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ORIGINAL ARTICLE

Gastric antral vascular ectasia in hepatitis C virus related liver cirrhosis: Fetching for predictors

Magdy Fouad,* Hanaa Khalaf Fath-Elbab,* Alaa Mohamed Mostafa,* Hend M Moness,† Nashwa Mohamed Adel‡ and Elham Ahmed§

*Gastroenterology and Hepatology Unit, Department of Tropical Medicine, †Department of Clinical Pathology, ‡Department of Radiology and §Department of Internal Medicine, Faculty of Medicine - Minia University, Minia, Egypt

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Correspondence
Magdy Fouad, Gastroenterology and Hepatology Unit, Department of Tropical Medicine, Faculty of Medicine - Minia University, Cornish Al-Nile Road, Minia University Hospital, 61111 Minia, Egypt.
Email: drmagdyf@yahoo.com

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Author contribution: Magdy Fouad and Hanaa Khalaf Fath-Elbab shared suggestion of the study design. Magdy Fouad interpreted the results. Alaa Mohamed Mostafa and Elham Ahmed shared patient recruitment. Elham Ahmed wrote the manuscript. Hend M Moness performed the laboratory investigation, and Nashwa Mohamed performed the ultrasonography.

Abstract

Background and Aim: Gastric antral vascular ectasia (GAVE) is observed in patients with liver cirrhosis and portal hypertension. The exact pathophysiologic mechanism that underlies this condition is unknown. In our study, we estimate the prevalence of GAVE in hepatitis C virus (HCV) cirrhosis and attempted to determine if any of the hepatocellular manifestations, liver functions, serum gastrin, abdominal ultrasound and endoscopic picture have a relation to, or could predict, the occurrence of GAVE in cirrhotic patients.

Methods: This study includes 500 HCV-related liver cirrhosis patients. According to endoscopic assessment, we detected 30 patients with GAVE (Group 2). From the 470 patients without GAVE, we randomly selected 120 patients (Group 1), to avoid statistical bias, for comparison with Group 2. Comparison included clinical manifestations, laboratory findings, serum gastrin, ultrasound findings, and endoscopic findings (esophageal and/or gastric varices and gastropathy).

Results: The percentage of GAVE in HCV-related liver cirrhosis is 0.06%. We can predict GAVE by platelets, palmer erythema, diabetes mellitus (DM), marked ascites > with area under the curve of 0.67, 75.5, 0.62, and 0.40%, and accuracy of 82.5, 72, 70.7, and 79.3%, respectively. There was no correlation found between occurrence of GAVE and endoscopic findings. Also, there was no correlation found between occurrence of GAVE and serum gastrin levels, which reflect another pathophysiology, and we found no statistically significant correlation with GAVE.

Conclusions: Palmer erythema, low platelets, DM, and ascites might help in the prediction of GAVE. GAVE is not linked to the presence, type or grade of varices, and gastropathy.

Introduction

Gastric antral vascular ectasia (GAVE) has a special picture in endoscopy: it is characterized by red spots (without a background mosaic pattern) either organized in a linear distribution radially departing from pylorus or arranged in a diffused way.[1] Although GAVE is observed in patients with liver cirrhosis and portal hypertension, the pathophysiology apparently does not involve portal hypertension: it is not detected in about 70% of portal hypertension patients, and the decrease of portal hypertension does not influence the progression of the disease. The exact pathophysiologic mechanism that underlies this condition is unknown.[2] Antro-pyloric dysfunction with irregular antral motor reaction to meals has been demonstrated in cirrhotic patients with GAVE. Humoral variables such as gastrin, vasoactive inhibitory peptide, 5-hydroxytryptamine, glucagon, catecholamine, and other undefined vasoactive substances have been suggested in the pathogenesis of GAVE.[3] It appears that liver transplantation, despite persistent portal hypertension, causes a complete vanishing of GAVE. Therefore, liver failure and not portal hypertension might play a role within the pathogenesis of GAVE by alternating the metabolism of certain unknown substances.[4]

The aim of this study is to estimate the prevalence of GAVE in hepatitis C virus (HCV) liver cirrhosis patients and to identify whether hepatocellular manifestations, liver functions, serum gastrin, abdominal ultrasonic and endoscopic picture have a relation to the occurrence of GAVE, whether these factors can predict the occurrence of GAVE in cirrhotic patients, contributing to an improved awareness of the pathogenesis of this commonly misdiagnosed disease, and whether these items can predict the occurrence of GAVE in cirrhotic patients.

