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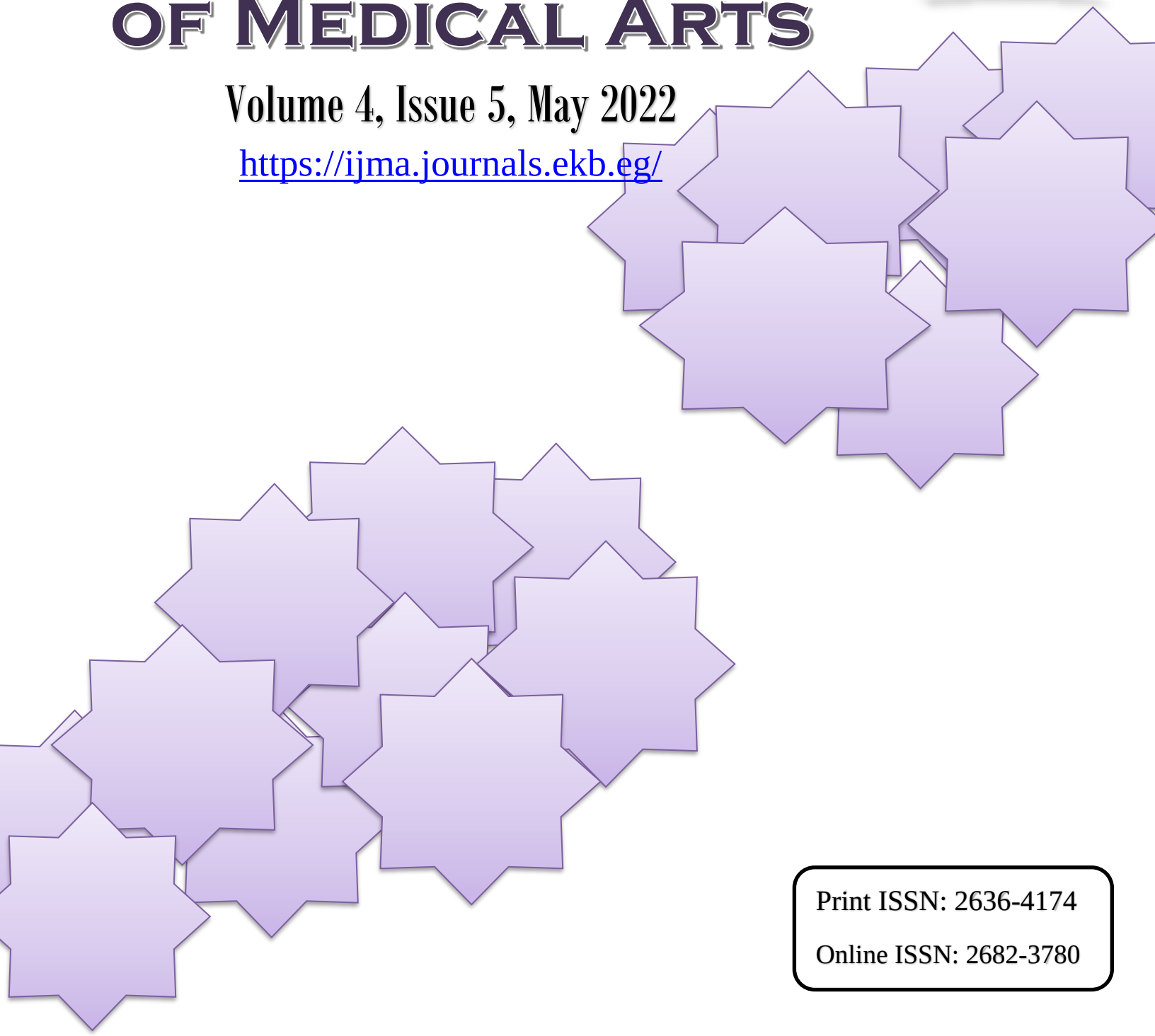

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Original Article

Correlation of Upper and Lower Endoscopic Changes in Patients with Liver Cirrhosis and Portal Hypertension

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ABST RACT

Background: It is a great challenge and complex process to manage complications in liver disease. Endoscopy was introduced and gained wide acceptance in the management of patients with liver disease.

