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Hospital Topics

Emergency Endoscopy after Gastrointestinal Haemorrhage in 50 Patients with Portal Hypertension

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Summary

Endoscopy was carried out in 50 patients with oesophageal varices within 24 hours of a major haematemesis or melaena. Sources of bleeding were identified in 42 of the cases and in only 19 patients was bleeding due to oesophageal varices. Bleeding from gastric varices was present in 11 patients, and a variety of acute and chronic lesions made up the remainder. In contrast with previous series haemorrhage from erosive gastric lesions was seen in only five patients and was no more common in 23 patients with alcoholic cirrhosis than in the group as a whole.

Introduction

Emergency endoscopy is a safe and reliable means of diagnosing the source of upper gastrointestinal bleeding.^{1,2} Accurate diagnosis of the bleeding site is particularly important in patients with portal hypertension since reports from Europe and the U.S.A. of emergency endoscopy have shown a high incidence of bleeding from lesions other than oesophageal varices. These have included erosive gastritis, chronic peptic ulcer, and Mallory-Weiss tears.³⁻⁹ There has been no detailed study of patients admitted to a British unit and we describe here the findings in 50 patients with portal hypertension in whom emergency endoscopy was carried out within 24 hours of a major haematemesis or melaena.

Patients and Methods

The 50 patients came from a series of 55 consecutive patients admitted between November 1972 and March 1974. Four patients considered too ill for endoscopy and one violent alcoholic patient were excluded. Thirty-one of the patients were male with a mean age of 50 years (range 20-64) and 19 were female with a mean age of 49 years (range 8-68). Portal hypertension was secondary to chronic liver disease except in two patients with portal vein thrombosis (table I).

TABLE I—Aetiology of Portal Hypertension in 50 Patients

Diagnosis	No of Patients
Cirrhosis:	
Alcoholic	23
Cryptogenic	8
Active chronic hepatitis	8
Primary biliary cirrhosis	6
Wilson's disease	1
Schistosomiasis	1
Extrahepatic portal vein obstruction	2
Budd-Chiari syndrome	1

Emergency endoscopy was carried out within three hours of haematemesis or melaena in 24 of the patients and within 24 hours in the remainder. An end-viewing ACMI 7089P endoscope was used with the patient in the left lateral position. Most patients were examined under sedation with intravenous diazepam (5-25 mg), but in 17 patients with signs of moderate or severe encephalopathy sedation was withheld. In no case was premedication used. A lesion was recorded as the bleeding site if it was seen to be bleeding, if blood clot was adhering to it, or if no other abnormality was seen during the course of a complete examination, which occurred in only four patients.

Results

Oesophageal varices were seen in all the patients, and in 17 cases gastric varices were also present. Oesophageal varices were identified as the sole source of bleeding, however, in only 16

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TABLE II—Endoscopic Diagnosis of Lesions Responsible for Bleeding in 50 Patients

	No. (%) of Patients
<i>One Bleeding Site</i>	
Oesophageal varices	16 (32)
Gastric varices	9 (18)
Oesophagitis	2 (4)
Gastritis	2 (4)
Duodenitis	2 (4)
Acute gastric ulcer	1 (2)
Acute duodenal ulcer	2 (4)
Chronic gastric ulcer	1 (2)
Chronic duodenal ulcer	3 (6)
<i>Two Bleeding Sites</i>	
Oesophageal varices and chronic gastric ulcer	1 (2)
Oesophageal varices and gastritis	1 (2)
Oesophageal varices and gastric varices	1 (2)
Gastric varices and gastritis	1 (2)

*In eight patients the cause of bleeding was not found.

patients, including the four patients in whom the diagnosis was made by exclusion. Eleven patients were bleeding from gastric varices and if four patients who were bleeding from more than one site are included (table II) oesophageal or gastric varices were responsible for bleeding in 29 patients. Duodenal varices were seen in two patients but were not bleeding.