Methods

All patients signed an informed written consent form to participate in our research; the study protocol was approved by the Ethics Committee of the Faculty of Medicine, Minia University, Egypt. The study was conducted in accordance with the ethical guidelines of the 1975 Helsinki Declaration. Our study was a cross-sectional one.

We recruited all patients with HCV-related liver cirrhosis attending the outpatient gastroenterology and hepatology clinics of Minia University Hospital. Patients with other causes of liver cirrhosis, for example, chronic hepatitis B, hemochromatosis, Wilson's disease, and autoimmune liver diseases, were excluded from the study.

Diagnosis of liver cirrhosis was made by using standard clinical, laboratory, radiological, and Fibro-Scan criteria. At the end of December 2019, we included 500 HCV-positive cirrhotic patients.

All included patients underwent a full clinical assessment and laboratory investigation including a complete blood count carried out by automated cell counter, Sysmex KX-21N (TAO Medical Incorporation, Osaka, Japan), and fasting blood glucose and liver function tests carried out by auto-analyzer Konelab i60 (thermo-electro, clinical chemistry automation systems, Finland). International normalized ratio (INR) was measured using the STAGO COMPACT CT Coagulation Analyzer (Diamond Diagnostics, Holliston, MA, USA). HBsAg and HCV Abs were tested by fully automated chemiluminescence technology (Cobas E

Table 1 Demographic, clinical data, and ultrasonic findings for the studied groups

	Patients without GAVE ($n = 120$)	Patients with GAVE ($n = 30$)	*P* value
Age (mean ± SD)	55.7 ± 10.4	56.1 ± 9.8	0.8
Sex			
Male	76 (63.3%)	23 (76.7%)	0.1
Female	44 (36.7%)	7 (23.3%)	
Hematemesis			
No	20 (16.7%)	4 (13.3%)	0.6
Yes	100 (83.3%)	26 (86.7%)	
Melena			
No	39 (32.5%)	5 (16.7%)	0.08
Yes	81 (67.5%)	25 (83.3%)	
Other bleeding tendency			
No	41 (34.2%)	8 (26.7%)	0.4
Yes	79 (65.8%)	22 (73.3%)	
DM			
No	91 (75.8%)	15 (50%)	0.005*
Yes	29 (24.2%)	15 (50%)	
HTN			
No	111 (92.5%)	26 (86.7%)	0.3
Yes	9 (7.5%)	4 (13.3%)	
Palmer erythema			
No	84 (70%)	6 (20%)	<0.001*
Yes	36 (30%)	24 (80%)	
Liver size			
Average	35 (29.2%)	4 (13.3%)	0.1
Shrunken	82 (68.3%)	24 (80%)	
Enlargement	3 (2.5%)	2 (6.7%)	
Spleen diameter, mean ± SD (cm)	14.85 ± 1.6	14.03 ± 3.2	0.05
Hepatic focal lesion			
No	103 (85.8%)	27 (90%)	0.5
Yes	17 (14.2%)	3 (10%)	
Portal vein			
Patent	117 (97.5%)	28 (93.3%)	0.2
Thrombosed	3 (2.5%)	2 (6.7%)	
Ascites			
No	58 (48.3%)	8 (26.7%)	0.001*
Mild	20 (16.7%)	10 (33.3%)	
Moderate	33 (27.5%)	4 (13.3%)	
Marked	9 (7.5%)	8 (26.7%)	

*Significant level at *P* value < 0.05.

Chi-square test for qualitative data between the two groups and independent sample *t*-test for parametric quantitative data between the two groups.