The aim of the work: The study aimed to assess the prevalence and clinical value of various forms of colonic mucosal changes in patients with liver cirrhosis and portal hypertension [PHT]. In addition to correlate these changes with oesophageal varices [OV] and portal hypertensive gastropathy [PHG].

Subjects and Methods: This prospective observational study included 50 adult patients with established diagnosis of liver cirrhosis and PHT. They were evaluated clinically, and then submitted to abdominal ultrasound, upper and lower gastrointestinal endoscopy.

Results: Esophageal varices and portal hypertension gastropathy, significantly associated with colonic hyperemia [colonic hyperemia was reported among 57.5% of esophageal varices compared to 20.0% of cases without varices; and reported with 60.0% of patients with compared to 26.7% of those without PHG].

Conclusion: The prevalence of hyperemia [colopathy] is associated with esophageal varices and portal hypertensive gastropathy.

Keywords: Liver Cirrhosis; Esophagogastroduodenoscopy; Colonoscopy; Portal Hypertension: Colopathy.

INTRODUCTION

Liver cirrhosis is defined as a development of regenerative nodules [recognized in histological examination]. These nodules are surrounded by fibrous bands. The condition is developed in response to chronic injury of the liver with consequent portal hypertension and end-stage liver disease [1]. Liver cirrhosis is the commonest cause of portal hypertension, while alcoholism and viral hepatitis – B and C are the commonest causes of cirrhosis worldwide [2].

Portal hypertension is a devastating complication of decompensating liver cirrhosis and it is responsible for the formation of ascites and development of portosystemic collateral veins. Esophageal varices are the most dangerous collaterals as it may be ruptured leading to hemorrhage which may be fatal [3].

In patients with cirrhosis, gastrointestinal bleeding is responsible for 25 % of overall mortalities [4]. Portal hypertensive gastropathy [PHG] is the commonest form of mucosal damage in patients with cirrhosis. The definite PHG pathogenesis is unclear. However, local or general changes in vascular hemodynamics may play a role [5]. PHG is characterized by typical lesions of the gastric mucosa, mainly in the fundus and upper body of the stomach. However, it could be recognized at any part of the stomach and even other parts of the GIT [e.g., small intestine, or the colon] [6].

Portal hypertension [PH] lead to hemodynamic and mucosal alterations in the entire upper GI tract as well lower GI tract. The PH related changes in the colon described as portal hypertensive colopathy [PHC], colonic varices, rectal varices, vascular ectasia in colon & rectum and hemorrhoids [7]. PHC is one of the commonest complications of chronic liver disease. Clinically, it could be manifested as lower GIT bleeding or unidentified chronic anemia in patients with severe PH [8].

The features of PHC include different vascular lesions [e.g, telangiectasia, cherry red spots and angiodysplasia- like lesions], colitis- like lesions [granularity, erythema, edema, friability] [9].

The PHC prevalence in patients with cirrhosis varies widely from 25% to 70%. Rectal or colonic varices are also varying widely, from 4% to 40% of patients. It is evident that colonoscopy is very important investigation for defining lower GI features of portal hypertension [10].

THE AIM OF THE WORK

The current work aims to estimate the prevalence and clinical features of different types of colonic mucosal alterations in patients with liver cirrhosis and portal hypertension [PH] and examine the possible correlation with esophageal varices and PHG.

PATIENTS AND METHODS

This prospective observational study included 50 adult patients with established diagnosis of liver cirrhosis and PHT, who attended Al-Azhar University Hospital [Damietta] for scheduled follow up. We excluded clinically unstable patient such as on vaso-pressure support, on ventilator support or in hepatic encephalopathy. In addition, patients with inadequate bowel preparation, and/or recent myocardial infarction and major co-morbidities, were excluded from the study.

