Haematemesis in an African Medical Ward

BY

MICHAEL GELFAND, C.B.E., M.D., F.R.C.P.
Professor of University College of Rhodesia and Nyasaland (with special reference to Africa).

Between 1957 and 1963, 63 male patients were admitted under my care to Harare African hospital. They were suffering from acute haematemesis from the upper gastrointestinal tract. The bleeding in each case was sufficient to necessitate the patient’s early admission to hospital. In some cases it stopped quickly, but in others it was so severe that the patient succumbed rapidly or, in the case of those with liver disease, some passed into coma and died.

The diagnosis was chiefly made on clinical grounds, which included a history, a physical examination and, in a certain number of cases, a radiological examination. In some the cause was established only at autopsy.

**Table giving causes in 63 adult males admitted with acute haematemesis to the medical male ward**

<table>
<thead>
<tr>
<th>Cause</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cirrhosis of liver</td>
<td>33</td>
</tr>
<tr>
<td>Duodenal ulcer</td>
<td>12</td>
</tr>
<tr>
<td>Acute erosion (acute duodenal ulcer or duodenitis)</td>
<td>4</td>
</tr>
<tr>
<td>Primary carcinoma of the liver</td>
<td>2</td>
</tr>
<tr>
<td>Cirrhosis and gastric ulcer</td>
<td>1</td>
</tr>
<tr>
<td>Polypoid growth of the stomach</td>
<td>1</td>
</tr>
<tr>
<td>Carcinoma of stomach</td>
<td>1</td>
</tr>
<tr>
<td>Leiomyosarcoma</td>
<td>1</td>
</tr>
<tr>
<td>Gastric ulcer</td>
<td>1</td>
</tr>
<tr>
<td>Unknown causes</td>
<td>7</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>63</strong></td>
</tr>
</tbody>
</table>

The results are interesting, as the series reveals how commonly cirrhosis of the liver is responsible for haematemesis, occurring almost three times as frequently as peptic ulcer. This is in keeping with the very high incidence of cirrhosis in the general population. Many patients with cirrhosis develop portal hypertension, which in turn is followed by oesophageal varices. It is important to realise that whereas in most of those cases the liver is enlarged and its surface irregular and hard, there are not a few patients who present only with a splenomegaly. When the enlargement is gross, portal hypertension is frequently considered, but when it is slight it is apt to be overlooked and the splenomegaly attributed to chronic malaria.
The usual age when haematemesis was seen was between 31 and 40 years, but there were a few cases over 50 years of age. There was one child of eight years in the series. This would correspond with the usual age of incidence of cirrhosis.

It will be observed that in contrast to the frequency of cirrhosis there were only two cases who had primary carcinoma of the liver. Admittedly cirrhosis of the liver is about ten times as common as primary cancer of the liver and therefore we should expect to find many more cases of haematemesis due to cirrhosis. Nevertheless one's impression remains that haematemesis due to cancer of the liver is not common. The occurrence of only two cases might tend to support this impression gained by clinical experience.

After cirrhosis of the liver, peptic ulcer was the next most important cause of haematemesis. Duodenal ulcer appeared to be by far the more common, there being 12 cases of duodenal ulcer compared with one of gastric ulcer.

Acute bleeding occurred in four patients in whom a definite cause was not found. They admitted to having epigastric pain for a few days before the bleeding commenced, but in none of them was there any previous history or suggestion of ulcer. The pain was irregular in onset, but in some it appeared to occur after eating, and for want of a better diagnosis I considered them to have acute gastric erosion or acute duodenitis. Their progress was good and X-ray generally revealed nothing of note. There was one African whose bleeding may be related to his having taken acetylsalicylic acid.

As opposed to what might be an acute gastric erosion (or duodenitis), there were seven patients who had no previous history of gastric upset, the first symptom being the acute bleeding. As they had no pain or prior dyspepsia, I have separated them for convenience from acute gastric erosion. Of the remainder, one patient had a carcinoma and another a polypoidal growth of the stomach.

Grieve et al. (1961) analysed 149 cases of haematemesis admitted to the Coronation Hospital, Johannesburg, but it is not clear from the study how many were not Africans, as patients who were Coloured could also have been included in the series. One may, however, presume that the majority were of African stock. They found the most frequent condition to be "acute gastritis and gastric liver," which they considered was usually related to an alcoholic bout. After a heavy bout of drinking the patient would start to vomit, and this act would appear to have precipitated the bleeding (Mallory-Weiss syndrome).

Grieve et al. (1961) found 32 patients in whom the bleeding was attributed to cirrhosis of the liver (32/136). This is a high figure, although not as high as in my series. It serves to confirm the importance of liver disease as a cause of haematemesis in Africans.

Following closely upon cirrhosis of the liver, they found that 25 cases (25/136) of peptic ulcer were responsible for the haematemesis. The difference in the incidence of peptic ulcer and cirrhosis is not significant and it would be fair to assume that peptic ulcer occurs in much the same frequency as does cirrhosis of the liver.

