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Oesophagogastroduodenoscopy in patients with cirrhosis: Extending the range of detection beyond portal hypertension

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ABSTRACT

Background: Oesophagogastroduodenoscopy is currently recommended for the screening of varices in cirrhosis. In addition to the assessment of varices, oesophagogastroduodenoscopy can detect conditions that, while unrelated to portal hypertension, may require treatment.
Aims: We evaluated in a large cohort of cirrhotic patients the prevalence of upper digestive findings other than oesophagogastric varices, the associations between upper gastrointestinal findings, portal hypertension and features of cirrhosis, and the incidence of new lesions in the course of a surveillance program.
Methods: Analysis of the records of 611 consecutive cirrhotic patients undergoing oesophagogastroduodenoscopy for screening and surveillance.
Results: 232 patients (38%) presented endoscopic lesions not related to portal hypertension: peptic diseases ($n = 193$), proliferative diseases ($n = 27$) and vascular diseases ($n = 12$). In the screening group, 127 patients (39.4%) had pathologic lesions. At multivariate analysis, an association was found between peptic diseases and the absence of portal hypertensive gastropathy (RR 3.3; 95% CI 2.2–4.8); vascular diseases were associated with endoscopic signs of portal hypertension ($p = 0.01$). During surveillance, 9/55 patients (16.3%) in the group without previous pathologic findings developed new lesions.
Conclusions: Oesophagogastroduodenoscopy in patients with cirrhosis undergoing endoscopy for screening diagnosed pathologic lesions unrelated to portal hypertension requiring a change in management in 39.4% of asymptomatic subjects.

1. Introduction

Oesophagogastroduodenoscopy (EGD) is the gold standard for the diagnosis of oesophageal varices (EV), and is currently recommended by guidelines as a screening tool for portal hypertension once a diagnosis of cirrhosis is made. The size of varices and the presence of red wale marks on varices are used to predict the need for the prophylaxis of beta-blockers for bleeding. The presence and size of varices, and the stage of cirrhosis, dictate the intervals for repeating EGD [1–5].

Screening all patients with cirrhosis by endoscopy detects medium/large varices, i.e. those at significant risk of bleeding, in 15–25% of patients [6]. EGD implies an economic burden and has a degree of invasiveness. In recent years, the growing interest in non-endoscopic screening of portal hypertension has led to the development of several alternative models for predicting the presence of high risk varices in order to avoid EGD in those patients who do not require primary prophylaxis for bleeding. Earlier studies evaluated the role of biochemical and sonographic parameters, such as platelet count/spleen diameter ratio [7], and Fibrotest [8]. More recently, transient elastography (Fibroscan), computerized tomography (CT) and video capsule endoscopy have been assessed. Fibroscan measurements establish a good correlation between liver stiffness and portal hypertension as measured by the hepatic venous pressure gradient (HVPG), but no correlation with variceal size [9,10]. Multidetector CT (MCT) oesophagography can detect the presence of EV and measure their size, with good agreement between radiologists and endoscopists, and generates better patient compliance than that found with unsedated EGD [11]. A further cost-effectiveness analysis of MCT oesophagography in comparison to EGD in cirrhotic patients concluded that MCT is more cost-effective than EGD for screening [12]. A recent multicenter trial that compared video capsule endoscopy to EGD suggested that the former has higher sensitivity and specificity in detecting varices [13], and it has been shown to be cost-effective compared to EGD [14]. Its availability is, however, currently limited in most clinical settings, and its inability to sample tissues may be a limitation in diagnosis.

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In the setting of cirrhosis, in addition to oesophageal and gastric varices, lesions of the upper digestive tract are frequently found. Some, such as portal hypertensive gastropathy (PHG), are common, and are clearly relevant to prognosis and treatment of the liver disease [15–17]. Others, such as peptic ulcers and gastric cancer, have a higher prevalence than that found in the general population [18,19].

Should non-invasive diagnostic techniques became the screening tool for clinically significant portal hypertension in cirrhosis, the number of EGDs performed in these patients would dramatically decrease. It is therefore important to know the actual prevalence of pathologic findings of the upper digestive tract other than oesophagogastric varices, which would determine a change in management of cirrhotic patients. In this study, the aim was to evaluate: (a) the prevalence of non-variceal pathologic findings in a large cohort of cirrhotic patients undergoing EGD for screening and surveillance, (b) the possible associations among upper gastrointestinal findings, portal hypertension and liver disease characteristics, and (c) the incidence of new lesions not related to portal hypertension during the endoscopic surveillance program.

