

## Narrow Band Imaging: A New Tool for Diagnosis of Portal Hypertensive Gastropathy

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**Background:** Patients with cirrhosis are at an increased risk of gastrointestinal hemorrhage, with the most common source being gastroesophageal varices. However, there are gastrointestinal mucosal lesions typical of cirrhosis that may also bleed in these patients, namely portal hypertensive gastropathy (PHG). **The aim of this study** was to evaluate the endoscopic microvascular architecture of the gastric mucosa in patients with liver cirrhosis by using the magnifying narrow band imaging system (NBI) and to evaluate the different non invasive markers for prediction of PHG. **Material and Methods;** 100 Helicobacter pylori-negative cirrhotic patients; 50 with clinical or radiological evidence of portal hypertension (groupA), 50 without portal hypertension (groupB) were enrolled in this study. Relevant clinical parameters assessed included ascites and splenomegaly. Laboratory parameters like hemoglobin level, platelet count,WBC count, prothrombin time, serum bilirubin, albumin and ultrasonographic characteristics like splenic size, portal vein diameter were assessed, as well as upper gastrointestinal endoscopy using white light endoscopy (WLE) and Narrow band imaging technique for assessment of esophageal varices and PHG. **Results:** (i) Abnormality of reddening mucosa (mild PHG), red spots (severe PHG) and mosaic-like pattern were observed on the gastric mucosa. By WLE, mild PHG was observed in 24 patients (48%) of group A and also group B patients. Severe PHG (24 patients (48%) vs 8 patients (16%)) and normal gastric mucosa (2 patients(4%) vs 18 patients (36%)) were observed in group A and group B respectively. (ii) On magnifying endoscopy with NBI, reddening mucosa was observed as extended and swollen gastric pits and various degrees of dilated and convoluted capillaries surrounding the gastric pits. Red spots were demonstrated as extended and swollen gastric pits, dilated and convoluted capillaries surrounding the gastric pits, and intramucosal hemorrhage around these capillaries. (iii) 10 patients with normal gastric mucosa by WLE showed mild PHG by NBI (2 patients from group A and 8 patients from group B).**Conclusion:** NBI is more sensitive than conventional WLE for detection of mild PHG. Thrombocytopenia and platelet count/splenic diameter are considered to be good non invasive predictors for presence of PHG rather than other parameters like WBC's count, portal vein diameter and splenic diameter.

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**Key words:** Narrow band imaging, portal hypertensive gastropathy.

### Introduction:

Portal hypertension is one of the main consequences of cirrhosis. It can result in severe complications, including bleeding of esophagogastric varices as well as spontaneous bacterial peritonitis or hepatorenal syndrome as complication of ascites (*Dib et al., 2006*).

The gastric mucosa of patients with portal hypertension is frequently subjected to many endoscopic alterations, including portal hypertensive gastropathy (PHG) and gastric antral vascular ectasia (GAVE), and these findings may cause gastrointestinal bleeding in those patients (*Hayashi and Saeki, 2007*).

PHG is the term used to describe the endoscopic appearance of gastric mucosa with characteristic mosaic-like pattern with or without red spots; and the pathogenesis of PHG was proven to involve venous congestion with gastric mucosal capillary dilation and

these findings was difficult to be seen by conventional endoscopy (*Hayashi and Saeki, 2007*).

The narrow band imaging system is an endoscopic imaging technique for the enhanced visualization of mucosal microscopic structure and capillaries of the superficial mucosal layer, by changing the spectral features of the illumination used in the video endoscope system. The narrow band imaging system obtain its images by using narrower bands of red, blue and green filters (R/B/G), which are different from conventional red, blue and green filters (*Tahara et al., 2009*). The depth of penetration into the mucosa depends on the wave length used superficial for blue band and deep for red band and intermediate for green band (*Sambougi et al., 2000*). In endoscopic examination, lesions are identified by changes in color and irregularity of mucosal surface (*Tajiri et al., 2002*).

Combining narrow band imaging with magnifying system allow very clear images of the capillaries of the mucosal surface and microvascular architecture of the gastric mucosa in patients with liver cirrhosis (*Sano et al., 2004*).

**Aim of the Work** was to evaluate the endoscopic microvascular architecture of the gastric mucosa in patients with liver cirrhosis by using the magnifying narrow band imaging system and to evaluate the different non invasive markers for prediction of PHG.

