

Impact of Oesophageal Variceal Injection on Gastric Varices in Cirrhotic Patients

Mohamad R El-medammes, Amany M Ibrahim, Ahmad M Basha

Mohamad R El-medammes, Amany M Ibrahim, Ahmad M Basha, Department of Internal Medicine, Faculty of Medicine, Zagazig University, Egypt

Correspondence to: Amany M Ibrahim, Department of Internal Medicine, Faculty of Medicine, Zagazig University, Egypt

Email: ammibrahim@zu.edu.eg

Telephone: +201001855393

Received: April 8, 2014 Revised: May 5, 2014

Accepted: May 11, 2014

Published online: June 21, 2014

© 2014 ACT. All rights reserved.

Key words: Oesophageal; Variceal injection; Gastric varices; Cirrhotic patients

El-medammes MR, Ibrahim AM, Basha AM. Impact of Oesophageal Variceal Injection on Gastric Varices in Cirrhotic Patients. *Journal of Gastroenterology and Hepatology Research* 2014; 3(6): 1114-1116 Available from: URL: <http://www.ghrnet.org/index.php/joghr/article/view/737>

ABSTRACT

AIM: Gastric variceal (GV) bleeding has the characteristics of more severe blood loss and higher mortality and is a more difficult management problem than oesophageal variceal (OV) hemorrhage. Therefore, it is important to identify patients with 'high risk' GV and it is important to knowing the effect of sclerotherapy on appearance of gastric varices. This study aim to show impact of esophageal variceal injection sclerotherapy on gastric varices appearance in cirrhotic patients.

METHODS: Included 100 cirrhotic patients presented with upper gastrointestinal bleeding due to oesophageal varices. All patient were subjected to history, clinical examination, laboratory investigation and upper GIT endoscopy: to detect size, shape, site and grades of oesophageal varices. After follow up of those patients for 6 months: we divided them into group 1 and group 2 according to presence or absence of gastric varices.

RESULTS: Gastric varices appeared after injection sclerotherapy of oesophageal varices in 10 patients (10%). There were no statistical significant differences between group 1 and group 2 as regard age, sex, clinical data and laboratory data. On the other hand there was no statistical significant difference between group 1 and group 2 patients as regard grade of esophageal varices

CONCLUSION: There is little relation between OV injection sclerotherapy and appearance of gastric varices.

INTRODUCTION

Gastro-esophageal varices are the most relevant porto- systemic collateral because their rupture results in variceal hemorrhage and it's the most common lethal complication of cirrhosis. Varices and variceal hemorrhage are the complication of cirrhosis that results most directly from portal hypertension^[1]. Portal hypertension is a life-threatening complication of cirrhosis and results in the development of portosystemic shunts comprising oesophageal varices^[2]. Gastric varices (GV) have been increasingly recognized as a major cause of gastrointestinal bleeding in patients with portal hypertension. Compared with oesophageal variceal (OV) bleeding, hemorrhage caused by gastric varices is usually more severe and hemostatic control is reported to be more difficult^[3]. Management for gastric varices usually includes vasoactive agents, endoscopic therapy and surgery^[4]. Rupture of gastric fundal varices is often lethal because of massive bleeding^[5]. The bleeding from GV is more severe, requiring more blood transfusions, and has a higher mortality rate than oesophageal variceal bleeding^[6]. Bleeding from GVs is generally thought to be more severe than bleeding from OVs but it occur less frequently^[7]. Endoscopic therapy has been considered the mainstay of specific therapy for acute variceal bleeding. Endoscopic sclerotherapy has been shown to reduce bleeding and increase survival^[8]. According to oesophago-gastric varices grading system of Japan society of portal hypertension; the varices are evaluated based on color white [(cw) and blue (cb)], form [small and straight (F1)

nodular (F2) and large or coiled (F3)] and the red color sign (Rco3). Gastric varices are commonly classified based on their relationship with oesophageal varices as well as their location in stomach. Gastro-oesophageal (GOV) are an extension of oesophageal varices and are categorized into two types. The most common is type one (GOV1) which extend along lesser curvatures and they are considered extension of esophageal varices and should be managed similarly. Type two gastric varices (GOV2) extend along fundus and tend to be longer and more tortuous. Isolated gastric varices occur in absence of oesophageal varices are classified into two types IGV1 which is located in the fundus and tend to be tortuous and complex and type two IGV2 which are located in body, antrum and around pylorus^[3]. In this research we will study of the impact of oesophageal variceal injection on gastric varices in cirrhotic patients.