2. Materials and methods

Data from all consecutive patients with liver cirrhosis undergoing EGD at our tertiary Academic Liver Unit, from January 2006 to April 2009, were collected in an *ad hoc* database. The diagnosis of cirrhosis was based either on a combination of clinical, biochemical and imaging methods ($n = 474$) or on liver biopsy ($n = 137$).

The following epidemiological and clinical data were registered: age, gender, aetiology of cirrhosis, time of diagnosis, previous variceal bleeding and treatments for portal hypertension, previous endoscopic findings, gastrointestinal symptoms and current therapy, including proton pump inhibitors (PPI), nonsteroidal anti-inflammatory drugs (NSAIDs), anticoagulants and beta-blockers. Biochemical data (total bilirubin, albumin, international normalized ratio (INR), haemoglobin and platelets) were also included. Spleen bipolar diameter was measured by standard B-mode ultrasonography.

An aetiology of cirrhosis was defined as viral (HBsAg positive and/or anti-HCV positive), alcoholic (current or historical ethanol intake of over 60 g/day), autoimmune according to defined criteria [20–22], post non-alcoholic fatty liver disease (NAFLD) if metabolic syndrome was present [23], and cryptogenic when no possible cause was identified. The severity of liver disease was classified according to the Child-Pugh score at the time of EGD [24].

All EGDs were performed by three endoscopists with experience in portal hypertension.

The timing for EGD was established according to the current guidelines [2,4].

The presence and size of EV were graded as small, medium or large according to the system proposed by the North Italian Endoscopic Club for the Study and Treatment of Esophageal Varices [25]. Classification of gastric varices was based on their relationship with EV, as well as their location in the stomach [26]. The presence and severity of PHG were scored according to the New Italian Endoscopy Conference criteria as either mild or severe [27]. Presence and severity of oesophagitis were graded according to the Los Angeles classification [28]. Gastric and duodenal ulcers were diagnosed as the presence of a crater >3 mm; the presence of bleeding and the base characteristics were graded according to the Forrest classification [29]. Gastritis and duodenitis were defined as the presence of erythema, oedema and mucosal friability with or without mucosal erosions. Rapid urease test and/or histology from both antral and gastric body biopsies were performed in patients with endoscopic findings suggestive of *Helicobacter pylori* (*H. pylori*) infection. Gastric antral vascular ectasia (GAVE) was diagnosed by the presence of flat, or slightly raised, red stripe-like lesions radiating from the pylorus to the antrum [30]. Gastric angiodysplasia was not reported because of the high risk of confusion with mucosal changes occurring in PHG. Gastric and duodenal polyps were defined as circumscribed, pedunculated or sessile lesions $\geq$5 mm.

Biopsies were performed when coagulation parameters were not severely impaired.

As recommendations for medium size EV are the same as those for large varices, we considered two categories of EV: small and medium/large. Upper digestive findings not related to portal hypertension were grouped according to their pathogenesis in: peptic diseases (oesophagitis, gastritis, duodenitis and peptic ulcers), proliferative diseases (gastroduodenal polyps and cancer), and vascular diseases (GAVE syndrome and duodenal angiodysplasias).

2.1. Statistical analysis

Data were obtained through retrospective review of the database and of clinical records. The demographics and clinical characteristics were analysed using the Student's *t*-test, analysis of the variance, and χ^2 test, as appropriate. Multiple logistic regression analysis was used to identify predictive variables of upper digestive lesions not related to portal hypertension. The Kaplan–Meier method was applied to examine the incidence of new lesions over the course of the surveillance program.

All tests were two-sided, and a value of $p < 0.05$ was considered statistically significant. Statistical calculations were made with the SPSS statistical package for Windows, version 13.0 (SPSS Inc., Chicago, IL, USA).

3. Results

611 patients underwent a single EGD between January 2006 and April 2009; 346 (56.6%) were male, and the mean age was 63.3 years. Hepatitis C was the leading cause of cirrhosis (73.8%). The majority of patients (71.1%) had cirrhosis Child-Pugh A at the time of EGD, 41 patients (6.7%) had a history of previous upper digestive bleeding. Dyspeptic symptoms were present in five patients (0.8%), four of them were receiving PPI at the time of EGD. Other indications to PPI therapy were a history of acid-related diseases found at previous EGD ($n = 22$) and gastrooesophageal reflux disease ($n = 1$).

