DUODENAL ULCERS IN CHILDREN

BY

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The occurrence of peptic ulcers in infancy and childhood has been known for over 100 years. Its frequency has not been realized until recently and does not seem to be generally recognized. Several specialized textbooks on children's diseases give it scant mention, and only discuss the acute perforating and bleeding ulcers of the neonatal period.

Apart from its surprisingly high incidence in children, when looked for, peptic ulcers in childhood differ in a number of respects from the textbook picture of ulcers in adult life. Below, therefore, some of the literature on this subject is reviewed and a series of 20 cases of duodenal ulcers in children between the ages of 7 and 15 seen at Booth Hall Hospital in the last five years is presented.

Incidence and Classification

Proctor (1925) found only 2 children among 8,260 cases of peptic ulcers in the records of the Mayo Clinic, and Guthrie (1942) found 9 cases in 6,059 post-mortem examinations at the Glasgow Children's Hospital between 1914 and 1941. Karlström (1949), reviewing the incidence of peptic ulcers in 26 paediatric clinics in Sweden, found 30 cases in a five-year period. On the other hand, Girdany (1953) reported 45 children with ulcers between the ages of 1 and 11 during only one year at the Children's Hospital, Pittsburg, and Alexander (1951) found 30 duodenal ulcers among 254 x-ray studies of the gastro-intestinal tract in children.

The history of ulcer pain in adults may go back to childhood. Proctor found in a series of 1,000 adult patients with duodenal ulcers that 26 dated the onset of their symptoms to childhood, even to the age of 4 or 5. Our series in Booth Hall shows a suspicious yearly increase in the incidence of duodenal ulcers since our interest in the problem. It appears that "those who seek shall find."

Cases of peptic ulcer in children are classified into primary, or secondary to burns, infection, or other illnesses. Another classification divides them into neonatal, infantile, childhood (1-9), and adolescent types. Gastric ulcer is much less frequent. Its ratio to duodenal ulcer has been estimated between 1:7 and 1:3. Our cases were all primary. They include only childhood and adolescent cases, and the younger age group is therefore not discussed. Only cases of duodenal ulcers are included.

Although from the literature the sexes appear to be equally susceptible to duodenal ulcers, 14 of our 20 cases

were males. At the time of diagnosis four were aged 7, one 8, one 10, five 11, three 12, and two aged 13, 14, and 15 respectively (Fig. 1). The length of the history varied from 2½ years to one day, with an average of 10 months. The delay was longest when the presenting symptom was pain and shortest when haematemesis was early. In four of our patients there was a family history of duodenal ulcer in one of the parents.

Clinical Picture

In infancy duodenal ulcer is an acute condition characterized by rapidly developing pathological changes and showing little tendency to repair. Perforation and haemorrhage are therefore commoner in this age group. In older children the disease tends to conform more and more to the adult pattern. As in 10% of adults the clinical picture is atypical, a much higher incidence of obscure presentations must be expected in children.

In our 20 cases the presenting symptom was pain in 14. In five of these the character, location, and time relation to meals or relief by milk suggested duodenal ulcer. In nine the pain was not typical of duodenal ulcer. Five patients gave a history of haematemesis on admission and one was admitted with a perforated ulcer.

In children the course of duodenal ulcer can be divided into three phases: acute, atypical, and typical. At the onset ulcers commonly present as acute conditions. The abdominal pain may be severe, generalized, or localized to the umbilicus, right hypochondrium, or right iliac fossa. The children may scream and double up with pain. Vomiting is usually associated with it and occasionally the temperature is elevated. It is therefore not surprising that the literature abounds with reports of patients who had an appendicectomy prior to the establishment of the correct diagnosis. The severe colicky pain may also raise the suspicion of intestinal obstruction. Haematemesis and perforation may occur at this stage—as they may at any stage—and so help in diagnosis. We wrongly operated under the mistaken diagnosis of appendicitis three times.

Case 1.—A boy was first admitted to hospital when he was 14 with a year's history of colicky central abdominal pains which were very severe while they lasted. They occurred at infrequent intervals, at first lasting several hours. Later they occurred almost daily. There was definite tenderness in the right iliac fossa. Subacute appendicitis was diagnosed. At laparotomy through a Battle incision, the appendix was removed. A Meckel's diverticulum 2 in. (5 cm.) long was found and excised. Immediately after he left hospital the colicky abdominal pain recurred. It was now centred just above McBurney's point and was so severe that it made him roll about in bed. A diagnosis of subacute obstruction was entertained but could not be substantiated. Hospitalization again led to quick subsidence of the pain. When, however, the pain soon recurred at home he was admitted for laparotomy, when a large duodenal ulcer of the anterior wall was found. Medical treatment was instituted with temporary success. Frequent relapses necessitated vagotomy and gastro-enterostomy at the age of 18, since when he has been symptom-free.