#### Subjects & methods:

100 patients with liver cirrhosis recruited from the endoscopy unit of Ain Shams University Hospital from December 2010 to September 2012 were enrolled in this study. Diagnosis of cirrhosis was based on clinical, biochemical, and ultrasonographic findings. All patients with Helicobacter Pylori infection and gastrointestinal tract bleeding were excluded from the study. Patients were divided into two groups: Group A, 50 cirrhotic patients with clinical or radiological evidence of portal hypertension like splenomegaly, dilated portal vein, and low platelet count of less than 100.000 or ascites. And group B, 50 cirrhotic patients without clinical or radiological evidence of portal hypertension.

Informed consent was obtained from all participants before enrollment in the study and they were subjected to the following:

- 1- Relevant history and physical characteristics including symptoms and signs of liver failure (spider angioma, palmar erythema etc.), hepatomegaly, splenomegaly, ascites and abdominal vein collaterals were recorded.
- 2- Blood tests: Hematological and biochemical workup included measurement of hemoglobin, total leukocyte count, platelet count, prothrombin time, and serum concentrations of bilirubin (total and conjugated), protein, albumin, alanine aminotransferase and aspartate aminotransferase.
- 3- Ultrasound Doppler: All patients underwent ultrasonography after overnight fast and the following details were recorded: liver size, echogenicity, portal vein (PV) diameter & patency, splenic size and presence of ascites.
- 4- Endoscopic evaluation: All patients underwent conventional white light endoscopy using Pentax EPM-3500 videoscope system whereas esophageal varices were classified into **Small varices**: < 5mm, minimally elevated veins above the esophageal mucosal surface, **Large varices**: > 5mm, tortuous veins occupying more than one-third of the esophageal lumen and it encompassing medium sized varices (*Garcia-*

*Tsao et al., 2007*). Also, the finding of gastric mucosa was classified according to (*Perini et al., 2009*) into **Normal mucosa** orange to light red colored mucosa with some small sub-mucosal veins in fornix and gastric body, **Mild PHG** mosaic-like pattern which appear as Small, polygonal areas surrounded by a whitish-yellow depressed border "snake-skin appearance", **Severe PHG** mosaic-like pattern is superimposed by red signs or if any other red signs are present.

- 5- Those patients were re-evaluated by using the NBI technique using Pentax EPK-i video processor which is a computer controlled processor that provides a spectacular image with an unrivaled of 1.25 megapixels which is approximately 50% higher than any other endoscopy using a sophisticated soft ware that enhances and analyse the image area providing detailed imaging of mucosa topography and vascularity.

#### Statistical Analysis:

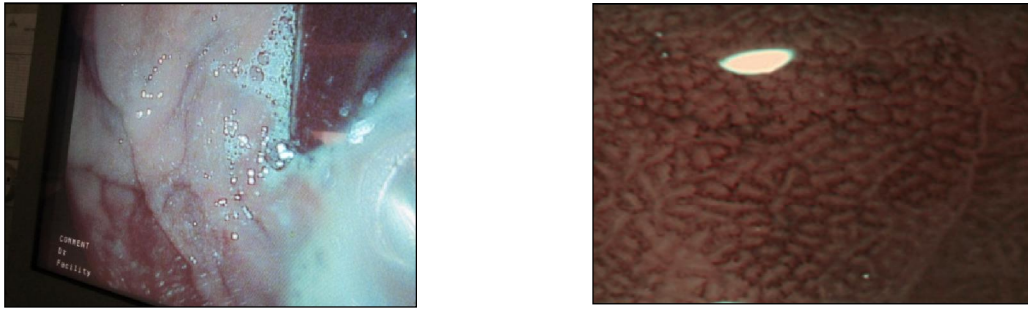
All collected data were expressed as mean± SD and analyzed by using SPSS version 12 using the following tests: Student t test, Chi-square test, Fisher test, Receiver operating curve (ROC) to detect area under curve (AUC), cut off value (COV) for best sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV) and efficacy. P<0.05 was considered significant and at P<0.001 was considered highly significant, while at P>0.05 was considered not significant.

#### Results:

Of the 100 cirrhotic patients included in the study; group A, 31(62%) were males and 19(38%) were females, with a mean age of 55.6± 8 years, WBC's count of (3.2 ± 1.4 x10<sup>3</sup>/mm<sup>3</sup>), platelet count of (66.5± 23 x10<sup>3</sup>/mm<sup>3</sup>), serum albumin level of (2.7 ± 0.5 g/dL), INR was 1.5 ± 0.2, mean splenic diameter was (169.6 ± 15 mm), portal vein diameter was (15.3 ± 1.2 mm), ascites was present in 37 patients (74%).

