www.jmscr.igmpublication.org
Impact Factor (SJIF): 6.379
Index Copernicus Value: 71.58
ISSN (e)-2347-176x ISSN (p) 2455-0450
crossref DOI: https://dx.doi.org/10.18535/jmscr/v6i4.111

IGM Publication
Journal Of Medical Science And Clinical Research
An Official Publication Of IGM Publication

Endoscopic and Histopathologic Features in Patients of Portal Hypertensive Duodenopathy

Authors
Dr Anil Sharma[1], Dr Mamta Sharma[2]
[1]Assistant Professor Gastroenterology, GMC Kota
[2]Assistant Professor Anesthesiology, GMC Kota
Corresponding Author
Dr Mamta Sharma
Assistant Professor Anesthesiology, GMC Kota

Abstract
Objective: *The aim of study is to describe the frequency of PHD in portal hypertension patients and its various clinical, endoscopic and histopathologic features.*
Methods: *Ninety four patients with cirrhosis and portal hypertention were studied. Two duodenal biopsies from first and second part were obtained during upper endoscopy and sent for histopathologic examination. Twenty five dyspeptic patients were taken as control.*
Results: *Endoscopic changes in duodenum during upper GI endoscopy were seen in 52 patients out of 94 patient (55.3%). These changes include erythema, erosion, ulcer and telangiectasia. Extending duodenal lesions were significantly higher in decompensated than in compensated cirrhotic patients. Erythema was the commonest lesion. ED (endoscopic duodenopathy) was significantly higher in patients of severe gastropathy (60.7%) than mild gastropathy (26.6%). Endoscopic Duodenopathy (ED) bleed was seen in 7.4%. ED was not related to variceal size and variceal bleed. Histopathologic changes includes capillary congestion, edema, apoptosis, fibrous changes and inflammation. These Histopathologic changes were not statistically significant between patients with or without ED.*
Conclusion: *Portal hypertensive duodenopathy is not related to variceal size or variceal bleeding but it is significantly higher in patient having severe gastropathy. Portal hypertensive duodenopathy can be a cause of overt or obscure bleeding. Histopathologic duodenopathy is more prevalent than endoscopic duodenopathy.*

Introduction

Well known gastrointestinal manifestation of portal hypertension includes esophageal, gastric and rectal varies. Portal hypertension is associated with development of mucosal changes in the gastrointestinal tract. McCormac et al[(1)] in 1985, gave detailed description of gastric mucosal changes associated with portal hypertension. There after it was shown that portal hypertension can affect all parts of the gastrointestinal tract and the entities have been given names according to regions involved - portal hypertensive gastropathy - PHG[(1)]; duodenopathy -PHD[(2)]; enteropathy-PHE[(3-5)] and colonopathy - PHC[(6)].

Despite the fact that upper endoscopy is a sensitive investigation in assessment of portal

hypertensive patients, only few studies in the literature has described lesions associated with the portal hypertension duodenopathy (PHD). Significance of PHD lesions lies in the fact that it can be a possible source of gastrointestinal bleeding.

A consensus definition of PHD is not available but various workers have considered many endoscopic and histologic features for diagnosis. Endoscopic finding of PHD can be classified after barakat et al[7] as - (a) mucosal erythema (patchy or diffuse) (b) mucosal edema (c) mucosal breaks (erosive or ulcers), and (d) vascular lesion (varices or telangiectasia). Vascular changes are the main histological features characterizing portal congestive process. They include capillary congestion/ dilatation and capillary angiogenesis [7,8]. Other changes are fibrous proliferation and increase apoptosis in a background of minimal or absent inflammatory cells.

The aim of study is to describe the frequency of PHD in portal hypertension patients and its various clinical, endoscopic and histopathologic features.

Patients and methods

Patients

The study included all patient with chronic liver disease and portal hypertension admitted to gastroenterology department, govt. medical college, kota between march 2017 to December 2017. They were total 94 patients (68 male and 26 female) with the age range of 18 to 70 yrs (42.86± 11.2). The study was approved by govt medical college ethics committee.

Methods

The diagnosis of chronic liver diseases was based on clinical data (*e.g.* ascites, jaundice, palmer erythema, muscle wasting and spider angioma), biochemical data (*e.g.* prolonged prothrombin time and decreased serum albumin) and ultra sonographic data (*e.g.* coarse bright liver echo pattern and nodular surface).

