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Evaluation of Esophageal Varices Band Ligation Effects on Rectal Varices and Hemorrhoids Development and Grading in Post-HCV Cirrhosis

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Abstract:

Background: Rectal varices and hemorrhoids are a gastrointestinal complication of portal hypertension. There is a large discrepancy in previous studies regarding prevalence of rectal varices and hemorrhoids and their correlation with other factors. **Aim of the work:** goal of our study was evaluation of the effect of band ligation of oesophageal varices (OVs) on the evolution and/or progression of rectal varices and haemorrhoids. **Patients and Methods:** This study conducted on 50 post-HCV cirrhotic patients screened for esophageal varices. They were divided into two main groups; Group I: It included 25 post-HCV cirrhotic patients, screened for OVs, band ligation was indicated in them, and they were banded for OVs. Group II: It included 25 post-HCV cirrhotic patients, screened for OVs, and band ligation was not indicated in them. All of them were submitted to oesophagogastrodudenoscopy (OGD) for assessment of OVs and short colonoscopy for assessment of rectal varices & haemorrhoids at the baseline and after 6 months. **Results**: Obliteration of esophageal varices by endoscopic variceal band ligation did not affect the incidence of hemorrhoids (38% before and after) and anorectal varices (12% before and after). **Conclusion**: It is concluded that esophageal variceal band ligation does not affect the incidence of hemorrhoids, or anorectal va1rices inpatients with liver cirrhosis.

Keywords: Liver cirrhosis, Portal hypertension, Oesophageal varices and Rectal varices.

Introduction

Cirrhosis is a chronic condition with a high mortality. It constitutes the fifth-leading cause of adult deaths and ranks eighth in economic cost among the major illnesses [(1)]. Cirrhosis is defined anatomically as a diffuse process with fibrosis and nodule formation. It is the end result of the fibrogenesis that occurs with chronic liver injury. Although the causes are many, without successful treatment or removal of the agent responsible, the end result of fibrogenesis is the same [(2).] Portal hypertension (PH) -defined as the elevation of hepatic venous pressure gradient (HVPG) above 5mmHg- is the initial and main consequence of cirrhosis and is responsible for the majority of its complications including variceal bleeding, spontaneous bacterial peritonitis (SBP) and hepatorenal syndrome (HRS), which represent the leading causes of death and of liver transplantation in patients with cirrhosis [(3)]. Esophageal varices is related to portal hypertension which commonly accompanies the presence of liver cirrhosis, with a prevalence that can range from 40 to 80% in patients with cirrhosis ***[(4)]*****.** The yearly rate of development of ‘‘new” varices is about 5–10% per year in patients with cirrhosis, and the progression from small to large varices occur in 10% to 20% of cases after 1 year. In the 2 years following the first detection of oesophageal varices, the risk of variceal bleeding ranges between 20% to 30% and results in 25% to 50% mortality within a week of the first bleeding episode***[(4)]*****.**The current recommendations are that all cirrhotic patients at the time of diagnosis should be screened with upper endoscopy to look for gastroesophageal varices and to eradicate them endoscopically by rubber band ligation or injection sclerotherapy. Follow-up endoscopy should be performed at 2-3 years intervals in compensated patients with no varices, and at 1-2 years intervals in compensated patients with small varices ***[(5)]*****.** The prevalence of colonic varices and rectal varices has been found to be 34-46%, and 10-20%, respectively, in patients with cirrhosis undergoing colonoscopy [(6).] Rectal varices are described and differentiated from hemorrhoids, as rectal varices extend more than 4 cm above the anal verge, are dark blue in color, collapse with digital pressure, and do not prolapse into the proctoscope on examination, whereas

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hemorrhoids do not extend proximal to the dentate line, are purple in color, do not collapse with digital pressure, and often prolapse into the proctoscope. They also classified rectal varices based on endoscopic grading (grade 1, <3 mm; grade 2, 3-6 mm; grade 3, >6 mm).

Patients and Methods

This is a prospective case-control study included a total number of 50 post-HCV cirrhotic patients screened for OVs at the Endoscopic Units of Al-Hussein and Bab El-Shariah University Hospitals at the period between October 2017, and June 2018. Approval of the ethical committee and a written informed consent from all the subjects were obtained. Patients were divided into two main groups:

Group I: It included 25 post-HCV cirrhotic patients, screened for OVs, band ``ligation was indicated in them, and they were banded for OVs.

