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Chapter 4

Gastric Antral Vascular Ectasia and Portal Hypertensive Gastropathy

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Abstract

Gastric antral vascular ectasia (GAVE) and portal hypertensive gastropathy (PHG) are mucosal lesions that can cause chronic gastrointestinal bleeding in the patients with cirrhosis. While PHG occurs exclusively in patients with liver cirrhosis, GAVE can also present in patients with systemic and autoimmune conditions. The need to accurately characterize these two conditions is dependent on clinical, endoscopic, and histological parameters. The management of GAVE utilizes endoscopic ablation techniques, while medical therapy is directed toward stabilizing portal pressure in patients with PHG. Herein, we review the epidemiology, diagnosis, pathophysiology, and medical, endoscopic, and surgical management of GAVE and PHG.

Keywords: stomach diseases, gastric antral vascular ectasia (GAVE), portal hypertensive gastropathy (PHG), portal hypertension, cirrhosis, management

1. Introduction

Gastric antral vascular ectasia (GAVE) and portal hypertensive gastropathy (PHG) are common gastric mucosal lesions that occur in patients with portal hypertension. These two conditions are responsible for acute on chronic gastrointestinal bleeding. While these two clinical entities share similar clinical presentations, their underlying pathophysiology, endoscopic features, and management options are different. The pathophysiology of GAVE is related to local changes in gastric mucosa, and management is aimed at endoscopic reduction of blood loss using thermal therapies. The pathophysiology of PHG is related to portal hypertension, and management is aimed at reducing portal hypertension using pharmacologic and in some

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cases portosystemic shunts. Thus, it is important to differentiate GAVE and PHG as their management options are different.

2. Epidemiology

GAVE accounts for approximately 4% of all upper gastrointestinal bleeding [1]. Approximately 40% of GAVE patients have cirrhosis of the liver, and 1 in 40 patients require liver transplantation [2]. Cirrhotic GAVE patients are predominantly males (75%; mean age 65 years), whereas noncirrhotic GAVE patients are predominantly females (71%; mean age of 73 years). GAVE has been associated with autoimmune disorders such as autoimmune connective tissue disorders (62%), Raynaud's phenomenon (31%), and sclerodactyly (20%) [3]. GAVE have also been reported in other medical conditions including scleroderma, bone marrow transplantation, chronic renal failure, ischemic heart disease, hypertension, valvular heart disease, familial Mediterranean fever, and acute myeloid leukemia [3–6].

The prevalence of PHG varies from 20 to 75% in portal hypertensive patients, and from 35 to 80% in patients with cirrhosis [7]. According to the HALT-C trial, approximately 37% of patients (364 of 1011) with biopsy confirmed cirrhosis or bridging fibrosis from hepatitis C had PHG [8]. While PHG can present at any age, its severity can vary from mild to severe. The severity of liver disease and severity of portal hypertension greatly influences the natural progression of PHG [9].

3. Pathophysiology

The pathophysiology of GAVE remains unknown; however, several mechanisms have been proposed including gastric dysmotility or autoimmune reactivity to gastric blood vessels [10–12]. A study on antral motility revealed an increase in antral area transit time with cirrhosis and GAVE when compared to controls [10]. Chronic recurrent trauma can lead to fibromuscular hyperplasia and vascular ectasia. Reduced gastrin levels have also been identified in GAVE patients when compared to patients with severe PTH and normal controls [13]. Prostaglandins E2 (PGE2) levels were found significantly elevated when compared to controls [14]. GAVE is not associated with portal hypertension and treatments aimed to decrease portal pressure have no role in treatment of GAVE [15].

The pathogenesis of PHG is related to increased resistance to portal blood flow in patients with liver disease, and concomitant elevation in portal pressure [16]. In patients with portal hypertension, approximately 70% develop PHG [17]. Resolution of PHG and its recurrence has been observed in patients with cirrhosis posttransjugular intrahepatic portosystemic shunt (TIPS) placement, and in noncirrhotic patients with postsurgical decompression of the portal system [17–19]. However, the linear correlation between the severity of portal hypertension and that of PHG is controversial. In a prospective study of 331 patients, it was reported that severe PHG showed a significantly shorter expected survival time than mild PHG (median survival

time, 77.6 ± 9.6 months in severe PHG) [20]. The study concluded that PHG was associated with severity of portal hypertension and prognosis in patients with cirrhosis. However, other studies have been unable to demonstrate a correlation between the severity of portal hypertension and that of PHG [21–23].

