suggested a dyserythropoietic basis for the anaemia. The finding
of a reduced red cell survival and the absence of iron in the bone
marrow fragments, however, indicated that blood loss was con-
tributing to the anaemia. These findings prompted an examination
of her personal possessions, and a number of syringes, needles,
and tourniquets were found. These were examined in a scintilla-
tion counter but they were free of radioactivity. The pyrexia was
shown to be factitious when she was seen rubbing the thermo-
meter on the sheets. The patient was isolated for 10 days, and
during this time the haemoglobin rose to near normal levels. At the
end of the period of isolation a new supply of tourniquets, syringes,
and needles were found in her locker; it was never fully established
how they were acquired.

The patient was told of the conclusions of our investigation and
sent back to Italy for psychiatric treatment.

Case 2
In November 1970 a 29-year-old Yugoslav technician was referred
to us for investigation of anaemia. He had been in good
health until 1964, when he experienced the first of many spon-
taneous, heavy nose bleeds. During the next three years he was
admitted to hospital on several occasions for blood transfusion and
nasal packing, and in 1967 a nasal septotomy was performed.
After this procedure nose bleeds were less frequent but he con-
tinued to be anaemic. In 1968 he was admitted to hospital with the
sudden onset of a left hemiplegia. He was severely anaemic.
No blood was seen in the cerebrospinal fluid and a moderate func-
tional recovery followed. Between 1968 and 1970 he was persistently
anaemic and required blood transfusions at monthly intervals. His
family and social history contained nothing of note.

On admission he complained of left-sided paraesthesiae. Physical
examination showed a well-built man with no anaemic stigmata and
a left hemiparesis.

Initial laboratory results were: haemoglobin 3·9 g/100 ml, M.C.H.C.
29%, reticulocytes 0·8%, platelets 282,000/mm³, and W.B.C. 5,000/mm³
(normal differential). On the blood film the red cells were noticeably hypochromic and microcytic. The bone mar-
row was hypercellular with severe erythroid hyperplasia. Dys-
erthropoietic changes were present in the developing erythro-
blasts. Iron was absent from bone marrow fragments. Serum iron
was 16 µg/100 ml and total iron-binding capacity 414 µg/100 ml.
Serum vitamin B₁₂ and folate levels were normal, as were blood
urea, bilirubin, and liver enzymes. Radiological examination of the
alimentary tract showed no abnormality. Acidified serum lysis and
direct antiglobulin tests were negative.

A ⁵¹Cr-labelled red cell showed a severely reduced lifespan
(⁵¹Cr half-life 10 days). No excess of ⁵¹Cr was detected in the urine
or stool. A ferrokinetic study with ⁵⁹Fe showed a rapid plasma iron clearance with a utilization of 80% day 8.

These results were consistent with a diagnosis of iron deficiency
due to blood loss. Intravenous and oral iron were given and this
was followed by a reticulocytosis of 17%. The bone marrow
appearances improved and stainable iron reappeared in marrow
fragments. The problem then resolved into one of establishing the
site of blood loss. For this a red cell survival test was repeated with ⁴¹Ca-labelled cells and a whole-body counting system was
used to measure the rate of isotope elimination. These studies
confirmed that the rapid disappearance of red cells from the circula-
tion and isotope from the body were of the same order. However,
no excess of isotope was detected in the urine or stool. A careful
watch on the patient disclosed that he spent long periods in the
toilets, and on several occasions fresh blood was found in the
bowel after these visits. Venepuncture marks in the left antecubital fossa
(hemiparetic side) were then noted. These had not been made by
medical personnel, and though no blood-letting apparatus was
found among his possessions all the evidence pointed to self-
induced bleeding.

The left hemiparesis was also investigated. A right carotid
arteriogram failed to fill a right middle cerebral artery, and it was
concluded that there was an occlusion of this artery at its origin.
The cause of this occlusion was presumed to be thrombotic.

Comment
Only a small number of cases of factitious anaemia have
been documented, the first two being reported in 1963 by
Dally et al. Bernard et al. (1967) described 12 cases where
anaemia was thought to be a consequence of self-induced
bleeding. As the two present examples of factitious anaemia
were identified during a six-month period it seems possible
that this diagnosis should be considered more often in patients
presenting with obscure anaemia.