METHODS

sive Care Unit of Internal Medicine Department, Zagazig University Hospitals. They were cirrhotics presented with upper gastrointestinal bleeding (hematemesis and or melena). All patients were subjected to clinical (general&local examination), laboratory tests(liver function tests-viral markers-kidney function tests), abdominal ultrasonography and upper endoscopy. All the above tests were also done after 6 months as a follow up examinations to discover the role of injection sclerotherapy of oesophageal varices in appearance of gastric varices. We divided patients into group 1 and group 2 according to presence or absence of gastric varices during follow up period.

RESULTS

There were 10 patients that gastric varices appeared after injection sclerotherapy of oesophageal varices (10%) (Table 1).Patients enrolled in study were 42 females and 58 males. Patients with gastric varices after injection sclerotherapy were 5 males and 5 females. Mean age of patients of group1 (gastric varices after injection sclerotherapy) was 54.58 years. Patient of group 2 (no gastric varices after injection therapy) were 53 males and 37 females and mean age was 54.58 years. There were no statistical significant differences between group 1and group 2 as regard age, sex and clinical data (P value >0.05). There were no statistical significant differences between group 1 and group 2 as regard liver function tests and viral markers (P value >0.05) (Table 2). As regard grades of OV they were: Grade I, II OV presented in 18 patients in patients of group 2. Grade I, III OV presented in 1 patient of group 2. Grade II OV presented in 27 patients of group 2 and in 4 patients of group1. Grade II, III OV presented in 39 patients of group 2, and in 5 patients of group 1. Grade III presented in 5 patients of group 2 and in 1 patient of group 1. There was no statistical significant difference between group 1 and group 2 patients as regard grades of OV (P value > 0.05) (Table3).

DISCUSSION

Hemorrhage from oesophageal or gastric varices is one of the main causes of death in patients with liver cirrhosis. The reported prevalence of esophageal varices in patients with cirrhosis ranges from 80% to 90%^[9], and 10-30% of patients with esophageal varices per year develop variceal hemorrhage^[4].

Endoscopic sclerotherapy is successful in controlling acute esophageal variceal bleeding in up to 90% of patients. In the United States, sodium tetradecyl sulfate or sodium morrhuate generally has been used as a sclerosant, while polidocanol or ethanolamine has been more popular in Europe. In Egypt we use ethanolamine oleate.

Table 1 Incidence of GV after injection sclerotherapy of patients with OV.

	No.	Percent
Studied cases	100	100%
Cases with gastric varices after sclerotherapy	10	10%

Table 2 Relation between group 1 and group 2 patients as regard laboratory data.

	Group 1		Group 2		t test	P
	Mean	SD	Mean	SD		
SGOT(iu/L)	30.20	9.82	43.51	25.03	1.660	0.100
SGPT(iu/L)	32.50	16.00	38.39	24.27	0.747	0.457
Albumin (g/dL)	2.620	0.485	2.740	0.544	0.669	0.505
Direct bilirubin	0.390	0.242	0.583	0.498	1.240	0.218
Total bilirubin (mg/dL)	0.990	0.498	1.076	0.542	0.477	0.634
PT(s)	14.230	2.055	14.794	3.444	0.507	0.613
PTT (s)	43.330	12.138	79.558	345.358	0.330	0.742
INR	1.380	0.244	1.423	0.255	0.507	0.610

Table 3 OV Grades during follow up.

OV grade	Total	Group 1		Group 2		P
		No	%	No	%	
Grade I and II	18	-	-	18	100	0.2
Grade I and III	1	-	-	1	100	1.0
Grade II	31	4	12.9	27	87.1	0.49
Grade II and III	49	5	11.9	39	88.6	0.79
Grade III	6	1	16.7	5	83.3	0.47

Complications of sclerotherapy may include mucosal ulceration, bleeding, oesophageal perforation, mediastinitis, and pulmonary complications. Long-term complications, such as oesophageal stricture formation, also may occur. Gastric varices are located deeper in the submucosa than oesophageal varices so sclerotherapy and ligation are usually ineffective in controlling acute bleeding from gastric varices and may be hazardous. N-butyl-2-cyanoacrylate (tissue glue) has been shown to be effective for bleeding gastric varices^[10]. Gastric variceal bleeding has the characteristics of more severe blood loss and higher mortality and is a more difficult management problem than oesophageal variceal hemorrhage. Therefore, it is important to identify patients with 'high risk' GV and to define successful approaches to prevent the first bleeding from large GV^[11].