DM, diabetes mellitus; GAVE, gastric antral vascular ectasia; HTN, hypertension.

Table 2 Laboratory investigation and child score of gastric antral vascular ectasia (GAVE) cirrhotic patients and non-GAVE cirrhotic patients

Laboratory data (mean ± SD)	Patients without GAVE ($n = 120$)	Patients with GAVE ($n = 30$)	*P* value
Total bilirubin	2.04 ± 1.73	2.74 ± 2.2	0.06
ALT†	60.3 ± 110.1	49.7 ± 34.2	0.6
AST†	96.6 ± 147.5	66.46 ± 40.71	0.3
Albumin	2.8 ± 0.6	2.7 ± 0.5	0.6
INR	1.5 ± 0.3	1.5 ± 0.4	0.8
Hemoglobin	9.3 ± 2.2	9.1 ± 2.01	0.5
Platelets	128.1 ± 52.7	97.5 ± 43.2	<0.001*
TLC	7.7 ± 3.8	9.1 ± 5.5	0.1
Gastrin, pg/mL	41.16 ± 30.90	39.24 ± 5.23	0.06
Child score			0.1
Class A	42 (35%)	6 (20%)	
Class B	51 (42.5%)	13 (43.3%)	
Class C	27 (22.5%)	11 (36.7%)	

*Significant level at *P* value < 0.05.

†Mann–Whitney test for nonparametric quantitative data between the two groups.

Independent sample *t*-test for parametric quantitative data between the two groups. Chi-square test for qualitative data between the two groups.

ALT, alanine aminotransferase; AST, aspartate aminotransferase; INR, international normalized ratio; TLC, total leucocyte count.

Table 3 Endoscope findings in our studied groups

Upper gastrointestinal endoscopy	Patients without GAVE ($n = 120$)	Patients with GAVE ($n = 30$)	*P* value
Esophageal varices			
No	5 (4.2%)	0 (0%)	0.4
Yes	110 (91.6%)	28 (93.3%)	
Eradicated	5 (4.2%)	2 (6.7%)	
Gastric varices			
No	84 (70%)	18 (60%)	0.2
Yes	36 (30%)	12 (40%)	
Both esophageal and gastric varices			
No	34 (28.3%)	14 (46.7%)	0.05
Yes	86 (71.7%)	16 (53.3%)	
Gastropathy			
No	51 (42.5%)	16 (53.3%)	0.3
Mild	43 (35.8%)	8 (26.7%)	
Moderate	9 (7.5%)	4 (13.3%)	
Severe	17 (14.2%)	2 (6.7%)	

Chi-square test for qualitative data between the two groups.

GAVE, gastric antral vascular ectasia.

411-Roche-Roche Diagnostics GmbH, Mannheim, Germany) and serum gastrin was assayed by the enzyme-linked immunosorbent assay technique (EIA) (the kit was supplied by Bioassay, catalog number E2035Hu).

Abdominal ultrasonography and upper gastrointestinal (GI) endoscopy were carried out in all cases. Upper GI endoscopy was done by using the oesophago-gastro-duodenoscope high-definition video by Pentax, EPK-i5000, Japan.

Examination of the esophagus, stomach, and duodenum was carried out to evaluate the presence, type, and the grade of varices, in addition to any relevant upper GIT (gastrointestinal) lesions related to portal hypertension. We used the Grade I–IV classification to classify the varices.[5]

According to endoscopic assessment of the 500 patients examined, we detected 30 patients with GAVE (Group 2). From the 470 patients without GAVE, we randomly selected 120 patients (Group 1), to avoid statistical bias, for comparison with Group 2.

Statistical analysis. The collected information was coded, arranged, and measurably analyzed utilizing the Statistical Package for Social Sciences software program, version 20. Descriptive statistics were created for the parametric quantitative data by mean, standard deviation, and minimum and maximum of the range; we used number and percentage for categorical data.

