

Incidence and Significance of Haematemesis in Cirrhosis

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SUMMARY

A retrospective study of 228 patients with cirrhosis admitted to Harare Hospital over a 3-year period was carried out to determine the incidence of haematemesis, and to assess the prognosis of patients with gastrointestinal haemorrhage as compared with those where liver failure had progressed to the extent that they had developed encephalopathy and coma. It was found that more patients fell into the latter group (50 cases) than into the group with haematemesis (with or without encephalopathy) (45 cases), but there was no significant difference in the mortality rate of the groups.

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It has for some time been a clinical impression at Harare Hospital that bleeding from the gastro-intestinal tract (oesophageal varices) is an uncommon cause of death in patients with cirrhosis and portal hypertension. The view was that death occurs from liver failure *per se* more often than from haemorrhage, although opinion was not available as to whether this was because of a low incidence of haemorrhage or because gastro-intestinal bleeding tended not to be severe. Clinical impressions, however, seldom reflect the true situation accurately and are often at considerable variance with the facts. In order to establish the relationship between cirrhosis and haematemesis and causes of death, it was decided to do a retrospective study of all patients in whom a diagnosis of cirrhosis was made in the professorial medical wards over a 3-year period from 1969-1971.

The study is open to all criticisms of retrospective surveys, particularly in that one is not able to plan with any great precision the analysis of the parameters in which one is interested, since invariably some investigations are incomplete or have been left out entirely, often for justifiable reasons.

However, notwithstanding these defects, we believe that we have been able to obtain some valid figures on the subject, which will at least put the incidence and significance of haematemesis as a cause of death in cirrhosis on a more factual footing. These results may provide a stimulus for the initiation of a well-designed prospective study, with clear terms of reference, into the problems and complications of liver disease in this country.

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METHODS

The notes of all patients with a diagnosis of cirrhosis who had been admitted to the professorial unit between 1969 and 1971 inclusive, were analysed. There were 228 such patients and there was no significant difference in the average yearly admission rate for cirrhosis during this period.

The group comprised those patients in whom the main diagnosis was one of cirrhosis with or without complications, and patients who were admitted primarily with other conditions, but who on examination were found incidentally to have cirrhosis.

It would appear apposite at this juncture to comment briefly on the type of cirrhosis seen at Harare Hospital. It has been established that most of the cirrhosis is of the cryptogenic variety, since in the majority of our patients no cause can be discovered. Histologically the cirrhosis is usually of the macronodular type. However, we were not concerned in this study with investigating this particular aspect of liver disease, and so details of this nature are not included.

Criteria for Diagnosis

The diagnosis of cirrhosis was primarily made on clinical grounds—the finding of an enlarged hard liver with or without an enlarged spleen or the presence of splenomegaly, with other stigmata of portal hypertension and abnormal liver function tests. There were often associated features of cirrhosis, such as porphyria cutanea tarda, and in a significant number of patients ascites was present. Serum protein estimations were done on the majority of patients (94%) and were only omitted in those patients who were admitted in a terminal state and who died before blood could be taken for investigation. Of those in whom the serum albumin was calculated, 28% had an albumin level of less than 2 g/100 ml and 49% had a level of between 2 and 3 g/100 ml. Only 23% of patients had a serum albumin within the normal range.

Liver biopsies were performed in a few patients but this was not considered necessary to establish the diagnosis. A distinction was not made between cirrhosis and bilharzial fibrosis for the purposes of this study, and most of the patients under 20 years of age probably had bilharzial fibrosis rather than cirrhosis.

A barium swallow examination was performed in 75 patients (33%) and the details are shown in Table I. Those patients who had haematemesis were assumed to be bleeding from oesophageal varices, although it must be admitted that this was not proved.

TABLE I. FINDINGS OF BARIUM SWALLOW STUDIES

	Haematemesis	Melaena only	No GiT bleeding
Total	45	17	168
No. of barium swallows	16	9	50
Varices	13	4	20

RESULTS

Fig. 1 gives an over-all analysis of the age and sex of all the patients in the study, together with the mortality in each age group. It can be seen that the maximum incidence of cirrhosis was in the 6th decade in men and in the 7th decade in women, but that all age groups were represented. This age distribution is similar to that recorded by Gelfand and Wall¹ in a survey of patients with cirrhosis at Harare Hospital in 1972. As could be expected, the mortality was higher in males (33%) than females (21%), and the over-all mortality was 30%.

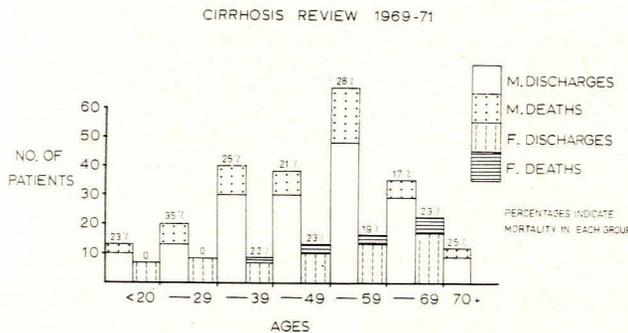


Fig. 1. Analysis of the age, sex and mortality of all the patients in the study.

