CASE REPORTS

TWO CASES OF DUODENAL ULCERATION IN CHILDREN

BY

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A growing number of articles (Paterson, 1922; Proctor, 1925; Kennedy, 1933; Craig, 1934; Mills, 1934; Burdick, 1940) show that peptic ulceration in children is not regarded seriously enough as a cause for puzzling gastro-intestinal symptoms. The admission of two cases of duodenal ulcer into a children's unit within a period of six months forms the occasion for this report.

Two groups of cases are likely to be met, a first broad division separating a 'primary' type in which the ulcer and its effects fill the foreground of the clinical and pathological picture, and a 'secondary' type, to be discussed no further, in which an ulcer develops in a child already suffering from some serious condition such as gastro-enteritis, peritonitis or a general infection.

Kennedy (1933) has classified the 'primary' cases into four with well-defined age incidence—neonatal, from a few weeks to twelve months, from one to nine years, and an adult type in later childhood. Cases since reported fit into this scheme.

Types of 'primary' ulceration

Neonatal type. In that miscellany of pathological conditions dubbed haemorrhagic disease of the newborn, gross melaena and haematemesis may arise from a bleeding ulcer. When death results from blood loss or from peritonitis following perforation, a large ulcer may be found. In one fatal case Kennedy (1924) reported microscopic ulceration of the duodenal mucosa where no macroscopic lesion was visible. If this is pathologically acceptable, recovery can be expected in some cases, and diagnostic difficulty in all, since a barium skiagram of stomach and duodenum would be normal and an autopsy without serial sections misleading. When there is serious bleeding from the gut with anaemia, without enlargement of spleen or liver and without erythroblastosis, peptic ulceration is a possible diagnosis, and treatment directed against the bleeding and the anaemia should be with vitamin K and repeated blood transfusions.

Infantile type. The diagnosis is rarely made until the onset of haemorrhage, and usually not until the post-mortem examination. A certain resemblance

between reported cases and case 1 shows that there are suggestive features in the history. A good start with feeding and weight gain is interrupted at the age of a few weeks or months by an infection, especially of the upper respiratory tract. Thereafter failure to thrive is associated with feeding difficulty, and there is unwillingness to take or to finish feeds, natural or artificial, with vomiting if feeds are pressed, and attacks of abdominal pain or discomfort. Changes in feeding may cause temporary improvement. The mother is accused of over-anxiety. In case 1 anxiety was increased by the laryngeal stridor which she understood to be due to a growth in the larynx capable of suffocating the baby at any moment. After further infections comes the terminal stage of bleeding, often mistaken for acute intestinal obstruction and treated by exploratory laparotomy. Whether or not parenteral blood and fluids with sedatives and alkalis by mouth would avert a fatal issue remains to be seen.

Childhood type. In children of between one and nine years vague gastro-intestinal symptoms, loss of appetite, failure to gain weight, if accompanied by abdominal pain, especially in the upper abdomen, or by epigastric tenderness, or by anaemia, should suggest an ulcer. Here again in many cases there is upper respiratory infection, and, in case 2 there was also evidence of psychological disturbance, with an unusually difficult domestic background. Without obvious or occult bleeding from the gut, or radiographic proof of an actual ulcer, the diagnosis is open to grave doubt. The statement of Moore (1941) that 'though desirable the demonstration of a crater is not always possible or necessary' may only confuse still further the already difficult subject of chronic gastro-intestinal disease in childhood. Fractional test meals are not apparently of value.

When the case presents as one of anaemia with gastro-intestinal symptoms and bleeding from the gut the differential diagnosis is from a bleeding ulcer in relation to ectopic gastric mucosa, ulceration from tuberculosis of the bowel, and intestinal polyps or parasites. Malignant neoplasms of the gut are exceedingly rare in the age period under discussion. In bleeding from an ulcer in a Meckel's diverticulum anaemia is characteristically acute and severe, and there is abdominal discomfort more than pain, without local tenderness. Appetite remains good throughout, there is no vomiting or haematemesis, weight gain is not affected. There are no symptoms between attacks. Diagnosis may be made by special barium meal examination or by laparotomy.

Adult type. In the later childhood years peptic ulceration produces a picture resembling that in adults.

Case reports

The following two cases represent the infantile and the childhood types.