Grading of the severity of the chronic liver disease was based on the child's classification modified by pugh et al. Accordingly 32 patient were categorized to class A, 33 in class B and 29 in class C. Confirmation of portal hypertension was based on demonstration of esophagogastric varices on Upper GI Endoscopy examination in 89 patients or collateral channel on abdominal ultrasound examination in 5 patients. The etiology of chronic liver diseases was alcohol in 42, hepatititis B in 34, cryptogenic in 11, hepatitis C in 4 and autoimmune in 3.

Upper GI Endoscopy examination of each patient was done for detailed evaluation of esophagus, stomach and duodenum. Upper GI Endoscopy was done under sedation given by anesthetist. Gastric lesions were described in relation to their location (i.e. fundus, body and antrum) and their types as follows (a) mucosal erythema including (i) mosaic like pattern (ii) patchy or diffuse mucosal erythema. (b) Mucosal breaks including erosions and ulcers. Duodenal lesions were described in relation to their location (i.e. first part, second part or both) and their type are as follows (a) mucosal erythema (patchy or diffuse) (b) mucosal edema, (c) mucosal breaks (erosion or ulcer) and(d) vascular lesion (varices or telangiectasia). Two duodenal biopsies were obtained from every patient using endoscopic biopsy forceps, one from first part other from the second part. These biopsies were submitted in a vial containing 10% formalin. Paraffin sections were prepared, stained by H&E stain and masson's trichrome stain and examined for mucosal capillary congestion, extravasation, fibrous proliferation, edema, inflammation and apoptotic figures.

Twenty-five patients with dyspeptic symptoms were used as control. They were 15 men and 10 women with an age range of 18 to 70 yrs (33.0± 11.4).Two duodenal biopsies (one from first and other from second part of duodenum) were obtained. These patient have normal ultra sonography and normal upper GI endoscopy examination. Duodenal biopsy samples were further processed in similar manner as for portal hypertensive patients.

Statistics

Stastical analysis was performed using chi-square ($\chi 2$) test. The value at 0.05 was set as the critical level of significance.

Results

Endoscopic changes in duodenum

Endoscopic changes in duodenum during upper GI endoscopy were seen in 52 patients out of 94 patient (55.3%). As shown in table 1 , 24 patient (25.5%) had lesions restricted to first part, 6 patient (6.3%) had lesions restricted to second part, 22 patients (23.4%) had extending lesion in both first and second part of duodenum. These extending lesions were significantly higher in decompensated (Child-Pugh B and C) than in compensated (CTP - A) patients; 19/62 (30.6%) versus 3/32 (9.3%) respectively ($p<0.05$).

Various types of endoscopic lesions are shown in table 2.Erythema was the commonest lesion seen in 59.6% of patient of endoscopic duodenopathy (31/52). Erosion (21.5%) and ulcer (9.6%) were other lesions visualized. Fifteen patient out of 94 had mild gastropathy (16.0%) , while 79 patient (84%) had severe gastropathy.

ED (endoscopic duodenopathy) was significantly higher in patients of severe gastropathy (48/79, 60.7%) than mild gastropathy (4/7, 26.6%), $p< 0.05$.

PHD bleeding - During endoscopic examination, 7 patient had blood clots overlying the duodenal erosion and ulcer as stigmata of bleed (7/94, 7.4%). The bleeding was manifested as melena in 4 patients and was occult in three patients. Bleeding was self limiting in all patients.

Variceal bleeding – ED was present in 4 out of 10 patients having no varices (40%), in 27 of 48 (56.2%) and 17 of 36 (47.2%) having large varices, proving that there was no relationship between size of esophageal varices and endoscopic duodenopathy (ED). Endoscopic duodenopathy was present in 19 out of 40 patients having bleeding varices (47.5%) and 23 of 44 patients having no variceal bleeding (52.2%), $p >0.05$.

Histopathologic Changes

Mucosal capillary congestion (dilated capillaries filled with red blood cells) was seen in 57.7% (26/45) of those having ED, and also in 55.1% (27/49) of those having no endoscopic duodenopathy. There was no statistically significant difference between these two groups, $p >0.05$. Capillary congestion was not seen in any of the biopsies from control subjects. In the second part of duodenum capillary congestion was seen similar to that of first part (table 4).