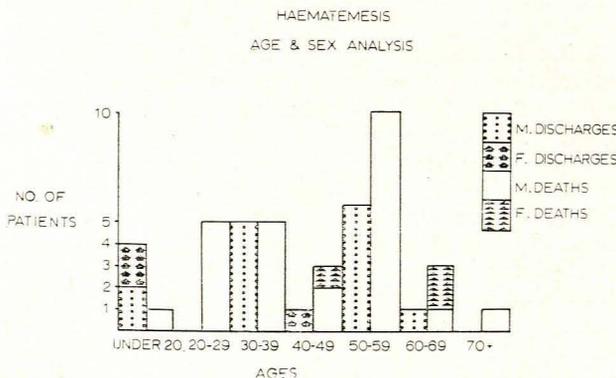


Fig. 2. Analysis of the age, sex and mortality of patients with haematemesis.

The number of discharges and deaths and the sex of patients with haematemesis in each age group are shown in Fig. 2. The total number of patients with haematemesis in the series is shown to be 45, an incidence

of 20%. It is obvious that haematemesis occurred in cirrhosis in all age groups, the maximum incidence being in the 6th decade. It was very much less common in women than men (6 as opposed to 39 cases) although the prognosis was equally poor.

CAUSES OF DEATH IN CIRRHOTIC PATIENTS

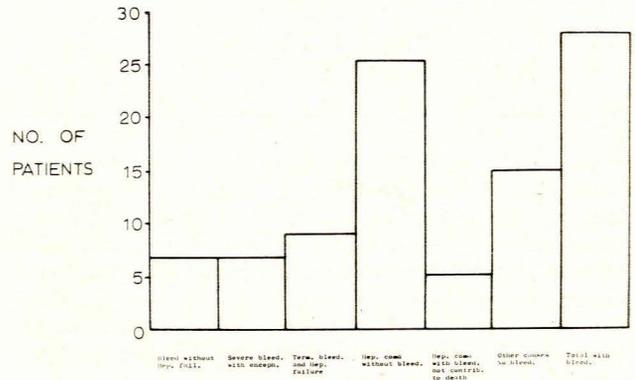


Fig. 3. Analysis of causes of death, with the number of patients in each group.

Fig. 3 shows the causes of death. This analysis proved to be a difficult task, as in a number of instances the precise cause of death was not clear. It was decided to divide the causes of death into 6 groups, depending upon the major contributory factors. The most important features, determining whether or not a patient died in this survey, were the occurrence of haematemesis and the presence of encephalopathy manifesting as hepatic coma. In a number of cases both were present in the same patient, and it was not always easy to assess which had played the dominant role in determining the prognosis in any individual patient. It can probably be fairly safely assumed that haematemesis had in fact precipitated coma in those patients in whom the two were associated temporally. Patients were assigned to the groups shown in Fig. 3 on the basis of what we judged to be the most significant factors leading to their deaths.

In Table II the causes of death are related to the total number of patients, with each of the major complications and the mortality of each group calculated. For completeness' sake the group with uncomplicated cirrhosis is also included.

The incidence of jaundice and ascites was determined, but, surprisingly, these factors were distributed fairly evenly throughout the whole group of cirrhotics and did not appear to correlate obviously with the development of coma and haematemesis. Since the significance of these clinical features was not relevant to the main purpose of the study, they are not presented here.

It can be seen that out of a total of 68 deaths, haematemesis was probably of major, though not necessarily of sole, significance in determining the deaths of 23 patients (34%), as opposed to 5 deaths (7%) in which haematemesis occurred but did not appear to be contributory. Twenty-five patients (37%) died of liver failure without any

TABLE II. INCIDENCE OF MAJOR COMPLICATIONS OF CIRRHOSIS WITH MORTALITY IN EACH GROUP

	Haematemesis only	Encephalopathy only	Haematemesis and encephalopathy	Total involving haematemesis	No complications
Number	14	50	31	45	133
Incidence	6%	22%	14%	20%	58%
Deaths	7	25	21	28	15
Mortality	50%	50%	68%	62%	11%

evidence of bleeding from the gastro-intestinal tract. The remaining 15 patients (22%) died of causes not directly related to liver cell failure, such as tuberculosis, pneumonia and renal disease.

a worse prognosis, but this might merely be a reflection of the severity of the underlying liver disease rather than an indication of a greater tendency to bleed on account of diminished clotting function.

HAEMATEMESIS - LENGTH OF HISTORY

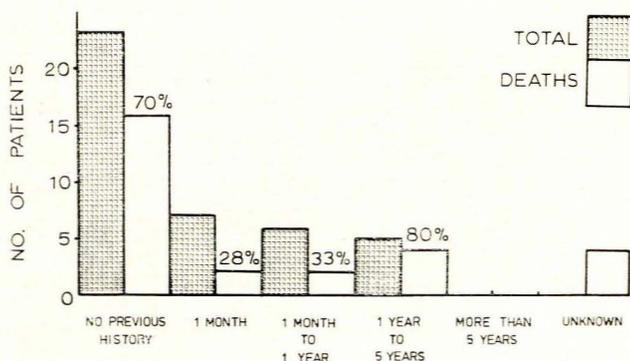


Fig. 4. The groups refer to the occurrence of haematemesis before admission in the periods indicated, and not to other symptoms associated with cirrhosis, which were often absent.