During the second phase the history is still atypical but symptoms are less acute. The abdominal pain is often generalized, central in position, or in the right hypochondrium. It may be associated with vomiting, but is frequently not relieved by vomiting, food, or alkalis. Although there is no time relationship to meals the pain is often worse at night. The attacks may last from a few minutes to several hours and may occur at odd and sometimes infrequent intervals. Direct questioning may occasionally elicit a more typical history. The association of the pain to food may not have been noticed but may be present. Particularly important is the pain which wakes the child at night or gives rise to distress in the morning. Other symptoms include pallor, loss of appetite, and loss of weight. Local tenderness may be absent, particularly if the ulcer is on the posterior wall. The suggested diagnosis during this phase is commonly mesenteric adenitis, periodic disease, food allergy, cyclical vomiting, etc.

FIG. 1.—Age incidence of the series.

Case 2.—A boy aged 14 was sent to us with a provisional diagnosis of *tabes mesenterica*. He gave a 4-months history of central abdominal pains of constant nagging type which bore no relation to meals. They tended to come on at no particular time but were worse at night. Recently, vomiting occurred with the attacks and he had had some diarrhoea. Although his appetite was good he had lost weight. Apart from his unhealthy appearance physical examination was negative. The night pains suggested the possibility of a peptic ulcer. Barium-meal examination showed some pylorospasm. The duodenal cap was irritable, with a slight deformity of its lesser curve. There was a suspicious fleck of barium in some of the serial films. There was occult blood. His haemoglobin was 64%. A fractional test meal was normal. He was treated medically in hospital for five weeks with gratifying result. He has had no recurrence for the past two years.

During the third phase symptoms more closely resemble the adult pattern. It is the older child who is more commonly seen in this phase.

Case 3.—This boy was first seen in 1949 at the age of 7 with a severe attack of pain in the right hypochondrium and vomiting. There was diffuse tenderness in that area. A diagnosis of hepatitis was made but could not be proved. Short attacks of abdominal pain continued. In 1953 he had a severe attack of right-sided abdominal pain and tenderness when a normal appendix was removed. Intermittent attacks of pain lasting about 10 minutes continued. In May, 1955, when he was first admitted to Booth Hall Hospital in a severe attack, it was noticed that these pains were eased by milk, and the diagnosis of duodenal ulcer was established. Five months later the attacks became typical. There was a definite relation to meals, and symptoms were relieved by food and alkalis. It was only now that tenderness over the duodenum was noticed. When last seen, 18 months later, there was still duodenal tenderness and occasional pain.

Case 4.—This case shows well the progression from atypical to more typical symptomatology and the difficulty of diagnosis. In May, 1955, a girl aged 10 first attended hospital with a fortnight's history of severe recurrent pains in the right iliac fossa associated with vomiting. Her temperature had been raised at the beginning of the attacks. As there was tenderness in the right iliac fossa appendicitis was diagnosed and a normal appendix was removed. A month later the pain recurred. This time it was situated higher up in the right side of the abdomen, and was on occasion so severe that it made her roll on the floor. Full investigations including barium meal, intravenous pyelography, cholecystogram, etc., were negative. Her symptoms improved on propantheline. In September, 1956, the pain was first noticed to be present mainly in the mornings. She felt better when she had frequent meals. There was now tenderness in the epigastrium. Barium-meal examination showed an irregularity of the duodenal cap with a small ulcer.

Complications

Children are liable to the same complications as adults, apart from malignancy, which has not been reported. Haematemesis is more common than melaena. Melaena without haematemesis is rare, and occurred only twice in our series, although occult blood is frequently present in the faeces. In spite of the absence of overt bleeding the children can be very anaemic. Haematemesis can be massive.

A boy of 12 who had a six-months history of typical abdominal pain had no haematemesis. The benzidine test was negative. His haemoglobin on admission was 50%. Another small boy of 7 who had a two-weeks history of abdominal pain and a haematemesis three days before admission had a haemoglobin of 30%.