I found peptic ulcer to be the cause of haematemesis about as often as did Grieve and his colleagues, but cirrhosis of the liver was met twice as often by me.

In the series by Grieve and his colleagues it is interesting to note that of seven tumours (four being gastric carcinoma), one was a leiomyosarcoma. Leiomyosarcoma seems to be one of the types of malignant tumour found in the African, though it is rare. In the list of "other causes," in two cases the haemorrhages followed rupture of an aortic aneurysm into the oesophagus.

There are several papers which deal with the different causes of haematemesis. Little purpose would be served by dealing with these in turn, but rather to mention two good examples and thereby to compare them with my findings in Africa.

Gates (1959) investigated patients admitted to the Bristol Royal Infirmary with haematemesis or melaena, and of these only 10 (3 per cent.) were due to portal hypertension. Gastric disturbance amounted to 203 (67.6 per cent.). There were 40 cases with acute erosion or acute ulcer (13.3 per cent.). In a much more recent analysis of patients treated for haemorrhage from

---

**Table**

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-10</td>
<td>1</td>
</tr>
<tr>
<td>11-20</td>
<td></td>
</tr>
<tr>
<td>21-30</td>
<td>9</td>
</tr>
<tr>
<td>31-40</td>
<td>18</td>
</tr>
<tr>
<td>41-50</td>
<td>4</td>
</tr>
<tr>
<td>60</td>
<td>1</td>
</tr>
</tbody>
</table>

---

367
in the upper alimentary tract, MacCaig et al. (1964) findings tally very closely with those of Gates, namely, a high incidence of bleeding from a peptic ulcer in contrast to portal hypertension. Out of 587 patients seen with haematemesis of the upper gastrointestinal tract, 62.1 per cent. were caused by a bleeding peptic ulcer, whereas there were only 11 (19 per cent.) subjects who were found to have oesophageal varices. The acute group in whom no lesion could be demonstrated on barium meal was the second most important and accounted for 26.8 per cent. of the patients.

CONCLUSION

In Africa there seem to be the same causes of haematemesis as are found in Europe, but with the difference that cirrhosis of the liver is far more important as a cause. This is in keeping with the high incidence of cirrhosis in the African. On the other hand, whilst peptic ulcer is seen in the African, yet it is seen far less often than in the European. Malignant disease of the liver appears to be an uncommon cause of haematemesis.

REFERENCES


A Short Note on the Findings of Schistosomes in Baboons (PAPIO RHODESIAE)

BY

A. J. PURVIS,
I. R. ELLISON
AND
E. L. HUSTING, B.SC.
Research Laboratory, Salisbury.

A number of workers have commented on the presence of schistosomes in baboons—Nelson (1960), Strong et al. (1961)—and it appears from these sources that the finding of S. mansoni is a fairly common occurrence.

Autopsies were performed on seven baboons (Papio rhodesiae) which were killed within half a mile of Lake McIlwaine, 20 miles from Salisbury, Rhodesia. The largest baboons were found to have unidentified schistosome worms in the mesenteric veins, and one of them had terminal-spined ova in the brain.

METHOD

An incision was made from the sternum to the symphysis pubis. The small intestine, bladder and liver were removed.

The small intestine was examined immediately by manual displacement of the blood from the mesenteric vessels. This made the worms easier to see. The bladder and the liver were removed for examination in the laboratory. Liver crushes and scrapings from bladder lesions were examined microscopically for ova.

In the case of the largest baboon, small pieces of tissue from the brain, bladder and liver were digested for 18 hours in 10 per cent. potass. hydroxide at 52° C. The digested material was centrifuged and examined for ova.

RESULTS

Seven baboons ranging in estimated weight from 15 lb. to 75 lb. were examined. Two of these weighed 15 lb., two 25 lb. and a third 55 lb. The larger two weighed 65 lb. and 75 lb. The four lightest baboons, which were presumed to be young animals, showed no evidence of schistosome infection, as did the animal weighing 55 lb. The 65 lb. baboon, however, had one pair of worms in the mesenteric blood vessels. The 75 lb. baboon had two pairs of worms in the mesenteric veins. The brain of the larger one, examined by the digestion technique, was found to contain three terminal-spined ova identified as those of S. haematobium. The schistosome worms themselves could not be identified due to distortion.

Since evidence of schistosomiasis was found in two of a small sample of wild baboons, it seems that wild baboon populations may constitute a host reservoir for schistosomes capable of infecting humans in Rhodesia. The significance of this possibility should not be overlooked in the planning of research and control of bilharziasis. Of particular interest is the finding of S. haematobium in Papio sp., particularly since the ova appeared in the brain. A long-term quantitative study seems indicated and it is intended to pursue this investigation.

REFERENCES


Acknowledgments

We express acknowledgment to Dr. V. de V. Clarke for encouraging this study and to Dr. Burnett Smith, Acting Secretary for Health, for permitting the publication of this note.