1. D. B., male, born 1/3/41, was admitted to hospital 17/7/41 and died 8/8/41 (aged 5 months). Birth was three weeks early, but otherwise normal, and birth weight was 6 lb. 12 oz. He seemed healthy, although jaundiced from the first to the twenty-first day. Breast feeding was established and progress was good until the age of four weeks, when he had a 'cold' with nasal discharge and a cough. He was unhappy, refused feeds, slept badly, cried much and

became difficult to handle. At six weeks he was weaned on to a 'humanized' dried milk (2½ level teaspoons in 3 oz. with ½ level teaspoon of glucose, three-hourly). At eleven and a half weeks (21/5/41) he was taken to a clinic where the feed was changed to a half-cream dried milk (5 oz. feeds four-hourly with later addition of 1 level teaspoon of glucose to each). His weight was then 10 lb. 7 oz., giving an average gain of over 6 oz. a week from the second week. This proved the maximum recorded weight. A vitamin A and D concentrate was added on 4/6/41 and on 18/6/41 the feed was changed again to another humanized dried milk (4 scoops to 5 oz. four-hourly). With each change he improved, but soon would lose his appetite and either vomit after or regurgitate curds between feeds. He would begin the feed with gusto, taking the first two or three ounces greedily, and then would take slowly and with much persuasion. The average time was half an hour varying from twenty minutes to one hour. Bowels were opened normally until 7/7/41, when he began green frothy motions, three or four each day. The strength of the feed was lowered to three scoops in five ounces and the motions improved.

Breathing had been noisy from birth, worse since the 'cold.' A loud croaking accompanied inspiration and expiration, the intensity varying, almost disappearing at night, becoming worse after feeds. Respirations were never laboured nor rapid, and he was never blue.

Examination on admission (17/7/41). A four months' old well baby, with mucous membranes of good colour, not cyanosed. Weight 10 lb. 4½ oz. Laryngeal stridor is present, and is unaffected by change of position of the head. There is suprasternal inspiratory recession but normal expansion of the lower ribs. Skull normal and general examination otherwise normal.

Diagnosis. Congenital laryngeal stridor; feeding difficulty associated with over-anxious mother and poor management (gross libels as was later apparent).

Progress was at first good. He was fed part separated and part full cream dried milk with added sugar, 5 oz. four-hourly ($\times 5$). The separated milk was gradually reduced and the sugar increased, but after eight days (25/7/41) motions increased to six and were looser, so that sugar was reduced. He began to vomit 29/7/41, temperature rose to 99.8° F., and pulse to 150, and thereafter he refused feeds, which were reduced in strength until on 31/7/41 he was put on to boiled water. Later in the day he was dehydrated and was given a slow sub-cutaneous injection of normal saline (300 c.c.). A left myringotomy for a red bulging drum produced blood-stained fluid, but on the next day a discharge of pus. Though there was slight improvement, he remained a sick baby with irregular fever (up to 100° F.), poor appetite, vomiting if feeds pressed, and one to three stools a day, not particularly loose. No non-lactose fermenting organisms were grown on stool culture. Two stools were streaked with dark blood and some mucus on 7/8/41, and later he looked ill, frowning as if in pain, the abdomen being soft, not distended, and tender over the right upper quadrant, where the liver was enlarged to two fingers breadths. In one vomit was a small streak of bright blood. Stool culture, repeated, was negative, and blood count showed Hb. 94 per cent., R.B.C. 4,880,000 per c.mm., W.B.C. 31,600 per c.mm. 8/8/41 at 3.0 a.m. he passed much bright blood without faeces per rectum. At 5.30 a.m. he was moaning and frowning in pain, T. 97° F., P. 130, R. 36.

He appeared extremely ill, with upper abdominal distension and tenderness under the liver edge. No tumour was felt, but as no faeces had been passed for twenty-four hours, and there was distension, pain and bleeding from the gut, a diagnosis of intestinal obstruction was made, possibly due to intussusception. At operation (Mr. J. P. Hosford) a clear fluid was found in the peritoneal cavity. The small intestine and colon up to the hepatic flexure

were distended and contained dark fluid. No obstruction and no Meckel's diverticulum could be seen. The baby died twelve hours later.

Post-mortem examination (Dr. Joan Ross) revealed a wide duodenal ulcer on the posterior wall with an eroded vessel in the base. There was altered blood in the duodenum. The liver was yellow, necrotic, and greatly enlarged, and showed in sections intense fatty degeneration, only a few cells being recognizable. The kidneys showed cloudy swelling. The larynx did not differ from that of a normal baby of the same age, and on section the ary-epiglottidean folds appeared normal.

