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EARLY COMPLEX DIAGNOSTIC APPROACH TO HAEMATEMESIS AND/OR MELAENA

By

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Early aetiologic diagnosis in cases of massive haemorrhage from the upper alimentary tract has been attempted in 126 cases. From the evidences of coagulogram, immediate oesophagoscopy, gastroscopy, and early or semi-early X-rays a correct diagnosis has been achieved in 89 per cent. Six cases were misdiagnosed. In eight cases the source of bleeding remained unidentified. The finding of gross rugal congestion and hypertrophy connected these haemorrhages with a minor alcoholic gastritis which may have healed by the time of endoscopy. Influenza constituted no precipitating factor, the influenza complement fixation test being positive in one case only.

The early complex diagnostic procedures had no adverse effects, haemorrhagic shock or advanced age are no contraindications.

Early aetiologic diagnosis of haematemesis and/or melaena provides for aetiologic treatment which is the most efficient means of avoiding unnecessary partial gastrectomy, on the one hand, and lethal haemorrhage from gastro-oesophageal varices on the other.

Correct management of massive haemorrhage arising in the upper gastrointestinal tract represents a difficult task. The pitfalls of the conservative methods are as hard to avoid as those of emergency surgery. This calls for the mobilization of every diagnostic possibility to find the cause of the haemorrhage. Our first results have been reported in 1959 [10]; they essentially agree with those advocated by PALMER and SCOTT (1955) [16], AVERY JONES (1959) [1], and KRENTZ (1960) [15].

The diagnostic lines laid down by us have not found general acceptance. The adepts of the policy of active diagnosis generally concentrate upon one single method, for instance on early gastroscopy or early radiography while taking a definite line against the other procedures. UNGEHEUER [18] went so far as to assert that in the case of massive gastrointestinal haemorrhage any diagnostic attempt represents an unnecessary risk, apart from causing loss of time and yielding false results. In opposition to these unitarian and nihilistic attitudes the present study intends to give voice to the conviction that the very danger of death by blood loss calls for utmost immediate diagnostic activity.

Our observations relate to 126 patients referred to our Department for haematemesis and/or melaena from 1958 onward. The diagnostic methods included

1. History and physical examination;
2. Investigations for haemorrhagic diathesis;
3. Immediate oesophagoscopy;
4. Immediate gastroscopy;
5. Early and semi-early X-rays.

It will now be attempted to outline the advantages and shortcomings of all these methods.

History and physical examination

History reveals the fact of haemorrhage and offers some information about its degree. The usual pitfalls are "pseudohaematemesis" and "pseudomelaena". Some patient while relieving his stomach of an overload of red wine in the poorly lit toilet of the public house, may mistake the vomit for blood. Some other who has had recurrent haemorrhages from a duodenal ulcer, may over-indulge in blood-sausage and run to the physician on the next day in alarm with a sample of black stools. The reverse may, however, also occur. In one of our patients the only complaint had been palpitation interpreted as being due to acute myocarditis. But for the presence of stains of tarry stools on his underclothes, the cause of the symptoms would have remained undetected. Many semi-educated people will connect their dark stools with over-indulgence in black coffee.

The history will reveal the possible precipitating factors, such as stress situations, alcoholism, ulcerogenous drugs, salicylates, reserpine, butazolidine, corticosteroids, certain antibiotics. A particularly valuable clue is a history of peptic ulcer or its perforation. A palpable mass in the abdomen or enlargement of both liver and spleen leave little doubt about the origin of the haemorrhage.

In our experience history is uninformative, or conflicting or even misleading in 50 per cent of the cases. Physical examination is negative in more than 75 per cent.

Investigations for haemorrhagic diathesis

At admission the following examinations are carried out: bleeding time, clotting time, platelet count, retraction of blood clot, Quick's prothrombin time, complete coagulogram according to GERENDÁS; capillary function tests (BORBÉLY, GÖTHLIN, tourniquet test), liver function tests (thymol turbidity, gold sol flocculation, alkaline phosphatase), non-protein nitrogen, and blood samples are taken for the influenza complement fixation test.