The ulcer perforated in two children in this series. One was a boy of 15 who had a year's history of attacks of colicky abdominal pain. Closure of the perforation resulted in cure. The other was a girl of 11 who was first admitted with haematemesis. She gave a year's history of abdominal pain unrelated to food. The barium meal examination showed a deformed and tender duodenal cap but no ulcer. She settled on medical treatment. She was readmitted nine months later with a perforation following recurrence of the pain for three weeks. Closure of the perforation again resulted in loss of symptoms.

Pyloric stenosis has been reported in about 25% of cases, more frequently in older children. There is, however, a

large spastic element which is reversible. One gets the impression from the literature that some of these cases have been operated on after too short a trial of medical treatment. Gross pyloric obstruction can usually be relieved medically, as it is caused more by massive oedema or spasm than fibrosis.

Case 5.—A boy of 12 had a 2½-year history of intermittent intense epigastric pains and vomiting two hours after meals. I first heard of him when our radiologist rang me up to tell me that he had done a barium-meal examination requested by the child's practitioner. He found a large duodenal ulcer and about 80% residue of barium in the stomach after six hours. After six weeks' treatment in hospital the residual barium in the stomach was negligible at 1½ hours, although tenderness and pain were still present.

Diagnosis

The realization that a duodenal ulcer is not a rare condition in children may be the first step in arriving at this diagnosis. In the acute and atypical phase there may be

FIG. 2.—Free acid curves of 10 children with duodenal ulcers. Shaded area represents the average normal limits of hydrochloric acid.

no indication in the history or from the physical signs that the underlying condition is an ulcer. In following up these patients with recurrent abdominal pains it is important to go into the symptomatology again at each interview, and sometimes ask leading questions about the time of day the pain mostly starts.

The only aid which established the diagnosis beyond doubt was a barium-meal examination. The fractional test meal is of little help, as it is usually normal in these cases. Ten of our cases had this investigation. Only one patient (Case 5) showed marked hyperacidity (Fig. 2). The benzidine test is often positive but is not diagnostic. Even at laparotomy an ulcer, even when specifically looked for, can easily be overlooked. There is very little fibrosis and the outer coat of the duodenum may appear normal. It is impracticable and probably unwise to carry out a barium-meal examination on every patient with obscure or recurrent abdominal pain. It is commonly of no help in the early phases of the disease and becomes positive only after several months, usually when the clinical picture contains some suggestive features. A premature barium-meal examination may therefore lead to failure to find the correct diagnosis.

The demonstration of the niche of a duodenal ulcer in children is difficult. Barium frequently passes the duodenal cap so rapidly that a niche cannot be demonstrated. The gross deformities of adults are rarely seen. Some bulbar deformity can usually be shown. This should, I believe, be accepted as confirmation of the diagnosis as it has been

borne out by the subsequent course of our patients. Often there is irritability of the cap and intermittent pylorospasm during the early part of the examination. This may be present in children, anyhow, and cannot be accepted as diagnostic evidence unless the clinical picture and course strongly suggest an ulcer. A repeat barium-meal examination a few months later may establish the diagnosis. The x-ray changes appear to be related to the chronicity of the lesion. In this series the average length of the history of cases showing a definite ulcer was 23 months; of those showing a deformed duodenal cap 7 months; and of those showing only irritability 4 months.

Two of our 20 patients did not have a barium-meal examination, one because the ulcer was found at laparotomy, and the other because of admission with a perforated ulcer. Four showed a definite ulcer; two of these, however, had a negative examination six months previously. Nine patients showed deformity of the duodenal cap. Five had pylorospasm and an irritable cap only. These had or developed a typical ulcer history later, as well as epigastric tenderness. One had haematemesis as well.

Treatment

Except for closure of perforations all our patients were controlled on medical treatment during childhood and adolescence. One required surgery at the age of 19 for frequently recurring attacks of pain. Two more may require surgery later. The indications for surgical treatment are, according to various writers, the same as for adults. Cameron (1953) found only 13 examples recorded in the American literature up to 1953. This small number is to be expected, as the bleeding vessel is not sclerosed and fibrosis of the ulcer bed is not callous and should respond to medical treatment. After the age of 17 stenosis or frequently recurring attacks of pain may occasionally demand surgery. At that age vagotomy and gastro-enterostomy may result in fewer growth and nutritional disturbances than partial gastrectomy. Gastro-enterostomy alone has been followed by an anastomotic ulcer in several instances. Medical treatment to be effective must be prolonged with four to six weeks' stay in hospital, not too frequently interrupted by such "terrifying investigations" as fractional test meals or barium meals in a dark room. Apart from routine treatment of rest, alkalis, sedatives, and propantheline, this should include, where necessary, psychiatric treatment.