After giving consent for participation, all patients were submitted to detailed history, clinical evaluation, laboratory investigations [complete blood count, liver & kidney function tests, co-agulation profile, and alfa-fetoprotein]. Then, abdominal ultrasonography [US] was performed by Toshiba Ultrasound [the Aplio 500 Toshiba, Japan], to estimate the liver and spleen size, detect collaterals, ascites, focal lesions, or signs indicative of cirrhosis [coarse echopattern, surface nodularity, and caudate lobe hypertrophy], and to assess the collaterals, and portal vein shape and diameter [11].

Upper endoscopy: Varices were assessed and graded 1 to 4 [grade 1, mucosal varices; grade 2, varices < 5 mm and fulfilling < $^1/_3$ of the esophageal lumen; grade 3, varices > 5mm and fulfilling > $^1/_3$ of the esophageal lumen; grade 4, varices occupying > 2/3 of the esophageal lumen [12]. In addition, recognition of PHG was done based on the two-category classification system, by Baveno III consensus. This classification assigned mild PHG for a snakeskin mosaic pattern changes, and severe when in addition to the mosaic pattern, there is flat or bulging red or black-brown spots are seen, and/or when there is active hemorrhage [13].

Colonoscopy: The colonoscopy was performed after standard bowel preparation by administration of 4 L of polyethylene glycol. The colon was investigated after washing of any obscured mucosa. PHC was graded according to the classification of Bini into three grades: grade 1, erythema of mucosa; grade 2, erythema of mucosa with a mosaic-like pattern; and grade 3, vascular lesions in the colon [e.g., cherry red spots, telangiectasias, or angio-dysplasia-like lesions] [14].

Statistical analysis: The collected data were, tabulated, and statistically analyzed using SPSS program [Statistical Package for Social Sciences] software version 26.0, Microsoft Excel 2016 and MedCalC program software version 19.1. Descriptive statistics were calculated for parametric data, and presented as mean and standard deviation, minimum & maximum and median was calculated for non-parametric data. The categorical data were presented as frequency and percentages. P value < 0.05 was considered significant.

RESULTS

The age of studied patients ranged between 55 and 77 years [68.50±6.61 years]. They were 36 males and 14 females. Their hemoglobin level ranged between 8.90 and 10 g/dl [9.78±0.20 g/dl]. The white blood cell count ranged between 3700 to 1000 cell/cc, while platelet count ranged between 53000 to 122000/cc. Their INR ranged between 1 and 1.4 [1.14±0.07], PT ranged between 1.80 and 20.0 seconds [6.92±3.16]. ALT ranged between 13 and 41 U/L, while AST ranged between 18 and 63 U/L. Serum albumin ranged between 2.5 to 3.80 g/dl [3.18±0.21 g/dl]. Bilirubin ranged between 0.7 to 3.0 mg/dl and AFP ranged between 2.20 to 1200 ng/ml [median, IQR 7.15 [5.0-8.5]. The mean values of s. creatinine and blood urea was 1.02± 0.14 mg/dL and 29.89± 3.44 mg/dL respectively. Ultrasonographic data of the studied patients revealed that, 17 [34%] patients had mild ascites, 14 [28%] patients had moderate ascites and 13 [26%] patients had marked ascites. Regarding liver size, the majority of patients [94%] had shrunken liver. All patients had coarse liver echo pattern and attenuated hepatic vein. The mean spleen size was 19.17± 1.62 cm with 46 [92%] had splenomegaly. The mean PV was 12.37± 0.61.