2. A. P., male, unaccompanied evacuee, born 9/3/32, was admitted to Cell Barnes Hospital 21/1/41 from his billet on account of dizziness and abdominal pain. He had been sent home from school as he was dizzy, felt sick, had sharp upper abdominal pain and a bad appetite.

He was born at term, weighing 7 lb. 2 oz., and was blue for two weeks. After two weeks of breast feeding he was fed artificially without cod-liver oil, but with orange juice. He had nasal catarrh and attended the Belgrave Hospital. At one year he was admitted to Lambeth Hospital for pneumonia, later to St. Giles' Hospital with chest trouble, then to Goldie Leigh and Homerton with chickenpox. He was always in and out of hospital, sometimes with cough and chest trouble, sometimes on account of sickness. He used to have poor appetite, would be coaxed and then would vomit at table. There were always rows over meals and his mother lost her temper and smacked him. He had some abdominal pain, at first attributed to constipation and treated with paraffin. He always liked meat and milk but not vegetables; he took eggs and butter, but was not fond of fried food.

October 1938, as he was pale and had bronchitis, he attended St. Thomas's Hospital for sun-ray. He made no progress, and 23/11/38, aged 6½ years, he was admitted to Lambeth Hospital on account of sore throat and purpuric spots on legs. (Blood count Hb. 68 per cent., R.B.C. 4,230,000 per c.mm., W.B.C. 10,000 per c.mm. Normal differential count.) Progress was satisfactory until 19/10/38, when he vomited blood-stained matter and his haemoglobin fell to 32 per cent. He was given a blood transfusion and treated as if he had a peptic ulcer. A platelet count at this time was normal. There is no record of a barium meal examination. The diagnosis made on discharge was follicular tonsillitis and haematemesis (probably due to a peptic ulcer). (P. J. Watkins, Medical Superintendent 8/3/41). Since then he has been in fair health except for poor appetite until just before admission, although he confessed to occasional upper abdominal pain unrelated to meals or exertion. Latterly he has felt a little dizzy and short of breath on exertion.

He sleeps well, became clean and dry early and without difficulty. Measles at 2 years. Chickenpox at 5 years.

Domestic history. The mother lived with his father for only six weeks and then left him. He knows of his real father but has seen him seldom. In 1934 the mother married again, and the stepfather gets on well with the boy. There was a miscarriage at six or seven months in 1938. There has never been a settled home: at first mother and baby lived with mother's mother and mother worked. Later mother left him entirely with grandmother but would have him with her for occasional weekends. At other periods he has been in charge of a foster mother, and then on many occasions in various hospitals. Mother does not think she was missed. Mother's sister was fond of him: she had pneumonia and pleurisy and had an operation, after which she died suddenly in June 1940. Shortly after mother's father died. The boy was strongly attached to both and was extremely upset. Then the 'blitz' started and he was evacuated to St. Albans.

On examination. Pale, ill boy with yellow tint of skin. Mucous membranes markedly pale. Sclerotics not yellow. (T. 99° F., P. 100, R. 24.) Weight 65½ lb. Eyes and fundi normal. Teeth good. Tonsils large, not inflamed. No enlarged lymph nodes. Systolic murmur at base of heart but otherwise normal. Blood pressure 106/60 mm. Hg. Abdomen soft, liver and spleen not enlarged. No tenderness, no tumour. Limbs normal. Urine clear, no albumin, no blood, no abnormal pigment. Stool formed, dark and contains obvious altered blood. Wet films showed R.B.C. in considerable numbers. Afebrile, ill, lassitude, anorexia.

After about a month general condition improved greatly and appetite became good.

