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Peptic ulcer in children

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I. INTRODUCTION

Peptic ulcer as a clinical entity appears to be as old as man himself. Ancient writings, dating back as far as Aesculapius and Celsus, indicate the cognizance of ulcer and even suggestions for therapy. Even the great Napoleon was known to have had an ulcer. However, the concept of peptic ulcer in children is a rather recent idea. The first reported case was published in 1821. Later, in 1918 a study was presented by Theile in which he reported 248 cases. He states in his paper that acute ulcers develop in the newborn and in nurslings while the chronic ulcers develop between the ages of 10 and 14 years. This would indicate that the first two years, the years from 10 through 14, and the third and fourth decades are ulcer years. This is true when speaking in generalities, but it has been shown since that ulcers, both acute and chronic, can occur at any age.

While the majority of cases of recognized peptic ulcer seen by the practicing physician will be in the age group over twenty, the primary purpose of this paper is to show that ulcers in children 16 years or under are not the rarity they were once thought to be. Benner, in the Journal of Pediatrics in 1943, said, "For many years it was the opinion of most physicians that gastric and

duodenal ulcers occurred so rarely in young children that they could be disregarded in diagnosis." It also, therefore, will be the aim of this paper to emphasize the necessity for consideration of peptic ulcer in cases of vague abdominal pain, irritability, anemia, digestive complaints, feeding problems and marasmus of obscure nature. This is doubly important when recognized that many children lose their lives through hemorrhage and peritonitis following perforation of an ulcer or ulcers that had remained unrecognized.

We will present a number of case histories and try to correlate age, symptoms and duration, sex, adequate management and etiologic features that may have a common foundation or similarity.

II. ANATOMY AND PHYSIOLOGY

Perhaps in discussing the various phases of peptic ulcers it would be well to review briefly the anatomy and physiology of the area concerned, because it is with a knowledge of these features that one can understand the mechanisms of the abnormal and normal functions.

The stomach, of course, is the dilated part of the gastrointestinal tract under the diaphragm with the cardiac sphincter above and the pyloric sphincter distally. This cavity is divided into cardia, corpus, and the antrum,

which is distal to the incisura angularis. The wall is made of serosa, muscular layer with three constituent layers, glandular layer and mucosa with epithelium of columnar cells. The duodenum has four parts. The first is that which includes the duodenal bulb and angles upward and to the right; the second or descending portion receives the bile and pancreatic ducts; the third passes to the left in front of the vena cava and joins the fourth passing upward to join the jejunum. The wall is comparable to the stomach wall but also contains Brunner's glands in the submucosa which secrete a thin alkaline mucus.

These organs are supplied with vagal parasympathetic and splanchnic sympathetics from T-6,7,8,9. It has been thought that the vagal impulses were motor and splanchnic inhibitory, but it is better to think of them as dual mechanisms whereby nervous impulses serve to modify the reactivity of the local neuromuscular mechanisms in the stomach itself (1). Sensations such as pain from tension or muscle spasm are transmitted through visceral afferents. These fibers pass without synapse to the spinal nerve root where ganglion cells are located. The sympathetic efferent nerves synapse in a ganglion and thus the use of ganglionic blocking agents have no effect on the sensory fibers.

The blood supply is from the celiac axis with numerous anastamoses arising from the three main arteries;

the hepatic, left gastric and splenic. Changes in vascularity have been noted with psychic influences and there has also been noted a shunting mechanism to a deep arteriovenous shunt system (2). Ulcers have been shown to be frequent in areas of poor vascularization and maybe a necessary pre-requisite to ulcer formation.

