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Esophageal Strictures Following Endoscopic Variceal Sclerotherapy

Antecedents, Clinical Profile, and Management

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We have evaluated 169 patients with portal hypertension receiving endoscopic variceal sclerotherapy in order to assess the predisposing factors, clinical profile, and treatment response of sclerotherapy-induced esophageal strictures. Of the 129 patients included in the final analysis, 20 (15.5%) developed persistent esophageal stricture. No significant difference was found with respect to age, nature of sclerosant (absolute alcohol, ethanolamine oleate, or sodium tetradecyl sulfate), etiology of portal hypertension, Child's class, initial variceal score, or intensity of sclerotherapy schedule between the patients who developed strictures and those who did not. However, female sex ($P < 0.01$) and persistent esophageal ulceration ($P < 0.05$) did predispose to stricture formation. Sclerotherapy-induced strictures presented with a variable grade of dysphagia, were always solitary, and were localized to the lower end of esophagus. Most of these could be dilated rapidly using Eder-Puestow metal olives (3.15 ± 0.80 dilatation sessions per patient). Stricture formation did interrupt an effective sclerotherapy program but only temporarily, and successful variceal obliteration could be obtained after stricture dilatation.

KEY WORDS: portal hypertension; sclerotherapy; varices; esophagus; stricture.

Esophageal stricture formation is one of the major sequelae of endoscopic variceal sclerotherapy (EVS). The incidence of stricture formation following EVS varies in different series from 0 to 41% (1–13). Although a number of factors influencing the formation of EVS-induced strictures have been listed (1), the data are meagre and conflicting, as most of the available studies have evaluated the role of one or two variables only (7–10). Moreover the clinical profile and treatment of EVS-induced strictures is not well documented. We have studied the relationship of EVS-induced strictures with age and sex of patients, etiology of portal hypertension and Child's class, nature of the sclerosant, and intensity of EVS schedule, as well as with ulcer formation and efficacy of EVS. The outcome of these strictures is also discussed.

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MATERIALS AND METHODS

Study Population. A total of 169 patients with portal hypertension and a history of upper gastrointestinal hemorrhage were initiated on an elective EVS schedule between January 1984 and September, 1988. Fifteen patients who died (cirrhosis: 13; extrahepatic portal venous obstruction: 2) due to hepatic encephalopathy and/or gastrointestinal bleed and 20 patients who dropped out before the third session of EVS were excluded from final analysis. Five patients who received a different sclerosant during subsequent EVS sessions were also excluded. The remaining 129 patients (age range 6–70 years, 84 men and 45 women) consisted of 68 cirrhotics (alcoholic: 27; postnecrotic: 33; others: 8), 33 with extra-

hepatic portal venous obstruction (EHPVO), and 28 patients with noncirrhotic portal fibrosis (NCPF). The patients were classified into A, B, and C classes using Pugh modification of Child-Turcotte criteria for severity of liver disease (14). Esophageal varices were graded from 1 to 4 using Conn's criteria (15). Informed consent was obtained from all patients included in the study.

Techniques. Patients received EVS with one of the three sclerosants—absolute alcohol (AA), 5% ethanolamine oleate (ETH), or 3% sodium tetradecyl sulfate (STD). While 90 consecutive patients presenting between January 1985 and December 1987 were randomized to receive one of the three sclerosants, the remaining patients received the sclerosant according to the availability of the agent. EVS was performed using a straight-viewing flexible fiberoptic endoscope (Olympus, GIF-Q or GIF-Q10) and a standard injector needle (Olympus, NM-3K) after premedication with intravenous hyoscine *N*-butyl bromide (20 mg) and pentazocine (30 mg). In addition, diazepam (5–10 mg) was given intravenously in children and apprehensive patients. The injections were given circumferentially by the intravariceal route when 0.5 to 2.0 ml of the sclerosant was injected at each site, depending on the grade of the varix. Starting from close to the cardia, one to three injections were made in each varix spaced approximately 2 cm apart. "Feel" of puncturing the varix and some oozing of blood after withdrawal of the needle were taken as indicators of the intravariceal nature of the injection. EVS was repeated at intervals of three weeks, and at each session evaluation of variceal grading was done and presence of dysphagia, esophageal ulceration, and/or stricture was noted. Details of ulcers were recorded; injection into the varix with an ulcer was deferred until the next session, and the patient was started on oral sucralfate suspension (1 g 6 hourly, 14 patients) or ranitidine (150 mg twice a day, 26 patients), which was continued until the end of EVS.