Table 4 Multiple logistic regression analysis for prediction of patient with gastric antral vascular ectasia (GAVE)

	AOR	95% CI	*P* value
DM			
No	1		
Yes	3.1	1.3–7.1	0.007*
Platelet	0.98	0.97–0.99	0.004*
Ascites			
No	1		
Mild	3.6	1.2–10.4	0.01*
Moderate	0.87	0.24–3.1	0.8
Marked	6.4	1.9–21.5	0.002*
Palmer erythema			
No	1		
Yes	9.3	3.5–24.7	0.001*

*Significant level at *P* value < 0.05.
AOR, adjusted odds ratio; CI, confidence interval; DM, diabetes mellitus.

Table 5 Multiple stepwise logistic regression analysis

	AOR	95% CI	*P* value
DM			
No	1		
Yes	4.2	1.3–13.02	0.01*
Platelets	0.98	0.97–0.99	0.01*
Ascites			
No	1		
Mild	3.2	0.88–12.05	0.07
Moderate	1.2	0.22–6.7	0.8
Marked	7.4	1.6–33.1	0.009*
Palmer erythema			
No	1		
Yes	16.06	4.6–56.5	0.001*

*Significant level at *P* value < 0.05.
AOR, adjusted odds ratio; CI, confidence interval; DM, diabetes mellitus.

Analyses were carried out for the parametric quantitative data between the two groups using the independent sample *t*-test, and for nonparametric quantitative data using the Mann–Whitney test.

Analyses were carried out for the qualitative data by usage of the chi-square test and Fisher's exact test. Logistic regression analysis was carried out to determine the independent predictors of GAVE. An receiver operating characteristic (ROC) curve was produced to determine the cutoff point, area under the curve (AUC), sensitivity, specificity, PPV, NPV, and the accuracy of variable predicting GAVE. *P* value <0.05 was significant.

Results

The percentage of GAVE in HCV-related liver cirrhosis is 0.06%. The mean age of patients with GAVE was 56.1 ± 9.8 years; most of them were male (76.7%).

In terms of the clinical data of our studied groups, palmer erythema was significantly higher in cirrhotic patients with GAVE than in cirrhotic patients without GAVE (*P* value = 0.001).

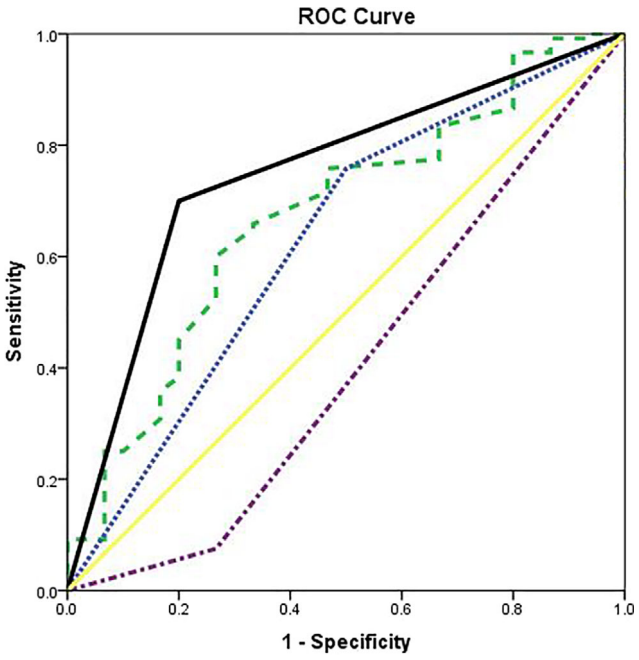

Figure 1 Prediction of Gastric Antral Vascular Ectasia in Hepatitis C Virus. Source of the curve: (——), DM, diabetes mellitus; (——), PLT, platelet; (——), PE, palmer erythema; (——), MA, marked ascites; (——), RL, reference line. ROC, receiver operating characteristic.

Diabetes mellitus (DM) was significantly higher in Group 2 than in Group 1 (*P* value = 0.005). The degree of ascites differed significantly between Group 1 and Group 2; where marked, it was significantly higher in Group 2 than Group 1, with a *P* value = 0.01 (Table 1).