The destructive or eroding factors of the stomach, important in ulcer formation, are propagated in the proximal two-thirds where are located tubular pepsin and hydrochloric acid producing cells: pepsin by chief and HCl by parietal cells. The distal one-third secretes mucus as does the surface epithelium. The HCl producing mechanism is not clear, but three theories are prevalent. One is the "membrane hydrolysis" theory of Hollander (3); a second of Davenport (4) is that carbonic anhydrase catalyzes the reaction of carbon dioxide plus water forming carbonic acid which dissociates into free hydrogen ions and the carbonate radical and this theory is supported by decreased production after giving carbonic anhydrase inhibitors; the third uses the "redox cycle" (5). The pure secretion is 166 milliequivalents chloride, 159 of hydrogen and 7 of potassium. But it is diluted quickly, although clinical reports of 110 units have been noted. Pepsinogen from the chief cells is quickly converted to pepsin in acid and is produced by stimulation of the Vagus. Mucus from

epithelial and gland cells coats the stomach and seems to inhibit pepsin and neutralize HCl.

Gastric secretion may be divided into interdigestive or basal secretion phase and digestive secretion which has three subphases--cephalic, gastric, and intestinal. Cephalic refers to that induced by sight, smell, etc.; gastric to that as a result of stimuli within the stomach and is the largest in amount. Intestinal phase concerns secretagogues when in the intestine rather than in the stomach.

Gastrin secreted in the antrum produces gastric secretion but results are variable in relation to postoperative results concerning hypersecretion.

Inhibitory effects on secretion may be shown after sympathetic stimulation and ingestion of fats and sugars which produces enterogastrone, a hormone. The acidity of gastric contents in addition are varied by the emptying time of the stomach, ingestion of food and extrinsic secretion.

III. ETIOLOGY

An ulcer may be defined as a local manifestation of a group of causes resulting in loss of lining mucosa. There are three main factors: (a) chemical and mechanical making up the eroding element; (b) tissue resistance and

defense mechanisms; (c) what some people call the systemic and constitutional diathesis factor (6).

The chemical factor is the one found in all cases of ulcer. Excessive acid is usually present in most patients but not necessarily the causative agent. Some evidence shows that duodenal ulcer patients have increased acid secretion, while gastric ulcers have lower secretion (7). While free HCl must be present, pepsin also is a factor, not as important, however, since it must work in an acid medium.

Mechanical factors are brought into the picture when you realize the majority of ulcers occur at sites of most trauma. For instance, the lesser curvature and the duodenum where the contents are "squirted" from the stomach.

There are many theories concerning localization of ulceration. One postulates that there is a mucus deficiency either from drugs, e.g., cincophen, or multiple deficiencies. Other defense mechanisms are the living nature of the cells, alkaline blood in the capillaries and urease which forms ammonia and carbamate. Depression of mucosal resistance may also be caused by ischemia from poor vascularization of the areas or arterio-venous shunts which may be hormonally or neurogenically controlled.

Systemic and constitutional factors may be classified as hereditary, hormonal, neurogenic, and those concerning personality interplay. Heredity may be a predisposing factor rather than predisposing to a diagnosis of hereditary illness. Draper and Touraine say it is "selective, environmental action on a favorable constitutional terrain" (8).

Hormonal factors are not well understood. G.I. hormones, gastrin and enterogastrone seem to play no part in ulcer etiology. Also there does not seem to be an increased incidence of ulcer in patients with endocrine imbalance. However, Shay (9) suggests two methods of hormonally induced hypersecretion. The first is by stress mediated through the hypothalamus to vagus to stomach. The second concerns stress acting on the hypothalamus stimulating the posterior pituitary causing increased adrenalin output and thus giving increased ACTH and secondary increased corticosteroid production. The sex difference in ulcers cannot be explained but it is known that men are under more stress and that women in the active reproduction period have low ulcer incidence.

That the G.I. tract responds to stress is well known. Beaumont and Wolf and Wolff have made intensive studies in this line. It is known that hemorrhage and perforation occur after intracranial surgery. Also

erosions may occur secondary to autonomic discharge as in the Curling ulcer (10). Emotional trauma leading to perforation and hemorrhage was seen with bombing in World War II (11).

