EXPERIENCES IN THE MANAGEMENT OF GASTRODUODENAL HAEMORRHAGE*

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This paper constitutes the preliminary report and some facets of an investigation into the causes, natural history, methods and results of treatment of gastroduodenal haemorrhage as met with in the Addington Hospital, Durban. It represents a personal experience in 82 cases.

DEFINITION

Gastroduodenal haemorrhage means the vomiting and/or defaecation of blood, the source of bleeding being either in the duodenum or stomach, including the lower oesophagus. The bleeding is usually of such amount as to produce the systemic changes associated with a large haemorrhage.

Incidence of gastroduodenal haemorrhage. The white population of Durban is at present ±150,000. The number of patients with gastroduodenal haemorrhage admitted to Addington Hospital (European section) were: 122 in 1958, 133 in 1959, and 65 in the first 6 months of the year in 1960. Though initial impressions suggested a seasonal variation of the number of cases, with a maximal incidence in the winter months, our analysis of the admission on a monthly basis did not entirely support this (Fig. 1).

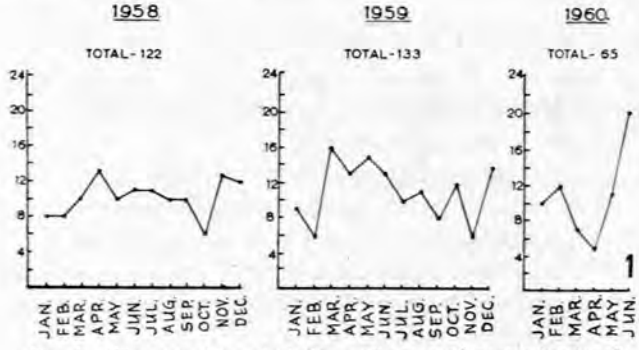

Fig. 1. The monthly admissions of cases of gastroduodenal haemorrhage at Addington Hospital, Durban.

FACTORS PRECIPITATING HAEMORRHAGE

The purposeful direct questioning of patients on the ingestion of drugs, e.g. phenylbutazone, cortisone, anticoagulant drugs (in particular acetylsalicylic acid), showed that many people were taking these drugs. This applies in particular to the 40+ age group and the ingestion of acetylsalicylic acid. In this regard we record the conclusions of Avery Jones *et al.*[1] that aspirin plays a definite part in precipitating gastroduodenal bleeding, confirming the previous studies of Muir and Cossar.[2] The pharmaceutical claims for soluble aspirin as either sodium acetyl salicylate or calcium acetyl salicylate must be critically considered. Both these salts may be soluble in water as claimed, but in hydrochloric acid and gastric juice they are precipitated as the crystalline acetylsalicylic acid.

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DIAGNOSIS OF THE SOURCE OF THE HAEMORRHAGE

An attempt to diagnose the source of bleeding is made as soon as possible and preferably on *clinical grounds* without exhausting special investigations. Infrequently, urgent barium examination in the bleeding phase has been undertaken, and Hampton's[3] 'non-touch technique' has been used. As the number of patients so examined have been few, the efficacy or safety of the procedure cannot be commented upon. We have not used the bedside barium examination using standard portable apparatus as reported by Cantwell.[4] This author states that the diagnosis has been accurate, as confirmed later in 82% of 200 cases. Furthermore, the procedure is stated to be non-disturbing and has been entirely free of complications. As radiology is unlikely to influence the selection of cases for urgent operation, we have not considered this procedure as essential and have reserved it for the investigation of those patients in whom the bleeding has ceased.

Gastroscopy has not been employed.

In arriving at a *clinical* diagnosis of the cause of the haemorrhage, we therefore consider the following:

1. *A Radiologically-established Diagnosis of Peptic Ulceration (Currently or Previously)*

Patients' affirmation that they have a peptic ulcer must be supported by radiological evidence of ulcer. Many have had a clinical diagnosis of ulcer made that was never supported by further investigations, and cannot therefore be considered diagnostically proved. Furthermore, we have been astounded by the number who have bled from a source other than the proved ulcer.