Other molecular mediators at the mucosal level have been implicated in the development of PHG including tumor necrosis factor (TNF)-α, endothelin-1 (ET-1), nitric oxide (NO), and prostaglandins [21, 24]. Interestingly, patients with cirrhosis and PHG have abnormal blood circulation, which makes them susceptible to reduced delivery of oxygen to the gastric mucosa [21, 25]. This phenomenon modifies blood circulation which enables reduced resistance of gastric mucosa to irritants in patients with cirrhosis and portal hypertension [26].

4. Diagnostic evaluation

GAVE and PHG can be encountered during upper endoscopy in both symptomatic and asymptomatic patients with liver cirrhosis. GI bleeding is the common significant complication of GAVE and PHG. PHG is responsible for about 8% of nonvariceal upper GI bleeding, while GAVE accounts for up to 4% [27, 28]. Both GAVE and PHG may have similar endoscopic appearances and require further histological analysis. In 1995, Payne et al. established that portal hypertensive gastropathy (PHG) and GAVE are distinct clinical entities that require different forms of treatments [13]. Thus, it is incumbent on clinicians to be able to differentiate both diseases.

GAVE is a disease limited to the stomach and is almost exclusively noted in the gastric antrum on endoscopy [29]. GAVE was first reported in 1984 and initially termed 'watermelon' stomach in three patients with iron deficiency anemia [30]. In their report, they described visible convoluted and tortuous columns of ectatic vessels along rugal folds of the antrum, which converged at the pylorus, resembling stripes of a watermelon. In more severe cases, GAVE can present as more punctate lesions or more diffusely, extending to the gastric body, which is most commonly associated with GAVE in cirrhotics than other etiologies [31]. Interestingly, GAVE patients have been reported to have more severe liver disease, greater blood loss, lower serum gastrin levels, and a higher incidence of previous sclerotherapy [13].

Histologically, GAVE is characterized by dilated mucosal capillaries and venules with intimal thickening, fibrin thrombi, spindle cell proliferation, and fibromuscular hyperplasia of the lamina propria [13, 32]. The presence of these histological features is used to calculate a GAVE score which has 80% diagnostic accuracy. This can be used to distinguish GAVE from PHG with a GAVE score equal or greater than three [13].

PHG lesions are typically seen in the gastric fundus unlike GAVE which is commonly found in the antrum. Endoscopically, PHG appears as a mosaic-like pattern or a diffuse, erythematous, and reticular cobblestone pattern of gastric mucosa consisting of small polygonal areas

with superimposed red punctate lesions >2 mm in diameter and a depressed white border [33–35]. Severe PHG is associated with flat or bulging red spots, resembling a scarlatina rash with friability or diffuse hemorrhagic gastropathy [36–38].

5. Management of GAVE

5.1. Endoscopic management

The treatment of choice in managing patients with GAVE is endoscopic ablation of the lesions. Pharmacologic or surgical intervention should be considered when endoscopic therapy has failed. Argon plasma coagulation (APC) has become the method most utilized by endoscopist. APC is a noncontact technique that uses argon gas to equally distribute thermal energy. High-frequency current is applied to the tissue with controllable depth of coagulation (roughly 2–3 mm) [39]. Its efficacy ranges from 90 to 100% [40]. Endoscopic band ligation (EBL) and radiofrequency ablation (RFA) are newer and promising techniques in the treatment of GAVE; however, RFA requires additional training and is not readily available in all endoscopic centers [41].