Certain personalities seem to be susceptible. A true ulcer personality is impossible to pin down because of variability and lack of controlled studies. He is often described as tense, anxious, and driving. He may be a promoter, manager, or executive, and may hold more than one job. They are ambitious, driving, and willing to shoulder responsibility. On the whole, sexual adjustment is good. Recurrences may be governed by uncontrollable circumstances.

The search for etiologic agents for pediatric ulcers is also a vague one. Many are the same in children as in adults, but we must disregard as a rule such factors as alcohol and tobacco, but must add a few others such as a prematurity, infection, and trauma, which have greater effect on the younger cases. They are also more common in children with hepatic and pancreatic diseases and erythroblastosis. In the neonatal period ulcers are many times the result of tremendous stress involving birth trauma or congenital abnormalities producing stress. Cases are reported in combination with congenital hydrocephalus, cystic disease of pancreas, Banti's Syndrome,

malnutrition, and cerebral lesions. These usually are the acute ulcer terminating in death.

Many stress the importance of familial evidence in children with ulcer and some say it should be one of the diagnostic features (12). It is said that more than one-fourth of all cases of ulcer show earlier generation involvement. Usually in those cases showing familial background the symptoms and severity are usually greater. Also in families with ulcer history there is an increased incidence of functional gastric disturbances found in the offsprings which may or may not lead to ulcer formation. It is said this familial incidence is five times that in the general population. It is apparently a transmitted lability of the stomach called "constitutional organic gastric deficiency" rather than an ulcerous tendency. J. Soler-Roig et al. in 1945 state that "the present conception is that of considering this constitutional and hereditary pathogenic mechanism is the most likely one in preference to localistic theories of increased secretion, gastritis or neurovegetative factors which consider only a single conditional factor, not demonstrable in all cases, and often related to the constitutional predisposition" (13).

An interesting feature of children's ulcers is the sex ratio. While in adults the sex ratio is 4:1 for

the males, in children the ratio is about even with only a slight male preponderance of 3:2. Some writers feel this is on an endocrinological basis. The male hormone may be secreted as early as three years of age, while the female hormone is lacking up to the age of ten years. After this they increase rapidly till puberty. At this time the ratio of ulcers becomes that seen in adults. Therefore, some postulate that as puberty approaches and the estrogen titer increases, these hormones exert an effect in protecting females from the occurrence of ulcers (14).

Dietary deficiencies may play a role. Some feel that ulcer is so frequent in malnourished infants that careful inquiry should be made concerning abdominal pain in these cases (15). One series reviewed showed that six of eight cases belonged to a group classified as under weight, members of families of low income and with evidence of deficient diets, notably protein (16). Some treatment has been instituted fairly successfully with high protein diet in adults. Also in this same series were two cases giving a family history. One definitely had an ulcer, while one had much indigestion but not under treatment.

A review of older literature shows a tendency to disregard the emotional factor in children. The later briefs show much more concern over the psychiatric angle.

It is now firmly believed by many that the younger ulcer patients have the same type personalities as do their older counterparts (17,18,19). They are described as tense, bright, and appear to lack "overt emotional lability." They are worriers and tend to keep their emotions suppressed. Most of them are described as "nervous" by their parents. This is a marked similarity to the adult patient who "keeps the lid on." A roentgenologist made a note that most of the children during fluoroscopy were usually tense and quiet and reserved, whereas the average child would be expected to be more noisy and uncooperative. A large group of the patients seem to have histories of broken or unhappy homes. Many show an incompatible school environment. Several cases have been described in which the symptoms first started following the birth of a sibling or an unpleasant episode in school or neighborhood. Several apparently cured cases had exacerbation of symptoms during febrile illnesses or following emotional disturbances or psychic trauma. Adequate evaluation cannot be made for the lack of satisfactory psychiatric control studies of comparable colleagues.

IV. PATHOGENESIS

The usual sequence of events in formation of peptic ulcers is not exactly known. It has been shown that

there are acute, subacute, and chronic ulcers. As far as is known now, most chronic ulcers are derived from acute ulcers unless the balance of eroding factors and defense factors is restored.