The simple clinical tests usually suffice for the detection of clotting disturbance; identification of its character requires a full coagulogram. For instance, in one of our cases it was on this evidence alone that we were able to identify haemophilia type B as the cause of the haemorrhage. Obviously haemophilia, as well as thrombopathic anomalies, exclude any surgical intervention and call for conservative treatment. Fig. 1 shows the gastric mucosa of a patient with myeloid leukaemia who died in consequence of thrombocyto-

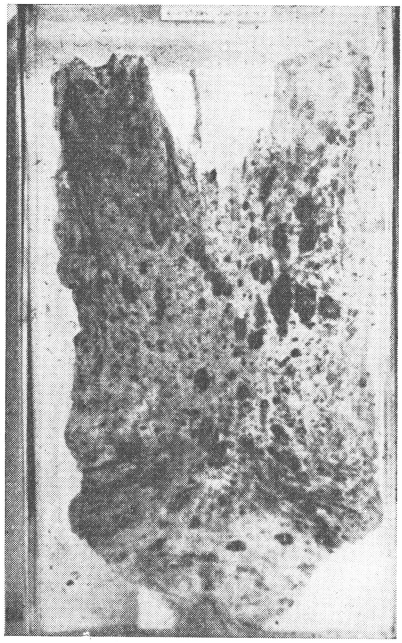

Fig. 1. G. L., 45-year-old ♀. Gastric manifestation of thrombocytopenic haemorrhage, chronic myeloid leukaemia

pathic haemorrhage. Obviously any further diagnostic procedure would have been futile.

The prothrombin test often presents problems. The level may vary in cases of massive haemorrhage and there are no means to decide when it is necessarily pathologic. At 20 per cent it is most certainly so, on the other hand, even values of 50 or 60 per cent may be connected with gastric haemorrhage in the case of hepatic cirrhosis where portal stasis constitutes an additional precipitating factor. Conversely, prothrombin activity may be nil, yet the patient may bleed from gastro-oesophageal varices, as it did in one of our cases. Haemorrhages in hepatic cirrhosis may have various causes, e.g. low prothrombin level, varices, exulcerated tumour, hepatogenic ulcer, etc. For this reason, even with low prothrombin levels it is imperative to pursue the investigations for other possible causes of the bleeding.

Immediate oesophagoscopy

The decision to pass down a straight inflexible oesophagoscope with the patient bleeding is not all too easily made. When we did it for the first time it was in the face of deep-rooted traditions. Even today the procedure has very few adepts all over the world. KATZ et al. [14] recommend a flexible oesophagoscope, DESNEUX [7] advocates intubation anaesthesia to facilitate introduction. We use an inflexible tube introducing it in local anaesthesia. Paradoxical as it may seem, haemorrhagic shock makes this procedure easier owing to the patient's apathy. Advanced age is no obstacle, in fact it makes — again quite paradoxically — the task even easier by the reduction of reflex irritability with advancing years. The procedure is carried out in the endoscopic laboratory under the cover of blood transfusion, all preparations being made for operation.

Oesophagoscopy is invaluable in the detection of oesophageal varicosity. Various other methods have been advocated for this purpose, such as X-rays, spleno-portography, spleen-pulp manometry, fluorescein-thread-test, estimation of blood ammonium level, etc., but no indirect procedure can be compared in its informative value to the direct visualization of varices where a clear picture may be gained about the length, number, and course of the distended veins. Oesophagoscopy is, furthermore, the only means of detecting superficial oesophageal ulcers as it did, in fact, in two of our cases. The very negativity of the oesophagoscopic finding provides a valuable clue, directing the search for the source of the bleeding to the gastrointestinal regions situated below the cardia.

A source of error lies in the fact that gastro-oesophageal varices generally rupture inside the stomach. This was the case in all of our six patients bleeding from ruptured varices. Consequently, we could not identify the site of rupture though we duly visualized the distended veins. This illustrates that the diagnosis of bleeding from varices rests in the majority of the cases on logical conjecture rather than on direct identification. This is why we were misled by the finding of oesophageal varicosity in two cases connecting it erroneously with the haemorrhage. In fact, its source had been a tumour of the gastric fundus in the first case and a gastric ulcer in the second.