Acute erosive lesions of the mucosa in the oesophagus, stomach, and duodenal bulb were observed in 20 patients and were the source of bleeding in 11, including five patients who were bleeding from acute erosive gastric lesions. There was no difference in the frequency of bleeding from acute lesions in the 23 patients with alcoholic cirrhosis and the remaining 27 (table III), but acute mucosal lesions were the cause of the bleeding in six of the eight patients with active chronic hepatitis in the series, all of whom were receiving prednisone. Only five patients had taken compounds containing aspirin before their haemorrhage, and in only one of them were acute mucosal lesions the site of bleeding. Chronic gastric ulcers were seen in two patients and in both were the source of bleeding. Five patients had chronic duodenal ulcers, but in only three of them was this the cause of the bleeding. In eight patients it was not possible to identify the source of bleeding, in five because of the volume of blood retained in the stomach and in three because more than

one lesion was present with nothing to indicate which had bled. In one of the patients a chronic gastric ulcer was obscured by blood but was found when the examination was repeated the next day.

Endoscopy was generally well tolerated and complications were rare. One patient examined early in the series, who was given 15 mg diazepam, did not regain consciousness for 72 hours. One seriously ill patient who was examined without sedation died shortly after endoscopy, probably from exsanguination, and in another patient fresh bleeding from a fundal varix was seen as the endoscope was being withdrawn, which had not been present when the instrument was being passed.

A barium meal examination was carried out in 27 of the patients within two weeks of the haemorrhage and oesophageal varices could be shown in 25. The presence of gastric varices was queried in five patients, in three of whom gastric varices had also been seen at endoscopy. There were 12 patients, however, in whom gastric varices were seen at endoscopy which could not be detected on the barium meal examination. The findings on barium meal examinations and on endoscopy agreed in two patients with gastric ulcers and five with chronic duodenal ulcers though endoscopy had shown the ulcer not to be the site of bleeding in two of the latter. As expected, no radiological abnormalities could be identified in association with acute superficial lesions which were the cause of bleeding in 11 patients.

Of the 50 patients in this series 13 were managed conservatively and survived to leave hospital, six of these subsequently having a successful portosystemic anastomosis. Thirteen patients were submitted to emergency surgery and seven of these survived. The remaining 24 patients never became fit for surgery and died from uncontrollable haemorrhage or hepatic failure. A necropsy was carried out one to 25 days after the admission in 20 cases, but in none was any chronic lesion discovered which had not been seen at endoscopy. We did not attempt to correlate the presence of acute superficial lesions at endoscopy and necropsy since such lesions may appear and heal within a few days.

Discussion

The frequency of bleeding from oesophageal varices in the various endoscopic studies reported of patients with cirrhosis admitted with gastrointestinal bleeding has been variable (table IV). Some of the series, however, included patients who did not have portal hypertension. In the two studies reported from the U.S.A., in which all the patients had oesophageal varices, the frequency of bleeding from oesophageal varices was 27%⁵ and 18%.⁹ There has been a strikingly high incidence of bleeding from erosive gastritis in all previous reports. Though we often observed patchy or diffuse erythema in the fundus, body, or antrum of the stomach, erosive gastritis was an uncommon source of haemorrhage in our patients. Almost all of

TABLE III—Comparison of Bleeding Sites in 23 Patients with Alcoholic Cirrhosis and in Patients with Other Diagnoses

Site	Alcoholic Patients	Other Patients
Oesophageal varices	9	10
Oesophagitis	0	2
Gastric varices	5	6
Gastritis	2	1
Acute gastric ulcer	0	1
Chronic gastric ulcer	2	0
Duodenitis	1	1
Acute duodenal ulcer	1	1
Chronic duodenal ulcer	2	1
Unknown	3	5

TABLE IV—Sites of Bleeding in Series of Patients with Cirrhosis as Reported by Previous Authors and Compared with Present Series

	No. of Patients	% Alcoholic Patients	Site of Bleeding (%)						Unknown	
			Oesophageal Varices	Oesophagitis	Mallory-Weiss Tears	Gastric Varices	Erosive Gastric Lesions	Chronic Peptic Ulcer		
<i>Oesophageal Varices not always Present</i>										
Europe:										
Paris <i>et al.</i> (1972) ^a	?	?	30				50	12		8
Peeters and Cremer (1972) ^a	86	53	25		5		23	14		5
U.S.A.:										
Bonanno <i>et al.</i> (1972) ^a	77	?	43	17	18		21	19		
Mobarhan <i>et al.</i> (1972) ⁷	30	100	63				27	6		3
Khadoost <i>et al.</i> (1971) ^a	167	94	51				38			
<i>Oesophageal Varices always Present</i>										
U.S.A.:										
Da Gradi <i>et al.</i> (1970) ^a	121	*	27	2	11	12	31	8		5
McCray <i>et al.</i> (1969) ^a	27	100	18				59	15		7
Present series	50	46	38	4		22	10	10		16