Group B included 28 (56%) males and 22 (44%) females. Their mean age was (52 ± 8 years), WBC's count was (3.9 ± 0.6 x10<sup>3</sup>/mm<sup>3</sup>), platelet count was (156.8 ± 32 x10<sup>3</sup>/mm<sup>3</sup>), serum albumin level was (3.5 ± 0.4 g/dL), INR was 1.1 ± 0.08, mean splenic diameter was (141.4 ± 14 mm), portal vein diameter was (11.1± 1.3 mm) with absence of ascites. Presence of PHG and its severity was significantly higher in group (A) patients.

The endoscopic examination findings of both groups are shown in table (1).



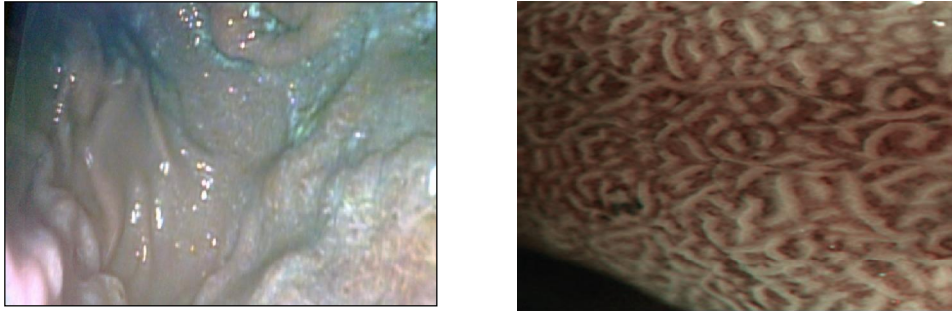
**Figure (1):** Normal gastric mucosa (a) NBI. (b) Magnified view.

**Table (1):** Comparison between both studied groups as regard UGI finding and PHG.

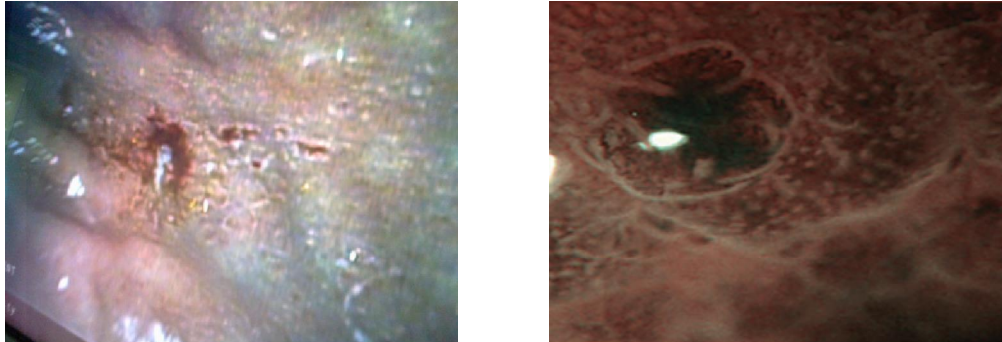
Variables	Group A N=50	Group B N=50	X <sup>2</sup>	P
<b>PHG by WLE</b>				
No PHG	2(4%)	18(36%)	<b>20</b>	<b>&lt;0.001 HS</b>
Mild PHG	24(48%)	24(48%)		
Severe PHG	24(48%)	8(16%)		
<b>PHG by NBI</b>				
No PHG	0	10(20%)	<b>18</b>	<b>&lt;0.001 HS</b>
Mild PHG	26(52%)	32(64%)		
Severe PHG	24(48%)	8(16%)		
<b>Oesophageal varices</b>				
No varices	4(8%)	23(46%)	<b>41</b>	<b>&lt;0.001 HS</b>
Small varices	19(38%)	27(54%)		
Large varices	27(54%)	0		