Immediate gastroscopy

Gastroscopy is by no means free from hazards. In the 49,000 cases collected by AVERY JONES et al. [2] from the world literature there were 50 oesophageal and 9 gastric perforations, beside other complications. The flexible gastroscope devised by WOLF and SCHINDLER greatly reduces these dangers. By its aid various authors have carried out early gastroscopy, in Hungary

BENKŐ et al. [4, 5, 6] in the first or second week following massive haemorrhage. Personally we undertook it under the cover of antishock measures in the very first hours after admission of the bleeding patient. In support of this activity, we may allege today quite a number of observations.

What has this examination to offer? First of all, it permits direct visualization even of widespread alterations of the gastric mucosa, e.g. multiple bleeding erosions, and it makes it possible to differentiate benign from malignant ulcers. It can be seen whether the lesion is still bleeding or whether a clot has already formed on its surface. In one of our cases, the bleeding was seen to pulsate synchronously with the arterial pulsations.

The method has the following limitations.

(1) Obliteration of the front lens by blood or mucus;
(2) Air-intolerance;
(3) Over-flexion of the instrument;
(4) The site of the lesion may be outside the visual field of the WOLF—SCHINDLER gastroscope;
(5) The gastric mucosa may be covered with blood.

One of the major shortcomings of gastroscopy is that it does not cover the region of the cardia, the preferential site of gastric varices and of acute ulcers with prevalently fatal haemorrhages. The fiberscope is designed for the visualization of these areas. As to duodenal ulcer, though regurgitation of blood through the pylorus is suggestive of its presence, its verification rests on X-ray evidence.

In one case, a blood clot covering the exulcerated area led us to misdiagnose a malignant tumour for a benign ulcer. Visualization is facilitated by previous cautious lavage of the stomach with ice-water. PALMER and SCOTT [16] use water for this purpose, personally we prefer saline.

Early and semi-early X-rays

At first, X-ray examination was attempted on the tenth day after haemorrhage, but the patients collapsed when standing upright, so it was given up. In his handbook SCHINZ [17] also warns against early diagnostic X-rays after gastrointestinal haemorrhage and reserves this procedure for exceptional cases. HAMPTON [13] finds it safe enough when the haemorrhage has ceased, with the patient in the recumbent position, but compression on the abdomen should be avoided. In Hungary, ERDÉLYI [8] does not warrant the procedure before the fourteenth day while FIGUS and IVÁNYI [9] are slightly less cautious. Personally we do not hesitate to carry out diagnostic radiography within the first 24 hours when blood volume has been restored by continuous transfusion and the erythrocyte count approximates 4 million. The patient is reclined to

45°, or, if necessary, made to stand in the upright position avoiding, however, abdominal compression. The X-rays permit to detect circumscript changes first of all in the duodenum. By asserting (1) that the blood clot generally does not blur the radiographic evidence by filling out the ulcer, and (2) that X-ray examination does not provoke a recurrence of bleeding we combate dogmatic beliefs which die hard.