The most common factor seems to be the decreased resistance of mucosal cells to HCl. A second important factor is alteration of blood supply. Increased blood supply by histamine and caffeine and lowered vascularity causing necrosis are methods. This may manifest itself in two ways: hemorrhage and necrosis, which may occur in that order or simultaneously. Functional alterations such as hypersecretion, spasm, hypermotility, and increased blood supply are all present with ulcer. What is not known, though, is whether these are causes or results of the ulcer.

Peptic ulcer is defined as a benign, nonspecific ulcer which penetrates the muscularis mucosa and is bathed by acid gastric secretion. Acute ulcers are those of short duration, under thirty days, and which may be multiple, variable in size, and occurring anywhere in the stomach or duodenum. These are also commonly found in association with infections, burns, brain surgery, or trauma and stress of any nature. The majority of these heal with or without treatment and a few result in chronic ulcerations. These may perforate without warning and are probably the

most common cause of undetermined G.I. bleeding.

Chronic ulcers are the bulk of those seen clinically. When in the stomach, 85 per cent are on the lesser curvature, 60 per cent being in the distal 6 centimeters. Duodenal ulcers are about 95 per cent in the first 3 centimeters of the duodenum. They are usually solitary but may be multiple. Microscopic features of the crater from within out are (a) purulent exudate, (b) thin coat of fibrinoid necrosis, (c) granulation tissue, and (d) an outer dense layer of scar tissue. The blood vessels at the base may be obliterated as a result of ulceration and scarring.

In the healing phase, signs of inflammation about the ulcer disappear and the crater is reduced in size. The bases and sides are filled in with granulation tissue and puckering of the serosa is seen due to shrinkage. This scarring may lead to deformity of the area and in the stomach may produce enough stenosis to give symptoms of gastric retention or radiologic evidence such as the hour glass deformity.

Healing time ranges from 14 to 100 days with the average being 40 days (20). This is all dependent on rigidity of management, size of ulcer, duration of the lesion, amount of scarring, and accurate classification into chronic ulcer.

V. SYMPTOMATOLOGY

The most outstanding symptom of ulcer is pain. In the great majority of cases of uncomplicated ulcer this is fairly characteristic and indicates a diagnosis immediately. It is described as a gnawing, aching, or burning type of distress, located in the epigastrium between the xyphoid and umbilicus in a relatively small area, with radiation a rare occurrence, usually relieved by food or alkali, coming on a various length of time following a meal, and has a tendency to a spring and fall periodicity. Other symptoms are nausea and vomiting or constipation. Hematemesis and melena may occur in 3 to 50 per cent of cases as a first symptom (21).

Theories as to the cause of ulcer pain are many. Pain may arise from excess acid (22), motor disturbance such as spasm (23), increased intragastric pressure (24), compression of the ulcer or serosal irritation (25), acid producing increased motor activity (26), acid irritation of nerve endings (27), or engorgement of the mucosa, thus lowering the pain level to intragastric pressure or tension (28).

In children, however, there is, unfortunately, a much lower incidence of "typical" ulcer pain. These bizarre presenting symptoms and the idea that ulcer is a rarity oftentimes obscure the diagnosis. Some authors

describe all those in their series as having ulcer symptoms and pain identical with those seen in adults. On the other hand, one series was made up of cases all of which were first seen with a diagnosis of acute appendicitis and subjected to laparotomy which revealed normal appendices.

It is, perhaps, best to subdivide the children into three groups in which the symptoms are relatively similar. In the first we may put those of the infantile group. The ages here run from the newborn to one or two years of age. As a general statement it is said that ulcers in this age group will bleed seriously or perforate. One group of authors in reviewing the literature found little in the history that was significant until the hemorrhage or collapse occurred (29). Another states that not infrequently there may be symptoms such as refusal of feedings, evidence of abdominal pain, vomiting, with or without streaking of blood, that sometimes occurs for months before the onset of more dire symptoms (30).

