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Duodenal Ulcer in Childhood

A Study of Predisposing Factors

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Duodenal ulcer has been regarded in the past as a rare disease in children, and in the period 1930–1958 only 3 cases were diagnosed at the Royal Hospital for Sick Children in Glasgow, in children over the age of 1 year. The diagnosis of childhood duodenal ulcer is now being made more frequently (Fig.), and while there is a more general awareness of the problem, a real increase in incidence, as suggested by Fällström and Reinand (1961), may also be responsible. Goldberg (1957) has reported 20 cases from the Manchester area, and Milliken (1965) in Dublin has reviewed 35 cases seen at one hospital in the period 1953 to 1962. In neonates and infants the disease carries a high mortality, often presenting as severe alimentary haemorrhage of uncertain origin, and in these patients it is probable that the causative factors are fundamentally different from those operative at a later age. This group is not discussed here. Among children and adolescents, with whom this communication is concerned, the disorder often resembles in its clinical presentation that occurring in adults, and is subject to the same complications (Hutchison, 1964). Our aim in studying the present series of cases has been to try to ascertain whether the factors known to predispose to duodenal ulcer in adults are also evident in children. Psychosomatic factors are also likely to be of importance in the aetiology of duodenal ulcers in children, as emphasized by Chapman, Loeb, and Young (1956), and these are the subject of a separate investigation.

Material and Methods

Thirty-six patients were interviewed, of whom 27 were male and 9 were female. All had previously suffered from duodenal ulcer and full clinical data of the original illness were available. None of the patients exceeded the age of 16 years by the time of diagnosis, which had been established either by barium meal examination at which a duodenal ulcer crater had been demonstrated unequivocally, or by laparotomy at the time of perforation. A brief clinical history was obtained, information being sought especially about continuing or recurring dyspeptic symptoms. A family history of duodenal ulcer was recorded, and was regarded as positive if one or other parent or at least one sib was known to have the disease on the basis of evidence such as previous gastric surgery or confirmatory *x*-ray evidence which left no doubt of its existence. A sample of blood was taken for ABO blood group determination. An augmented histamine test meal (Kay, 1953) was carried out on 19 of the 36 patients, and was also performed on 6 children who were in hospital and who suffered from a variety of disorders other than peptic ulcer. In none of the patients with duodenal ulcer had the disorder followed the administration of steroids, aspirin, or other drugs.

The mean age of the patients at the time of investigation was 12·6 years (range, 7–23 years) and their mean age at the time of diagnosis was 10·3 years (range, 4–16 years).

Results

The results of acid secretory studies are presented in Tables I and II. The mean age of the duodenal ulcer group of children and adolescents was 12·5 years (range, 7–17 years) and their mean weight was 41·2 kg. The mean maximum acid output was 13·6 mEq/hr., and the over-all acid output expressed as mEq/hr./10 kg. wt. was 3·30. When the maximum acid outputs are considered in relation to the ages of the patients, a positive correlation can be demonstrated which is statistically significant ($r = 0{\cdot}51$, $0{\cdot}02 < p < 0{\cdot}05$). The mean age of the non-ulcer children was 9 years (range, 7–12 years) and their mean weight was 30·5 kg. Mean maximum acid output was 9·7 mEq/hr. and over-all acid output in mEq/hr./10 kg. wt. was 3·19.

The ABO blood group distribution of the 36 patients is given in Table III and is contrasted with the ABO blood group frequency in a control group of 5898 blood donors as supplied to us by the

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† Dr. Melrose died on January 26, 1967.

TABLE I

Details of 36 Children with Duodenal Ulcer

Case No.	Sex	Age at Diagnosis (yr.)	Present Age (yr.)	Presenting Symptoms	Continuing Activity	Blood Group	Family History	Weight (kg.)	Maximum Acid Output (mEq/hr.)
1	M	14	15	Pain	+	–	+	55·5	25·6
2	M	9	10	Pain	+	O	–	26·0	2·4
3	M	11	17	Pain	+	B	+	58·0	41·9
4	M	11	13	Haematemesis	+	O	+	48·0	13·5
5	M	13	14	Pain	+	O	+	51·0	5·9
6	M	11	16	Haematemesis	–	O	–	51·0	3·8
7	M	12	14	Vomiting	–	O	–	50·0	10·3
8	F	6	11	Pain	+	O	+	54·0	7·6
9	F	4	9	Haematemesis	+	O	–	28·1	4·7
10	M	13	13	Anaemia	–	O	–	43·1	32·6
11	M	7	7	Pain	+	A	–	24·5	5·9
12	M	10	10	Pain	+	O	–	27·7	18·0
13	M	10	10	Pain	+	O	–	27·3	3·1
14	M	11	13	Pain	+	O	–	36·5	12·0
15	M	8	9	Pain	+	B	–	35·0	13·5
16	M	10	10	Pain	+	O	+	27·0	11·4
17	M	12	17	Vomiting	+	–	–	58·6	10·8
18	M	15	17	Perforation	–	O	+	52·6	28·6
19	M	12	12	Pain	+	O	+	29·0	6·8
20	M	9	21	Pain	+	A	–		
21	F	12	17	Vomiting	+	O	–		
22	F	10	11	Pain	+	–	–		
23	M	12	13	Pain	+	–	–		
24	M	11	23	Haematemesis	–	O	–		
25	M	14	17	Perforation	+	O	–		
26	F	7	7	Pain	+	O	+		
27	M	9	10	Haematemesis	+	O	+		
28	M	8	12	Pain	–	AB	+		
29	M	9	9	Pain	+	A	+		
30	F	11	12	Pain	–	O	+		
31	M	11	13	Haematemesis	+	A	–		
32	F	4	7	Haematemesis	+	O	+		
33	F	10	15	Pain	+	A	–		
34	M	8	14	Haematemesis	+	B	–		
35	F	7	12	Haematemesis	–	O	+		
36	M	12	15	Haematemesis	–	O	+		