Group II: It included 25 post-HCV cirrhotic patients, screened for OVs, and band ligation was not indicated in them.

Inclusion criteria: Patients with post-HCV liver cirrhosis, portal hypertension and OVs. None of the study patients received or had been receiving beta-blockers and / or ARBs. All study subjects were normotensive.

Exclusion criteria: Patients who are co-infected with HBV, portal vein thrombosis, hepatocellular carcinoma, other co-morbidity that might affect portal pressure (e.g. co-existing cardiac disease, and co-existing chronic obstructive airway disease), patients with history of treated OVs; either by band ligation or sclerotherapy, patients requiring sclerotherapy for gastric varices, patients in whom porto-systemic shunt surgery had been done, patients with history of treated haemorrhoids and /or rectal varices; either by band ligation or surgical intervention, patients with prior total colectomy, patients who refused to participate in the study and dropped out patients.

All patients were subjected: -at the beginning of the study- to complete history taking, full clinical examination; laboratory tests which included HCV IgG antibodies (HCV Ab), Hepatitis B surface antigen (HBsAg), complete blood picture (CBC), alanine aminotransferase (ALT), aspartate aminotransferase (AST), serum albumin, total & direct bilirubin, prothrombin time and INR, serum creatinine , alpha feto protein (AFP) and abdominal ultrasonographic examination. Also all of them were submitted to OGD for assessment of OVs and short colonoscopy for assessment of rectal varices & haemorrhoids at the baseline and after 6 months.

Statistical analysis:

Data were analyzed using Statistical Program for Social Science (SPSS) version 15.0. Quantitative data were expressed as mean± standard deviation (SD). Qualitative data were expressed as frequency and percentage.

The following tests were done:

1. Independent-samples t-test of significance: was used when comparing between two means.
2. Chi-square test: was used when comparing between non-parametric data.
3. Pearson's correlation coefficient (r) test: was used for correlating data.
4. Linear regression: It is used to test and estimate the dependence of a quantitative variable based on its relationship to one or more independent variable.
5. Probability (P-value) :
    - P-value < 0.05 was considered significant.
    - P-value < 0.001 was considered as highly significant.
    - P-value > 0.05 was considered insignificant.

Results

All patients submitted to OGD and short colonoscopy at the baseline and after 6 months and the following results were obtained.

Table (1): Comparison between studied groups as regard endoscopic findings at the baseline.

Variables	Group	Group I (N = 25)	Group II (N = 25)	ANOVA p-value
OVS	**No OVS**	0 (0%)	0 (0%)	**< 0.001***
	OVS grade I	0 (0%)	11 (44%)	
	OVS grade II	2 (8%)	12 (48%)	
	OVS grade III	15 (60%)	2 (8%)	
	OVS grade IV	8 (32%)	0 (0%)	
PHG	**No PHG**	5 (20%)	8 (32%)	**0.02****
	Mild PHG	9 (36%)	13 (52%)	
	Severe PHG	6(24%)	4 (16%)	
RVS	**No RVS**	20 (80%)	24 (96%)	0.2
	RVS grade I	1 (4%)	1 (4%)	
	RVS grade II	3 (12%)	0 (0%)	
	RVS grade III	1 (4%)	0 (0%)	
Piles	**No piles**	15 (60%)	16 (64%)	0.9
	Internal piles grade I	1 (4%)	1 (4%)	
	Internal piles grade II	7 (28%)	5 (20%)	
	Internal piles grade III	2 (8%)	3 (12%)	
	Internal piles grade IV	0 (0%)	0 (0%)	

***: p-value < 0.001 is considered highly significant.**
***: p-value < 0.05 is considered significant.**

This table shows highly statistical significant difference **(p-value < 0.001)** between group I & group II as regard OVS, statistically significant difference **(p-value < 0.05)** as regard PHG and no statistically significant difference **(p-value > 0.05)** as regard RVs & hemorrhoids at baseline.

Table (2): Comparison between studied groups as regard endoscopic findings at 6 months.

