Case reports

Table 2. Maternal and neonatal plasma 11-hydroxycorticoids
"Cortisol" concentrations (µg/100 ml)

<table>
<thead>
<tr>
<th>Age (days)</th>
<th>Number of cases</th>
<th>Range in plasma concentrations</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Basal</td>
<td>Post-Synacthen</td>
</tr>
<tr>
<td>Normal Infants</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>25</td>
<td>11-77</td>
</tr>
<tr>
<td>3</td>
<td>25</td>
<td>6-29</td>
</tr>
<tr>
<td>5-7</td>
<td>20</td>
<td>6-31</td>
</tr>
<tr>
<td>Maternal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Third trimester</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>Fourth day</td>
<td></td>
<td>✓ 52</td>
</tr>
<tr>
<td>Post-natal vaginal delivery</td>
<td>8</td>
<td>✓ 55</td>
</tr>
<tr>
<td>Post-natal Caesarean section</td>
<td>4</td>
<td>✓ 73</td>
</tr>
</tbody>
</table>

Recovery from severe paraquat poisoning

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Summary
Recovery from poisoning with the herbicide, paraquat (20% concentrate—'Gramoxone') is rare (Malone et al., 1971). A patient is reported who survived despite ingesting an apparently fatal dose. Treatment consisted of forced diuresis and a single haemodialysis. Oxygen, steroids and immunosuppressives were not given.

References

Case report

A 28-year-old peat worker accidentally took from a lemonade bottle a large mouthful of 'Gramoxone' on an empty stomach. He noted the bitter taste and spat out what was left in his mouth. He did not vomit. Over the next 48 hr he developed a sore throat and severe dysphagia.

On admission, 52 hr after ingestion, there was superficial ulceration of the soft palate and pharynx. Scattered rhonchi were heard on auscultation. The previous medical history was unremarkable. He smoked twenty cigarettes a day.

Investigations. The abnormal laboratory findings were a urinary paraquat level of 6800 μg/100 ml; PaO₂ 75 mmHg (normal 85–95); D₂CO 26 ml/mmHg/min (predicted normal 33); FEV₁% 68.5 (predicted normal 83); blood urea 68 mg/100 ml; GFR 60 ml/min; excretion of renal tubular cells 350,000/hr (normal 14,000–57,000); urinary lysozyme 2.5 μg/ml (normal 0.63 ± 0.43). The chest X-ray showed increased vascular markings. (Fig. 1).

Treatment. Forced diuresis was begun with 200 ml of 20% mannitol and a high fluid intake, mainly oral, and output (10–24 l/day) was maintained throughout the admission. A 5-hr haemodialysis was carried out 67 hr after admission, but no paraquat was recovered from concentrates of the dialysing fluid. Oxygen was withheld, although the PaO₂ fell to 66 mmHg on the sixth day after ingestion. D₂CO did not fall below 25 ml/mmHg/min (day 10).

The renal tubular cell excretion was maximum on day 11 (735,000/hr). There was proteinuria (100 mg/100 ml), but no glycosuria, phosphaturia or amino-aciduria.

Hepatic damage developed and the bromsulph-
Paraquat has been described as a 'hit and run' poison (Barnes, 1968), but in view of recent reports of the apparent value of forced diuresis, the severity of pulmonary fibrosis and the prognosis in paraquat poisoning would appear to depend not only on the quantity ingested but also on the rate of removal. This case shows that even when a delay occurs before the patient is seen that the outcome of poisoning with 20% paraquat is not inevitably fatal.

Acknowledgements

We wish to thank Dr P. D. Bewsher for permission to report this case and all the staff who contributed to the care of the patient. We also wish to thank Dr D. M. Ferguson (ICI Ltd) for his advice and co-operation.

References


Primary haemorrhagic thrombocythaemia with Philadelphia chromosome

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Summary

A case of primary haemorrhagic thrombocythaemia with the Ph1 chromosome is described and its relation with myeloproliferative disorders is discussed.

Persistently raised platelet counts may be associated with polycythaemia vera, agnogenic myeloid metaplasia (Linman & Bethell, 1957), chronic myeloid leukaemia (Minot & Buckman, 1925), after splenectomy (Hardisty & Wolff 1955), and sometimes with no obvious cause (Ozer et al., 1960), called primary haemorrhagic thrombocythaemia.

The Philadelphia (Ph1) chromosome was discovered by Nowell & Hungerford (1960) in patients with chronic granulocytic leukaemia. Since then the Ph1 chromosome has been demonstrated in patients who initially presented as polycythaemia, in eosinophilic leukaemia (Gruenwald et al., 1965) and in primary haemorrhagic thrombocythaemia (Tough et al., 1963; Dougan, Woodliff & Onesti, 1967).

I present here a further case of primary haemorrhagic thrombocythaemia with the Ph1 chromosome.

Case report

A thinly-built married female aged 75 years was admitted to hospital in May 1970 for pain in the upper abdomen, particularly in left hypochondrium, bruises on the legs, a history of haematemesis, moderate anaemia and weight loss. The liver and spleen were not palpable but there was some tenderness in the splenic region. There was a past history of similar recurrent attacks of abdominal pain mainly in the splenic region and repeated gastro-intestinal haemorrhage for many years.

Investigations in a different hospital in 1959 revealed hypochromic anaemia with haemoglobin 10·5 g/100 ml, and normal white cell count, but a platelet count was not performed. Various biochemical investigations and radiological investigations including barium studies of the gastro-intestinal tract at that time did not reveal any
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