Bradlow (14) goes deeper yet and subdivides into four more infantile sub-groups. In the first are those without symptoms, the diagnosis being made at autopsy. Here are seen the occasional concealed intestinal hemorrhage. These are the acute type ulcers, however. Group two includes those which suddenly develop hemorrhage,

hematemesis, perforate, and commonly die. The symptoms of the third group resemble pylorospasm. There is much vomiting and it may be projectile. In the fourth group he places those with a history similar to the following. The child is apparently normal till it encounters some type of stress like an upper respiratory infection. Thereafter it may show lack of weight gain, feeding difficulties, vomiting, attacks of abdominal distress, loose stools, evidence of dehydration with acidosis and streaking with blood of vomitus or stool. There is also described a puppy-like cry simulating the cry of colic. These may be diagnosed as various other G.I. ailments until perforation and commonly death occur.

The second major group is the childhood group containing the ages from 2 to 9 or 10 years. Here we find a rather latent period from ages 2 through 7, in which are very few reported ulcers. It is well to note, however, that most of these children finally diagnosed at ages 7 through 9 have had rather vague symptoms for months and even years.

It is in this group that we find a higher incidence of pyloric stenosis and perforation. Hemorrhage is now an uncommon presenting symptom. In this group the symptoms may be so typical that diagnosis is relatively easy or so vague and confusing that diagnosis is not made

till an operation is performed. The younger the child the more varied the symptoms. Symptoms such as loss of weight and appetite when accompanied by upper abdominal pain or tenderness or anemia should suggest ulcer. The pain may be recurrent or chronic and may occur typically at night or before breakfast in the older children. Abdominal tenderness is the most common physical finding, being epigastric or periumbilical (in the younger children usually). A note of caution should be remembered in talking to the children. They are usually very susceptible to suggestion and many of them show evidence of psychological abnormalities. Nausea and vomiting are uncommon (14), pyrosis, flatulence and relief with alkali are not frequently mentioned although some children will volunteer that milk or food gives relief of symptoms. Constipation, vomiting, night pain, and localized tenderness are noted much more frequently in some series (17, 19,29,31,32).

The third or late childhood group 9-16 years of age shows a fairly easily recognized group of symptoms. These tend to have the same characteristics as the adult ulcers, but investigators may easily be misled by the lack of adequate description or misunderstanding on the part of the patient. It is also in this group that a higher incidence of psychological imbalance and emotional

stress are found to play an important role, because in this period a maximal amount of adjustment and exposure to previously nonexistent stress situations occurs.

VI. DIAGNOSIS

The diagnosis of ulcer in the adult patient as stated before is, in the majority of cases, relatively simple after obtaining the typical history. Physical examination usually adds little to the diagnosis but is very helpful in ruling out other abdominal conditions. Laboratory aids to diagnosis other than the routine blood and urine work-up include gastric analysis, stool examination, and gastroscopy. Gastric analysis may furnish valuable information, but in many cases the findings are variable and provide at best only presumptive evidence of ulcer. Findings indicating retention are suggestive of old or chronic ulcer with scarring and deformity resulting from it. Stool examination may show occult blood periodically. Again only indicative evidence. Gastroscopy is of little value in duodenal ulcer and even in skillful hands has questionable value in gastric ulcers. Biopsy is valuable only in adults with a differential diagnosis including neoplasm of some sort.

The best diagnostic aid to detection of ulcers remains the x-ray examination of a barium swallow. Ulcers

above the incisura angularis usually show the fundamental ulcer crater which may be smooth or irregular in outline. Secondary signs include spasm, alteration of rugal pattern, and localized tenderness during fluoroscopy. Ulcers below the incisura are difficult to demonstrate and are more likely to be associated with alterations of contour. The most characteristic sign is persistent spasm of the antrum with a delayed emptying time.