Investigations :

- 22 1/41 R.B.C. 2,340,000 per c.mm. Hb. 48 per cent. (6.62 gm.). C.I. 1.12.
W.B.C. 7000, polymorphs 4760 (68 per cent.), eosinophils 70 (1.5 per cent.), basophils 70 (1 per cent.), lymphocytes 1750 (25 per cent.), mononuclears 350 (5 per cent.), aniso- and poikilocytosis, and polychromasia. No nucleated cells.
- 25 1/41 Reticulocytes 2.4 per cent. Fragility of R.B.C.: haemolysis begins at 0.45 per cent., complete at 0.4 per cent. Fouchet negative. Van der Bergh direct negative. Indirect less than 0.4 units.
- 25 1/41 Wassermann, Kahn and Meinicke Reactions negative.
- 26 1/41 Occult blood (Benzidene) strongly positive.
- 27 1/41 " " " negative.
- 31 1/41 Bleeding time: 1 min. 30 sec. (Duke).
Coagulation time: 6 min. (Lee and White 4-7 min.).
Reticulocytes 2.4 per cent.
Platelets 196,000 per c.mm.
R.B.C. 2,640,000 Hb. 50 per cent. (6.9 gm.), C.I. 0.94.
- 12 2/41 R.B.C. 3,660,000, Hb. 70 per cent. (9.7 gm.), C.I. 0.97.
Reticulocytes 2.5 per cent.
- 1/4/41 Occult blood: 3 specimens positive.
- 19/5/41 R.B.C. 4,740,000, Hb. 84 per cent. (11.6 gm.), C.I. 0.89.
W.B.C. 9500.
- 24/5/41 Fractional test meal (7 per cent. alcohol) 30 c.c. at 11.50 a.m.

	2/10 HCl	FREE HCl GM. PER CENT.	TOTAL ACIDITY		BILE	MUCUS
			2/10 HCl	GM. HCl PER CENT.		
Resting juice ..	36	0.131	60	0.219	0	0
12.05 0 ..	23	0.084	40	0.146	0	0
.40 35 minutes ..	0	0	19	0.069	0	0
1.15 70 minutes ..	22	0.080	40	0.146	0	0
	No blood					
	No starch					

Radiography (reports by Dr. G. Simon)

26/1/41 Plain abdomen: normal.

AFTER BARIUM—stomach shows normal peristalsis and emptying.

Duodenum: fairly gross deformity of the duodenal cap with projection. Diagnosis: duodenal ulcer.

- 20/2/41 AFTER BARIUM: Stomach still contains fluid (last food 12 hr. before). There is deep peristalsis with intermittent emptying.
Duodenum: There is still deformity of the cap with projections. The ulcer crater does not appear to be healed.
- 24/3/41 AFTER BARIUM: Deformity of the duodenal cap is present as previously, in fact a small diverticulum appears to be forming near the lower part of the cap (fig. 1).
- 20/5/41 Duodenal cap now smooth and regular in outline. The ulcer crater appears to have healed.

FIG. 1.—24.3.41. At A pseudo-diverticulum of the duodenal cap.

Intelligence tests (Dr. Millman): Showed a high standard of mental ability in all the tests used.

Psychological examinations: May, 1941 (Dr. Lovel Barnes):

'In examining this child I endeavoured to estimate two important problems: (a) was there an emotional factor in the commencement of his illness; and (b) to what extent his progress was influenced by his psychological condition. I found it extremely difficult to form an estimate of (a). It was necessary to work back from the state in which I found the child to-day and a subsequent interview with the mother did not clarify the position. With regard to (b) I considered that he was an intelligent but insecure child. There was a considerable amount of anxiety about his illness which he managed to cover to quite an extent. The anxiety, however, did not all centre on the illness; a great deal of it was concerned with the idea of separation from his mother and could be considered a true separation anxiety. In my interview with the mother this was discussed and I was so impressed by the importance of it that I agreed to the mother taking the child back to Clapham (although he was exposed to the risk of air raids) rather than produce further anxiety by a prolonged separation. It is evident, therefore, that there is considerable emotional instability in this case which has obviously been there since early childhood. It is now undoubtedly influencing his health, and it cannot be disregarded as a possible precipitating cause although there is of course no proof of this. I do consider, however, that it would play a strong part in producing a recrudescence of the symptoms at a future date.'

Treatment. Bed for ten weeks on post-Lenhartz diet with ascorbic acid 50 mgm. and a vitamin A and D concentrate, 15 minims a day and liquid paraffin, 30 minims, three times a day after meals. Two tablets of iron sulphate twice a day from 4/2/41.

Weight on discharge was 70 lb.; he was eating well. Since then he has been free from symptoms, and weight (6/3/42) at ten years is 81 lb. He has been living with his mother but attends school regularly.

Summary

Two cases of duodenal ulcer are reported in detail. One (infantile type) died at five months. The other (childhood type) bled first aged $6\frac{1}{2}$ years, was diagnosed at 8 years 10 months, and made a good recovery.

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