A stricture was defined as inability to negotiate a GIF-Q endosoope (outer diameter 11 mm) and was confirmed by barium swallow examination. Patients with stricture who were symptomatic for more than three weeks were treated by dilatation with Eder-Puestow metal olives. During each session up to four olives of increasing diameter were passed. Dilatation sessions were repeated every three weeks until the 45 F olive was negotiable and the patient had relief of symptoms. In patients with unoblitered varices at the time of stricture formation, EVS was resumed after full dilatation was achieved.

Statistical Evaluation. Data were analyzed to assess stricture formation in relation to age and sex of the patients, etiology of portal hypertension, Child's class, nature of sclerosant, initial variceal score (obtained by adding the grades of all the varices), intensity of EVS schedule (ie, number of EVS sessions and volume of sclerosant), and occurrence of ulcers. To analyze the differences between the three sclerosants, the incidence of stricture formation with each agent and the number of EVS sessions and the total volume of sclerosant injected till the formation of strictures were compared. The success rate of EVS was compared between patients developing stricture and those without it. For the purpose of this analysis, EVS was considered successful when there was total obliteration of varices or ≥50% downgrading of variceal score (16). Variables for the severity of EVS-induced strictures were evaluated by comparing the etiology of portal hypertension, Child's class, nature of sclerosant, and intensity of EVS schedule in patients requiring ≤3 dilatations with those requiring ≥4 dilatations. The χ^2 method with Yates' correction was used to compare dichotomous variables. Comparisons between groups of parametric data were made using Student's two-tailed *t* test for unpaired data and between groups of nonparametric data with Mann-Whitney U test. With all analyses, an associated probability (P value) of <5% was considered significant. Data are expressed as mean ± SEM.

RESULTS

Of the 129 patients included in the study, 25 patients developed an esophageal stricture. Five of these patients with strictures had resolution of dysphagia within three weeks of diagnosis. Twenty patients with a stricture (15.5% of all the patients on EVS), who were symptomatic for more than three weeks, were subjected to dilatation. The number of EVS sessions received by these 20 patients prior to stricture formation ranged from 2 to 15 with a mean of 4.5 ± 0.71, while the total volume of sclerosant received ranged from 8 to 142 ml with a mean of 40.98 ± 7.16 ml.

Stricture Formation with Different Sclerosants. Table 1 gives the patient characteristics in the three sclerosant groups—AA, ETH and STD. The three groups were comparable in all respects except that the age of patients in the STD group was significantly older ($P < 0.05$) as compared to the ETH group. Table 2 gives the results with individual sclerosants for incidence of stricture formation, and the number of EVS sessions as well as total volume of sclerosant injected prior to stricture formation. There was no significant difference in these three parameters between any two of the three sclerosants when compared to one another ($P > 0.05$).

Stricture Formation and Age and Sex of Patients. The patients who developed a stricture had a mean age of 30.2 ± 3.83 years (range 6–65 years) compared to 35.67 ± 1.50 years (range 7–70 years) for the patients who did not develop strictures ($P > 0.05$, unpaired *t*-test). While only 34.9% (45/129) of total patients were women, among the patients developing strictures, 60% (12/20) were women. There was a significant sex difference between patients with strictures and those without it, with more women in stricture group ($P < 0.01$, χ^2 method).

Stricture Formation and Etiology and Severity of Liver Disease and Initial Variceal Score. The incidence of stricture formation in patients with cirrhosis was 10.29% (7/68), in NCPF 21.43% (6/28), and in EHPVO 21.21% (7/33). The incidence of strictures in relation to Child's class was 17.86% (15/84) in Child's A as compared to 11·11% (5/45) in Child's B and C together. The differences

TABLE 1. CHARACTERISTICS OF PATIENTS IN DIFFERENT SCLEROSANT GROUPS

	Absolute alcohol (AA)	*Ethanolamine oleate (ETH)*	*Sodium tetradecyl sulfate (STD)*
No. of patients	93	20	16
Age (years)*,†	34.19 ± 1.60	32.0 ± 3.55	41.69 ± 4.07
Sex (M/F)‡	61/32	13/7	10/6
Etiology of portal hypertension‡ (No. of patients)			
Cirrhosis	48	10	10
NCPF§	21	4	3
EHPVO¶	24	6	3
Child's class‡ (No. of patients)			
Class A	60	14	10
Class B	22	4	3
Class C	11	2	3
Initial variceal score**	11.7 ± 0.41	12.45 ± 1.05	12.12 ± 0.84