Tables 1 and 2 show demographic and clinical features. EGD was performed as screening in 322 patients (52.7%) and as a surveillance examination in 289 patients (47.3%).

3.1. Portal hypertension related findings

EGD revealed the presence of EV in 413 patients (67.6%): small varices in 232 patients (38%), and medium/large varices in 181 (29.6%). Red signs were present in 111/413 of patients with EV (26.9%), and were correlated with variceal size: 94% versus 6% ($p < 0.001$) respectively in patients with medium/large EV and small varices. In the group of patients undergoing EGD for screening ($n = 322$), 183 subjects (57%) had EV, and medium/large EV were identified in 57 patients (18%), red signs were described in 35/183 of patients with EV (19%).

Gastric varices were observed in 41 patients (6.7%); 13/322 subjects (4%) were in the screening group.

PHG was reported in 448 patients (73.3%), with the similar rates for mild (37.3%) and severe PHG (36%). Among the 322 patients who underwent EGD for screening, 217 (67%) showed evidence of PHG, mild in 36% and severe in 31% of the cases.

Table 1
Demographic features of the 611 patients.

Parameter	
Age (years)	63.3 ± 10.3
Male/female	346/265 (56.6%/43.4%)
Aetiology of liver disease	
Hepatitis C	451 (73.8%)
Alcohol	53 (8.7%)
Hepatitis B	50 (8.2%)
AIH/PBC/PSC[a]	16 (2.6%)
Cryptogenic	13 (2.1%)
Post-NAFLD[b]	27 (4.4%)
Wilson's disease	1 (0.1%)
Child-Pugh class	
A	435 (71.1%)
B	140 (23%)
C	36 (5.9%)

Data presented as mean ± standard deviation or *N* (%).
[a] AIH = autoimmune hepatitis; PBC = primary biliary cirrhosis; PSC = primary sclerosing cholangitis.
[b] Non-alcoholic fatty liver disease.

Table 2
Clinical features of the 611 patients.

Parameter	
Haemoglobin (g/dL)	12.6 ± 2.2
Platelets ($\times 10^3$/mL)	107 ± 66
Bilirubin (mg/dL)	1.6 ± 2.1
INR[a]	1.1 ± 0.2
Albumin (g/dL)	3.7 ± 0.6
Spleen diameter (cm)	14.7 ± 3.5
PPI[b] at EGD	113 (18.5%)
NSAIDs[c] at EGD	9 (1.5%)
Anticoagulants at EGD	5 (0.8%)
Beta-blocker at EGD (%)	56 (9.2%)

Data presented as mean ± standard deviation or *N* (%).
[a] International normalized ratio.
[b] Proton pump inhibitors.
[c] Non-steroidal anti-inflammatory drugs.

The prevalence rates of EV, gastric varices and PHG were significantly higher in patients in Child-Pugh classes B and C than in patients in class A; moreover, as expected, significative associations were found among EV size, gastric varices, severity of PHG and impaired liver function tests, anaemia, low platelet count and enlarged spleen diameter (data not shown).

3.2. Endoscopic findings unrelated to portal hypertension (Table 3)

Overall, 232 patients (38%) presented with endoscopic lesions not related to portal hypertension: peptic diseases ($n = 193$), proliferative diseases ($n = 27$) and vascular diseases ($n = 12$). In the screening group, 127/322 patients (39.4%) had pathologic lesions: among the 322 patients, 42/75 subjects (56%) presented with upper digestive lesions, without endoscopic findings of portal hypertension.

Acid-related conditions: Oesophagitis was reported in 32 patients (5.2%). Gastritis was reported in 138 patients (22.6%), 58 of whom presented with single or multiple erosions. Gastric inflammation of any degree was histologically diagnosed in all 43 cases in which biopsies were performed. Complete intestinal metaplasia was found in 3 patients, 1 of whom underwent EGD for screening. 39 patients (6.4%) received a diagnosis of duodenitis, confirmed by histology in all 8 cases in which biopsies were performed; in 14 patients single or multiple erosions were found. In 23 patients (3.8%), EGD revealed a gastric ulcer, multiple ulcers were found in 11 patients. A duodenal ulcer was reported in 11 patients (1.8%).

Table 3
Endoscopic findings not related to portal hypertension.

