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Efficacy of argon plasma coagulation for gastric antral vascular ectasia associated with chronic liver disease

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Abstract

Gastric antral vascular ectasia (GAVE) is a rare cause of chronic gastrointestinal bleeding. The aim of this study was to evaluate the relationship between GAVE with cirrhotic patients and liver dysfunction, portal hypertension and the safety and efficacy of argon plasma coagulation (APC) in treating GAVE with cirrhotic patients. Eight cirrhotic patients with the characteristic endoscopic findings of GAVE were registered. In this study, APC was performed for GAVE in all eight patients. The patients-liver function was classified by Child-Pugh classification and classifications were: two class A, five class B and one class C (mean score: 7.8). Five patients had previously received prophylactic endoscopic injection sclerotherapy for esophageal varices and one had esophageal varices. Balloon-occluded retrograde transvenous obliteration (B-RTO) for gastric varices had been performed in other one patient. Portal hypertensive gastropathy (PHG) was recognized in only one case. APC was performed in all eight patients and one to three treatment sessions were needed (mean: 1.8 sessions). No complications were observed in the initial treatment. During follow-up, endoscopies revealed the recurrence of GAVE in two patients requiring further treatment by APC (recurrence rate: 25%). After APC treatment, the recurrence of GAVE was not observed with endoscopy in the other six patients. The results suggest that GAVE is related to severe liver damage and portal hypertension in cirrhotic patients. APC is a safe and effective treatment against GAVE.

Keywords: Argon plasma coagulation; Gastric antral vascular ectasia; Esophageal varices; Liver cirrhosis; Portal hypertension

1. Introduction

Gastric antral vascular ectasia (GAVE) is a rare cause of chronic gastrointestinal bleeding, often presenting as iron-deficiency anemia, melena or rarely, hematemesis [1–4]. The endoscopic findings are found almost exclusively in the antrum and red spots are either aggregated in linear stripes or diffusely spread. Histologically, dilated mucosal capillaries with fibrin thrombi and fibromuscular hyperplasia of the lamina propria are seen without inflammation [5]. The diagnosis of GAVE is achieved on the basis of endoscopic and histological appearance. GAVE has been associated with cirrhosis, renal failure, bone marrow transplantation, scleroderma. The majority of GAVE patients have underlying cirrhosis and portal hypertension. However, Spahr et al. have reported that GAVE is not directly related to portal hypertension [6].

The optimal treatment of GAVE is not known. Argon plasma coagulation (APC) is a relatively new non-contact electrocoagulation technique. Grund et al. have reported on the first experiences with APC as an option in the treatment of gastrointestinal hemorrhage [7].

The aim of this study was to evaluate the relationship between GAVE with cirrhotic patients and liver dysfunction, portal hypertension and the safety and efficacy of APC in treating GAVE with cirrhotic patients.

2. Patients and methods

Between January 2001 and December 2003, eight patients with the characteristic endoscopic findings of GAVE were

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registered. The subjects included three males and five females ranging in age from 57 to 78 years (mean: 67.9). The underlying pathologies of chronic liver diseases were liver cirrhosis in seven patients and cirrhosis associated with hepatocellular carcinoma in one. The etiologies of their liver diseases were as follows: hepatitis B surface antigen (HBs Ag) positive in one case, anti-HCV antibody positive (HCV) in four, alcoholic liver diseases in one and unknown in two. All eight patients had iron-deficiency anemia and five patients also had a history of melena.

In this study, APC was performed for GAVE in all eight patients. The unit used in this study was the standard APC equipment consisting of a high-frequency electrosurgical generator (ICC 350, Tuebingen, ERBE, Germany) an automatically regulated argon source (APC 300) and a flexible APC probe. The APC probe was a 2.3 mm Teflon-coated catheter with a heat-resistant ceramic tip, which could be passed through the working channel of an endoscope. The 60 W electrical power and argon gas flow was 2 L/min. Coagulation time was under 2 s. Follow-up periods after APC were from 12 to 47 months (mean 28 months).