X-ray study is the most dependable method of diagnosing duodenal ulcers also. These are usually in the first two or three centimeters of the duodenum and when on the lesser curvature wall are easiest to demonstrate. Anterior and posterior wall ulcers are more difficult to demonstrate. Aside from the niche, the most common sign is a pinching in of the sides of the duodenum. With pressure one can occasionally see folds radiating toward a crater. Other indirect manifestations include irritability, active gastric peristalsis, and reflex gastric spasm. If an ulcer niche has been demonstrated, x-ray is valuable in following the patient and plotting cure or healing. If none has been demonstrated you may still see signs that an ulcer has been present in the form of scarring and deformity of the bulb.

There are many variations in criteria used in diagnosing ulcer by x-ray, and also there are many nonspecific

changes seen in the duodenum which is sometimes called duodenitis. Whether this is a precursor of ulcer or an actual inflammation of the duodenum is not certain. Many times, of course, it takes a resume of the clinical history for the roentgenologist to make a presumptive x-ray diagnosis of ulcer. Sometimes a clinical history along with secondary signs is enough to make a diagnosis in some instances.

In making the diagnosis in children the most important factor is the realization that ulcer does occur in this age group, both acute and chronic. As mentioned before, the history, especially in the younger patients, is far from typical and an adequate physical examination is many times impossible because of the misleading signs that may be encountered. In children even more than adults, the physician must turn to the x-ray for dependable diagnosis. However, there are more pitfalls in reading children's films than in adults. Many times there will be overlooked ulcers because of the age of the patient. Borderline situations will occur in which the film may show pylorospasm and delayed emptying, or in which the duodenal bulb may reveal irritability and irregularity without the actual formation of a crater. In these cases it is urged that atropine be administered and, when such deformities persist, a presumptive diagnosis

only may be made. This may be checked by follow-up films after medical therapy has been initiated to see the response and to follow the cure (14,18,19).

It still remains that the only positive diagnosis of ulcer rests with the finding of a crater and associated signs. These, as in the adult, are pylorospasm, deformity of the bulb, folds radiating toward a crater, and a crater niche with a radioluscent area surrounding it. Tenderness over the bulb on fluoroscopy is usually a consistent sign. Irritability of the bulb cannot be used as being indicative in children because this is rather a common finding in normal children. Hyperperistalsis is not uncommon in children and often is hard to evaluate.

In the newborn period where there is bleeding from the G.I. tract with anemia, without large spleen or liver, and without erythroblastosis, peptic ulcer should be kept in mind. Hematemesis in a child with stenotic symptoms, indefinite abdominal discomfort, especially epigastric, even if occurring alone, may be caused by ulcer and an x-ray examination is indicated.

In considering a differential diagnosis one must ascertain that blood in the gut has not come from elsewhere such as nose or mouth or from the nipple of the mother. Melena neonatorum is caused 50 per cent of the time by peptic ulcer (14). The physician must eliminate

hemorrhagic disease of the newborn, syphilis, and septicemia. Blood dyscrasias are rare in the neonatal period. Hemophilia is rarely seen here. Other unusual conditions are congenital leukemia, congenital thrombocytopenia, and congenital hypofibrinogenemia. In infants ulcer may simulate pylorospasm, pyloric stenosis, and congenital duodenal stenosis. Gastroenteritis may be an initial diagnosis and terminally bleeding may be diagnosed as acute intestinal obstruction. In older children other causes of hemorrhage may be colitis, intussusception, polyps, Meckel's diverticulum, or parasites. Neoplasms are very rare. Frequent false diagnoses are appendicitis, mesenteric lymphadenitis, neuroses, and allergy states.

VII. MEDICAL MANAGEMENT

The aims of treatment are to relieve symptoms, heal the ulcer, and prevent recurrences. It has been stated that with optimal conditions the healing of an ulcer takes on the average of forty days. Optimal conditions are very rarely obtained and recurrences are difficult to control. The original etiological factors continue plus the presence of lowered resistance from the previous ulcer. Some authorities say that medical management is useful only in relieving symptomatology until a spontaneous remission can occur.