	All subjects ($n = 611$) *N* (%)	Screening group ($n = 322$) *N* (%)
Oesophagitis	32 (5.2%)	19 (6%)
Grade A	17	10
Grade B	13	8
Grade D	2	1
Gastric ulcer	23 (3.8%)	16 (5%)
Forrest IIb	1	1
Forrest IIc	5	4
Forrest III	17	11
Gastritis	138 (22.6%)	84 (26%)
Gastric polyp	20 (3.3%)	9 (2.8%)
Gastric cancer	2 (0.3%)	1 (0.3%)
GAVE[a]	11 (1.8%)	3 (0.9%)
Duodenal ulcer	11 (1.8%)	5 (1.5%)
Forrest IIb	2	2
Forrest IIc	2	1
Forrest III	7	2
Duodenitis	39 (6.4%)	23 (7.1%)
Duodenal polyp	5 (0.8%)	3 (0.9%)
Duodenal angiodisplasia	1 (0.2%)	–
Total number of lesions	282	163
Total number of patients	232/611 (38%)	127/322 (39.4%)

[a] Gastric antral vascular ectasia.

H. pylori was checked for in 68 patients with peptic ulcer, gastritis and/or duodenitis: infection was diagnosed in 51 patients (75%), and in 37/44 patients (84%) in the screening group. *H. pylori* was detected in 47% of 34 peptic ulcers, and in 48% and 45% of gastric and duodenal ulcers, respectively. No significant associations were found among *H. pylori* infection, sex, age, Child-Pugh class and endoscopic signs of portal hypertension. Only 9 patients (4.7%) with peptic diseases had alcoholic cirrhosis ($p = 0.01$); no association were found neither with other aetiologies, nor with NSAIDs or PPI intake. Screening for EV was the most frequent indication for EGD, involving 61% of the patients with peptic diseases.

Acid-related conditions were significantly correlated with compensated liver disease, no endoscopic signs of portal hypertension, higher platelet count and lower spleen diameter (Table 4). On multivariate analysis, only the absence of PHG (RR 3.3; 95% CI 2.2–4.8) was independently associated with peptic diseases, while gastric varices, Child-Pugh class and spleen diameter had no impact on peptic diseases.

Proliferative diseases: A gastric cancer (histology: adenocarcinoma) was found in two patients (0.3%). One of them underwent EGD for screening. Gastric polyps were reported in 20 patients (3.3%), with a mean size of 9 mm. Histology revealed 7 hyperplastic polyps, 3 xanthomas and 1 serrated adenoma, the last 1 found in a patient in the screening group. Multiple polyps were found in 7 patients. 5 patients presented duodenal polyps (0.8%), with a mean size of 11 mm. In one of these patients who underwent EGD for screening, a polypoid lesion of the major ampulla was found, and a histological diagnosis of ampullary adenoma was made. On univariate analysis no significant associations were found with liver disease characteristics, biochemical parameters and signs of portal hypertension.

Vascular alterations: 11 patients (1.8%) had GAVE at EGD confirmed by histology, three of them were in the screening group; one patient presented with duodenal angiodysplasia (0.2%). Vascular lesions were seemingly correlated with EV and severe PHG (Table 5). When multivariate analysis was carried out no significant associations were found.

Operative EGDs were performed in 96/232 patients (41.4%) with lesions not related to portal hypertension: biopsies were performed in 81 patients, polipectomy in 12 patients, injective haemostasis in 3 patients with ulcer lesions and adherent clots. Overall, a medical

Table 4
Univariate analysis of factors associated with peptic diseases.

Variable	Peptic disease present ($n = 193$)	No peptic diseases ($n = 418$)	p value
Oesophageal varices	111 (57.5%)	302 (72.2%)	<0.001
Gastric varices	6 (3.1%)	35 (8.4%)	0.01
PHG[a]	109 (56.5%)	339 (81.1%)	<0.001
Class A Child-Pugh	152 (78.7%)	283 (67.7%)	0.005
Haemoglobin (g/dL)	12.8 ± 2.2	12.5 ± 2.2	0.07
Platelets ($\times 10^3$/mL)	115 ± 83	103 ± 56	0.029
Bilirubin (mg/dL)	1.5 ± 1.7	1.7 ± 2.3	0.289
INR[b]	1.1 ± 0.2	1.1 ± 0.1	0.499
Albumin (g/dL)	3.8 ± 0.6	3.6 ± 0.6	0.003
Spleen diameter (cm)	14.2 ± 3.2	14.8 ± 3.6	0.039

Data presented as mean ± standard deviation or N (%).
[a] Portal hypertensive gastropathy.
[b] International normalized ratio.