Within the laboratory investigations of our studied groups, platelets were significantly lower in patients with GAVE. There was no significant difference in serum gastrin levels between Group 1 and Group 2 (Table 2).

Table 3 shows that there is no significant difference between the two groups with respect to the presence, type, or grade of endoscopic findings (esophageal and/or gastric varices and portal gastropathy).

As it uses a multiple logistic regression analysis, our study detected that the predictors for GAVE in HCV-related liver cirrhosis were DM, reduction of platelets, palmar erythema, and marked ascites (Tables 4 and 5).

The ROC curve (Fig. 1) was used for the prediction of GAVE in HCV cirrhotic patients by palmar erythema with an AUC of 0.75 and accuracy of 72%, DM with an AUC of 0.62 and accuracy of 70.7%, marked ascites with an AUC of 0.40 and accuracy of 79.3%, and platelets with an AUC of 0.67 and accuracy of 82.5% (Table 6).

Discussion

GAVE is a rare etiology of occult GI bleeding and iron-deficiency anemia, comprising 4% of cases of non-variceal upper GI hemorrhage.[6]

Table 6 Sensitivity, specificity, PPV, NPV, and accuracy % for diabetes mellitus (DM), ascites, platelets and palmar erythema

Variable	AUC	*P* value	Sensitivity	Specificity	PPV	NPV	Accuracy
Palmer erythema	0.75	<0.04*	80	70	40	93.3	72
DM	0.62	0.05	50	75	34.1	85.8	70.7
Marked ascites	0.40	0.06	26.7	92.5	47.1	83.5	79.3
Platelets	0.67	0.05	66.7	65.8	32.8	88.8	82.5

*Significant level at *P* value < 0.05.

AUC, area under the curve; PPV, positive predictive value; NPV, negative predictive value.

GAVE's clinical appearance ranges from chronic occult GI bleeding demanding multiple transfusions to acute GI bleeding.[7] It is usually related to liver cirrhosis and, specifically, portal hypertension.[8]

GAVE may be mixed with portal hypertensive gastropathy. Indeed, the pathophysiology of the two diseases is completely distinct, as GAVE may be caused by neurohumoral substances such as prostaglandin E, possibly gastrin, vasoactive intestinal peptide, and/or 5-hydroxytryptamine, leading to vasodilation and disturbed motility.[3]

This study was conducted to identify factors associated with GAVE and predict risk factors with HCV-related cirrhotic patients. Our study reveals that there is no relation between age and sex and GAVE. This is in line with Wang *et al*.,[9] who found no age or sex significance in HCV-related liver cirrhosis patients having GAVE. In contrast, Foutch *et al*.[10] suggested that vascular ectasia is acquired as a result of a degenerative process associated with aging.

Based on past history, our study denies any relation between systemic hypertension with GAVE in HCV-related liver cirrhosis, while diabetes is one of the predictors for GAVE. Wang *et al*.[9] proved that diabetes is the most common concomitant disorder related to vascular ectasia.

Our study also revealed that palmer erythema has a highly significant relation (P value = 0.001) with GAVE among HCV-related liver cirrhosis and is one of the predictors for GAVE. This can be explained by the vasodilatation that may occur due to the vasoactive substances, which play an important role in the etiology of vascular ectasia. This is in line with Kamath *et al*.,[11] who claimed that GAVE is due to the shunting of blood and the altered metabolism of vasoactive substances in the presence of liver disease.

Child C score was the commonest score among patients with GAVE. This is in line with reference 12, which proved that GAVE is most frequently detected in patients with the furthest progressed liver illness. Our study shows no significant relation between INR and vascular ectasia.

In contrast, Abd El-Ghany *et al*.[13] found a significant value between INR and vascular ecstatic patients. Our study shows no relation between hemoglobin level and the presence of vascular ecstatic patients (P value < 0.001). This study is in contrast with Yamada *et al*.,[14] who found that hemoglobin was significantly decreased in cirrhotic patients with vascular ectasia than in those without. In addition, Poll *et al*.[8] studied cirrhotic patients with or without vascular ectasia and noticed that blood transfusions markedly increased for patients with vascular ectasia-related liver cirrhosis than for without ectasia.