Edema was the commonest change seen in 76.6% of biopsies from first part and 82.9% of biopsies from second part .There was no statistically significant difference seen between patient with or without ED ($p> 0.05$). Apoptotic figures were reported in 15.95% of biopsies from first and second part. There was no statistically significant difference between patients with or without ED. Minimal edema and apoptotic figures were seen only occasionally in control subjects.

Fibrous proliferation in lamina propria was seen less frequently in biopsies from first (5.3%) and second part (7.4%). There was no statistically significant difference between presence or absence of ED. Fibrous change was not seen in any of control patients.

Villous changes including shortened villi with a decreased or even reversed villous/crypt ratio were evaluated in second part only. These villous changes were seen in 7.4% of portal hypertensive patient with no statistically significant difference between patient with or without ED.

Inflammation was seen in 9.5% and 6.4% of patients from first and second part of duodenum. Inflammation was absent in all biopsies from control patients.

Table 1 Location of endoscopic duodenopathy in relation to CTP class

CTP class	Lesion only in 1st part	Lesion only in 2nd part	Lesion in both part
A (N = 32)	14 (43.7%)	3 (9.3%)	3 (9.3%)
B (N = 33)	5 (15.1%)	1 (3.0%)	9 (27.2%)
C (N = 29)	5 (17.2%)	2 (6.8%)	10 (34.4%)
TOTAL (N = 94)	24 (25.5%)	6 (6.3%)	22 (23.4%)

Table 2 various types of endoscopic lesions in the different part of the duodenum in 52 patients

Type of lesion	1st part only	2nd part only	Both parts
Patchy erythema	11	1	9
Diffuse erythema	6	2	2
Ulcer	3	1	1
Erosion	6	2	3
Telangiectasia	2	1	2
Total	28	7	17

Table 3 Histopathologic changes versus endoscopic changes in the duodenum first part in portal hypertensive patients

Histopathologic changes	Endoscopic duodenopathy (n=45)	No endoscopic duodenopathy (n=49)	Total (n=94)
Capillary congestion	26(57.7%)	27(55.1%)	53(56.3%)
Edema	34(75.5%)	38(77.5%)	72(76.6%)
Apoptosis	6(13.3%)	9(18.4%)	15(15.9%)
Fibrous changes	3(6.7%)	2(4.1%)	5(5.3%)
Inflammation	4(8.8%)	5(10.2%)	9(9.5%)

Table 4 Histopathologic changes versus endoscopic changes in the duodenum second part in the portal hypertension patients.

Histopathologic changes	Endoscopic duodenopathy (n=24)	No endoscopic duodenopathy (n=70)	Total (n=94)
Capillary congestion	13 (54.2%)	43(61.4%)	56(59.5%)
Edema	18 (75%)	60(85.7%)	78(82.9%)
Apoptosis	7 (29.1%)	8(11.4%)	15(15.9%)
Fibrous changes	4 (16.6%)	3(4.3%)	7(7.4%)
Villous changes	2(8.3%)	5(7.1%)	7(7.4%)
inflammation	2 (8.3%)	4(5.7%)	6(6.4%)

Discussion

Duodenal mucosal changes due to Portal hypertention have been described in different reports. In this study ED was reported in 55.3% of portal hypertention patients. Different figures have been reported in literature ranging from 8.4%[10] to 60%[11], which may be due to different patient selection criteria. Various authors described the occurrence of duodonopathy with worsening liver fuction. This study also shows that extending lesion (across first and second part of duedonum) have higher frequency in decompensated than compensated cirrhotic patients.

Different types of endoscopic duodenopathy lesions were seen. No lesion is specific for portal hypertensive duodonopathy. Commonest lesion were erythematous lesions. Next common lesions were erosions. Erythema was also commonest duodenopathy lesion in some other studies [12,13]. Varying types of lesions and patchy nature of lesions is because of varying local mucosal microcirculatory changes in portal hypertensive duodenopathy (PHD).

In this study PHD bleeding was seen in 7.4 % patient (overt or ocult). Episodes of overt bleeding were self limited. some case report[14] have presented massive bleed because of PHD lesions.

Endoscopic duodenopathy (ED) was found neither related to the esophageal varices size nor variceal bleeding. ED was related to severity of gastropathy. This means that factor which govern the development of portal hypertensive duodenopathy is not the high portal pressure itself but the point at which high portal pressure start to produce congestive changes.