Table [1] shows the results of upper endoscopy. Briefly, 40 [80%] patients had esophageal varices with 15 [30%] patients had grade I, 7 [14%] patients had grade II and 3 [6%] patients had grade IV. 35 [70%] patients had portal. hypertension gastro-pathy. One [2%] patient had gastric antral vascular ectasia and 3 [6%] patients had duodenitis. Lower endoscopic findings of the studied patients are shown in table 7. Two [4%] patients had internal piles. 28 [56%] patients had hemorrhoids. Half [50%] patient had hyperemia, 18 [36%] patients had angiodysplasia, 7 [14%] patients had rectal varices and one [2%] patient had telangectasia [Table 2].

In the current work, there was a statistically significant relation between esophageal varices with hyperemia as 23 [57.5%] patients with esophageal varices had hyperemia. In addition, there was statistically significant relation between PHG with hyperemia [p= 0.031] as 21 [60%] patients with portal hypertension gastropathy had hyperemia and 14 [40.0%] portal hypertension gastropathy patients with no hyperemia. However, there was no statistically significant relation between gastric varices with any of the lower endoscopic findings [Table 3]. There was no statistically significant relation between gastritis, GAVE, or duodenitis with any of the lower endoscopic findings [Table 4].

Table [1]: Distribution of the studied patients regarding upper endoscopic findings

Parameters		Studied patients [n=50]	
		N	%
Esophageal varices		40	80.0%
Grades of esophageal varices	I	15	30.0%
	I-II	6	12.0%
	II	7	14.0%
	III	2	4.0%
	IV	3	6.0%
Portal hypertensive gastropathy [PHG]		35	70.0%
Gastric varices		4	8.0%
Gastritis		1	2.0%
Gastric antral vascular ectasia [GAVE]		1	2.0%
Duodenitis		3	6.0%

Table [2]: Distribution of the studied patients regarding lower endoscopic findings

Parameters	Studied patients [n=50]	
	n	%
Normal	9	18.0%
Internal Piles	2	4.0%
Hemorrhoids	28	56.0%
Hyperemia	25	50.0%
Angiodysplasia	18	36.0%
Rectal varices	7	14.0%
Telangectasia	1	2.0%

Table [3]: Relation between esophageal varices, portal hypertensive gastropathy and gastric varices with lower endoscopic parameters

Variables	No esophageal Varices [n=10]		Esophageal variance [n=40]		Test	p
	n.	%	n.	%		
Internal piles	1	10.0%	1	2.5%	1.172	0.363
Hemorrhoids	3	30.0%	25	62.5%	3.43	0.084
Hyperemia	2	20.0%	23	57.5%	4.50	**0.034***
Angiodysplasia	4	40.0%	14	35.0%	0.087	0.768
Rectal varices	2	20.0%	5	12.5%	0.374	0.541
Telangectasia	0	0.0%	1	2.5%	0.225	1.00
	No PHG [n=15]		**PHG [n- 35]**			
	n.	%	n.	%		
Internal piles	1	6.7%	1	2.9%	0.397	0.529
Hemorrhoids	6	40.0%	22	62.9%	2.23	0.136
Hyperemia	4	26.7%	21	60.0%	**4.67**	**0.031***
Angiodysplasia	5	33.3%	13	37.1%	0.066	0.797
Rectal varices	2	13.3%	5	14.3%	0.008	1.00
Telangectasia	0	0.0%	1	2.9%	0.437	1.00
	No gastric varices [n=46]		**Gastric varices [n=4]**			
	n.	%	n.	%		
Internal piles	2	4.3%	0	0.0%	0.181	1.00
Hemorrhoids	25	54.3%	3	75.0%	0.637	0.627
Hyperemia	21	45.7%	4	100.0%	4.35	0.11
Angiodysplasia	16	34.8%	2	50.0%	0.370	0.612
Rectal varices	6	13.0%	1	25.0%	0.437	0.464
Telangectasia	1	2.2%	0	0.0%	0.089	1.00