2. *A History of Dyspepsia*

Most patients over the age of 45 admitted to some form of dyspepsia and this was also a frequent feature in the younger patients with a history of alcoholism. The presence of indigestion as a symptom could therefore not in any way serve to establish the presence of peptic ulceration, and was always found in gastritis from alcohol or aspirin.

3. *The Mode of Onset of Bleeding*

To this we have attached great significance.

(i) The onset of a large, painless haemorrhage, particularly in a young patient and in the absence of any previous dyspeptic history, should suggest an ulcerating benign tumour of the stomach. Fig. 2 shows such a large leiomyoma of the stomach.

Case 1. Mrs. M, 42 years, April 1959. Sudden, large silent bleed of bright blood. No previous history of bleeding or dyspepsia. Bleeding stopped after 24 hours. Barium meal revealed a tumour on greater curve of stomach with ulcer on apex. Pre-operative diagnosis—leiomyoma.

Operation: (Mr. A. Copley) Gastrotomy and sleeve resection of stomach including tumour undertaken.

Histology. Argentaffinoma.

(ii) Repeated bloodless vomiting over a short time, if followed by a sudden bloody emesis, is diagnostic of the

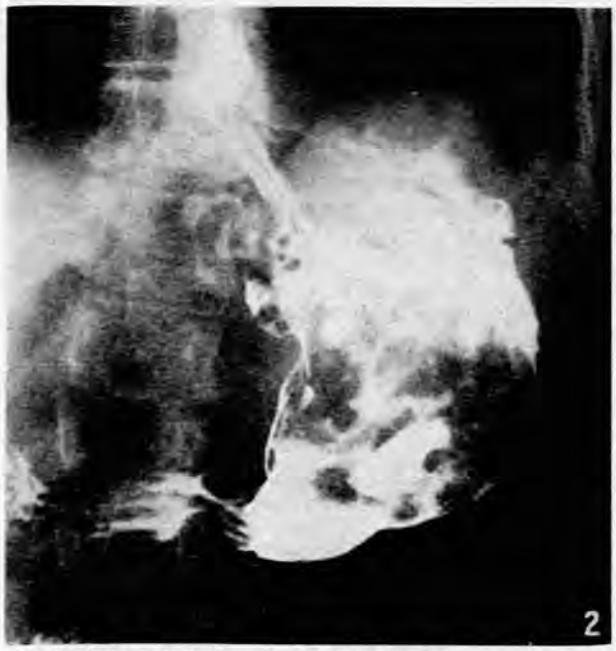

Fig. 2. X-ray of a leiomyoma of the stomach.

Mallory-Weiss syndrome[5] in which the gastro-oesophageal mucosa sustains a fissured tear from the effort of vomiting.

Case 2. Mr. P, 38 years, 17 April 1959. Arrested for insobriety. Chronic dyspeptic history. Whilst in the cells, vomited stomach contents frequently through the night, but did not bleed. 18 April 1959, sudden profuse haematemesis; bright red and altered blood. Patient admitted to Addington Hospital where transfused with 3 pints of blood. No hepatomegaly and no splenomegaly. 19 April 1959, melaena and fresh haematemesis. 2 pints of blood transfused.

21 April 1959, very large haematemesis requiring rapid transfusion—7 pints of blood: for immediate operation. Provisional diagnosis, chronic gastritis. At laparotomy there was no ulcer of stomach or duodenum. Lesser sac opened. Fresh subserous haemorrhage, extending as a flare from cardia of stomach, was noted in anterior wall. Gastrotomy performed over distance of 6 inches. Constant brisk oozing of blood seen to be rising at oesophagogastric junction from tear in mucosa of this region. Procedure: suture of fissure from within stomach after wedge biopsy.

No further haematemesis or melaena.

4. *The Volume of Blood Lost*

It has often been stated that carcinoma of the stomach does not produce a haematemesis or melaena of great amount. In 2 of our patients this has not been the case. The first patient, aged 31, with subsequently proved carcinoma of the pylorus, required rapid transfusion of 2 pints of blood; a female aged 39, with carcinoma of the body of the stomach, had to be similarly transfused.

5. *The Nature of the Blood Loss*

Patients with *bleeding varices* had an acute, massive, bright-red, bloody emesis. Melaena then followed. Patients with *bleeding duodenal ulcers* often had both haematemesis and melaena, but often melaena alone occurred. If the haemorrhage was rapid, the blood in the rectum was bright red and unaltered.