*Mean ± SEM.
**$P > 0.05$ for any two groups; unpaired t test.
†$P < 0.05$ for ETH versus STD, $P > 0.05$ for any other two groups; unpaired t test.
‡$P > 0.05$; χ^2 method.
§NCPF, noncirrhotic portal fibrosis.
¶EHPVO, extrahepatic portal venous obstruction.

in the incidence of stricture formation in respect to etiology or the Child's class (A vs B + C) were not significant ($P > 0.05$, χ^2 method). There was no difference in the initial variceal score between the patients developing a stricture (12.8 ± 0.82) and those who did not develop stricture (11.68 ± 1.04) ($P > 0.05$, Mann-Whitney U test).

Stricture Formation and Intensity of EVS Program. There was no difference between patients with strictures and those without them, in any of the three sclerosant groups, with respect to the total volume of sclerosant injected (Table 2). However, a significantly lower number of EVS sessions had been performed with AA in patients with strictures as compared to those without it ($P < 0.01$). The number of EVS sessions was not different in those with a stricture as compared to those without it for the other two sclerosant groups.

Stricture Formation and Variceal Ulceration. A total of 40 patients had esophageal ulceration overlying the variceal column three weeks after the last EVS session. The ulcers were always seen in the lower 5–6 cm of the esophagus and were oval in shape, vertical diameter being greater than the horizontal one. Circumferentially, individual ulcers did not involve more than approximately 15% of the esophageal mucosa. Of the 20 patients with a symptomatic esophageal stricture 12 (60%) had preceding ulceration, while of the 104 individuals without a stricture 28 (26.9%) had preceding ulceration ($P < 0.05$, χ^2 test). The ulcer size (greatest diameter) was more than 10 mm in six (50%) of the 12 patients with ulceration and

TABLE 2. EFFECT OF INTENSITY OF SCLEROTHERAPY IN RELATION TO SCLEROSANT AND DEVELOPMENT OF STRICTURE

	Patients with persistent stricture (N = 20)	*Patients without stricture (N = 104)*	P *value*
Absolute alcohol			
No. of patients	15	76	
No. of EVS* sessions†	3.53 ± 0.36	4.95 ± 0.24	<0.01‡
Volume of sclerosant† (ml)	32.17 ± 4.67	36.28 ± 1.84	>0.05‡
Ethanolamine oleate			
No. of patients	3	14	
No. of EVS* sessions†	8.67 ± 3.48	8.5 ± 1.19	>0.05§
Volume of sclerosant† (ml)	84.67 ± 32.63	82.89 ± 14.09	>0.05§
Sodium tetradecyl sulfate			
No. of patients	2	14	
No. of EVS* sessions†	5.5 ± 3.5	6.21 ± 1.17	>0.05§
Volume of sclerosant† (ml)	41.5 ± 28.5	39.04 ± 8.18	>0.05§

*EVS, esophageal variceal sclerotherapy.
†Mean ± SEM.
‡Mann-Whitney U test.
§Unpaired t test.

TABLE 3. NUMBER OF DILATATIONS REQUIRED IN PATIENTS WITH EVS-INDUCED STRICTURES

Dilatation sessions (N)	*Patients* (N)
1	11
2–3	3
4–5	3
>5	3

subsequent strictures and in four (14.3%) of the 28 individuals with ulceration but no subsequent strictures ($P < 0.05$, χ^2 test). Multiple ulcers were also seen more frequently in patients developing strictures (four of 12) than in those not developing strictures (four of 28), although this difference did not achieve statistical significance ($P > 0.05$, χ^2 test).

The time elapsed between detection of esophageal ulceration and diagnosis of strictures varied from three to six weeks (ie, at one of the next two endoscopy sessions). Four of the 12 patients with ulceration and strictures still had active still ulceration at the time of diagnosis of strictures. Ulcer healing occurred within the next three to nine weeks, and no difference was observed in this regard between those who subsequently developed strictures and those who did not. Neither was any difference regarding stricture formation observed among patients with ulceration who received ranitidine (eight of 26) or sucralfate (four of 14) ($P > 0.05$, χ^2 test).