Variables	Group	Group I (N = 25)	Group II (N = 25)	ANOVa p-value
OVS	**No OVS**	4 (16%)	0 (0%)	0.1
	OVS grade I	7 (28%)	15 (60%)	
	OVS grade II	12 (48%)	9 (36%)	
	OVS grade III	2 (8%)	1 (4%)	
	OVS grade IV	0 (0%)	0 (0%)	
PHG	**No PHG**	2 (8%)	11 (44%)	**< 0.001***
	Mild PHG	10 (40%)	12 (48%)	
	Severe PHG	13 (56%)	2 (8%)	
RVS	**No RVS**	20 (80%)	24 (96%)	0.2
	RVS grade I	1 (4%)	1 (4%)	
	RVS grade II	3 (12%)	0 (0%)	
	RVS grade III	1 (4%)	0 (0%)	
Piles	**No piles**	15 (60%)	16 (64%)	0.9
	Internal piles grade I	1 (4%)	1 (4%)	
	Internal piles grade II	7 (28%)	5 (20%)	
	Internal piles grade III	2 (8%)	3 (12%)	
	Internal piles grade IV	0 (0%)	0 (0%)	

***: p-value < 0.001 is considered highly significant.**

This table shows highly statistical significant difference **(p-value < 0.001)** between group I & group II as regard PHG and no statistically significant difference **(p-value > 0.05)** as regard OVs, RVs & hemorrhoids at 6 months.

Table (3): Correlation between RVs and OVs (base line)

Variables / Baseline		RVs (N=6)	No RVs (N=44)	p-value
OVs	0	0 (0%)	0 (0%)	0.4
	I	0 (0%)	11 (25%)	
	II	1 (16.7%)	13 (29.5%)	
	III	3 (50%)	14 (31.8%)	
	IV	2 (33.3%)	6 (13.6%)	

***: p-value > 0.05 is considered insignificant.**

This table shows no statistical significant difference **(p-value > 0.05)** between RVs and No RVs groups as regard OVs (at base line).

Table (4): Correlation between RVs and OVs (6 months).

Variables / 6 months		RVs (N=6)	No RVs (N=44)	p-value
OVs	0	2 (33.3%)	1 (2.3%)	0.02*
	I	2 (33.3%)	19 (43.2%)	
	II	1 (16.7%)	21 (47.7%)	
	III	1 (16.7%)	2 (4.5%)	
	IV	0 (0%)	0 (0%)	

***: p-value < 0.05 is considered significant.**

This table shows statistically significant difference **(p-value < 0.05)** between RVs and No RVs groups as regard OVs (6 months).

Table (5): Correlation between RVs and PHG (base line)

Variables / Base line		RVs (N=6)	No RVs (N=44)	p-value
PHG	No	1 (16.7%)	7 (15.9%)	0.9
	Mild	3 (50%)	24 (54.6%)	
	Severe	2 (33.3%)	13 (29.5%)	

***: p-value > 0.05 is considered insignificant.**

This table shows no statistical significant difference **(p-value > 0.05)** between RVs and No RVs groups as regard PHG (at base line).

Table (6): Correlation between RVs and PHG (6 months)

Variables / 6 months		RVs (N=6)	No RVs (N=44)	p-value
PHG	No	0 (0%)	9 (20.5%)	0.5
	Mild	2 (33.3%)	20 (45.5%)	
	Sever	4 (66.7%)	15 (34%)	

***: p-value > 0.05 is considered insignificant.**

This table shows no statistical significant difference **(p-value > 0.05)** between RVs and No RVs groups as regard PHG (at 6 months).

Discussion

Cirrhosis is a chronic condition with a high mortality. It constitutes the fifth-leading cause of adult deaths and ranks eighth in economic cost among the major illnesses [(1)]. Portal hypertension (PH) -defined as the elevation of hepatic venous pressure gradient (HVPG) above 5mmHg- is the initial and main consequence of cirrhosis and is responsible for

the majority of its complications including variceal bleeding, spontaneous bacterial peritonitis (SBP) and hepatorenal syndrome (HRS), which represent the leading causes of death and of liver transplantation in patients with cirrhosis [3].