In our study the prevalence of GV in patient with OV after injection sclerotherapy were 10%. The prevalence of GV in patients with portal hypertension varies from 18% to 70%^[12], although the incidence of bleeding from gastric varices is relatively low ranging from 10% to 36%^[13].

Sclerotherapy and band ligation have changed the outlook for patients with upper GI bleeding. Sclerotherapy was the initial available modality that led to marked reduction in immediate mortality of cirrhosis due to upper GI bleeding. It is now gradually being replaced by esophageal variceal band ligation which has shown better results in terms of variceal obliteration and fewer side effects like ulceration, perforation and strictures formation than sclerotherapy^[14].

From a clinical point of view, it is essential to recognize that only fundal varices that are located within the gastric wall (submucosal location) may bleed^[15]. In contrast, fundal varices that are located along the outside border of the gastric fundal wall (adventitial type) may not bleed. The diagnosis of fundal varices, in particular the submucosal fundal varices, is challenging, using endoscopy, submucosal fundal varices often appear as mass-like nodular and tortuous winding elevations of the mucosa of the fundus^[16]. But with increasing use of esophageal variceal band ligation and sclerotherapy for oesophageal varices, incidence of fundal varices had increased. Some studies concluded that sclerotherapy results in worsening of fundal varices^[17,18]. Others showed that band ligation of esophageal

varices was associated with more frequent development of fundal varices and worsening of portal hypertensive gastropathy compared to sclerotherapy in retrospective study^[19]. There are studies which show that sclerotherapy can lead to worsening of portal hypertensive gastropathy. One such study by Sarin *et al* found that over a 52 month follow up period, portal hypertensive gastropathy increased dramatically following sclerotherapy^[3]. Another study by Gupta *et al*^[20] showed a marked increase in portal hypertensive gastropathy during a two year follow up period after sclerotherapy. Portal hypertensive gastropathy was present in 33% of patients prior to sclerotherapy compared with 79% following sclerotherapy.

In our study the change in the degree of oesophageal varices during follow up is reported. Grade I, II presented in 18 patients without GV, grade I, III presented in 1 patient without GV, grade II presented in 27 patients without GV, and in 4 patients with GV, grade II, III presented in 39 patients without GV and in 5 patients with GV and grade III presented in 5 patients without GV and in 1 patient with GV. On the other hand other studies showed that Type 1 (GOV1) varices, which extend along the lesser curvature, are considered extensions of esophageal varices and should be managed similarly. Type 2 (GOV2) gastric varices extend along the fundus and tend to be longer and more tortuous. Isolated gastric varices (IGV) occur in the absence of esophageal varices and are also classified into 2 types. Type 1 (IGV1) are located in the fundus and tend to be tortuous and complex, and type 2 (IGV2) are located in the body, antrum, or around the pylorus. The presence of IGV1 fundal varices requires excluding the presence of splenic vein thrombosis^[3].

CONCLUSION

There is little relation between oesophageal variceal injection sclerotherapy and appearance of gastric varices. On the other hand we recommended other studies to deal with the impact of band ligation of esophageal varices on gastric varices.

CONFLICT OF INTERESTS

There are no conflicts of interest with regard to the present study.

REFERENCES

- 1 Groszmann RJ. Beta-adrenergic blockers and nitrovasodilators for the treatment of portal hypertension: the good, the bad, the ugly. *Gastroenterology* 1998; **113**: 1794-1797
- 2 D'Amico G, Luca A. Natural history. Clinical-haemodynamic correlations. Prediction of the risk of bleeding. *Baillieres Clin Gastroenterol* 1997; **11**: 243
- 3 Sarin SK, Lahoti D, Saxena SP, Murthy NS, Makwana UK. Prevalence, classification and natural history of gastric varices: a long term follow-up study in 568 portal hypertension patients. *HEPATOLOGY* 1992; **16**: 1343-9
- 4 Garcia-Tsao G, Sanyal AJ, Grace ND, Carey WD. Prevention and management of gastroesophageal varices and variceal hemorrhage in cirrhosis. *Am J Gastroenterol* 2007; **102**: 2086-2102
- 5 Akahoshi T Hashizume M, Shimabukuro R, Tanoue K, Tomikawa M, Okita K, Gotoh N, Konishi K, Tsutsumi N, Sugimachi K.