3. Results

In two patients (cases 1 and 2), active oozing bleeding from GAVE had been observed endoscopically. In three patients (cases 4–6), marked anemia was recognized and gastrointestinal bleeding (melena) was observed in five cases (cases 1, 2, 4, 6 and 8). The liver functioning of the patients was classified by Child-Pugh classification and the classifications were: two class A, five class B and one class C (mean score: 7.8). Five patients (cases 1, 2, 5, 6 and 7) had previously received prophylactic endoscopic injection sclerotherapy (EIS) for esophageal varices. Case 4 had esophageal varices (Lm, F2, Cw, RC(−)) according to The General Rules for Recording Endoscopic Findings of Esophageal Varices prepared by the Japanese Research Committee on Portal Hypertension [8]. Balloon-occluded retrograde transvenous obliteration (B-RTO) for gastric varices had been performed in other one patient (case 8). Portal hypertensive gastropathy (PHG) was recognized in only one case (case 3) and this case showed mild PHG (Table 1). Antral motility in these GAVE was frequent and intense in all eight patients. Hypergastrinemia was discerned in three of the five patients tested.

Table 1
Characteristics of GAVE patients

Case	Gender	Liver disease	C-P score	EV	PHG
1	F	LC	B (8)	(−)	(−)
2	F	LC	B (7)	FORC (−)	(−)
3	M	LC	A (5)	(−)	(+)
4	F	LC	B (8)	LmF2CwRC (−)	(−)
5	F	LC	B (9)	LmF1CbRC (−)	(−)
6	M	LC	C (10)	F0RC (+)	(−)
7	M	LC	A (6)	(−)	(−)
8	F	LC + HCC	B (9)	(−)	(−)

LC: liver cirrhosis; C-P: Child-Pugh; EV: esophageal varices; PHG: portal hypertensive gastropathy.

APC was performed in all eight patients and treatment was commenced at the pylorus, moving proximally. The goal of treatment was the formation of pale yellow coagulum over the vascular lesions. One to three treatment sessions were needed (mean: 1.8 sessions). No complications were observed in the initial treatment. Endoscopic findings after APC revealed both the small ulcers and the improvement of GAVE in all eight patients. During follow-up, endoscopies revealed the recurrence of GAVE in two patients requiring further treatment by APC (recurrence rate: 25%). However, further treatment by APC was effective in these two cases. On the other hand, after APC treatment, the recurrence of GAVE was not observed with endoscopy in other six patients.

4. Case presentation (a 69-year-old female with C type liver cirrhosis)

The fibergastroscopic examination revealed active oozing bleeding from mucosal diffuse spotty redness spreading over the antrum of the stomach (Fig. 1). The mucosal spotty redness revealed telangiectasia with endoscopic findings (Fig. 2) and an ultrasonic microprobe (UMP) using 20 MHz under constant filling of deaerated water in the stomach showed small low echoic spots within the second (mucosa) and third (submucosa) sonographic layers (Fig. 3). We diagnosed GAVE based upon these findings. She was treated with APC and the probe was applied to the lesion beginning at the pylorus and proceeding proximally. The sessions of APC were repeated 1 week later and three sessions were performed in this patient. The fibergastroscopic examination 20 days after treatment showed the improvement of GAVE (Fig. 4). The additional APC was performed 3 months later on the recurrent GAVE in this patient. After the additional APC treatment, the recurrence of GAVE was not observed with endoscopy and the low echoic spots within the second sonographic layer disappeared via UMP.

5. Discussion

The etiology of GAVE remains unknown and it is thought to encompass a variety of conditions including autoimmune disease, liver cirrhosis, portal hypertension, chronic renal failure, cardiovascular disease, achlorhydria, severe hypochlorhydria, hypothyroidism and diabetes mellitus [9–16]. In Gostouts article, atrophic gastritis occurred in 19 of 19 (100%) with biopsies and hypergastrinemia was observed in 25 of 33 (76%) tested [9]. Gastrin may play a role in the pathogenesis of GAVE. In our cases, hypergastrinemia was discerned in three of five patients tested. However, it may not be fundamental and also, Ikeda et al. have reported that the development of GAVE associated with cirrhosis was concerned with the time courses [17,18].