Pyrexia and elevated erythrocyte sedimentation rate in acute gastro-duodenal haemorrhage

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Summary
Rises in the body temperature and the erythrocyte sedimentation rate (ESR) in the absence of significant associated disease, were each observed in one-third of a large series of patients with acute gastro-duodenal haemorrhage.

While only 15% of the patients showed both a pyrexial reaction and a rise in the ESR, fully 55% of the cases showed one or the other abnormality.

There was no significant difference in the incidence of these abnormalities in haematemesis from chronic duodenal ulcer, chronic gastric ulcer or acute gastric erosion and the rise in the ESR showed no significant correlation with the severity of bleeding.

Introduction
Despite the fairly early recognition of fever in gastro-intestinal haemorrhage (Ewald, 1897; Kroner, 1926; Bang, 1928; Purjesz, 1931; Dill & Isenhour, 1939), standard textbooks of medicine and monographs on gastroenterology generally omit reference to this and there is an even greater paucity of information on the frequency of elevation of erythrocyte sedimentation rate in these cases. As these abnormalities, when present, may on occasion lead to difficulty in diagnosis, a group of patients hospitalized on account of haematemesis has been studied to obtain information on the incidence of these abnormalities.

Materials and methods
Case records of all patients admitted to one general medical ward of the Royal Infirmary, Aberdeen, on account of upper gastro-duodenal bleeding during the years 1962–65 were examined. There was a total of 155 patients, comprising 103 patients with chronic duodenal ulcer, sixteen with chronic gastric ulcer and thirty-six patients who were thought to have bled from gastric erosions or acute gastric ulcer. The last group is designated as ‘acute gastric erosion(s)’ in this report; many had ingested drugs amongst which salicylates figured prominently. Forty-four cases were excluded on account of previous gastric surgery or associated diseases likely to influence the ESR and the body temperature.

The remaining 111 patients had 121 episodes of bleeding (Table 1). Where patients underwent a blood transfusion or gastric surgery, only the information prior to these procedures was used except in respect of the ESR which, as will be shown later, was uninfluenced by blood transfusion. As this, however, led to a considerable reduction in the number of patients in whom adequate temperature readings prior to transfusion were available, a retrospective random sample of 144 episodes of bleeding admitted to

<table>
<thead>
<tr>
<th>Source of bleeding</th>
<th>No. of patients</th>
<th>No. of episodes of bleeding</th>
<th>No. with adequate data</th>
<th>No. of episodes of bleeding with ESR (mm)</th>
<th>Total with ESR &gt; 15 mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic duodenal ulcer</td>
<td>78</td>
<td>88</td>
<td>83 (94%)</td>
<td>49 (34%)</td>
<td>34 (41%)</td>
</tr>
<tr>
<td>Chronic gastric ulcer</td>
<td>11</td>
<td>11</td>
<td>11 (100%)</td>
<td>5 (6)</td>
<td>6 (55%)</td>
</tr>
<tr>
<td>Acute gastric erosion(s)</td>
<td>22</td>
<td>22</td>
<td>20 (91%)</td>
<td>13 (7)</td>
<td>7 (35%)</td>
</tr>
<tr>
<td>Total</td>
<td>111</td>
<td>121</td>
<td>114 (94·5%)</td>
<td>67 (47)</td>
<td>47 (41%)</td>
</tr>
</tbody>
</table>
the hospital group during 1957–61 was examined. Data in respect of both the ‘current study’ and the ‘retrospective sample’ are described in this report.

**Erythrocyte sedimentation rate**

ESR was estimated by the standard Westergren method with 3.8% sodium citrate as diluent (Westergren, 1921). Sedimentation rates in excess of 15 mm in the 1st hour were regarded as abnormal.

**Body temperature**

Body temperature was measured by the ‘½-min’ clinical thermometers, at least once every 12 hr and in many instances at 4-hr intervals. Because of the vomiting, some patients had axillary temperature readings taken instead of the standard mouth recordings, but for the purpose of this study, only readings in excess of 99°F (37.2°C) were regarded as abnormal.

**Results**

**Erythrocyte sedimentation rate (ESR)**

Sufficient information on the ESR in the ‘current study’ was available in 114 of the 121 episodes of gastro-duodenal bleeding (Table 1). The overall incidence of raised ESR in this group was 41%. When the figures for the retrospective study are included (Table 2), the ESR was raised in 36% of 258 cases. There was no significant difference in the relative incidence of raised ESR between the various aetiological groups (0.70>P>0.50).

Where the ESRs were elevated, in three-quarters of the instances peak levels were recorded during the 1st week. In the majority of the patients the ESRs remained elevated for 7–10 days. Several patients had a moderately raised ESR at the time of discharge from the ward, but in all such cases normal readings were obtained at a subsequent outpatient visit 4–6 weeks later.

Sixty-eight of the 114 patients in the ‘current study’ required a blood transfusion. Of these twenty-seven showed a rise in the ESR as compared with twenty of the forty-six non-transfused cases. The difference between the two groups failed to show any significant influence of blood transfusion on the ESR (0.50>P>0.30).

**Body temperature**

Of the total of fifty-seven episodes of acute blood loss in the ‘current study’ in whom sufficient data were available, body temperatures higher than 99°F were recorded in nineteen patients (33.3%). When the figures for the retrospective sample are included (Table 3), pyrexia was observed in 37% of 171 patients. There was no significant difference in the relative incidence of pyrexia between the various aetiological groups (0.70>P>0.50).