**Examination of liver cirrhosis patients with NBI revealed:**

- Normal mucosa by WLE appeared as rounded gastric pits with honey comb like capillaries surrounding the pits (figure 1).
- Mosaic like pattern appeared as extended and swollen gastric pits, with various degrees of dilated and convoluted capillaries surrounding the gastric pits.
- Reddening mucosa appeared as extended and swollen gastric pits, with various degrees of dilated and convoluted capillaries surrounding the gastric pits (figure 2).
- Red spots appeared as extended and swollen gastric pits, dilated and convoluted capillaries surrounding the gastric pits plus intra-mucosal hemorrhage around capillaries.
- 2 patients in group A and 8 patients in group B had normal gastric mucosa by WLE and on *switching to NBI technique* the capillaries surrounding gastric pits on the antral mucosa were dilated and convoluted which classify them as mild PH (table 1).
- NBI detects more cases of mild PHG



**Figure (2):** Mild PHG (a) NBI. (b) Magnified view.



**Figure (3):** Severe PHG (a) NBI. (b) Magnified view.

Comparing patients with and without PHG, it was found that patients with PHG had lower hemoglobin, albumin, platelet count, PC/SD and higher PT, INR, spleen diameter than patients without PHG and these differences were statistically significant (table 2).

**Table (2):** Comparison between cases with and without PHG by NBI as regard laboratory and sonographic data.

Variables	PHG by NBI		t- test	P
	No (10)	Yes (90)		
WBCs ( $\times 10^3/\text{mm}^3$ )	3.9 $\pm$ 0.2	3.6 $\pm$ 1	0.9	>0.05 NS
HB (g/dl)	11.6 $\pm$ 2	10.2 $\pm$ 2	2.2	<0.05 S
Platelets ( $\times 10^3/\text{mm}^3$ )	194.6 $\pm$ 34	102 $\pm$ 49	5.2	<0.001 HS
Albumin (g/dL)	3.4 $\pm$ 0.3	3 $\pm$ 0.6	2.2	<0.05 S
PT(second)	13.1 $\pm$ 3	15.5 $\pm$ 2	2.7	<0.05 S
INR	1.1 $\pm$ 0.2	1.3 $\pm$ 0.4	1.9	>0.05 NS
Spleen diameter (mm)	130 $\pm$ 18	158 $\pm$ 24	4	<0.001 HS
PC/SD	1501 $\pm$ 650	698 $\pm$ 320	5.1	<0.001 HS

The best cut off value of platelet count for prediction of PHG was  $135 \times 10^3$  when NBI was the detection method of PHG with better sensitivity and specificity than that of WLE (table 3)(figure 4, 5).

Also, when NBI is used to diagnose PHG, the best cut off value of platelet count / splenic diameter for prediction of PHG was 960, while on using WLE (the cut off value was 640) with higher specificity and overall diagnostic accuracy than WLE (table 3) (figure 4, 5).

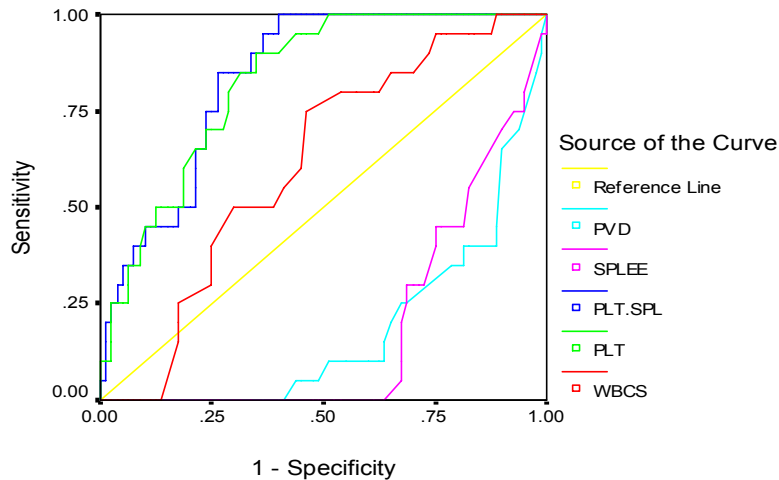
**Table (3):** Validity of different laboratory markers in prediction of PHG with WLE and NBI.

Variables	NBI			WLE		
	WBCs	platelets	PC/SD	WBCs	platelets	PC/SD
Best cut off	3.6x10 <sup>3</sup>	135 x10 <sup>3</sup>	960	3.5 x10 <sup>3</sup>	100 x10 <sup>3</sup>	640
AUC (area under curve)	0.68	0.91	0.92	0.61	0.82	0.84
Sensitivity	80%	92%	90%	80%	90%	90%
Specificity	50%	87%	80%	47%	72%	67%
PPV	72%	96%	93%	78%	93%	92%
NPV	51%	85%	83%	56%	75%	70%
Accuracy	60%	84%	80%	62%	76%	72%

Splenic diameter and portal vein diameter were poor predictors of PHG as shown in table (4) & (figure 4, 5).

