AN UPPER GASTROINTESTINAL ENDOSCOPIC STUDY OF FIFTY CASES OF CIRRHOSIS LIVER

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SUMMARY

To find out the incidence of different upper gastrointestinal (UGI) lesions like esophageal and or gastric varices (EV or GV), congestive gastropathy (CG) and peptic ulcer (PU) in patients with cirrhosis liver and their association amongst themselves and the degree of hepatic dysfunction, 50 cases with proven cirrhosis were subjected to UGI endoscopy. 32(64%) patients had EV (10 grade I, 17 grade II, and 5 grade III). Grade II and III EV were associated with Child's grade B and C liver disease ($p<0.01$). 16/22 (72.72%) cases of the grade II & III EV had red colour sign (RCS) ($p<0.01$). 14(28%) patients had history of haematemesis or melena in the past and out of them 12 cases had RCS ($P<0.01$). GV were seen in 14(28%) cases and all of them had EV as well but there was no association between the grades of GV and EV. 28(56%) cases had CG which was independent of the grades of EV or liver disease. No significant increase in the incidence of PU was seen in the cases studied. There was a good correlation between the grades of EV and RCS and the degree of hepatic dysfunction while gastric signs were independent of either of them.

INTRODUCTION

Cirrhosis liver is a common, often progressive and usually fatal disorder. Upper gastrointestinal (UGI) bleeding is a leading cause of death in patients with liver cirrhosis. In most cases the hemorrhage originates from EV or from CG.[1] The average risk of bleeding in cirrhotic patients with EV who have not bled previously is 30%.[2] The mortality rate is twice of an EV bleed than that of a non-EV UGI bleed in cirrhotics[3] and there is 40% risk of death in the initial episode[4] with 50% mortality at 6 weeks[2] and only 30% survival at one year.[4] This high mortality rate is the rationale for the prophylaxis. UGI lesions lend themselves to an easy diagnosis and management by UGI endoscopy. A lot of emphasis is being placed on both primary prophylactic sclerotherapy[5,6,7] or sclerotherapy after the first bleed to prevent the rebleed.[8,9]

Besides, pharmacotherapy in the form of beta blockers[10,11] nitrates[12] or both in combination[13] has also been shown to be very effective in reducing the incidence of primary or secondary bleed by reducing portal hypertension. As sclerotherapy is not an innocuous procedure, it becomes highly prudent to detect patients at high risk of bleeding and only then subject them to prophylactic sclerotherapy.[9]

UGI endoscopy alone or in combination with clinical data may be utilized to identify patients at high risk of bleeding. This study

was done to correlate the endoscopic findings with the clinical stage of the disease and to identify the endoscopic signs in patients at risk of bleeding.

MATERIAL AND METHODS

This study was conducted in Hayat Shaheed Teaching Hospital from September 1991 to March 1992. All patients seen and suspected of having cirrhosis were admitted. Detail history and meticulous clinical examination was followed by liver functions tests, prothrombin time, ascitic fluid examination (when present) and abdominal ultrasound studies. The diagnosis was confirmed by liver biopsy in all except those who had either a prolonged prothrombin time (not correctable by Vitamin K treatment) or had tense ascites. Uncooperative or hepatic coma patients were excluded from the study due to inability to perform endoscopy. Patients underwent routine UGI endoscopy and findings were recorded with the following protocol.

1) The size of the Esophageal Varices (EV).[14]

a) Grade I: Flattened by insuflation, separated by areas of normal mucosa.

b) Grade II: Not flattened by insuflation but separated by areas of normal mucosa.

c) Grade III: Confluent varices not flattened by insuflation and not separated by normal mucosa.

2) The red colour sign (RCS).[15,16]

a) Cherry red spots (small red spots about 2mm in diameter).

b) Haematocystic spots (large round crimson projections resembling blood blisters more than 4mm in diameter).

c) Red wale markings (longitudinal dilated venules resembling whip marks).

d) Diffuse redness.

3) The size of Gastric Varices (GV).[15]

a) Grade I: Suspicion of varices (dilated or unusually visible veins and or enlarged tortuous gastric folds)

b) Grade II: Obvious varices with cerebriform appearance.

TABLE – I

GRADE OF VARICES VS. LIVER DISEASE

GRADE OF LIVER DISEASE	(N)	GRADE OF ESOPHAGEAL VARICES		
		I	II	III
CHILD GROUP A	18	3	—	—
CHILD GROUP B	27	7	15	2
CHILD GROUP C	05	0	2	3
TOTAL	50	10	17	5

TABLE – II

RED COLOR SIGN

GRADE OF EV	(n)	RED COLOR SIGN	
		PRESENT	ABSENT
I	10	—	10
II	17	11	6
III	5	5	0
TOTAL	32	16	16

4) Congestive gastropathy (CG).[17]

   a) Mild congestive gastropathy:

      i) A fine pink speckling or scarlatina type rash.

      ii) Superficial reddening, particularly on the surface of the rugae giving a striped appearance.

      iii) Raised red edematous mucosa resembling snake skin separated by a fine white reticular pattern.

   b) Severe congestive gastropathy:

      i) Discrete red spots (resembling cherry red spots on EV) which may become confluent and bleed.

      ii) A diffuse hemorrhagic gastritis.