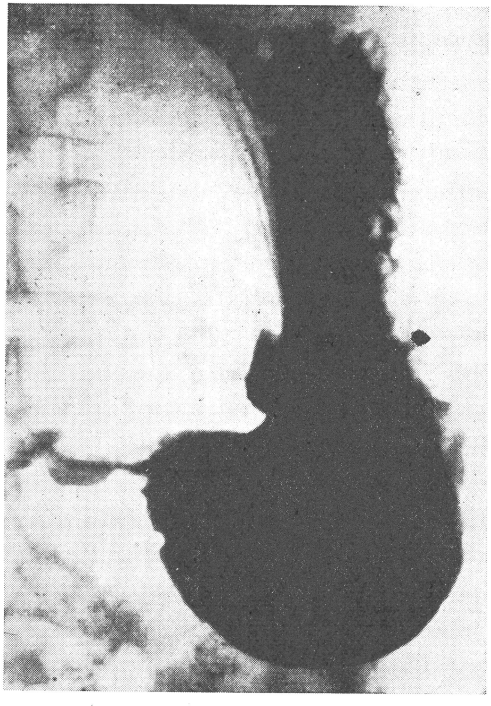

Fig. 2. V. L., 66-year-old ♀. Gastric ulcer. Early X-rays

The ulcer of the lesser curvature seen in Fig. 2 has been identified by early radiography. The patient, a 66-year-old woman, had been unfit for operation because of mitral stenosis and died with circulatory failure 24 hrs. after the onset of haemorrhage. Confrontation of X-rays and post-mortem findings seen in Fig. 3 shows that the radiographic evidence was fully congruent with the morphology of the ulcer and that the gastric mucosa suffered no damage by the procedure.

Since the use of a distinctor is not permissible, negative X-ray findings call for a repeated study in 10 to 14 days. This we call "semi-early radiography". The procedure has failed us in the following three cases.

1. Duodenal ulcer had been suspected on indirect evidence. Autopsy revealed two small ulcers of the fundus as the sources of haemorrhage.

2. A duodenal tumour was suggested by disturbances of passage, later a postbulbar ulcer was found to be responsible for the haemorrhage as well as for the disturbance of passage.

3. Duodenal deformity interpreted as duodenal ulcer was found at surgery to be due to duodenal polyposis, this having been responsible for the haemorrhage.

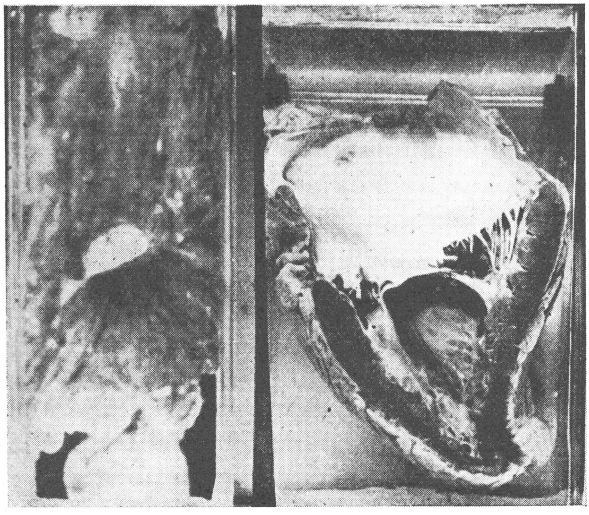

Fig. 3. Post-mortem finding of gastric ulcer seen in Fig. 2 (left side). Buttonhole stenosis (right side)

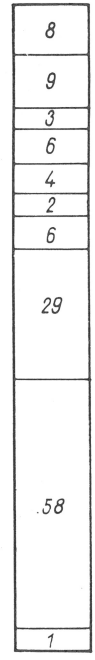

Fig. 4. Cause of upper gastrointestinal haemorrhage in 126 patients.

8 Unidentified — 9 Haemorrhagic diathesis — 3 Multiple haemorrhagic erosions of gastric mucosa — 6 Gastro-oesophageal varices — 4 Gastric tumour — 2 Erosions of oesophagus — 6 Jejunal ulcer — 29 Gastric ulcer — 58 Duodenal ulcer — 1 Duodenal polyposis

Results

Results are seen in Table I. The methods outlined in the foregoing do not replace each other. In some cases it is endoscopy, in others X-rays which help to detect the source of haemorrhage.

Oesophagoscopy reveals varicous veins, gastroscopy permits to visualize gastric changes of widespread nature whereas focal lesions, first of all gastric or duodenal ulcers, are best studied by means of X-rays.