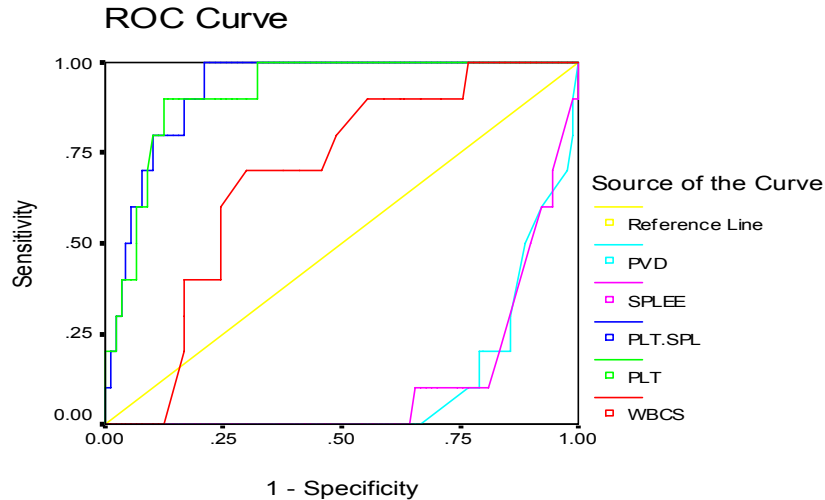
**Table (4):** Validity of radiological and endoscopic markers in prediction of PHG with WLE and NBI.

Variables	NBI			WLE		
	Splenic diameter	PVD	Eosophageal varices	Splenic diameter	PVD	Eosophageal varices
Best cut off	130	11		140	12	
AUC	0.11	0.18		0.19	0.19	
Sensitivity	44%	42%	85%	50%	47%	83%
Specificity	49%	52%	60%	40%	54%	65%
PPV	46%	50%	83%	45%	49%	85%
NPV	50%	53%	64%	51%	56%	69%
Accuracy	52%	49%	75%	55%	50%	73%



**Figure (4):** ROC curve to determine cut off value for best sensitivity and specificity of different markers in differentiation between patients with and without PHG when WLE is used to diagnose PHG.





**Figure (5):** ROC curve to determine cut off value for best sensitivity and specificity of different markers in differentiation between patients with and without PHG when NBI is used to diagnose PHG.

#### Discussion:

Portal hypertensive gastropathy (PHG) occurs as a complication of cirrhotic or non-cirrhotic portal hypertension. PHG is clinically important because it may cause acute (and even) massive, or insidious, blood loss. It is characterized by an endoscopic abnormality of the gastric mucosa that is classically described as a mosaic-like pattern that resembles the skin of a snake, with or without red spots (*Cubillas and Rockey, 2010*).

Abnormalities in the gastric microcirculation seem to be responsible for the congestion seen in PHG. However, some controversy remains as to whether this congestion is active or passive. Some data suggest that gastric mucosal blood flow might be decreased, but the total blood flow (blood flow in mucosa and submucosa of the stomach) may in fact be increased in PHG suggesting that PHG develops because of congestion caused by blockade of gastric blood drainage rather than by hyperemia (*Cubillas and Rockey, 2010*).

This study revealed that magnifying NBI technique by enhancing examination of microvascular architecture of gastric mucosa allow detection of more cases of PHG especially mild PHG

Regarding endoscopic NBI picture of PHG our results were in agreement with *Hayashi and Saeki. (2007)* who found that reddening mucosa was observed in 49% of cirrhotic patients with clinical or radiological evidence of portal hypertension, and on switching to NBI the gastric mucosa showed extended and swollen gastric pits, with various degrees of

dilated and convoluted capillaries surrounding the gastric pits.

Also Red spots, especially on the corpus mucosa and fornix mucosa, were observed by them in 36% of cirrhotic patients with clinical or radiological evidence of portal hypertension, and on switching to NBI the gastric mucosa showed extended and swollen gastric pits, dilated and convoluted capillaries surrounding the gastric pits plus intra-mucosal hemorrhage around capillaries (*Hayashi and Saeki, 2007*).