Table 5
Univariate analysis of factors associated with vascular diseases.

Variable	Vascular diseases present ($n = 12$)	No vascular diseases ($n = 599$)	p value
Oesophageal varices	12 (100%)	401 (67%)	0.01
Gastric varices	2 (16.7%)	39 (6.5%)	0.16
Severe PHG[a]	9 (75%)	211 (35.2%)	0.01
Class A Child-Pugh	8 (67.7%)	427 (71.3%)	0.72
Haemoglobin (g/dL)	11.7 ± 2.6	12.6 ± 2.2	0.13
Platelets ($\times 10^3$/mL)	115 ± 52	107 ± 66	0.68
Bilirubin (mg/dL)	1.5 ± 1.1	1.6 ± 2.1	0.84
INR[b]	1.1 ± 0.2	1.1 ± 0.2	0.95
Albumin (g/dL)	3.3 ± 0.6	3.7 ± 0.6	0.07
Spleen diameter (cm)	15.2 ± 2.7	14.7 ± 3.6	0.61

Data presented as mean ± standard deviation or N (%).
[a] Portal hypertensive gastropathy.
[b] International normalized ratio.

treatment with PPI was started in 159/232 patients (68.5%) and 1 patient with gastric cancer underwent to surgery.

3.3. Development of new lesions in the course of endoscopic surveillance

300 patients (49.1%) had no upper digestive lesions unrelated to portal hypertension at the first endoscopic examination. 55 of them underwent a second EGD during the follow-up (mean 13.3 ± 11.24 months). Nine patients (16.3%) developed new lesions: one oesophagitis, six gastritis, one gastric polyp (histology: hyperplastic) and one duodenitis. In all these subjects EGD was performed for endoscopic surveillance or treatment of EV.

4. Discussion

The prevalence rates of oesophagogastric varices and PHG in our study were comparable to those reported in the literature [16,26,31]. Likewise, the association between endoscopic findings of portal hypertension and severity of liver disease confirms previous data [6,16]. An interesting finding was the significant correlation between presence of PHG and anaemia, not found in a previous study [17], probably because of the higher prevalence of severe PHG in our study than in the HALT-C cohort.

Endoscopic findings unrelated to portal hypertension were reported in 38% of all patients with cirrhosis, and in 39.4% of those undergoing EGDs for the screening of portal hypertension. It is important to note that the majority of patients did not have at the time of endoscopy, symptoms specifically suggestive of upper digestive lesions and the 76% of patients receiving PPI did not present acid-related diseases at the time of EGD. The overall prevalence of peptic diseases was 31.6% and, in contrast to previous reports [32,33], there was no significant association with alcoholic cirrhosis or with NSAIDs intake, probably because of the small number of patients with active alcohol consumption and of patients receiving these drugs at the time of EGD. Among patients with peptic diseases, the most frequent indication for EGD was screening for portal hypertension.

Even if the way to diagnose gastroduodenitis could be questionable, it is worth to note that this is a descriptive endoscopic study aimed to identify lesions requiring a therapy in a clinical setting where is essential to weigh the risk associated to a biopsy versus the benefit of a definite pathologic diagnosis.

Univariate analysis suggested that peptic diseases were significantly associated with compensated cirrhosis and no endoscopic signs of portal hypertension. Moreover, multivariate modelling confirmed that patients without PHG had a 3.3-fold higher rate of peptic diseases.

We speculated about a possible selection bias to explain these findings, but patients were specifically asked about gastrointestinal disorders at the time of EGD, and only 3/101 patients with no endoscopic findings of portal hypertension at a previous EGD referred symptoms.

These results confirm earlier studies concerning the lack of association among peptic diseases, the severity of liver disease and HVPG values [32,34]. However, other studies have reported the existence of such relationships [35,36] and the pathogenesis of peptic lesions in cirrhosis is still controversial.

In our cohort the prevalence of peptic ulcers was 5.6% consistent with earlier reports [17,37] and higher than in normal volunteer populations [38]. Interestingly, we found a higher prevalence of gastric ulcers than duodenal ones, differently from the majority of the studies in this setting [17,32,35,37], but consistent with the results of an Italian series of 226 cirrhotic patients [39].