The following characteristics were found in the studied groups:

- **Liver function tests;** revealed that hyperbilirubinemia, hypoalbuminemia and low prothrombin activity were more common in group I, giving the assumption that the degree of OVs are associated with more severe liver affection, which was in agreement with [8,9], who report that presence of such findings could be related to the disturbed liver function in patient with advanced cirrhosis.

- **Regarding platelet count;** there was a highly statistical significant difference **(p-value < 0.001)** between the two groups (65.2 +15.9 in group I & 101.6 + 17.4 in group II), that was in agreement with **[10]** who states that a low platelet count has been constantly found to be related to the degree of OVs.
- **Regarding hemoglobin level and pallor**, there was a statistically significant difference between the two studied groups (40% vs 24% for pallor, and 8.2 + 0.7 vs 9.7 + 0.4 for hemoglobin) that can be explained by the more severe hepatic decompensation - as a cause of coagulopathy- and the prescense of significant thrombocytopenia in group I which considerd as a risk factor for bleeding tendency (proved by the significant difference between INR in the two groups; 1.7 + 0.5 vs 1.3 + 0.2) which participate in presence of anemia. This was in agreement with **[11]**.

- **As regard Child score of the studied groups** which reflect the degree of hepatic decompensation, we found a positive correlation between the degree of OVs and Child score of the patient, this was in agreement with **[10,12]** who reported that patients with Child class B and C had higher incidence of esophageal varices compared to Child A patients (39.8%,43.4%,16.8%) respectively with p-value=0.004. Most of our patients had Child-Pugh score (B and C) and this goes with **[13,14]** who report that OVs are frequently observed in patients with more severe liver disease. Other as **[15]** had controversy results as they reported that there is no relationship was found between hepatic function, as assessed by Child-Pugh score and OVs.

- **Regarding U/S findings;** there was a statistically significant difference as regard spleen size (mild splenomegaly in 16% vs 40%, moderate splenomegaly in 60% vs 32% & huge splenomegaly in 20% vs 16%), this was in agreement with **[16].** Also there was a positive correlation had been found between portal pressure, portal vein diameter and degree of OVs (where portal vein dilatation was 92% vs 80%) that was in agreement with**[17]** who reported that a direct relationship between portal vein diameter and degree of OVs.

- **At our initial upper GI endoscopy**; The prevalence and relationship of various endoscopic features at enrollment were shown in Table ([1]), majority of patients had OVs grade III & IV in group I, and OVs grade I & II in group II, portal hypertensive gastropathy was seen in twenty (80%) of patients in group I, and seventeen (68%) of patient in group II. It was mild in most of cases. This means a positive correlation between degree of OVs& PHG and Child score of the patient.

- **At initial short colonoscopy;** Rectal varices were present in six of (12%) patients, three of them in group I and the other three in group II, while hemorrhoids were noted in nineteen (38%) of patients, which is the same prevalence in non hepatic population.

Ten of them were in group I & nine in group II, indicating that there was no relationship between the presence or absence of rectal varices or hemorrhoids and the Child-Pugh score, cirrhosis grade, OVs or portal hypertensive gastropathy. These findings of our initial upper GI endocopy& short colonoscopy were in agreement with **([18])** who studied 60 patients and reported that portal hypertensive gastropathy was seen in 48(80%) of patients and it was mild in all cases. Hemorrhoids were noted in 22 (38%) of patients. There was no relationship between the presence or absence of hemorrhoids and the Child-Pugh cirrhosis grade, PHG, gastric varices, anorectal varices or portal hypertensive colopathy. Anorectal varices were present in seven (12%) of patients.

- **After 6 months of EVL upper GI endoscopy** revealed that OVs are obliterated in 12% of patients in group I, where median number of sessions for obliteration of OVs is 3 (range 2-5), While in group II no OVs have been obliterated, with majority of patients had OVs grade I & II in both groups . As regard PHG there was increase in frequency (80% vs 92%) & severity (24% severe PHG vs 48%) in group I after band ligation**,** in contrary to group II where mild improvement was seen in frequency & severity, that was in agreement with **([18]).**

- **After 6 months of EVL short colonoscopy**; revealed no increase in incidence of rectal varices and hemorrhoids, that was in agreement with **([18]).**

Conclusion: It is concluded that oesophageal variceal band ligation does not affect the incidence of hemorrhoids, or anorectal varices in patients with liver cirrhosis and portal hypertension.

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