5) Peptic ulcer (PU):

   a) Gastric ulcer.

   b) Duodenal ulcer.

The procedure was performed in left lateral position without any premedication under pharyngeal local anaesthesia with 2% lignocaine using Olympus GIF instrument. To assess the degree of hepatic dysfunction Child-Turcotte classification was used (18).

RESULTS

Fifty patients were included in the study. Mean age was 42.4 years ranging from 20 to 65 years. There were 32 males and 18 females. 6 patients were Afghan refugees. 20(40%) were positive for Hepatitis B surface antigen while two cases were alcoholics. Only 16(32%) patients volunteered history of Jaundice and 14(28%) patients history of haematemesis and or melena in the past. Jaundice was present in 18(36%) on presentation while 4(8%) were in hepatic precoma with confusion, disorientation and asterexis.

Ascites was present in 32(64%) of these patients and later on all those 22 patients who had moderate to tense ascites were found to have EV ($p < 0.01$). 18(36%) patients were in Child's group A, 27(54%) in Child's group B and 5(10%) patient were in Child's group C. 32(64%) cases had EV (10 Grade I, 17 Grade II and 5 Grade III varices). There was no difference in age and sex in the distribution of EV. In Child's group A, 15 patients did not have EV while 3 patients had grade I varices. In Child's group B, 24 patients had EV ($p < 0.01$), 7(14%) cases of grade I, 15(30%) of grade II and 2(4%) of grade III esophageal varices] while all 5 patients with Child's group C disease had EV (2 had grade II and 3 had grade III EV).

16/22 (72.72%) of the total patients with grade II and III EV had RCS (P <0.01), which was highly associated with grade II (70% i.e 12 out of 17 cases) and grade III (80% i.e 4 out of 5 cases) EV. No RCS was present on grade I EV.

GV could be demonstrated in 14(28%) of the cases and all of them were associated with EV. 9(18%) were of grade-I and 5(10%) were of grade II. No RCS was seen on GV.

28(56%) patients had CG and half of these had none or grade-I EV.

8(16%) patients had PU [5(10%) Duodenal and 3(6%) Gastric]. No relationship was found between the presence of PU and severity of EV or degree of hepatic dysfunction.

No complications were encountered during endoscopies thus proving it to be a safe procedure.

DISCUSSION

The results of this study differ a little from other studies. In this study EV were observed in 64% of the cases [vs 80% in Cales et al[15] study] and RCS in 72.72% cases [vs 40% in Cales et al[15] and 76% in Beppu et al[19] studies]. CG in 56% patients [vs 98% in Cales et al[15] series and 53% in McCormack et al[17] study] and GV in 28% as compared to 31% by Cales et al[15] and 16% in Palmer's study.[20] The difference in the percentages is not wide as far as the incidence of EV and RCS is concerned. However the difference is very wide between our study and Cales et al study in the detection of CG. This difference may be related to the intake of alcohol which is the main cause of cirrhosis in western society. Alcohol is a gastric secretagogue and irritant[21] and it has been found that the incidence of gastritis is higher in alcoholics than in non-alcoholic cirrhosis.[22] Secondly the difference may be due to interobserver variability which may become pronounced and significant for signs of lesser grade.[15]

We have found a very high incidence of RCS on grade II (70%) and grade III (80%) EV. This is in confirmation with international studies which have shown that the prevalence of red wale marking doubles as the grade of varices increased.[23] Several studies have also confirmed in prospective controlled trials the importance of RCS in the prediction of EV bleed.[19,23,24] There was a positive correlation between hepatic dysfunction and grade of EV as 70% of grade I and 100% of grade II and III EV were in Child's group B or C. There are various studies, both in favour[15,23] and against[24,25] this positive correlation between the grade of EV and the degree of hepatic dysfunction, and the actual relationship between these factors is still obscured. Mild (in 38%) or severe CG bore no relationship to the presence of EV nor to hepatic dysfunction. According to Iwato et al[26] they are of low sensitivity and specificity in the diagnosis of portal hypertension as compared to the presence of EV and GV). We found PU in 16% (10% duodenal and 6% gastric) and considering the fact that the incidence of duodenal ulcer in normal population may be 15%[27] its association with the cirrhosis cannot be stressed upon.

In conclusion, it can be stated that expertly performed UGI endoscopy appears, in most instances, to offer the best available method for identifying the lesions in UGI tract in cirrhotics. Keeping in view the prophylaxis for bleeding one can offer to cirrhotics with EV in the form of prophylactic sclerotherapy or pharmacological agents, early diagnosis becomes imperative and important. Therefore every diagnosed case of cirrhosis must undergo UGI endoscopy as early as possible to determine and plan further management.