Medical treatment tries to equilibrate the eroding and defense factors by neutralizing the excess acidity and putting the insulted area to rest. Symptomatic relief can best be obtained by putting the patient at rest, preferably bed rest. This is most satisfactorily done in the hospital with sedation of some sort.

The diet must be both functional and palatable. It serves three main purposes: (a) avoidance of acid stimulators, (b) usage of antacid foods, and (c) decreasing the possibility of mechanical, chemical, or thermal irritative agents. Milk is satisfactory in satisfying all three requirements. It tends to lower gastric secretion through the enterogastrone mechanism, its casein acts as an antacid, and it is liquid and non-irritating. With a diet restrictive in many areas a good vitamin supplement should be used with emphasis on ascorbic acid.

Antacids reduce gastric acidity by a chemical reaction and are preferably insoluble. Complete neutralization of the gastric contents is impractical and oftentimes impossible even with massive doses (33). The antispasmodics are usually of the belladonna alkaloids, atropine being the standard, and depress the postganglionic parasympathetics.

The anti-cholinergic drugs act at the effector

site and at the ganglionic level. The most promising are those of the Banthine series. They appear to have an inhibitory effect on G.I. motility, gastric secretion, and mucosal engorgement (34).

Although there may be some question as to how much of a role psychological factors play in the etiology of ulcer, there is no doubt that psychotherapy is of value in the treatment. Good informal psychotherapy by the general practitioner or pediatrician established through good doctor-patient relationship is oftentimes the most important item both in cure and prevention of recurrences. Even with this type of therapy the patient may be obliged to forsake all prevailing environmental exposures for a while, or alter them appreciably, in order to have maximal protection against recurrences.

The patient or guardian should be educated as to the complications of inadequate management or recurrent attacks of ulcer distress. It is suggested that the patient seek medical aid when (a) symptoms recur and don't respond promptly to management, (b) unusual symptoms appear in regard to duration or severity, (c) there are frequent recurrences, (d) vomitus is bloody or coffee ground, (e) stools are bloody or tarry, (f) sudden unexplained weakness occurs (35).

Simple uncomplicated peptic ulcer is treated

medically in children even more than in adults. Without bleeding or perforation, due to the more superficial nature of most, the ulcers may very well be treated with diet alone with milk between meals and at bedtime. This may be done by the use of diets without spicy, fried, or fatty foods. Good chewing is essential and hygienic measures must be watched closely. More specific dietary measure with utilization of the Sippy or Meulengracht diets may be used.

In cases in which these measures are not effective or in which the duration and severity of the symptoms warrant, alkalis and spasmolytics may be used. In some cases Banthine has been used without dietary restriction with favorable results, usually in a dose of 25-50 mgm two to three times daily.

One case is reported in which the child had been vomiting after every meal and on whom a duodenal ulcer was demonstrated. Due to the extreme dehydration and inability to retain oral fluids the patient was given electrolyte solutions and 5 per cent glucose water I.V. for several days until the vomiting ceased. Hypodermic atropine and nembutal suppositories were used for the spasm and relief of pain and rest. Continuous drip of milk by Levine tube and Amphogel ounces one every four hours were used. After the vomiting stopped the tube

was removed and a Sippy diet was started with Amphogel and magnesium oxide. Recovery was uncomplicated.

Henrickson reports a case in more severe circumstances (17). The patient is put at strict bed rest and the stomach is gavaged with normal saline. Sustagen supplying the daily requirement of carbohydrate, fat, protein, and minerals is given through the naso-gastric tube. Twelve hundred calories per day are supplied this way by continuous drip. Vitamin K 5 mg/day, probanthine 15 mg every 6 hours, Kolantyl ounces 2, two or three times daily, ascorbic acid and a polyvitamin mixture are given. Change from tube feeding is made to a bland diet with probanthine and Kolantyl and then later maintenance is by diet alone.