In Fig. 4 the significance of a previous attack of haematemesis is considered. The patients were divided into 5 groups on the basis of their history as indicated. The only facts which emerge are, firstly, that there were no patients alive in this study who had had a haematemesis more than 5 years before the present admission, and secondly, that the mortality rate of the patients with no previous haematemesis was 70%, which was considerably higher than in the patients who had had a bleed, which they had obviously survived, sometime during the previous month or year.

Table III relates the prothrombin index to the mortality in the patients with haematemesis. It is clear that those patients with a prothrombin index of less than 90% had

TABLE III. PROTHROMBIN INDEX IN PATIENTS WITH HAEMATEMESIS

	Prothrombin index			Not done
	90 - 100%	60 - 89%	<60%	
Discharged	8	4	—	7
Died	6	13	1	8

DISCUSSION

The results indicate that in no less than 60% of the patients there were no major sequelae as shown by encephalopathy or haematemesis. The majority of the patients in this group were discharged reasonably well, the mortality being only 11%. This contrasts sharply with the mortality in the groups of patients in whom major complications were present. Hepatic coma or pre-coma were seen in 50 patients (22%), and the mortality in this group was 50%. The patients in this group did not have significant haematemesis, and in most cases it was unfortunately not possible to determine the factors precipitating the coma, if indeed there were any, apart from progressive decline in liver function. Only 14 patients (6%) had a pure haematemesis with no evidence of encephalopathy and of these 50% died. The cause of death in this group can thus be considered to be exsanguination. Falling between these categories is the group in which haematemesis and coma occurred together, and this combination featured in 14% of the total. The mortality was 68%, indicating that this was a particularly ominous association.

By combining the abovementioned categories in which haematemesis played a major part in the illness, it can be seen that this applied to 45 patients and that the mortality was 62%. This compares with a mortality of 66% of cirrhotic patients dying of haematemesis in Boston, and a 33% mortality in London.²

Unfortunately it was not possible to correlate the quantity of blood lost or the frequency of the haematemesis with mortality, as these factors were not usually recorded in any detail in the notes, often because the patients were unable to supply this information.

It is of interest to discuss briefly the findings of the barium swallow. This investigation was performed in 75 patients in the series, and varices were detected in 37 of them. The indication for doing a barium swallow, however, was not clear, and in 50 of the 75 patients there was no history or clinical evidence of gastro-intestinal tract bleeding. Of the 50 patients 20 had oesophageal varices. Contrast studies were done in only 16 of the 45 patients with a history of haematemesis, and of these, as might have been expected, varices were demonstrated in a high proportion (80%).

In the series there were 17 patients who were reported as having melaena without haematemesis. Barium swallows were performed in 9 of these patients and varices were shown to be present in 4 of them. Significantly, only 1 patient in this group died. Melaena without haematemesis, even in the presence of oesophageal varices, therefore, appears to be relatively benign, and it may be inferred that severe upper gastro-intestinal haemorrhage is likely to produce haematemesis. On account of the relatively small number of barium studies performed, no conclusion can be drawn regarding the incidence of varices in cirrhotics and their tendency to bleed.

Sherlock³ stated that in cirrhosis the prognosis of bleeding oesophageal varices tends to be serious, depending on the severity and the extent of the underlying hepatocellular disease. She gave a figure of 44% as the immediate mortality. She found that the seriousness of bleeding in cirrhosis varied from place to place. For instance, she found that 67% of her London patients survived one year, in contrast to 34% in Boston. The patients in the Boston series had more severe signs, such as encephalopathy, jaundice and an ascites; also, 83% of the Boston patients were alcoholics, in contrast to 25% of the London

group. This may be due to the fact that alcoholic cirrhotics have more hepatocellular disease. With portal obstruction without cirrhosis the prognosis is better, since liver failure is mild.

Although this study leaves a number of questions unanswered, it can validly be concluded that haematemesis is an important contributory factor in the terminal illness of a significant number of patients with cirrhosis at Harare Hospital. Pure haematemesis was indeed an uncommon manifestation of cirrhosis, and thus an uncommon cause of death. However, haematemesis occurring *pari passu* with liver failure was not a rare event, and in many patients it was the factor precipitating their terminal liver failure, from which they died some time later. Haematemesis was the final insult in patients whose liver function was so compromised that they already had encephalopathy, and death occurred as a direct result of a haemorrhage which might not otherwise have been fatal.

REFERENCES

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2. Sherlock, S. (1964): *Brit. J. Surg.*, **51**, 746.
3. *Idem* (1971): *Disease of the Liver and Biliary System*. 4th ed., p. 216. Oxford: Blackwell.