6. *Bleeding Associated with Abdominal Pain*

The association of acute upper abdominal pain with haematemesis is usually considered to be indicative of haemorrhage plus perforation. We have not encountered such a case, but made the diagnosis in one patient who was subjected to laparotomy and gastrectomy without an evident cause of haemorrhage.

Histology of the resected stomach revealed acute gastric erosions of the type met with in 'aspirin' ulceration. On subsequent questioning the patient admitted to having ingested 4 aspirin tablets (20 gr., 1·3 G.) on an empty stomach, a few hours before onset of symptoms.

7. *A Palpably Enlarged Liver*

Marked liver enlargement below the costal margin occurred in 2 patients who were acknowledged alcoholics. Neither bled from oesophageal varices and indeed both had duodenal ulcers. In the first, congestive cardiac failure as a result of anaemia caused hepatomegaly, which receded on effective cardiac treatment. In the second a fatty liver was shown at autopsy.

One may be tempted to jump to the precipitate and erroneous conclusion that, because the patient is an alcoholic and because the liver is enlarged, portal hypertension is the background to the haemorrhage.

8. *A Palpable Spleen*

A history of chronic alcoholism cannot alone permit the apportionment of blame for the haematemesis to oesophageal varices. Chronic and acute gastritis are the more likely causes, and we are of the opinion that portal hypertension with oesophageal varices cannot be diagnosed in the absence of a palpable spleen. In those cases in which oesophageal varices have been the cause of the haemorrhage, a spleen was easily palpable.

9. *Gastroduodenal Bleeding Followed by Coma*

Although haematemesis may occur in the coma of uraemia, this has not been a surgical problem. Those patients who bleed first and then go into coma probably have impairment of liver function, but again we have found that in the absence of a palpable spleen a source of bleeding other than varices was present.

Case 3. Mrs. W., 45 years, 14 March 1960, admitted with melaena, in state of circulatory collapse. No haematemesis. The patient was conscious and known to be a chronic alcoholic.

A hard liver, three-fingers enlarged, was palpable, but a spleen was not felt. Six hours after admission patient went into a coma and her breath had the odour of foetor hepaticus.

Treatment failed. Autopsy showed a large bleeding duodenal ulcer.

10. *External Abdominal Trauma*

This was invoked, although not proved, as the cause of bleeding in one patient aged 17 who was struck a sharp blow across the upper abdomen and immediately vomited large amounts of fresh blood. Indeed, 7 pints of blood (transfused) were needed to restore circulatory equilibrium.

SELECTION OF CASES FOR EMERGENCY OPERATION

We have used as a yard-stick the expected results of treatment of haematemesis and melaena without surgery. The expected mortality in the treatment without surgery of patients younger than 45 years, is 0·5%. The expected mortality in the treatment without surgery of patients over 50 years, is 15%.[6]

As there are no effective means of inhibiting the bleeding without surgical aid, all patients over the age of 45 are admitted to a surgical ward and kept under most careful

observation, including gastric aspiration through a Ryle tube. In addition the physician's assessment of the patient's general state of health is obtained.

Emergency Surgery

Indications for emergency surgery, i.e. operation after restoration of blood pressure to the systolic of 100 mm. Hg with rapid transfusion of blood are as follows:

1. *Patients over 45 years of age, (group A)* with:

(*a*) Haemorrhage for a period longer than 24 hours and still actively bleeding.

(*b*) Haemorrhage that has temporarily stopped, but restarts.

(*c*) Haemorrhage plus abdominal pain that suggests perforation.

(*d*) Haemorrhage plus a chronic history of proved peptic ulceration.

(*e*) Haemorrhage and a history of previous haemorrhage.

Though the above criteria are used to decide on operation, we are naturally guided by the patient's associated biological and clinical state. In the aged or decrepit elderly, i.e. in patients of 70 years or more, we have tended to be more elastic in our absolute criteria for operation and often have risked conservative treatment. However, age alone is no contra-indication to surgery. Our oldest patient successfully submitted to emergency gastrectomy for haematemesis due to a chronic posterior duodenal ulcer, was 84 years.