PHG: portal hypertensive gastropathy

Table [4]: Relation between gastritis, with lower endoscopic parameters

Variables	No gastritis [n=49]		Gastritis [n=1]		Test	p
	n.	%	n.	%		
Internal piles	2	4.1%	0	0.0%	0.043	1.00
Hemorrhoids	27	55.1%	1	100.0%	0.802	1.0
Hyperemia	24	49.0%	1	100.0%	1.02	1.0
Angiodysplasia	17	34.7%	1	100.0%	1.81	0.63
Rectal varices	6	12.2%	1	100.0%	6.27	0.14
Telangectasia	1	2.0%	0	0.0%	0.021	1.0
	No GAVE [n=49]		**GAVE [n=1]**			
	n.	%	n.	%		
Internal piles	2	4.1%	0	0.0%	0.043	1.0
Hemorrhoids	27	55.1%	1	100.0%	0.802	1.0
Hyperemia	25	51.0%	0	0.0%	1.02	1.0
Angiodysplasia	18	36.7%	0	0.0%	0.574	1.0
Rectal varices	7	14.3%	0	0.0%	1.66	1.0
Telangectasia	1	2.0%	0	0.0%	0.021	1.0
	No duodenitis [n = 47]		**Duodenitis [n = 3]**			
	n.	%	n.	%		
Internal piles	1	2.1%	1	33.3%	7.15	0.118
Hemorrhoids	26	55.3%	2	66.7%	0.147	1.0
Hyperemia	24	51.1%	1	33.3%	0.355	1.00
Angiodysplasia	16	34.0%	2	66.7%	1.30	0.291
Rectal varices	7	14.9%	0	0.0%	0.520	1.0
Telangectasia	1	2.1%	0	0.0%	0.065	1.0

GAVE: gastric antral vascular ectasia

DISCUSSION

The main aim of the current study was to evaluate the prevalence and clinical significance of various forms of colonic mucosal changes in patients with liver cirrhosis and PH and correlating them with esophageal varices [OV] and PHG. This included 50 adult patients with established diagnosis of liver cirrhosis and PH attending to Al-Azhar university hospital, Damietta, for scheduled follow up.

In the current study, the age ranged from 55 to 77 years with a mean age ±SD was 68.50± 6.61 years, with male sex predominance [72% were males]. Shalaby *et al*. [15] studied portal hypertensive colopathy in Egyptian cirrhotic patients. They performed a cross-sectional study with the inclusion of 60 patients with liver cirrhosis. They revealed that; males constituted 75% of them. The ages of the enrolled patients ranged from 30 to 69 years with a mean age of 52.3±6.1 years. Also, Abd-Elsalam *et al*. [16] studied the correlation of platelets count with endoscopic findings in a cohort of Egyptian patients with liver cirrhosis they included a total of 110 cirrhotic patients were enrolled in the study with a mean age of 54.39±7.46 years; 73 [66.36%] of them were men.

Indicators of PH included clinical and Ultrasonographic manifestations [e.g., splenomegaly, vascular collaterals, ascites, and variceal bleeding]; and upper gastrointestinal endoscopic criteria [esophageal varices and portal hypertensive gastropathy]; as well as, low platelet counts [9].

The laboratory data of the current work are in line with previous literature. The mean hemoglobin level was 9.78± 0.20 g/dl. The mean WBCs count was 5.65± 1.35x10^9/L. The mean platelets count was 78.34± 12.09x10^9/L. Shalaby *et al.* [15] reported higher mean hemoglobin [11.57±1.86 g/dl], and mean platelet count [87.68±42.71x10^9/L]. The cause of the difference may be due to the variation of the studied age groups, and different sample size.

Regarding coagulation, liver and kidney function results, Shalaby *et al*. [15] reported comparable results with our results as they reported mean values of INR [1.47±0.44], ALT [43.88± 18.58], AST [48.13±24.09], albumin [g/dl] [2.76± 0.33] and Bilirubin [2±0.89] mg/dl, as well the mean value of creatinine [0.9±0.37] mg/dl.

The study by Abd-Elsalam *et al*. [16] found that the means albumin level was 3.16±0.63, mean bilirubin level was 2.18±2.1, and the mean INR was 1.42±0.43, these results were comparable to our findings.

