brief report

Prevalence of gastric varices and portal hypertensive gastropathy in patients with Symmers' periportal fibrosis

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BACKGROUND AND OBJECTIVE: Symmers' periportal fibrosis secondary to schistosomiasis is a common cause of portal hypertension worldwide. Data on the prevalence of gastric varices and portal hypertensive gastropathy in this group of patients with portal hypertension is relatively scarce. The aim of this study was to determine the prevalence of gastric varices and portal hypertensive gastropathy in patients presenting with portal hypertension secondary to Symmers' periportal fibrosis.

PATIENTS AND METHODS: In a prospective study, upper gastrointestinal endoscopy was carried out to determine the prevalence of gastric varices and portal hypertensive gastropathy in patients with portal hypertension secondary to Symmers' periportal fibrosis.

RESULTS: Of 143 patients studied, 24 patients (16.8%) had gastric varices (grade I in 10.5%, grade II in 6.3%) and 31 patients (21.7%) had portal hypertensive gastropathy (mild in 11.2%, severe in 10.5%). Gastric varices were more prevalent in patients with grade I and II esophageal varices and portal hypertensive gastropathy was more prevalent in those with grade III and IV esophageal varices, but the differences were not statiscally signifant.

CONCLUSION: We concluded that both gastric varices and portal hypertensive gastropathy seem to have a lower prevalence in patients with portal hypertension secondary to Symmers' periportal fibrosis when compared to reported data in patients with portal hypertension secondary to liver cirrhosis and non-cirrhotic portal fibrosis.

Schistosomiasis is endemic in 70 countries and affects more than 200 million people worldwide.[1] The mortality associated with *S. mansoni* infection is mostly due to the development of Symmers' periportal fibrosis (PPF), and subsequent portal hypertension and development of esophageal varices, which also cause significant morbidity.[2,3] In Sudan the prevalence of infection with *S. mansoni* in endemic areas such as the Gezira and White Nile regions may reach up to 70% and PPF up to 18% in areas not covered by control programs.[4] Data on the prevalence of gastric varices and portal hypertensive gastropathy (PHG) in patients with Symmers' periportal fibrosis, a common cause of portal hypertension worldwide, remains relatively scarce as most studies were conducted in patients with portal hypertension secondary to liver cirrhosis. The prevalence of gastric varices in cirrhotic patients varies from 20% to 57% in previous studies.[5,6] Gastric varices are associated with fewer but more severe episodes of bleeding than esophageal varices; they may bleed in up to 20% of patients and bleeding is more difficult to control.[5] The prevalence of PHG varies greatly, from 9% to 98%, and bleeding from PHG is generally uncommon and rarely severe and tends to be chronic rather than acute.[7] The prevalence of gastric varices and PHG tends to increase following esophageal sclerotherapy or band ligation.[7] This study was conducted to determine the prevalence of gastric varices and portal hypertensive gastropathy in patients with portal hypertension secondary to Symmers' periportal fibrosis.

PATIENTS AND METHODS

This was a prospective, descriptive, hospital-based study conducted at Soba University Hospital, Khartoum, Sudan, during the period from March 2003 to July 2004. All patients with portal hypertension secondary to Symmers' PPF referred for elective upper GI endoscopy were enrolled in the study if they fulfilled the

following criteria: 1) residence in an endemic area for intestinal schistosomiasis for more than 10 years, 2) a history of *S. mansoni* infection, 3) features of PPF on abdominal ultrasonography,[8,9] 4) a portal vein diameter of >13 mm on abdominal ultrasound and 5) a platelet count of <100 000. Exclusion criteria included the following: 1) biochemical or ultrasonographic features of portal hypertension due to causes other than PPF such as liver cirrhosis, 2) PPF with previous endoscopic esophageal therapy such as band ligation or sclerotherapy or prophylactic pharmacological therapy such as ß blockers, 3) positive serology for HBsAg or HCV antibodies or 4) malignancy. The study was approved by the medical research board, Faculty Of Medicine, University of Khartoum and all patients were included in the study after giving informed consent.

All patients underwent an upper GI endoscopy using an Olympus GIF XQ 240 video gastroscope to assess (a) the presence and grade of esophageal varices from grade I-IV according to Paquet et al,[10] and (b) the type of gastric varices from I-III according to Hosking and Johnson[11] with type I gastric varices having an inferior extension of esophageal varices across the squamocolumnar junction, type II gastric varices located in the fundus of the stomach and nearly always accompanied by esophageal varices and type III gastric varices located in the fundus or body of the stomach in the absence of esophageal varices , and (c) the presence and degree of portal hypertensive gastropathy as described by McCormack et al[12] into mild, those with fine pink speckling and a snake skin striped appearance and severe being those with discrete red spots and diffuse hemorrhagic gastritis.