Peptic ulcer account for 16% of overall upper digestive haemorrhages in cirrhotics [40] and assuming a 5-fold increase in risk of complications or death for patients with cirrhosis and bleeding peptic ulcer [41], early diagnosis and antisecretory

treatment of asymptomatic peptic ulcers could have major relevance.

Several studies have reported either increased or similar prevalence of *H. pylori* in cirrhotics when compared with controls [35,39,42,43]. In our study, overall prevalence of *H. pylori* infection was 75% consistent with literature data, but only 47% of peptic ulcers were related to *H. pylori*, such relatively low prevalence can be partially explained by the different methods used for the diagnosis of *H. pylori* infection, indeed, serologic studies reported higher prevalence than studies that used invasive methods [39,42–45]. Another reason could be that only 0.8% of our patients presented dyspeptic symptoms at the time of EGD. Moreover, history of antibiotics intake could affect the prevalence of *H. pylori*, but, due to its retrospective nature, the present study cannot assess this variable. Triple therapy for *H. pylori* in cirrhotic patients achieves high rates of eradication and prevents ulcer recurrence [46,47]. Since *H. pylori* is involved in gastric carcinogenesis, and the prevalence of gastric cancer in cirrhotics is higher than in the general population, its eradication could prevent development of gastric adenocarcinoma [19,48]. Of the two gastric cancer cases (0.3%) found in this cohort, one was seen as an incidental lesion in a patient underwent EGD for screening. We found an overall prevalence of gastric polyps of 3.3% in our patients, higher than in previously reported series in cirrhotics, which ranged from 1.3 to 2% [37,49] and in the general population [50]. Adenomatous polyps are premalignant lesions, and after excision an endoscopic surveillance program is recommended to exclude recurrence [51]. Hyperplastic polyps may have increased risk of cancer, and harbour dysplastic foci in up to 19% of cases. Some authors recommend polypectomy of all lesions of >0.5 cm [52]. In our study, histology revealed one incidental adenomatous polyp (5%) in one patient undergoing EGD for screening, and hyperplastic polyps in 35% of the cases. Other premalignant conditions, such as intestinal metaplasia associated with *H. pylori* ($n = 3$), and ampullary adenoma ($n = 1$), were diagnosed incidentally; two of the four patients underwent EGD for screening.

GAVE was the most frequent vascular disease, reported in 1.8% of patients. A significant correlation was found among vascular diseases, presence of EV and severe PHG. However multivariate analysis did not confirmed these findings. There was no significant difference in haemoglobin levels according to the presence of vascular diseases, probably because the majority of the lesions were not actively bleeding at the time of EGD. This result suggests that in cirrhotic patients vascular lesions can be present regardless of severe anaemia.

During surveillance, 16% of patients in the subgroup without previous lesions unrelated to portal hypertension, developed new lesions but considering the small size of this subgroup such result cannot support definite conclusions about incidence of new lesions.

This study involved more than 600 consecutive patients and it inspects the wide range of endoscopic findings in cirrhotics; however the sample size of some subgroups might be too small to allow robust conclusions.

A further limitation is represented by the retrospective design with no control population, though this study reflects the real clinical practice in a single, tertiary referral centre for liver cirrhosis. Finally factors as aetiology and severity of liver cirrhosis, diagnostic methods, geographic and racial differences may explain discrepancies in our results.

The methods proposed as alternatives to EGD for the screening of patients with cirrhosis and portal hypertension, based on ultrasound, biochemistry or transient elastometry, are in principle unsuitable for detecting, in this setting, pathologic findings unrelated to portal hypertension. MCT oesophagography cannot reveal EV bleeding stigmata, found in 19% of the patients in our screening group, vascular diseases and small sized proliferative or peptic lesions. Capsule endoscopy is the only technique capable of detecting EV red signs and other endoscopic lesions, though like CT scanning it lacks therapeutic potential.

Although only a minority of patients in the screening group had EV requiring prophylactic therapy (18%), in 39.4% of patients in this group, EGD resulted in changes in management, consisting in a new therapy, polypectomy or surgery for digestive cancer. In our opinion, EGD still plays a major role in a careful evaluation of the patient with cirrhosis, and offers the advantage over other diagnostic tools of allowing clinicians to perform biopsies and carry out therapeutic interventions in a timely manner.

Conflict of interest

The authors declare that they have no conflict of interest.

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