In agreement with our results regarding ultrasound findings, the study by Salama *et al*. [17] revealed that [31.4%] patients had mild ascites, [28.6%] patients had moderate ascites and [25.7%] patients had marked ascites. The majority of patients [88.6%] had shrunken liver. All patients had coarse liver echo-pattern and attenuated hepatic vein. The mean spleen size was 16.58±2.75 cm with [94.28%] had splenomegaly. The mean PV was 12.26±2.75mm. Shalaby *et al*. [15] revealed that [28.3%] of patients had mild ascites, [35%] of patients had moderate ascites, [15%] of patients had marked ascites and [21.6%] had absent ascites. All patients had coarse liver echo pattern and attenuated hepatic vein. The mean spleen size was 15.90±2.38 cm. The mean PV was 12.50±2.47mm.

Upper endoscopy is the gold standard for diagnosis and grading of esophageal varices, a dangerous complication of PH. The mortality rate after acute variceal bleeding has been reduced with introduction of new treatment modalities. However, it remains high [18]. Varices are graded by their size by the American Association for the Study of Liver Disease [AASLD] into small or large varices, with 5 mm diameter as the cutoff [19]. The Japanese Research Society developed an earlier grading system for PH and varices and graded varices into small, medium and large. However, the description does not include a cutoff [20]. Another study found no difference in observer agreement between the 2-grade and 3-grade systems [21].

Regarding the upper endoscopic data, 80% had esophageal varices [30% grade I, 14% grade II and 6% grade IV]. 70% of patients had portal hypertension gastropathy. One [2%] patient had gastric antral vascular ectasia and 3 [6%] patients had duodenitis. Salama *et al*. [17] revealed that 82.9% of patients had esophageal varices with 31.4% grade I, 17.1% grade II, 14.3% grade III and 8.6% had grade IV; and 11.4% had portal hypertension gastropathy. One [2%] patient had gastric antral vascular ectasia and 3 [5.7%] patients had duodenitis.

The results of lower endoscopy showed that 2 [4%] patients had internal piles, 28 [56%] patients had hemorrhoids. Half of our patients had hyperemia, 18 [36%] patients had angiodysplasia, 7 [14%] patients had rectal varices and one [2%] patient had telangiectasia. In line with our results the study by Salama *et al*. [17] revealed that [51.4%] patients had multiple lesions, [57.1%] of patients

had hemorrhoids, [45.7%] of patient had hyperemia, [34.3%] patients had angiodysplasia, and [14.3%] of patients had rectal varices.

Regarding the relation between esophageal varices with lower endoscopic parameters the current results revealed that there was a statistically significant relation between each of esophageal varices and portal hypertension gastropathy with hyperemia. No other significant associations were found. In line with these results, Salama *et al*. [17] reported that the presence of colonic hyperemia significantly correlated with the presence of gastroesophageal varices, whereas the presence of hemorrhoids, rectal varices and angiodysplasia did not correlate with the presence of gastroesophageal varices. In addition, Shalaby *et al*. [15] reported that there was no significant relation between gastric varices and portal hypertensive colopathy grade. In addition, Bresci *et al*. [22] detected that one of these colonic mucosal abnormalities significantly correlated with the presence of gastroesophageal varices and PHG.

In contrast, Ito *et al*. [23] detected that OV were not related to any of the colonic mucosal abnormalities. This could be explained by the possibility that increased portal pressure leading to gastroesophageal varices and PHG might deviate the main brunt of PHT towards the upper portion of the GI tract from its lower portion, thus decreasing the chance for the appearance of colonic mucosal abnormalities and vice versa.

Conclusion:

The prevalence of PHC and hemorrhoids increases with the progression of liver cirrhosis. Being a potential source of acute lower GI bleeding, PHC requires additional studies not only to determine their frequency, but also to understand their pathophysiology and establish proper universal endoscopic classification.

Conflict of interest: none

Financial disclosure: none

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