Psychiatric Considerations

The pros and cons of an emotional background have been hotly debated for some time. Proctor and Alexander state that worry and stress play no part in the aetiology of duodenal ulcers in children. Aye (1953) and Chapman *et al.* (1956) found emotional trauma in all their patients. Our psychiatric unit is at present engaged in the psychological evaluation of our patients, so that I only want to summarize some of our experiences. These agree with Girdany's statement that these patients "are usually bright and tense and characterized by their parents as nervous. They often seem to be lacking in outward emotional ability and tend to keep things to themselves."

A typical example was a girl of 11 whose mother told me that the girl did not show grief like her sister when their father died. According to her mother her worries go inwards.

Our experience also agrees with the conclusions of Chapman *et al.* that the common factors in these patients are: "(1) their marked inability to be comfortable with expressions of any hostile or aggressive feelings, and (2) their resultant passivity and subnormal assertiveness with a strong need to attempt to obtain endless amounts of affection and approval by being servile and obliging to all persons around them." They are the children who hand you their temperature charts during the ward round.

It seems that when circumstances develop which bring strain in family relationships in children of this mental

make-up, a duodenal ulcer may develop. In our experience two of the most important precipitating causes appear to be the death of the father and worry over school examinations, particularly if an exaggerated importance is instilled into them of the necessity of passing the 11+ examination.

Six of our 20 patients had lost their father. These children, although not unduly disturbed outwardly, try to make up to their mothers for their loss by attempting to take the paternal role.

In 7 of the 14 patients in whom, owing to age, worry over the 11+ examination could be operative we found a significant relationship of the development of symptoms to intensive preparation for the examination, worry over the approaching examination, or over failure to pass it.

Case 6.—A boy started intensive coaching 18 months before the examination. Three months later symptoms developed. Six months prior to the examination he had to be admitted to hospital with severe symptoms. Significant improvement occurred only when the scholastic pressure at home was relaxed, but minor exacerbations continued with each examination he had to sit.

Case 7.—This boy, the son of ambitious parents, failed his 11+ examination. The full implications of failing again were constantly explained to him. He started extra coaching at home. A month later symptoms developed.

Case 8.—A girl was expected to pass her examination but as the date approached her work fell off. Her parents kept on reiterating that she must do well and "this examination settles your entire future." While waiting for the result her symptoms developed.

Case 9.—A girl failed her 11+ examination. Within a fortnight of starting at a technical school her symptoms began. She told us she was very worried over her failure to pass, as this meant she would not be able to go on to the university and would have a poorer job in consequence.

Case 10.—A boy was coached for the examination. Symptoms began before he sat for it but became worse after he started in his new school, where he had difficulty in keeping up with the other children. Since transfer to an easier school symptoms have subsided.

Prognosis

The prognosis for duodenal ulcer cases appears to be good. Children of the 7-9 age group responded well to treatment and symptoms did not on the whole recur. In children aged 10 to 15 years symptoms tended to recur more frequently, but could be controlled at least over the adolescent period.

Summary

A series of 20 children between the ages of 7 and 15 with duodenal ulcers is presented. The clinical picture can be subdivided into an early acute and later atypical and typical phases. Barium-meal examination often becomes diagnostic only when the clinical picture shows some features suggestive of peptic ulceration. Surgical treatment is rarely required except for perforations. The influence of home stresses in the aetiology is discussed, particularly loss of the father and pressures put on the child with regard to school examinations.

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REFERENCES

- Alexander, F. K. (1951). *Radiology*, **56**, 799.
- Aye, R. C. (1953). *Ibid.*, **61**, 32.
- Cameron, A. L. (1953). *A.M.A. Arch. Surg.*, **66**, 827.
- Chapman, A. H., Loeb, D. G., and Young, J. B. (1956). *J. Paediat.*, **48**, 248.
- Girdany, B. R. (1953). *Pediatrics*, **12**, 56.
- Guthrie, K. J. (1942). *Arch. Dis. Childh.*, **17**, 82.
- Karlström, F. (1949). *Helv. paediat. Acta*, **4**, 455.
- Proctor, O. S. (1925). *Surg. Gynec. Obstet.*, **41**, 63.