Clinical Profile and Treatment of Strictures. Of the 20 patients with persistent strictures, 12 had dysphagia to solids alone, while eight had dysphagia to both solids and liquids. Stricture in each patient was solitary, localized within the distal 5 cm of the esophagus, and measured between 1–2 cm in length. A total of 63 dilatation sessions were carried out with a mean of 3.15 ± 0.80 (range 1–15) sessions per patient. However, only six patients required ≥4 dilatation sessions (Table 3). While 17 (85%) patients had complete relief of dysphagia following dilatation, three patients had only partial relief and continued to be symptomatic despite dilatation up to 45 F. Complications, which were minor, occurred during four sessions (6.35%) and consisted of bleeding ($N = 3$) and esophageal leak ($N = 1$). These complications were managed conservatively with nil orally, parenteral fluids, and mild sedation. Blood transfusion (1 unit) was given to one of the patients with bleeding while the patient with esophageal leak was treated with antibiotics. None of the patients had mediastinitis, fever, or septicemia after dilatation. Comparing the patients who required ≤3 dilatations and those who required ≥4 dilatations, there was no significant difference ($P > 0.05$) with respect to etiology of portal hypertension, Child's class, nature of sclerosant, number of EVS sessions, and the total volume of sclerosant injected prior to stricture formation (Table 4). The period of follow up after dilatation ranged from 8 to 59 months (mean 25.9 ± 3.18 months) with no recurrence of dysphagia or strictures in successfully treated patients.

Stricture Formation and Success of EVS. Of the 104 patients without a stricture 81 (77.9%) had successful EVS, while nine (45.0%) of the 20 patients with a stricture had successful EVS at the time of stricture formation; this difference between the two was significant ($P < 0.005$, χ^2 method). Eighteen patients with strictures had less than complete obliteration of varices at the time of stricture formation. Twelve of these 18 patients gave consent for further EVS after effective dilatation. EVS was successfully achieved in all these 12 patients without any further complications.

TABLE 4. COMPARISON OF CHARACTERISTICS IN PATIENTS REQUIRING ≤3 DILATATION SESSIONS WITH THOSE REQUIRING ≥4 SESSIONS

	≤3 dilatation sessions (N = 14)	*≥4 dilatation sessions* (N = 6)	P *value*
Etiology of portal hypertension (No. of patients)			
Cirrhosis	5	2	>0.05†
NCPF[a]	5	1	
EHPVO[b]	4	3	
Child's class (No. of patients)			
Class A	11	5	>0.05†
Class B + C	3	1	
Sclerosant used			
Absolute alcohol	12	3	>0.05†
STD[c]/Ethanolamine	2	3	
Volume of sclerosant used prior to stricture formation‡	35.82 ± 9.15	53.0 ± 10.03	>0.05§
No. of EVS[d] sessions prior to stricture formation‡	4.28 ± 0.92	5.0 ± 1.12	>0.05§

[a]NCPF, noncirrhotic portal fitrosis; [b]EHPVO, extrahepatic portal venous obstruction; [c]STD, sodium tetradecyl sulfate; [d]EVS, endoscopic variceal sclerotherapy.
†χ^2 method.
‡Mean ± SEM.
§Unpaired *t* test.

DISCUSSION

The exact pathogenesis of esophageal strictures following EVS remains unclear. Tissue reaction to the sclerosant is believed to be the major determinant (11), while sclerotherapy-induced alteration in esophageal motility (10) and acid reflux (4, 10) are presumed to be contributory. Factors influencing the tissue reaction, however, remain to be elucidated. The present study is an attempt to define these factors.

We did not find any correlation between stricture formation and Child's class and the etiology of portal hypertension. Severity of liver disease was found to be a risk factor in one study (6) but not in another (11). We admit that the number of patients with Child's class C in our study was small, and a larger number might have been more helpful in deriving a definite conclusion. Ours is the only study in which the influence of different causes of

portal hypertension on stricture formation is considered. In nearly all the available studies, patients with a single etiology have been studied and the incidence of strictures has varied between 0 and 17.6% among cirrhotics (2, 3, 12), between 0 and 18% among patients with EHPVO (17, 18) and between 1.7% and 8% among noncirrhotic intrahepatic portal hypertension (NCPF and schistosomiasis) (19, 20). Interestingly a majority (60%) of the patients who developed strictures were females. This subgroup of patients did not differ from rest of the patients in regard to severity of liver disease, nature of sclerosant, and technique and intensity of EVS. We cannot offer any explanation for the higher rate of stricture formation in females.