The χ^2 test with Yates' correction was used to compare proportions. The odds ratio (OR) with 95% confidence intervals were calculated either directly or by the Mantel-Haenszel method for stratified analysis.

RESULTS

A total of 200 patients with portal hypertension presented to the endoscopy unit during the study period, and 57 were excluded from the study as follows: 35 patients had liver cirrhosis, 12 patients had positive serology for HBsAg or HCV antibodies, 4 patients had hepatocellular carcinoma, 4 patients had previous endoscopic esophageal therapy, 1 patient had Budd-Chiari syndrome and 1 patient had splenic vein thrombosis. Only 143 patients presenting with portal hypertension secondary to Symmers' PPF were included in the study. The mean age of the study group was 42 years with an age range of 15 to 75 years and 82% were males (Table 1). All had esophageal varices on endoscopy, 5% had grade I varices, 31% had grade II varices, 58% had grade III varices and 6% had grade IV varices.

Gastric varices were detected in 24 patients (16.8%), 10.5% had type I varices (grade I) and 6.3% had type II varices (grade II) (Table 2). Patients with grade I and II esophageal varices had the highest prevalence of gastric varices but the differences were not statistically significant. Portal hypertensive gastropathy was detected in 31 patients (21.7%), and was of mild degree

Table 1. Demographic criteria and endoscopic findings in 143 patients with Symmers' periportal fibrosis.

Variable	Number of patients (%)
Gender	
Male	117 (82%)
Female	26 (18%)
Mean age, range (years)	42 (15-75)
Endoscopic findings	
Gastric varices	24 (16.8%)
Type I (grade I)	15 (10.5%)
Type II (grade II)	9 (6.3%)
Portal hypertensive gastropathy	31 (21.7%)
Mild	16 (11.2%)
Severe	15 (10.5%)

Table 2. Grading of esophageal varices in patients with gastric varices and portal hypertensive gastropathy.

Variable	Patients with grade I/II esophageal varices (n=52)	Patients with grade III/IV esophageal varices (n=91)	Odds ratio (95% confidence interval)	*P* value
Patients with gastric varices (n=24)	12 (23.1%)	12 (13.2%)	1.98 (0.75-5.23)	.20
Patients with portal hypertensive gastropathy (n=31)	8 (15.4%)	23 (25.3%)	0.54 (0.2-1.41)	.24

in 11.2% and severe in 10.5% of patients. The prevalence of PHG was more in those with grade III and IV esophageal varices.

DISCUSSION

Our study is one of few on the prevalence of gastric varices and PHG in patients with Symmers' periportal fibrosis. In this study gastric varices were detected in 16.8% of patients. The prevalence of gastric varices in patients with cirrhosis and in patients with noncirrhotic portal fibrosis varies greatly; Watanabe reported up to 57% in patients with cirrhosis[6] and Amarpurkarin reported up to 44% in noncirrhotic portal fibrosis.[13] In this study, 10.5% had grade I and 6.3% had grade II, and similar results were noted in cirrhotic patients in previous studies, where grade I were noted to be more prevalent than grade II.[5] It was also noted that gastric varices were more prevalent in those with grade I and II esophageal varices. Although this was not statistically significant, it was noted before by Watanabe[6] that in cirrhotic patients with advanced gastric varices, esophageal varices were either absent or minimal.

PHG was detected in 21.7% of patients. These findings are similar to those reported by Chavez,[14] who reported PHG in 33.3% of patients with PPF due to *S. mansoni* infection. A higher figure was reported in those with non- cirrhotic portal fibrosis,[13] in those with PPF secondary to *S. japonicum*[15] and in those with liver cirrhosis[7] with figures of 54%, 55.6% and 98%, respectively. In our study it was also noted that the prevalence of PHG increased in those with grade III and grade IV esophageal varices, observations similar to those by Amarpurkarin[13] in noncirrhotic portal fibrosis and by Lou[15] in PPF secondary to *S. japonicum*. There was no statistical significance between the presence of gastric varices or PHG and the grade of esophageal varices (Table 2).

We conclude that gastric varices and PHG in the study population with portal hypertension secondary to Symmers' PPF seem to have a lower prevalence when compared to reported data in patients with portal hypertension secondary to liver cirrhosis or noncirrhotic portal fibrosis.

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