Table I

Results of examinations

	Number of investigations			
	Total	Positive	Negative	Erroneous*
Coagulogram	76	11	65	
Immediate oesophagoscopy	88	8	78	2
Immediate gastroscopy	78	19	58	1
Early X-rays	79	59	17	3
Semi-early X-rays	27	22	5	
Influenza complement fixation test	70	1	69	

* Final diagnosis was based on data obtained during haemorrhage, the clinical course and the surgical or post-mortem findings.
Surgeon: Prof. T. G. KARLINGER
Pathologist: Prof. G. ROMHÁNYI

The columns of the diagram represent the distribution of cases according to aetiology.

The complex diagnostic approach has led to correct diagnosis in 89 per cent of the cases. It must be noted that indirect signs of ulcer were also evaluated. Six cases out of 126 were misdiagnosed. In 8 cases the source of haemorrhage was unidentifiable, but it is attributed to alcoholic gastritis. Haematemesis was shown by BENEDICT [3], but not duly realized ever since, to be common in alcoholics. Early gastroscopy shows unspecific changes of gastritis. The history of these patients often revealed over-indulgence in alcoholic drinks to which popular belief attributes a curative value in common cold. The folds of the gastric mucosa were grossly congested and thickened. This finding of alcoholic gastritis is definitely compatible with the occurrence of haemorrhage though being negative as concerns identification of its source.

No close correlation was found between influenza and haematemesis or melaena. The influenza complement fixation test was positive in one out of 70 cases only.

The complex diagnostic procedure had no adverse effect of any kind.

Diagnostic program

Today we have sufficient experience to lay down the following diagnostic program: Investigations for haemorrhagic diathesis, immediate oesophagoscopy and gastroscopy, and, under the cover of transfusion, early X-rays.

We might start with the X-rays reserving gastroscopy for the eventuality of a negative finding. Such a policy, while possibly saving the patient an un-

necessary instrumental intervention, may on the other hand cause a delay in diagnosis, jeopardizing the outcome. This strongly recommends oesophagoscopy as the first step and makes it even imperative when red blood suggestive of a ruptured varicosity has been vomited.

Endoscopy is contraindicated in 1. Haemophilia or thrombocytopathy; 2. lack of cooperation by the patient; 3. chest deformities; 4. intractable cough; 5. heart failure; 6. oesophagostenosis; 7. aneurysm of the abdominal aorta.

Early radiography has no contraindication if HAMPTON's [13] or our technique is observed. On the other hand it is as useless as any other diagnostic procedure if a life-saving intervention is outside the possibilities, for instance in semicomatose patients with hepatic cirrhosis. Over the age of 90 years we abstain from operation.

Technical details

At admission the immediate task is to carry out the capillary tests and to take blood samples for the detection of haemorrhagic diathesis. In the meantime transfusion of blood is started. Morphine 20 mg + atropine 1 mg is given. This is followed one hour later by oesophagoscopy after anaesthesia of the pharynx with 0.5 per cent tetracaine spray. Visualization of varicosity terminates the endoscopic procedure and calls for urgent operation consisting of ligature of the bleeding vein and of the V. coronaria gastrica. No shunt operation is considered at this moment. If no varicosity is seen, an Ewald—Boas-tube is passed with the patient in the left lateral position, careful gastric lavage with ice-cold saline is started and continued until the washing fluid returns relatively clear. The saline is infused into the stomach in 200 ml portions by the aid of a balloon serving for the removal of gastric contents. Then gastroscopy is performed. If the source of bleeding is still undetected, further 2000 to 3000 ml blood are transfused until the erythrocyte count has reached 4 million. Blood counts are being checked at close intervals so as to avoid over-transfusion. Next morning X-rays are made with the patient reclined to 45° instead of being placed in the recumbent position as suggested by HAMPTON [13]. He may even be made to stand upright for a short time if enough blood has been transfused. Palpation should be avoided under any circumstance. If the investigation has been uninformative, it must be repeated in 10 to 15 days, this time by the use of compression. Personally we prefer a compression tube to a distinctor.