On the other hand, the following case is an example of the considered need for conservative treatment in a patient who in all other respects is suitable for classification in the surgical group.

Case 4. Mr. E. G., age 50. Admitted on 20 August 1959 with weakness, epigastric pain and melaena of 5 days' duration.

There was a strong past history of alcoholism, but not indigestion.

On examination: Patient anaemic with haemoglobin of 6·5 g. per 100 ml. Pulse 100 per minute. Abdomen a little distended. A two-finger enlargement of the liver below the costal margin was palpable. Three pints of blood were slowly transfused and the patient went into acute left ventricular failure with pulmonary oedema. Treatment with digitalis and mercurial diuretics was necessary to bring him out of failure. The bleeding continued for several days, but the patient's general state could be maintained with slow transfusion of packed red cells. An electrocardiogram, done on 22 August 1950, revealed a fresh anterior infarct in the myocardium.

On X-ray, 1 October 1959, a healed duodenal ulcer with scarring of the duodenal cap was demonstrated.

2. *Patients under* 45 *years of age* (*Group B*) are generally treated without operation. However, the latitude allowed in the criteria for exclusion from emergency operation in the 45+ age group has a complementary group for operative inclusion in the under-45 group. This includes:

(*a*) The bleeding oesophageal varices.

(*b*) Continued major bleeding to third, fourth and fifth day.

(*c*) Continued slow bleeding as witnessed by persistent occult blood in the stool and a falling haemoglobin.

(*d*) Bleeding with perforation.

(*e*) Haemorrhage plus chronic history of proved peptic ulceration.

OPERATIVE MANAGEMENT

Patients selected for emergency surgery are operated upon under a general anaesthetic. We have not had recourse to the use of local anaesthesia as practised by Tanner.[7] The abdomen is opened through a left upper paramedian incision. We prefer the left-sided rather than the right-sided approach, since it affords better access to the cardia and fundus of the stomach, both of which have frequently been found to be the sites of origin of haemorrhage.

The Operative Routine

1. Careful palpation and inspection of the anterior gastric and duodenal walls (first and second part) and palpation of the posterior wall through the duodenum for evidence of induration and ulceration. The finding of a duodenal ulcer must not be taken as the established source of the bleeding. Up to 15% of duodenal ulcers are associated with gastric ulcers (Maingot[5]).

Case 5. Mr. C. Age 61. Clerk. Admitted to Addington Hospital in May 1960. Admitted history of duodenal ulcer of 15 years. Large haematemesis which required transfusion of 3 pints of blood. The haemoglobin and blood pressure were satisfactory until 10 May 1960 when a further acute haematemesis occurred. The patient was prepared for emergency operation with the following findings:

There was a chronic duodenal ulcer present on the first part of the duodenum.

Palpation of the anterior gastric wall revealed a hard thickening on the proximal portion of the lesser curvature. A 4-inch gastrotomy incision was then made through the anterior gastric wall. The stomach contained much fresh blood and blood clots which were sucked out. A finger in the stomach, introduced through the pylorus into the duodenum, was shown to be free of fresh blood; a finger introduced proximally in the stomach fell into a sharp, punched-out, deep, benign ulcer crater high on the lesser curvature. In the centre of this crater was a vibrissa-like artery from which the blood was spurting.

2. The gastrocolic omentum is divided allowing access to the lesser sac. The posterior surface of the stomach is now inspected and must be freely mobile on its omental bursa. The presence of adhesions between the posterior stomach wall and posterior peritoneum of the lesser sac is considered pathological and frequently the result of a posterior gastric ulcer, the presence of which may not otherwise be seen or felt. In 3 patients we have been led to a chronic posterior gastric ulcer, each the source of haematemesis, by observing the posterior fixity of the stomach.

3. The presence of a fixed hiatus hernia is sought for. To date, none of our operated cases have revealed this source of bleeding. We note with great interest, however, the large number encountered by Marchand.[9] We accept the possibility of not having detected the actual presence of a bleeding hiatus hernia, though it might have been present. In this respect, we should like to mention our experience in routine palpation in all abdominal operations of the oesophageal hiatus which allows the admission of 2 to 3 fingers in nearly all patients over the age of 55.