Our study detects a reduction in platelets in cirrhotic patients with GAVE, indicating that platelets are predictors for GAVE. This is in agreement with Wang *et al*.,[9] who found that there is a relation between a reduction in platelet level and the occurrence of vascular ectasia in HCV cirrhotic patients. On the other hand, albumin levels have no significant relation to the presence of vascular ectasia in HCV-related liver cirrhosis. This is in contrast with Wang *et al*.,[9] who claim that albumin levels in patients without cirrhosis were higher than in patients with cirrhosis in the presence of vascular ectasia. alanine aminotransferase and aspartate aminotransferase levels had no significant relation to vascular ectasia in HCV-related liver cirrhosis. This is in line with the findings of Abd El-Ghany *et al*..[13]

Multiple logistic regression analysis determined that marked ascites has a significant relation with GAVE. This is in line with Payen *et al*.[15] and Lee *et al*.,[16] who both described an increased incidence of GAVE in patients with chronic liver disease. This can be explained by the presence of ascites in patients with end-stage liver disease, which is associated with more liver failure and more disturbance in the metabolism of certain substances, which have a role in the pathogenesis of GAVE.

We therefore tried to detect the relationship between serum gastrin levels and GAVE occurrence in our study as an example of these substances, but it was insignificantly decreased in cirrhotic patients with GAVE than in cirrhotic patients without GAVE. This was the opposite finding to Quintero *et al*., who showed that cirrhotic patients with GAVE had significantly increased serum gastrin levels (872 ± 367 pg/mL) in comparison with cirrhotic patients without lesions (137 ± 253 pg/mL). Additionally, Quintero *et al*. showed that 73% of cirrhotic patients with GAVE had hypergastrinemia, while hypergastrinemia appeared in 11% of cirrhotic patients without lesions.[17]

Payen *et al*.[15] detected that serum gastrin levels were significantly lower in the cirrhotic group with GAVE than in the cirrhotic group without GAVE. Our results relating to serum gastrin may indicate that it has no role in the pathogenesis of GAVE, and other substances may be needed to be studied. Vasoactive substances have been mentioned as having a possible role in the pathogenesis of GAVE. Lowes and Rode[18] suggested that neuroendocrine substances such as 5-hydroxytryptaminne can cause vasodilation in the gastric mucosal wall.

The splenic size in our study has no significant relation with vascular ectasia in HCV-related liver cirrhosis. This is in line with the study by Djordjević *et al*.,[19] who showed that there is no relation between splenic size and the presence of GAVE.

Endoscopic findings (esophageal and/or gastric varices and portal gastropathy) for our studied groups show that the presence, type, or grade of endoscopic findings have no relation with vascular ectasia in HCV cirrhotic patients. This is agreed by Naidu *et al*.,[20] who claimed that despite GAVE being detected in many cirrhotic

patients, no rational link has been proved among esophageal, gastric varices, portal hypertension, and vascular ectasia and it has no role in the prediction of GAVE. This is in contrast with Abd El-Ghany *et al*.,[13] who found a relation between the presence of esophageal varices and vascular ectasia in HCV liver cirrhosis.

In our study, we used the ROC curves to specify the significance of palmer erythema, platelets, DM, and ascites to vascular ectasia. The ROC curve (Fig. 1) of palmer erythema can predict GAVE in cirrhotic patients with a sensitivity of 80% and a specificity of 70%, while the ROC curve of platelets showing an optimal cutoff value (>2.6) indicates a sensitivity of 66.7% and a specificity of 65.8%. Additionally, the ROC curve of DM can predict GAVE in cirrhotic patients with a sensitivity of 50% and a specificity of 75%, while the ROC curve of ascites shows a sensitivity of 26.7% and a specificity of 92.5%.

Therefore, the small number of patients with GAVE in our study was a limitation, so we need another study with larger number of GAVE patients in the future.

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Data availability statement

This published article contains all the knowledge produced or analyzed during this research.

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