In eighteen of the nineteen instances with pyrexia in the ‘current study’, the maximum temperature recorded was between 99 and 100°F, on the 1st day in eight cases, on the 2nd day in three cases, between the 3rd and the 5th day in five cases, and between the 6th and the 10th day in two instances. The last patient showed a peak temperature of 100.4°F on the 3rd day. The pyrexia lasted for 24 hr in fourteen instances, for 24–48 hr in two and for 72–120 hr in three instances.

**Correlation between pyrexia and the raised ESR**

Sufficient information on both parameters in the same patients was available in 160 cases in the two series (Table 4). Of these fifty-three (33%) showed a rise in the ESR and fifty-eight (36%) a pyrexial reaction.

During seventy-three (46%) episodes the patients were apyrexial and had a normal ESR. In twenty-nine instances the ESRs were elevated but the body temperature remained normal and in thirty-four cases a pyrexial reaction was noted with a normal ESR. In only twenty-four (15%)
of the 160 episodes, however, were both the ESR and the body temperature elevated. There was no correlation between the incidence of pyrexia and the raised ESR (0·20 > P > 0·10).

**Table 3**

<table>
<thead>
<tr>
<th>Source of bleeding</th>
<th>No. of episodes of bleeding with temperature &gt;99°F</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Current study (1962–65)</td>
</tr>
<tr>
<td>------------------------------------</td>
<td>-------------------------</td>
</tr>
<tr>
<td>Chronic duodenal ulcer</td>
<td>13 (45)</td>
</tr>
<tr>
<td>Chronic gastric ulcer</td>
<td>1 (4)</td>
</tr>
<tr>
<td>Acute gastric erosion(s)</td>
<td>5 (8)</td>
</tr>
<tr>
<td>Total</td>
<td>19 (57)</td>
</tr>
</tbody>
</table>

Figures in parentheses refer to total number of patients in each group.

**Table 4**

<table>
<thead>
<tr>
<th>Body temperature*</th>
<th>Erythrocyte sedimentation rate</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal</td>
<td>Raised</td>
</tr>
<tr>
<td>Raised</td>
<td>34 (21%)</td>
<td>24 (15%)</td>
</tr>
<tr>
<td>Normal</td>
<td>73 (46%)</td>
<td>29 (18%)</td>
</tr>
<tr>
<td>Total</td>
<td>107 (67%)</td>
<td>53 (33%)</td>
</tr>
</tbody>
</table>

* 0·20 > P > 0·10.

**Discussion**

A rise in the ESR or the body temperature was each noted in one-third of patients with acute gastro-duodenal haemorrhage in this series. There was no direct correlation between the two abnormalities as in only 15% of the episodes were both parameters elevated though fully 55% of the patients showed one or the other abnormality.

Kroner (1926) recorded pyrexia in 18% of 300 patients with peptic ulcer, the incidence in the small number of cases with haemorrhage (58%) being higher than in those without haemorrhage (16%). Bang (1928) noted an overall incidence of pyrexia of 50% in 358 patients with peptic ulcer, only 7% in those without bleeding but in 91% of 150 cases with haemorrhage. Comparable figures from Dill & Isenhour (1939) were 53% of 199 patients with peptic ulcer, 46% of 155 cases without haemorrhage and 79·5% of the forty-four patients with haemorrhage. Thus all authors are agreed that the incidence of pyrexia is higher when there is acute blood loss. The relatively higher incidence of pyrexia in the earlier series as compared with the present study may well have been due to failure to exclude patients who received blood transfusions and possibly also due to the increasing sophistication in the diagnosis of associated conditions likely to lead to pyrexia on their own account.

Comparable data on the incidence of elevated ESR in gastro-duodenal haemorrhage are scarce. Mathisen (1950) observed a rise in body temperature and/or the ESR in 160 (56%) of 285 cases of gastric and duodenal ulcer. Thirty-two of the cases had haemorrhages at the time of the study and a number of others were anaemic. Further breakdown of the figures is not possible from the published data.

While the diagnostic importance of these manifestations is clear, there is less certainty regarding their mechanism and significance. Hurst & Stewart (1929) concluded that the 'old view that the pyrexia is due to absorption of products of putrefaction of blood is generally correct'. Anemia itself has also been incriminated, though phlebotomies of 500–600 ml of blood in four patients were without significant influence on the body temperature (Dill & Isenhour, 1939). The latter workers also failed to obtain pyrexial reactions in dogs venedected of one-quarter of the total blood volume, even when the animals were fed the venedected blood by gavage. Similarly, Schiff, Shapiro & Stevens (1944) failed to obtain any rise in the body temperature in six patients who were given 750–1850 ml of citrated blood by gastric tube.

Mathisen (1950) has suggested that the pyrexia may be due to either an associated gastritis or localized peritonitis from penetration of the
Acute gastro-duodenal haemorrhage

ulcer. In the present study neither the incidence of the pyrexial reaction nor the raised ESR was significantly different between the three groups with blood loss from chronic duodenal ulcer, chronic gastric ulcer and acute gastric erosion(s). Thus, although in some patients localized peritonitis may have contributed to the rise in the ESR and the body temperature, it would seem that in the series as a whole an associated gastritis was the more likely aetiological factor. As routine gastroscopy with the fiberscope in the acute management of haematemesis is now standard practice in several large centres, this aspect of the problem should lend itself to a more direct examination.

It should be noted, however, that in the present study there was a dissociation between the incidence of pyrexia and the raised ESR. Further, although both Dill & Isenhour (1929) and Schiff et al. (1944) found pyrexial reactions to be more common following massive or moderately severe bleeding than with milder degrees of haemorrhage, in the present study there was a lack of correlation between the severity of bleeding, as assessed by the need to transfuse, and the rise in the ESR.

Acknowledgments
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References
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