4. The spleen is demonstrated and its size noted.

5. The mesenteric and gastro-epiploic veins are inspected for congestion as in portal hypertension. These may possibly be collapsed if shock is still a feature.

6. Whether an ulcer is present or not we have made a habit of opening the stomach in every case through a 3 - 4 inch anterior gastrotomy incision, in order to ensure that the bleeding is indeed coming from the pathological site visualized or palpated from the exterior of the stomach, and for examination of its interior where no cause of bleeding can be determined by examining its exterior. The interior aspect of the stomach is carefully visualized and palpated. A finger

is introduced into the duodenum, then up to the oesophagus. We presume that peptic ulceration of the oesophagus should be felt. Collapsed varices will not be palpable, but bleeding from within the lower oesophagus might be demonstrated on the gloved finger. We have learned to palpate the cardia especially well, because on two occasions we missed a carcinoma at this site. The visualization and palpation of the *interior* of the stomach has at times revealed unsuspected causes of haemorrhage, e.g. the Mallory-Weiss syndrome cited, and bleeding gastric ulcer in the presence of duodenal ulcer. One patient had a diffuse petechial haemorrhagic state of the gastric mucosa. We have not found this gastrotomy an added operative or postoperative hazard.

7. The problem of a 'negative' gastric and duodenal examination including gastrotomy. This occurred in just more than half the cases operated upon and is the surgeon's dilemma.

Guided by the experience of the Mayo Clinic[10] we have performed subtotal gastrectomy in the absence of overt pathology; of these one patient continued bleeding for 30 hours after subtotal gastrectomy, another bled again on the 7th postoperative day, but these survived. A third patient had a fatal haemorrhage on the third postoperative day. Nevertheless, the figures from the Mayo Clinic authority are significant.

Forty-eight patients were operated upon—the stomach and duodenum were opened, but no abnormality detected.

In 28 no gastrectomy was performed; two out of three had a recurrent haemorrhage. In 20 gastrectomy was performed, and in these 1 in 10 had recurrent haemorrhage. Eight showed an ulcer on histological examination.

THE DEFINITIVE OPERATIVE PROCEDURE USED

Apart from gastrotomy no single standard procedure is recommended; each condition being treated on its own merits. Gastrectomy has been performed for bleeding duodenal ulcer in all cases but one. This exception was a patient with a carcinoma of the bladder (with pelvic secondaries), who also presented with a history of chronic duodenal ulceration and haematemesis which would not stop. Duodenotomy revealed the bleeding posterior ulcer. Haemostasis was achieved by under-running the ulcer with suture. For gastric ulcer we have performed either wedge resection of the ulcer or gastrectomy, depending on the site of the ulcer and the condition of the patient. Where the ulcer was so high on the lesser curve as to make wedge excision hazardous and near-total gastrectomy unadvisable, we have occluded the ulcer by suture from the interior of the stomach. For the benign tumour, we have performed sleeve resections including the tumour growths. In cases with a tumour on a pedicle we have transected the stalk only. For carcinoma at the pylorus subtotal gastrectomy has been done. In the case of Mallory-Weiss syndrome, the tear in the gastric mucosa was sutured from within the stomach. In this case, as in that of the high gastric ulcer, we have not hesitated to use silk suture and trust that we have not provided a nidus for further ulceration.

FOLLOW-UP

In 3 years we have had no repeat haematemesis after operation. Further observation is obviously needed. Two problems remain a cause for anxiety with regard to the method of their surgical management. These are:

1. Bleeding oesophageal varices in portal hypertension.
2. Bleeding from carcinoma near the oesophago-gastric junction.

In the former we are employing Tanner's method of porto-azygos disconnection with gastric transection. We have not found a suitable form of treatment for the latter.

SUMMARY

1. The incidence of gastroduodenal haemorrhage in a general hospital (Addington), Durban, is shown and some precipitating factors are mentioned.

2. The claims for soluble aspirin are contested.

3. The diagnosis on clinical grounds of the source of bleeding is considered.

4. The criteria for emergency operation are discussed, together with the operative procedure.

5. Gastrotomy is considered an essential procedure in every single case before embarking on the remedial operation.

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