While the present study was in contradiction with *Hayashi and Saeki. (2007)* among group B patients as they found lower percentage of mild PHG (5%) and also lower percentage of severe PHG (5%) among cirrhotic patients without clinical or radiological evidence of portal hypertension and this could be attributed to the lower number of patients included in their study (22 patients) versus 50 patients in the present study and also to the difference in cirrhosis etiologies.

In the present study, the presence of PHG in cirrhotic patients without evidence of portal hypertension could be explained by the methods of diagnosis of portal hypertension based on clinical and radiological evidences only (like splenomegaly, dilated portal vein, thrombocytopenia less than 100,000 and presence of ascites) not on more invasive methods of diagnosis as measurement of hepatic venous pressure gradient. From this, the clinical and radiological parameters should be revised and more studies are needed to evaluate their accuracy and reliability in prediction of PHG.

In the present study, with NBI cirrhotic patients with PHG (90 patients) had lower platelet count than cirrhotic patients without PHG (10 patients) ( $102 \pm 49 \times 10^3$  versus  $194.6 \pm 34 \times 10^3$  with  $p$  value  $< 0.001$ ).

When NBI is used to detect PHG, the best cut off value of platelet count for prediction of PHG was (135.000) with sensitivity 92%, specificity 87%, PPV 96%, NPV 85% and efficacy 84%, while on using WLE the cut off value was (100.000) with sensitivity 90%, Specificity 72%, PPV 93%, NPV 75% and efficacy 76%.

Moreover, **Esmat and Omran, (2011)** put cut off value for platelet count ( $131 \times 10^3$ ) in prediction of portal hypertension, whereas sensitivity 84.15%, specificity 83.33 %. While **Mahassadi et al. (2012)** found that the best cut off value for platelet count in prediction of portal hypertension and oesophageal varices was (110.500) with sensitivity 80%, Specificity 69%, PPV 89.5%, NPV 51% and efficacy 77.5%.

The difference in values of platelet count between the present study and other studies could be explained by the difference in liver cirrhosis etiologies among the studied groups as noted by **Alempijevic et al, (2012)** who noted different platelet counts in alcoholic patients explained by the toxic effect of ethanol on bone marrow.

Thrombocytopenia is a common and highly specific manifestation of hypersplenism. It is suggested that the main mechanism of thrombocytopenia is splenic sequestration and pooling. Other mechanisms might be explained by insufficient synthesis of thrombopoietin (**Alempijevic et al., 2007, Sharma & Aggarwal, 2007**).

Moreover, cirrhotic patients with PHG had a statistically highly significant lower PC/SD than cirrhotic patients without PHG ( $698 \pm 320$  versus  $1501 \pm 650$  with  $p$  value  $< 0.001$ ).

Also, When NBI is used, the best cut off value of platelet count/ splenic diameter for prediction of PHG was (960) with sensitivity of 90%, specificity 80%, PPV 93%, NPV 83% and efficacy 80%, while on using WLE the cut off value was (640) with sensitivity 90%, Specificity 67%, PPV 92%, NPV 70% and efficacy 72%.

In a multicenter study using platelet count/spleen diameter ratio in the prediction of oesophageal varices, the cut-off value of 909 had sensitivity of 92% and specificity of 67% (**Giannini et al., 2006**).

Both portal vein diameter and splenic diameter were poor predictors of PHG.

The low sensitivity and specificity of the PVD in prediction of PHG could be explained by that the doppler haemodynamic parameters are affected by other factors such as the development of portosystemic collateral circulation (**Pozniak, 2002**).

Moreover, portal vein dilatation may occur in the absence of portal hypertension e.g. in response to huge splenomegaly or acute PV thrombosis (**Sabbá et al., 1992**).

Accordingly, the NBI with its higher accuracy than WLE in detection of PHG changed the cut off value of different non invasive markers in prediction of PHG.

By NBI, swelling of gastric pits with dilatation of capillaries surrounding the gastric pits and intra-mucosal hemorrhage were observed on the gastric mucosa in portal hypertension patients and so we concluded that NBI is considered as a novel technique that may enhance the accuracy of diagnosis of PHG in cirrhotic patients with portal hypertension and especially mild PHG and it changed the cut off value of different non invasive predictors of PHG so we recommend that not only cirrhotic patients with platelet count lower than  $100 \times 10^3$  but also those with platelet count lower than  $135 \times 10^3$  should undergo screening upper gastro-intestinal endoscopy so as not to miss early PHG changes.

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