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 contributing 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 scintillation counter but they were free of radioactivity. The pyrexia was shown to be factitious when she was seen rubbing the thermometer 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 spontaneous, 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 septostomy was performed. After this procedure nose bleeds were less frequent but he continued 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 functional 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 marrow was hypercellular with severe erythroid hyperplasia. Dyserythropoietic changes were present in the developing erythroblasts. 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% by 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 ⁵¹Cr-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 circulation 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 bowl 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 Daily *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 a 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).

We should like to thank Professor Dacie for his help and encouragement, and Professor Bianchini in Cremona and Dr. Rolovic in Belgrade for referring patients under their care.

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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 complication 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 gastroschisis, but here the rupture is paraumbilical 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 2½ 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

FIG. 1—Small intestine extruding through base of umbilicus.

FIG. 2—Findings at operation.

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 (Scalettar *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 with 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 gastroschisis 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.

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