Compared to older laser therapy methods, APC is more user-friendly, manageable, cheaper, and safer. The risk of perforation is very low and limited to very thin-walled structures [42]. The pooled recurrence rate of bleeding is estimated at 36% [43]. Cryotherapy has also been introduced as another means for managing GAVE. It makes use of nitrous oxide to freeze abnormal mucosa and causes superficial necrosis. A pilot study that assessed 12 patients with GAVE and anemia showed that 50% of patients achieved a complete response after cryotherapy [44]. The remaining patients achieved a partial response with decreased transfusion requirements. However, the optimal delivery mechanism and the number of treatments required remain unclear.

Overall, EBL seems to be the safest and has only been associated with minor complications such as abdominal pain [45]. An observational study of 22 patients (9 patients receiving endoscopic thermal therapy vs. 9 patients receiving EBL) reported fewer bleeding in the EBL cohort (67 vs. 23%), as well as fewer treatment sessions for EBL (4.9 vs. 1.9), and a decrease in EBL-related transfusions (−5.2 vs. −12.7) [46]. A prospective study of 21 patients reported a clinical response that was achieved in 19 patients (91%) after a mean of 2.28 endoscopic sessions and a mean of 16 bands applied [47]. Another study comparing the efficacy of EBL vs. APC reported a lower recurrence rate in the EBL cohort (8.3 vs. 68%) [48].

5.2. Medical treatment

While a variety of drugs have been used to manage GAVE-related bleeding, none has shown to be clinically effective and efficacious as an alternative to invasive methods. Pilot studies with estrogen-progesterone hormone therapy have been shown to control bleeding due to gastrointestinal vascular malformations, including GAVE, with side effects [49–51]. Despite bleeding cessation, GAVE lesions persisted. Reduction of treatment frequency resulted in

bleeding relapse, requiring reinstitution of daily therapy for hemostatic control [49, 52, 53]. However, this form of treatment is not well studied and patients are at risk for developing severe side effects, such as menorrhagia and gynecomastia, and increased risk of endometrial and breast cancer [54].

A long acting somatostatin analog, octreotide, has been reported as an effective drug in controlling chronic bleeding due to vascular abnormalities [55]. This may in part be due to the inhibitory effect on neuroendocrine cells, ectatic vessels, and smooth muscle cells [55, 56]. Octreotide also displays antiangiogenic effects and limits the growth of blood vessels [57]. However, octreotide treatment has been unsuccessfully replicated by other authors and thus necessitates further investigation [58]. Success has been reported from the use of corticosteroids, tranexamic acid, thalidomide, and serotonin antagonist [59–63]. However, these treatments have been reported in some case reports and the results have not been confirmed by controlled clinical trials.

5.3. Surgical intervention

Surgical intervention is reserved for patients who do not respond to medical and endoscopic therapies. Surgical approaches include gastrectomy and antrectomy, which may be the only reliable approach to achieving a cure. Antrectomy is more commonly used and has clinical efficacy in eliminating bleeding and transfusion dependency, as patients do not report postoperative recurrence of bleeding was associated with multiorgan failure [64]. Portacaval shunts and TIPS have no role in the management of GAVE [11]. In GAVE patients due to underlying cirrhosis, complete resolution of symptoms has been observed following liver transplant, despite persistent portal hypertension [15].

6. Management of PHG

6.1. Medical treatment

The management of PHG is focused on abating portal pressure, mainly through the use of medical therapy rather than endoscopic means. Similar to esophageal varices, management attempts to reduce hepatic venous pressure gradient (HVPG) to <12 mmHg or by 20% which correlates with a reduction in mortality in some studies [65]. A meta-analysis established that target HVPG is a valid marker to monitor drug efficacy for variceal bleeding and patient prognosis [65]. Beta blockers are first-line drugs used to reduce portal pressure and have the most benefit in patients with mild PHG [66]. Modest effects have been noted in patients with severe PHG [67]. It is unclear whether beta blockers are prophylactically effective in preventing bleeding from PHG [24]. However, in patients receiving propranolol or nadolol for esophageal variceal bleeding prophylaxis, beta blocker therapy showed a reduction in future PHG bleeding [68].