Glasgow and West of Scotland Blood Transfusion Service. Among the children with duodenal ulcer there is a significant excess of blood group O ($\chi^2 = 4{\cdot}2$, $p < 0{\cdot}05$).

In Table IV are listed the clinical features with which the patients presented at the time of initial diagnosis. One patient with persistent vomiting was found at laparotomy to have pyloric stenosis,

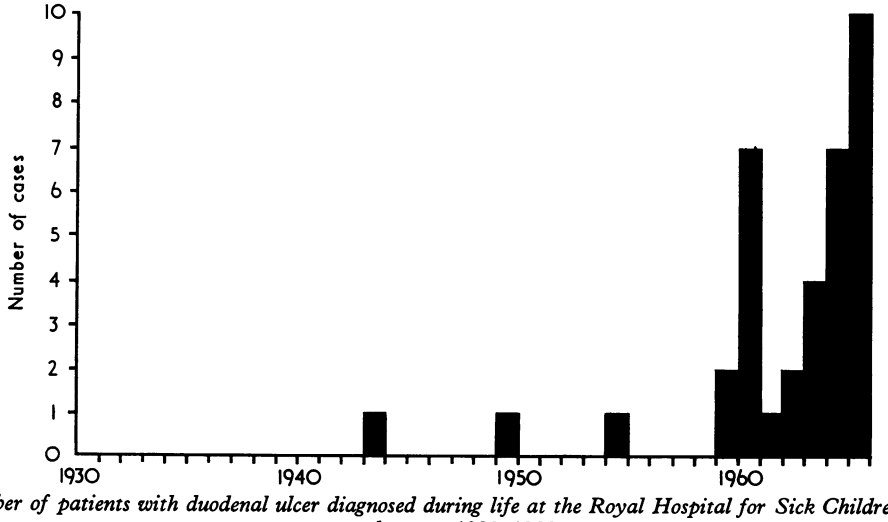

FIG.—*Number of patients with duodenal ulcer diagnosed during life at the Royal Hospital for Sick Children, Glasgow, between 1930–1966.*

TABLE II

Results of Gastric Secretory Studies in Non-ulcer Patients

Subject No.	Sex	Age (yr.)	Diagnosis	Weight (kg.)	Maximum Acid Output (mEq/hr.)
1	M	11	Undescended testis	30·0	13·5
2	M	5	? Appendicitis	23·0	7·5
3	M	7	Obesity	45·5	8·3
4	M	11	Undescended testis	31·0	4·5
5	M	12	Perthes disease	30·0	17·8
6	M	8	Aquired megacolon	23·5	6·8

and partial gastrectomy was carried out. The subsequent course of the illness in the 36 patients is also shown, and the presence or absence of continuing symptoms at different time intervals noted. Excluding the 9 patients in whom the diagnosis had been made during the past year, it was found on interrogation that 19 patients continued at intervals to have alimentary symptoms consistent with persistent duodenal ulcer and 8 patients were eupeptic.

There was a positive family history of duodenal ulcer in 17 patients, and a negative family history in 19.

Discussion

Acid hypersecretion was infrequently detected in the children suffering from duodenal ulcer studied by Goldberg (1957) and by McAleese and Sieber (1953), but their observations may be criticized on the grounds that maximum histamine stimulation was not employed. Ghai, Singh, Walia, and Gadekar (1965) performed augmented histamine test meals on duodenal ulcer cases and on a control group of normal children; there was no significant difference in basal acid output between the two

TABLE III

ABO Blood Group Distribution of 32 Duodenal Ulcer Patients and of Control Series*

	Blood Group			
	O	A	B	AB
Duodenal ulcer ..	23 (71·9%)	5	3	1
Controls	3177 (53·9%)	1906	637	178

* The blood groups of 4 of the patients were not known.