- Long-term results of endoscopic Histoacryl injection sclerotherapy for gastric variceal bleeding: a 10-year experience. *Surgery* 2002; **131**: S176-S181
- 6 Thakeb F, Salem SA, Abdallah M, el Batanouny M. Endoscopic diagnosis of gastric varices. *Endoscopy* 1994; **26**: 287-291
- 7 Tajiri T, Onda M, Yoshida H, Mamada Y, Taniyai N, Yamashita K. The natural history of gastric varices. *Hepatogastroenterology* 2002; **49**: 1180-1182
- 8 Hartigan PM, Gebhard RL, Gregory PB. Sclerotherapy for actively bleeding oesophageal varices in male alcoholics with cirrhosis. Veterans Affairs Cooperative Variceal Sclerotherapy Group. *Gastrointest Endosc* 1997; **46**: 1-7
- 9 D'Amico G, Pagliaro L, Bosch J. Pharmacological treatment of portal hypertension: an evidence-based approach. *Semin Liver Dis* 1999; **19**: 475-505
- 10 Huang YH, Yeh HZ, Chen GH, Chang CS, Wu CY, Poon SK, Lien HC, Yang SS. Endoscopic treatment of bleeding gastric varices by N-butyl-2-cyanoacrylate (Histoacryl) injection: longterm efficacy and safety. *Gastrointest Endosc* 2000; **52**: 160-167
- 11 Smruti Ranjan Mishra1, Barjesh Chander Sharma1, Ashish Kumar and Shiv Kumar Sarin1, Primary prophylaxis of gastric variceal bleeding comparing cyanoacrylate injection and beta-blockers: A randomized controlled trial. *Journal of Hepatology* 2011 vol. 54 j 1161-1167
- 12 Watanabe K, Kimura K, Matsutani S, Ohto M, Okuda K. Portal hemodynamics in patients with gastric varices. A study in 230 patients with esophageal and/or gastric varices using portal vein catheterization. *Gastroenterology* 1988; **95**: 434-440.
- 13 Sarin SK and Kumar A. Gastric varices: profile, classification, and management. *Am J Gastroenterol* 1989; **84**: 1244-1249.
- 14 Burak KW, Lee SS, Beck PL. Portal hypertensive gastropathy and gastric antral vascular ectasia (GAVE) syndrome. *Gut* 2001; **49**: 866-872
- 15 Sanyal AJ. The value of EUS in the management of portal hypertension. *Gastrointest. Endosc* 2000; **52**: 575-577
- 16 Boustière C, Dumas O, Jouffre C, Letard JC, Patouillard B, Etaix JP, Barthélémy C, Audigier JC. Endoscopic ultrasonography classification of gastric varices in patients with cirrhosis. Comparison with endoscopic findings. *J Hepatol* 1993; **19**: 268-272
- 17 De la Pena J, Rivero M, Sanchez E. Variceal ligation compared with endoscopic sclerotherapy for variceal hemorrhage: prospective randomized trial. *Gastrointest Endosc* 1999; **49**: 417-423
- 18 De BK, Ghoshal UC, Das AS, Nandi S, Mazumder DN. Portal hypertensive gastropathy and gastric varices before esophageal variceal sclerotherapy and after obliteration. *Indian J Gastroenterol* 1998; **17**(1): 10-12
- 19 Shahid Sarwar, Anwaar A Khan, Altaf Alam, Arshad Kamal Butt, Farzana Shafqat, Kashif Malik, Irfan Ahmad, Aamir Khan Niazi effect of band ligation on portal hypertensive gastropathy and development of fundal varices. *J Ayub Med Coll Abbottabad* 2006; **18**(1)
- 20 Gupta TK, Chen L, Groszmann RJ. Pathophysiology of portal hypertension. *Clin Liver Dis.* 1997; **1**: 1-12

Peer reviewer: Romani MP Romani, Department of Physiology and Biophysics, Case Western Reserve University, 10900 Euclid Avenue, Cleveland, OH 44106-4970, the United States.