Our results have shown that the nature of the sclerosant does not influence the chance of development of stricture formation. In an earlier study, we had shown that the three sclerosants used by us (AA, ETH, and STD) had similar efficacy and complication rates (16). A review of literature reveals that the rate of stricture formation does not differ with different sclerosants, being up to 24% with STD (3, 6, 7, 11), up to 16% with ETH (4, 12), up to 17.4% with AA (13, 16), and up to 41% with polidocanol (1, 18, 19).

Intensity of EVS, as judged by the number of EVS sessions and the volume of sclerosant injected, was not found to have any adverse bearing in this study. The observations of other workers (6, 11) are in accordance with ours, although Sorenson et al (1) had contradictory findings. Earlier studies have shown no influence of interval between EVS sessions (7, 13) as well as the route of EVS (intra- versus paravariceal) (9) on the stricture formation. It should be emphasized, however, that it may be difficult to control the exact delivery of the sclerosant, particularly when sclerosis starts setting in. Moreover, it has been shown that up to 25% of the intended intravascular injections are actually made paravariceally (8). The length of the injector needle also can affect the complication rate, depending upon the depth of injections made. However, this factor is difficult to control.

We have shown a correlation between esophageal ulcers persisting three weeks after the last EVS session and subsequent stricture formation. Moreover, ulcers tended to be larger in patients developing strictures. Some workers (1–3, 6) had earlier failed to find any correlation between ulcers and stricture formation. However, all these studies had evaluated ulceration within a week of last EVS session. Furthermore, the size of ulcers was not taken into consideration. We feel that, unlike the usual post-EVS ulcers, which heal spontaneously without sequelae (21), those that are persistent and larger are not so benign and they tend to heal with fibrosis and hence predispose to stricture formation. Another possible determinant is the depth of ulceration, which is difficult to assess. We have treated all our patients with EVS induced ulcers with anti ulcer drugs and found no difference in the stricture rate between patients receiving sucralfate and ranitidine. It is a moot point whether such treatment alters the natural history of EVS-induced ulceration (10, 22, 24), and our study, because of its design, cannot contribute on this aspect. Snady et al (10), in a recent study, did note a significantly lower stricture rate using vigorous acid protection regimen of antacid, cimetidine, and sucralfate.

There is limited information in the literature on the profile of EVS-induced strictures. In our observation strictures responded well to Eder-Puestow dilatation with a mean of 3.15 sessions per patient. Post-EVS strictures are generally reported to be easily dilatable (4, 6, 11). Some strictures, however, were found to be relatively refractory. When we analyzed the variables that may influence refractoriness of strictures, we found no differences in the patients requiring ≤ 3 dilatations and those requiring ≥ 4 dilatations (Table 4).

Dilatation of the strictures was effective, with complete relief of dysphagia in 85% of the patients. Persistent esophageal dysmotility, documented earlier in some of the patients undergoing EVS (25), may explain the failure of complete relief of dysphagia in some of our cases despite adequate dilatation. Although development of strictures did, at least temporarily, hamper the eradication of varices, EVS was performed successfully after stricture dilatation. Moreover, stricture dilatation did not precipitate bleeding from partially treated varices.

REFERENCES

1. Sorensen T, Burcharth F, Pedersen ML, Findahl F: Oesophageal stricture and dysphagia after endoscopic sclerotherapy for bleeding varices. Gut 25:473–477, 1984
2. Kitano S, Iso Y, Yamaga H, Hashizume M, Higashi H, Sugimachi K: Trial of sclerosing agents in patients with oesophageal varices. Br J Surg 75:751–753, 1988
3. Korula J, Balart LA, Radvan G, Zweiban BE, Larson AW, Kao HW, Yamada S: A prospective, randomised controlled trial of chronic esophageal variceal sclerotherapy. Hepatology 4:584–589, 1985