REFERENCES

1. Cioni G, Tincani E, Cristani A, et al. Does the measurement of portal flow velocity have any value in the identification of patients with cirrhosis at risk of digestive bleeding? Liver. 1996; 16(2): 84.
2. Burroughs AK. The natural history of Varices. J Hep 1993; 17(2): S10.
3. Thomas E, Rosenthal WS, Ryner W, Katz D. Upper gastrointestinal hemorrhage in patients with alcoholic liver disease and esophageal varices. Am J Gastroenterol. 1979; 72: 623.
4. Graham DV, Smith JL. The course of patients after variceal hemorrhage. Gastroenterology. 1981; 8: 800.
5. Witzel L, Wolberg E, Merki H. Prophylactic endoscopic sclerotherapy of esophageal varices: a prospective controlled study. Lancet. 1985; 1: 773.
6. Paquet KJ, Kalk JF, Klein CP, Gad HA. Prophylactic sclerotherapy for esophageal varices in high risk cirrhotic patients selected by endoscopic and hemodynamic criteria: a randomized, single center controlled trial. Endoscopy. 1994; 26(9): 729.
7. Koch H, Binmoeller KF, Grimm H, et al. Prophylactic sclerotherapy for esophageal varices: long term results of a prospective study. Endoscopy. 1994; 26(9): 729.
8. Kiire CF, Gangaidzo IT, Sitima J, Ndemera B. Endoscopic sclerotherapy in Zimbabwe. Cent Afr J Med. 1993; 39(9): 177.
9. Terblanche J, Borroughs AK, Hobbs KEF. Controversaries in the management of bleeding esophageal varices (second of two parts). N Eng J Med 1989; 320: 1469.
10. Qureshi H, Zuberi SJ, Najmuddin S, et al. Propranolol in bleeding esophageal varices. Pak Med Res 1989; 28: 16.
11. Avgerinos A, Reikomis G, Klonis C, et al. Propranolol in the prevention of recurrent upper gastrointestinal bleeding in patients with cirrhosis undergoing sclerotherapy: a randomised controlled trial. J Hepatol 1993; 19(2): 301.
12. Angelico M, Carli L, Piat C, et al. Isosorbide-5-mononitrate versus propranolol in the prevention of first bleeding in cirrhosis. Gastroenterology. 1993; 104(5): 1460.
13. Candid V, Joaquim B, Maria T, et al. Nadolol plus Isosorbide mononitrate compared with sclerotherapy for the prevention of variceal bleeding. N Eng J Med. 1996; 334: 1624.
14. Watanabe K, Kimura K, Matsutani S, et al. Portal hemodynamics in patients with gastric varices. Gastroenterology. 1988; 95: 434.
15. Cales P, Zabatto B, Meskens C, et al. Gastroesophageal endoscopic features in cirrhosis: observer variability, interassociation and relationship to hepatic dysfunction. Gastroenterology: 1990; 98: 156.
16. The North Italian Endoscopic Club for the study and treatment of esophageal varices: prediction of the first variceal hemorrhage in patients with cirrhosis of the liver and esophageal varices: a multicenter study. N Eng J Med; 319:983.
17. McCormack TT, Sims J, Eyre-Brook I, et al. Gastric lesions in portal hypertension: inflammatory gastritis or congestive gastropathy? Gut 1985; 26: 1226.
18. Child CG, Turcotte JG. Surgery and Portal Hypertension, in The Liver and Portal Hypertension. Child CG bed. Philadelphia, W B Saunders company 1964; 50.
19. Beppu K, Inokuchi K, Koyanagi N et al. Prediction of variceal hemorrhage by esophageal endoscopy. Gastrointest Endosc 1981; 27: 213.
20. Palmer ED. Endoscopic contribution to the understanding of portal hypertension. Ann NY Acad Sci 1970; 170: 164.
21. Kauer CM, Silver man S Jr. Alimentary Tract and Liver, in Current Medical

Diagnosis and Treatment. Schroeder SA, Krupp MA, Tierny LM, McPhee SJ (eds). Apleton and Lange 1990; 441.

22. Robinovitz M, Kumar S, Kajani M, et al. Combined upper and lower gastrointestinal endoscopy: a prospective study in alcoholic and nonalcoholic cirrhosis. Alcoholism (NY) 1989; 13: 790.

23. Colombo E, Casiraghi MA, Minoli G, et al. First bleeding episode from esophageal varices in cirrhotic patients: a prospective study of endoscopic predictive factors. Ital J Gastroenterol. 1995; 27(7): 345.

24. Sarin SK, Sundram KR, Ahuja RK. Predictors of variceal bleeding: an analysis of clinical, endoscopic and hemodynamic variables with special reference to intravariceal pressure. Gut 1989; 30: 1757.

25. Buset M, Marez BD, Baize M, et al. Bleeding esophagogastric varices: an endoscopic study. Am J Gastroenterol 1987; 82: 241.

26. Iwao T, Toyonaga A, Ikigami M, et al. McCormack's endoscopic signs for diagnosing portal hypertension: comparison with gastroesophageal varices. Gastrointest Endosc 1994; 40(4): 470.

27. Kumar PJ, Clark ML. Peptic Ulcer, in Clinical Medicine, 3rd ed. Bailliere Tindall, W B Saunders, London. P-191.