Therapeutic aspects

Early recognition of the cause of gastrointestinal haemorrhage is the prerequisite of aetiologic management. In our opinion, bleeding varices call for surgery which must be as urgent as possible if it is to be life-saving. In the case of a bleeding ulcer, on the other hand, we avoid immediate surgery if possible. Haemorrhagic diatheses, clotting disturbances call for blood transfusions, local application of thrombin, or platelet transfusions or intravenous administration of antihaemophilic globulin.

The failure of conservative treatment is no indication for surgery. Emergency operations carried out too late, apart from being unsuited, bear a sad

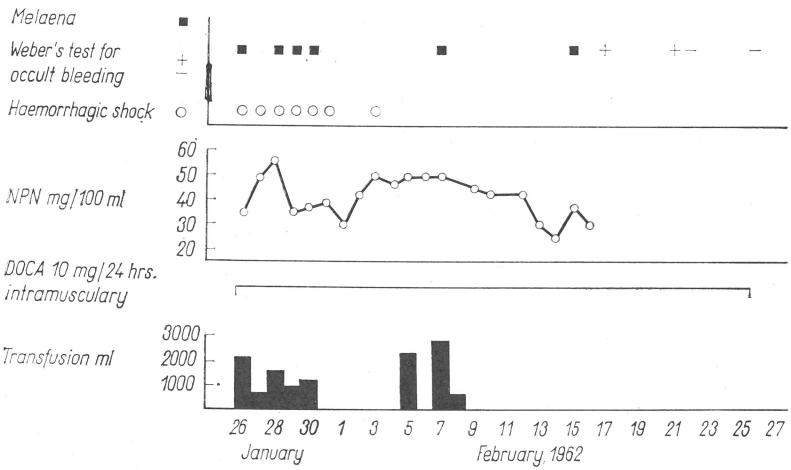

Fig. 5. S. I., 22-year-old ♂. Duodenal ulcer. Recurrent bleeding. Early diagnosis prompted a conservative attitude. Complete recovery was achieved

testimony to this. Injudicious application of partial gastrectomy is not only useless but also bound to increase the number of gastric invalids. The policy of random resection is indefensible.

Immediate endoscopy and early X-rays are particularly rewarding in the following cases: 1. Erosions; but for urgent endoscopy, these lesions would pass unrecognized owing to their rapid healing tendency. 2. Gastro-oesophageal varices. 3. Malignant tumours. 4. Peptic ulcers with recurrent haemorrhages. Surgery may possibly be avoided even after the second haemorrhagic shock if the site and character of the lesion warrant this attitude. The following case was illustrative.

A 22-year-old male was admitted to our department with massive haemorrhage from the upper gastrointestinal tract. On admission he was pale,

sweating, he had tachycardia and an erythrocyte count of 2,800,000. The coagulogram was normal. Immediate endoscopy showed occasional pinpoint bleedings over the antral mucosa. Early X-rays revealed ulcerogenic deformity of the duodenum. The site and character of the lesion as well as the age of the patient prompted a conservative treatment. Melaena recurred six times and required blood transfusions totalling 12.3 litres. The progress of the case is seen in Fig. 5. Control X-rays on the 2nd March 1962 revealed converging mucosal folds at the bulbar basis and in the oblique position an ulcer niche appeared in profile on the posterior wall of the duodenum. Four weeks later, the ulcer had dwindled to a minor duodenal deformity.

The leading article of Lancet of February 26, 1966, quotes the view of various authors that duodenal ulcer is less liable to death than gastric ulcer by blood loss. It is, however, difficult to substantiate this claim. At any rate, early diagnosis of either is of great help to the surgeon, as the exact location of the ulcer will save him the delay caused by gastrotomy and by finding the acute lesion.

It is also to be mentioned that early diagnosis of a bleeding peptic ulcer raises the possibility of its management with DOCA. HÁMORI et al. [11, 12] have found this drug completely to inhibit the formation of duodenal ulcer and, to a great extent, that of gastric ulcer induced by cinchophen in the dog.

The objective which we are pursuing by our complex diagnostic approach is briefly this: No patient should be exposed to unnecessary surgery, nor should he be sacrificed to an undue conservativism.

*

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