During histopathologic examination capillary congestion was seen in 56.3% patient with no stastical difference between patients having ED or not having ED. This implies that in portal hypertensive patients normal looking intestinal mucosa during endoscopy doesn't mean that it is not having the changes of PHD. Edema of lamina propria was very common (76.6%) with no relation to ED. Edema results from increased capillary hydrostatic pressure in portal hypertention. Increased apoptosis was also found which had no relation with ED. Increased apoptosis can be because of decreased mucosal O2 level in congested mucosa. Fibrous proliferation and duodenal villous change were less common histopathologic finding and can be because of mucosal hypoxia.

In conclusion endoscopic duodenopathy is a common complication of portal hypertension, giving rise to varying type of lesions like erythema, edema, erosion, ulcer and telangiectasia. Extending lesions (involving both part of duodenum) are significantly more common in decompensated cirrhotic patients. Portal hypertensive duodenopathy is not related to variceal size or variceal bleeding but it is significantly higher in patient having severe gastropathy. Portal hypertensive duodenopathy can be a cause of overt or obscure bleeding. Histopathologic duodenopathy is more prevalent than endoscopic duodenopathy with similar prevalance in patients with and without endoscopic lesions.

References

1. T. T. McCormack, J. Sims, I. Eyre-Brook et al., "Gastric lesions in portal hypertension: inflammatory gastritis or congestive gastropathy?" Gut, vol. 26, no. 11, pp. 1226–1232, 1985.
2. S. Vigneri, R. Termini, A. Piraino et al., "The duodenum in liver cirrhosis: endoscopic, morphological and clinical findings," Endoscopy, vol. 23, no. 4
3. A. S. Nagral, A. S. Joshi, S. J. Bhatia, P. Abraham, F. P. Mistry, and I. M. Vora, "Congestive jejunopathy in portal hypertension," Gut, vol. 34, no. 5, pp. 694–697, 1993.
4. S. P. Misra, M. Dwivedi, V. Misra, and M. Gupta, "Ileal varices and portal hypertensive ileopathy in patients with cirrhosis and portal hypertension," Gastrointestinal Endoscopy, vol. 60, no. 5, pp. 778–783, 2004.
5. G. D. de Palma, M. Rega, S. Masone et al., "Mucosal abnormalities of the small bowel in patients with cirrhosis and portal hypertension: a capsule endoscopy study," Gastrointestinal Endoscopy, vol. 62, no. 4,
6. R. A. Kozarek, V. A. Botoman, J. E. Bredfeldt, J. M. Roach, D. J. Patterson, and T. J. Ball, "Portal colopathy: prospective study of colonoscopy in patients with portal hypertension," Gastroenterology, vol. 101, no. 5, pp. 1192–1197, 1991
7. M. Barakat, M. Mostafa, Z. Mahran, and A. G. Soliman, "Portal hypertensive duodenopathy: clinical, endoscopic, and histopathologic profiles," American Journal of Gastroenterology, vol. 102, no. 12, pp. 2793–2802, 2007.
8. V. Misra, S. P. Misra, M. Dwivedi, and S. C. Gupta, "Histomorphometric study of portal hypertensive enteropathy," American Journal of Clinical Pathology, vol. 108, no. 6, pp. 652–657, 1997.
9. Pugh RNH, Murray-Lyon JM, Dawson JL, et al. Transection of oesophagus for

bleeding oesophageal varices. Br J Surg 1973;60:646–9.

10. Menchen L, Ripoli C, Martin-Jimenez I, et al. Prevalence of portal hypertensive duodenopathy in cirrhosis: Clinical and haemodynamic features. Eur Gastroenterol Hepatol 2006;18:649–53.
11. Boron-Kaczmarzka A, Hryniewicz A, Kemona A, et al. Morphologic changes of small intestine epithelium in the course of post-alcoholic liver cirrhosis. Drug Alcohol Depend 1990;25:299–303
12. Gupta R, Saraswat VA, Kumar M, et al. Frequency and factor influencing portal hypertensive gastropathy and duodenopathy in cirrhotic portal hypertension. J Gastroenterol Hepatol 1996;11:728–33.
13. Desai N, Desai D, Pethe V, et al. Portal hypertensive jejunopathy: A case control study. Indian J Gastroenterol 2004;23:99–101
14. Santoro GA, Aiello C, Galloro G, et al. Massive lower gastrointestinal haemorrhage in patients with portal hypertensive enteropathy: A report of two cases. Hepatogastroenterology 1997;44: 1029–32.