In a randomized controlled trial to investigate the efficacy of propranolol, 26 of 54 patients received propranolol and the rest placebo. Daily doses of 40–320 mg were used. In the cohort

receiving propranolol, patients reported significantly lower rates of rebleeding (38 vs. 65%) at 12 months and at 30 months (7 vs. 52%) compared with controls [67]. Similarly, a smaller study using a dose of 24–480 mg/day decreased the incidence of acute bleeding in 16 patients with PHG and also reduced the grade of PHG in 24 asymptomatic patients when given at a dose of 160 mg/day [68].

In unstable patients who have contraindications for beta blockers, other agents have been studied with varying efficacy including somatostatin, octreotide, terlipressin, and vasopressin [69–72]. Somatostatin and its analogs showed complete control of acute bleeding with 11% rebleeding after withdrawing infusion [69]. Octreotide controlled bleeding in 100% of patients within 48 h. Vasopressin controlled bleeding in 64% of patients over the same time [71]. Terlipressin, a vasopressin analog (not available in the United States), was similarly effective as vasopressin [72].

6.2. Endoscopic management

Acute bleeding in the setting of PHG rarely occurs. A large study reported an incidence of acute bleeding from gastropathy in 8 of 315 patients (2.5%), compared to chronic bleeding which occurred in 34 patients (10.8%) [73]; however, if it occurs, such bleeding episodes can be severe and challenging to manage. In addition to intravenous medical therapy with aforementioned agents aimed at reducing portal pressure and hemostatic control, appropriate antibiotic and resuscitation should be initiated and tailored to the patient's needs.

Endoscopic therapy for acute bleeding from PHG remains investigational and may provide temporary control. For patients with refractory bleeding who are not candidates for portosystemic shunting, limited data suggest that endoscopic thermal therapy may be efficacious. Similar to GAVE, APC has proven successful in controlling bleeding and reducing transfusion requirements [74]. Furthermore, hemostatic powder is emerging as a useful means for managing patients with acute bleeding. The powder acts by forming a barrier over the bleeding site and increasing the concentration of clotting factors [75].

6.3. Surgical intervention

In cases of failed medical or endoscopic therapy requiring increase blood transfusions, portosystemic shunt therapy should be considered through the placement of a transjugular intrahepatic portosystemic shunt (TIPS). Shunting works by relieving portal hypertension with the placement of a tube (shunt) between the portal vein which carries blood from the intestines to the liver and the hepatic vein which carries blood from the liver back to the heart. Patients who have the TIPS procedure show significant improvement in endoscopic appearance of PHG and number of transfusion requirements [76].

A prospective study of 30 patients with mild PHG and 10 patients with severe PHG with recurrent GI bleeding had a 75% reduction in endoscopic severity, a Childs-Pugh Score of 11.5, and a mean reduction in portacaval gradient from 20 to 12 mmHg following TIPS [17]. Patients typically show endoscopic improvement in 6 weeks for mild cases and up to 3 months for more severe cases of PHG [77]. A retrospective study of 40 Child-Pugh class A

and B cirrhotic patients comparing surgical shunting and TIPS found improved outcomes from surgical shunting with reduced 30-day mortality, reduced rebleeding events, and fewer shunt revisions and hospitalizations [78].

However, surgical shunting carries risks of substantial perioperative morbidity and mortality. In those who survive operation, accelerated hepatic decompensation and neuropsychologic deterioration (portosystemic encephalopathy) significantly diminish the overall benefit of the shunting procedure [78]. Similarly, TIPS carries a potential risk for rapid liver failure necessitating liver transplantation [79].

7. Conclusion

In summary, GAVE and PHG are two clinically distinct entities that present with gastrointestinal blood loss. Majority of patients with portal hypertension and cirrhosis will develop PHG; however, it can also occur in the setting of noncirrhotic portal hypertension. GAVE is associated with gastric dysmotility, autoimmune reactivity, reduced gastrin levels, and elevated prostaglandins. It is not associated with cirrhosis. Therapy of PHG is directed toward lowering and stabilizing portal pressure with beta blockers or shunt procedures. GAVE management mainly involves the use of endoscopic methods to ablate bleeding lesions. When GAVE is complicated by cirrhosis, it is incumbent on clinicians to differentiate it from PHG as GAVE does not respond to treatments aimed at reduction of portal pressure.

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