groups, but maximal acid output was found to be higher in the group of children suffering from duodenal ulcer than in the control group. These authors also found a correlation between high maximal acid output and activity of the ulcer at the time of the test. In the present investigation the acid output as expressed in mEq/hr. per 10 kg. wt. was insignificantly greater in the ulcer group as compared with the non-ulcer group (3·30 and 3·19, respectively). The non-ulcer group of patients cannot be regarded, however, as a control group; they were few in numbers, and had a lower mean age and a lower mean body weight. In addition, the mean body weight was distorted by obesity in one subject. At the time of the augmented histamine test only 4 of the 19 ulcer patients had had a prolonged spell of freedom from ulcer symptoms, so that any attempt to correlate maximal acid output with ulcer activity would be misleading. It is of interest that in the control group studied by Ghai *et al.* (1965) maximal acid output in the control group was 2·02 mEq/hr./10 kg. wt. which may be contrasted with the acid output of 3·30 mEq/hr./10 kg. wt. in our ulcer patients, but any comparison is invalidated by a difference of 15 kg. in mean body weight between the two groups. As we have shown, a positive correlation exists between age and acid output, and a similar correlation can

TABLE IV

Symptoms in 36 Children with Duodenal Ulcer

Clinical Presentation	No. of Cases	Continuing Activity					
		<1 yr.	>1 yr.		>3 yr.		>5 yr.	
			Present	Absent	Present	Absent	Present	Absent
Abdominal pain	20	8	10	2	4	2	4	0
Haematemesis	10	—	6	4	3	4	2	3
Vomiting	3*	—	2	1	2	0	2	0
Perforation	2	—	1	1	1	0	0	0
Hypochromic anaemia	1	1	0	0	0	0	0	0
Total	36	9	19	8	10	6	8	3

* One patient had pyloric stenosis; partial gastrectomy was performed.

also be demonstrated between body weight and acid output (Ghai *et al.*, 1965). It is evident, therefore, that to establish the existence of acid hypersecretion in childhood duodenal ulcer a control group of healthy children matched for age and weight are necessary, and we have not been able to obtain access to such a group. The evidence favouring acid hypersecretion as a factor in the causation of childhood duodenal ulcer is inconclusive, and further investigation is required.

In this series, as in adults, an excess of male over female patients was recorded and no associated diseases were present which might have predisposed to the occurrence of duodenal ulcer, nor was there a history in any case of the ingestion of ulcerogenic drugs.

Almost half of the cases had a family history of duodenal ulcer, an incidence exceeding that recorded by Muggia and Spiro (1959); the hereditary aspects of the disease have been emphasized by Doll and Kellock (1951).

The excess in blood group O observed in children in this series parallels that recorded in adults by Aird, Bentall, Mehigan, and Roberts (1954) and previously noted in this area by Brown, Melrose, and Wallace (1956).

The clinical presentation at the time of diagnosis differed in no important respect in children as compared with adults (Table IV). The symptoms were often periodic, and frequently the pain was of the typical duodenal ulcer type, being epigastric in site, coming on before meals and occasionally during the night, and being relieved by food or alkalis. The description of the symptoms depended on the ability of the child to communicate adequately and on accurate observation by the parents, and these factors may have been important in the patients in whom no set pattern of symptoms was noted. One child required partial gastrectomy on account of pyloric stenosis, and 6 years later at the age of 18 there was no detectable effect on subsequent growth and development. The weight of the patient was 56·7 kg., and tables of 'ideal' weight for a subject of similar age, sex, and height indicate a weight of 49·1 kg. The progress made by this patient after operation does not support the claims of Cameron (1954) and Muggia and Spiro (1959) that gastric surgery in childhood can retard later development.

The subsequent fate of the patients after duodenal ulcer had been diagnosed in childhood is of interest. Excluding those in whom the diagnosis had been made during the past year, 70% reported the presence of continued or recurring symptoms, though in the majority the period of follow-up was short. This incidence can be contrasted with the 50% recurrence rate observed on follow-up in a group of 92 patients studied by Michener, Kennedy, and DuShane (1960). Of the 11 patients who had been followed up for at least 5 years after diagnosis, 8 had continuing symptoms. These studies tend to confirm the clinical impression that duodenal ulcer in childhood carries a poor long-term prognosis in so far as permanent healing is concerned.

Summary

Follow-up studies were made on 36 children suffering from duodenal ulcer and particular reference was paid to gastric secretory capacity, blood group distribution, family history, and subsequent progress. Males were affected three times as often as females. Augmented histamine test meals were performed in 19 children, with results which suggested that acid hypersecretion might be a factor in causation, but the findings were inconclusive. Blood group O was present in excess. A positive family history was present in half of the patients, and a history of persistent recurrent ulcer symptoms was obtained in almost three-quarters. One patient was found to have sustained no adverse effects on growth and development following gastric surgery 6 years previously.

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