The bone marrow in both patients showed pronounced
dyserythropoiesis, a feature not generally regarded as
characteristic of iron deficiency. This probably explains why
the diagnosis was not made earlier and an alternative cause
sought to explain the bizarre bone morphology (Hill et al.,
1972).

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Extrusion of Small Intestine due to
Rupture of Previously Normal
Umbilicus

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Extrusion of the abdominal viscera can occur as a complica-
tion of several conditions. The commonest of these are
exomphalos, which is usually diagnosed at birth, and a patent
omphalomesenteric duct, which frequently declares itself by a
persistent umbilical discharge. A much rarer condition is
rupture of an umbilical hernia. Abdominal viscera are also
found outside the abdominal wall in gastrochisis, but here the
rupture is parambilical in site and the umbilicus itself is
entirely normal.

A case of primary umbilical rupture is presented which does
not fit into any of the above categories.

Case History
A female infant was born normally at term in a general
practitioner unit after an uneventful pregnancy. The birth weight
was 2,800 g. Progress was satisfactory, and the umbilical cord
dried and separated at the base on the sixth day. The umbilicus was
subsequently passed as normal by the district midwife, the general
practitioner, and the mother (who had two other children, both
under 2 years).

The mother had noticed some slight reddening of the skin around
the umbilicus three or four days before admission to this hospital

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at the age of 7 weeks. The umbilicus itself was still entirely normal, there was no umbilical discharge, and the baby was otherwise well.

On the day of admission the umbilicus suddenly ruptured after a bout of crying, and loops of small intestine prolapsed through the defect. There had been no vomiting or bowel disturbance. On examination of the abdomen it could be seen that about 2 ft. (60 cm) of small intestine had herniated through the base of the umbilicus. The bowel itself looked healthy and was peristalsing normally (Fig. 1). There was no obvious reddening of the skin around the umbilicus. The child was not shocked or hypothermic, and no other abnormality was detected.

At operation a short stump of umbilical cord was still present, and at the base of the cord there was a small oval defect through which the gut had protruded. The gut was congested but looked viable and was covered only by visceral peritoneum. The falciform ligament was above the defect and the lateral umbilical ligaments below it (Fig. 2). The urachus was obliterated, and no abnormality of the abdominal contents could be found; in particular, there was no Meckel's diverticulum or evidence of an omphalomesenteric duct. There was no umbilical sepsis.

In order to reduce the bowel the umbilical defect was extended transversely by incisions on either side, allowing the intestine to be returned into the abdomen without difficulty. The short stump of the umbilical cord, which was oedematous, was ligatured and resected just inside the abdominal wall. The defect in the linea alba and peritoneum was repaired in one layer with a purse-string suture, and skin closure was effected by interrupted silk sutures.

Postoperatively the baby made an uneventful recovery. Prophylactic ampicillin had been administered before surgery and was continued thereafter for seven days. Bowel sounds returned within a few hours and a normal oral feeding regimen was started within 24 hours of surgery. The abdominal wound healed satisfactorily. The baby was discharged from hospital after 10 days and has remained well.

Comment

Extrusion of the abdominal contents in infants is rare. Herniation of the small intestine through a patent omphalomesenteric duct has been described (Scalettet et al., 1952; Howard et al., 1953). In the present case there had been no umbilical discharge or umbilical polyp, and no evidence of a duct or duct remnant could be found at operation.

Rupture of an umbilical hernia without extrusion of the intestine has also been reported. This must be a very rare occurrence, with only three cases in the English literature (McLean, 1950; Strange, 1956; Harding-Jones and Robson, 1965). In all three instances, however, the umbilical hernia had been noted before rupture occurred.

Exomphalos and gastrochisis can be ruled out in this infant as both would have been obvious either at birth or shortly afterwards (Rickham and Johnston, 1970).

The reddening of the periumbilical skin noticed three or four days before rupture raises the possibility of sepsis being a predisposing factor here. Although a possibility, there was no evidence of sepsis found on examination or at operation. In any case we can find no reference to umbilical rupture in an infant after sepsis.

The present case does not fit into any of the above categories. We therefore suggest that it is a case of rupture of an apparently normal umbilicus, with sepsis as a possible predisposing factor.

We would like to thank Dr. A. D. Griffiths for his helpful advice in the preparation of this report.

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