gillespie PE, Murray MA, Kaffes AJ, et al. Argon plasma coagulation in the Management of Symptomatic Gastrointestinal Vascular Lesions: experience in 100 consecutive patients with long-term follow-up. Am J Gastroenterol. 2006;101:58–63.
39. Shah N, Cavanagh Y, Kaswala DH, Shaikh S. Development of hyperplastic polyps following argon plasma coagulation of gastric antral vascular ectasia. J Nat Sci Biol Med. 2015;6(2):479–82.
40. Farooq FT, Wong RCK, Yang P, Post AB. Gastric outlet obstruction as a complication of argon plasma coagulation for watermelon stomach. Gastrointest Endosc. 2007;65(7):1090–2.
41. Gross SA, Al-Haddad M, Gill KRS, 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(2):324–7.
42. McGorisk T, Krishnan K, Keefer L, Komanduri S. Radiofrequency ablation for refractory gastric antral vascular ectasia (with video). Gastrointest Endosc. 2013;78(4):584–8.
43. Markos P, Bilic B, Ivekovic H, Rustemovic N. Radiofrequency ablation for gastric antral vascular ectasia and radiation proctitis. Indian J Gastroenterol. 2017;36(2):145–8.
44. Wells CD, Harrison ME, Gurudu SR, Crowell MD, Byrne TJ, DePetris G, et al. Treatment of gastric antral vascular ectasia (watermelon stomach) with endoscopic band ligation. Gastrointest Endosc. 2008;68(2):231–6.
45. Keohane J, Berro W, Harewood GC, Murray FE, Patchett SE. Band ligation of gastric antral vascular ectasia is a safe and effective endoscopic treatment. Dig Endosc. 2013;25(4):392–6.

46. 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(4):237–42.
47. Zepeda-Gómez S, Sultanian R, Teshima C, Sandha G, Van Zanten S, Montano-Loza AJ. Gastric antral vascular ectasia: a prospective study of treatment with endoscopic band ligation. Endoscopy. 2015;47(6):538–40.
48. Novitsky YW, Kercher KW, Czerniach DR, Litwin DEM. Watermelon stomach: pathophysiology, diagnosis, and management. J Gastrointest Surg. 2003;7(5):652–61.