4. Howard ER, Stringer MD, Mowat AP: Assessment of injection sclerotherapy in the management of 152 children with oesophageal varices. Br J Surg 75:404–408, 1988
5. Sarin SK, Mishra SP, Sachdev GK, Thorat V, Dalal L, Broor SL: Ethanolamine oleate versus absolute alcohol as a variceal sclerosant: A prospective, randomized, controlled trial. Am J Gastroenterol 83:526–530, 1988
6. Haynes WC, Sanowski RA, Foutch PG, Bellapravalu S: Esophageal strictures following endoscopic variceal sclerotherapy: Clinical course and response to dilatation therapy. Gastrointest Endosc 32:202–205, 1986
7. Akriviadis E, Korula J, Gupta S, Ko Y, Yamada S: Frequent endoscopic variceal sclerotherapy increases risk of complications. Prospective randomized controlled study of two treatment schedules. Dig Dis Sci 34:1068–1074, 1989
8. Rose JDR, Crane MD, Smith PM: Factors affecting successful endoscopic sclerotherapy for oesophageal varices. Gut 24:946–949, 1983
9. Sarin SK, Nanda R, Sachdev G, Chari S, Anand BS, Broor SL: Intravariceal versus paravariceal sclerotherapy—a prospective, controlled, randomised trial. Gut 28:657–662, 1987
10. Snady H, Rosman AS, Korsten MA: Prevention of stricture formation after endoscopic sclerotherapy of esophageal varices. Gastrointest Endosc 35:377–380, 1989
11. Drell E, Prindiville T, Trudeau W: Post-sclerotherapy stricture: Etiology, incidence and therapy. Gastrointest Endosc 31:138, 1985 (abstract)
12. MacDougall BRD, Westaby D, Theodossi A, Dawson JL, Williams R: Increased long-term survival in variceal haemorrhage using injection sclerotherapy. Lancet 1:124–127, 1982
13. Sarin SK, Sachdev G, Nanda R, Batra SK, Anand BS: Comparison of the two time schedules for endoscopic sclerotherapy—a prospective randomized controlled study. Gut 27:710–713, 1986
14. Pugh RNH, Murray-Lyon IM, Dawson JL, Pietroni MC, Williams R: Transection of oesophagus for bleeding oesophageal varices. Br J Surg 60:646–649, 1973
15. Conn HO: Ammonia tolerance in the diagnosis of oesophageal varices. A comparison of endoscopic, radiological and biochemical techniques. J Lab Clin Med 70:442–451, 1967
16. Kochhar R, Goenka MK, Mehta S, Mehta SK: A comparative evaluation sclerosants for esophageal varices: A prospective randomised controlled study. Gastrointest Endosc 36:127–130, 1990
17. Kahn D, Terblanche J, Kitano S, Borman P: Injection sclerotherapy in adult patients with extrahepatic portal venous obstruction. Br J Surg 74:600–602, 1987
18. Bhargava DK, Dwivedi M, Dasarathy S, Arora A: Endoscopic sclerotherapy for portal hypertension due to extrahepatic obstruction: Long term follow up. Gastrointest Endosc 35:309–311, 1989
19. Bhargava DK, Dwivedi M, Dasarathy S, Sundaram KR: Sclerotherapy after variceal hemorrhage in noncirrhotic portal fibrosis. Am J Gastroenterol 84:1235–1238, 1989
20. El-Zayadi A, El-Din SS, Kabil SM: Endoscopic sclerotherapy versus medical treatment for bleeding esophageal varices in patients with schistosomal liver disease. Gastrointest Endosc 34:314–317, 1988
21. Sarin SK, Nanda R, Vij JC, Anand BS: Esophageal ulceration after sclerotherapy—a complication or accompaniment? Endoscopy 18:44–45, 1986
22. Roark G: Treatment of post-sclerotherapy esophageal ulcers with sucralfate. Gastrointest Endosc 30:9–10, 1984
23. Tabibian N, Smith JL, Graham DY: Sclerotherapy associated esophageal ulcers: Lessons from a double blind, randomized comparison of sucralfate suspension versus placebo. Gastrointest Endosc 35:312–315, 1989
24. Singal AK, Sarin SK, Misra SP, Broor SL: Ulceration after esophageal and gastric variceal sclerotherapy—Influence of sucralfate and other factors on healing. Endoscopy 20:238–240, 1988
25. Larson GM, Vandertoll DJ, Netscher DT, Polk HC: Esophageal motility: Effects of injection sclerotherapy. Surgery 96:703–710, 1984