*Almost exclusively alcoholic (A. E. DaGradi, personal communication).

the patients in previous series have had alcoholic cirrhosis compared with only 46% of our patients, but the distribution of lesions shown to be bleeding in this group was similar to that for the complete series. Few of our patients, however, were admitted after an acute drinking bout. Among previous authors only DaGradi *et al.*⁵ seem to recognize haemorrhage from gastric varices, which we found to be a relatively common site.

Failure to locate the source of bleeding was more common in our series than in previous reports, but our criteria may have been more stringent. Retained blood was a problem in only five cases and we did not use gastric lavage because it may produce artefactual bleeding.¹⁰ In 40% of those examined within three hours and 60% of those examined later the bleeding had already stopped and if multiple lesions are found then it may be impossible to decide which was bleeding. Thus endoscopy should be performed as soon as blood volume has been restored so that if bleeding continues the most appropriate treatment can be selected.

Moderate or large oesophageal varices can be shown without difficulty on barium meal examination, but it may be difficult to obtain the appropriate films in severely ill patients. In this series, however, though oesophageal varices were present in every case they were the source of bleeding in only 19 patients. So far we have been unable to establish any definite radiological criteria for the diagnosis of gastric varices, which were the source of bleeding in 11 cases. A double contrast technique may help with this problem. Though in this series chronic peptic ulcers were always correctly diagnosed from the barium meal examination it has been estimated that 20%-40% of duodenal ulcers are missed by standard barium meal examinations,¹¹ and acute superficial lesions, which were the source of bleeding in nearly a quarter of our patients, cannot be diagnosed at all. When

multiple lesions have been shown radiographically there is also no means of knowing which was responsible for the haemorrhage. In our view an emergency barium meal examination is not indicated when an adequate endoscopic examination can be made.

The main hazard of endoscopy in patients with liver disease is that the sedation required may precipitate hepatic encephalopathy, and diazepam should be given with the greatest caution. Though it is often possible to withhold sedation it is also important that the patient should not become agitated as this may precipitate further bleeding. In our experience alcoholic patients are particularly apt to become violent after diazepam. Endoscopy in patients such as ours requires considerable experience and should not be attempted by the novice. Though at present we cannot prove that the information gained leads to better management and a lower mortality we would suggest that the potential hazards of the procedure are more than outweighed by the advantages of obtaining accurate information about the source of bleeding.

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Today's Treatment

Blood and Neoplastic Diseases

Thrombosis: Treatment and Prophylaxis

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Thrombosis is the conversion of blood from a fluid to a gel-like state inside a blood vessel during life. Two important mechanisms are concerned: the conversion of fibrinogen to fibrin with the entrapment of red blood cells within the fibrin mesh, and the adhesion of platelets.

Effect of Thrombosis

Thrombosis in an artery will partially or totally occlude the lumen and so restrict the blood flow. This prevents blood reaching the tissue which the artery supplies. The survival

of the tissue depends upon the rate of occlusion, the presence or absence of collateral vessels, and the sensitivity of the tissues to anoxia. When the artery is an end artery the tissue will die, but in most places collateral vessels exist which save the tissue, though the blood flow rarely returns to normal. Some tissues, such as the brain and heart muscle, are very sensitive to ischaemia and will die after a few minutes of anoxia. The ischaemia caused by arterial thrombosis can be made worse by further thrombosis, blocking the entry of blood from collateral vessels, and by distal occlusion by small emboli from the primary thrombus.

Thrombus in a vein has three major effects. Firstly, blood flow is prevented, causing pain and swelling in the tissues being drained by the vein concerned. Secondly, the thrombus may fragment and move on through the heart to obstruct the pulmonary circulation, and thirdly, the thrombosis may destroy the valves. The effects of venous thrombosis are exacerbated by extension of the thrombus and the absence of collateral vessels.

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