As mentioned above, careful and thorough effort should be made to rectify the particular stress situation of this individual patient. If this cannot be adequately carried out by the family doctor, family or individual psychotherapy should be attempted by a specially trained clinician.

VIII. SURGICAL MANAGEMENT

The indications for surgical intervention in children are much the same as for adults. Prompt surgery is most often necessary in the younger age group below

six years, but many cannot be operated because of their very poor general condition. Treatment against bleeding in the newborn must be emphasized because of the inability of the child to tolerate blood loss.

These surgical indications for the most part are pyloric obstruction, perforation (especially in the very young), persistent hemorrhage, or intractable ulcer pain not responding to medical management. The mortality for children under one year old is very high. For those over six years it is about 5 per cent (14).

Perforation is a surgical emergency. Some writers indicate that equally satisfactory results are obtained by continuous gastric suction and supportive therapy such as is sufficient in some adult cases. In children, however, where the peritoneal defenses are poor, there should be no hesitancy in exploring. It is recommended that, in addition to the routine closure, the plugging of the orifice by the omentum be done to prevent later stenosis and obstruction.

Bleeding is a grave prognostic sign in children. Deaths from ulcers are usually preceded by 24-48 hours with hematemesis and/or melena. In some cases hematemesis may be controlled by conservative treatment. In a review of the literature, McAleese and Sieber point out that a high percentage of these cases will require operation at

a later date (36). They also note that severe G.I. hemorrhage not responding to transfusions and other supportive therapy within 24 hours should be operated. When during exploration the bleeding point is found, it may either be excised or controlled by suture ligature. There still remains a divergence of opinion as to whether further steps such as gastrectomy or gastroenterostomy should be done.

In the cases of intractable symptomatology and obstruction, the disagreement seems to be whether to do a gastroenterostomy or a gastric resection. Most pediatric surgeons in the past have recommended gastroenterostomy as the choice. More recent authors find no basis for this recommendation and see no reason why children should be any less susceptible to stomal ulcers than adults and therefore recommend as a desirable precaution the extensive resection of the stomach.

This again is open to debate because of the other types of postoperative syndromes such as are seen in adults. The frequency of these complications in adults ranges from 5 to 25 per cent (1,37). Some of these postgastrectomy syndromes are recurrent or persisting ulceration including gastrojejunocolic fistula, dumping syndrome, symptoms due to altered carbohydrate absorption, postgastrectomy anemia, and changes in weight.

Vagotomy is highly advocated in adults by Dragstedt (38) but condemned strongly in children by McAleese. His main objection is the diarrhea commonly seen as a side effect and which is so poorly tolerated even by the older children.

Copello (39) reports a case in a 15-year-old boy on whom a fundusectomy was done after finding a callous ulcer in the first portion of the duodenum. His reasons for doing so were: (a) the patient's age, (b) the nature of the ulcer, (c) the extent of the process which included the pancreas, (d) the high level of both free and total acid, and (e) the fact that there was no orificial stenosis. Thus he claims to remove the possibility of stomal ulcer and suppresses hyperacidity by eliminating the zone of secretion. If this fails another operation is necessary.

IX. CASE REPORTS

The following are some cases seen at The University of Nebraska Hospital, Omaha, Nebraska; Children's Memorial Hospital, Omaha, Nebraska; and The Spring Valley Hospital, Cherokee, Iowa. All but two of the cases have been seen within the last five years.

Case No. 1

A 9-year-old boy with three siblings, one older. No family or school difficulties apparent to the family physician but he is described as the "tense" type. He had a ruptured appendix about five years before the present illness and some "belly trouble" intermittently since then. It was thought to be due to adhesions. An upper G.I. series showed increased gastric peristalsis, spasm at the pyloro-duodenal segment, and delayed emptying time of the stomach. A crater was seen at the base of the duodenal cap with tenderness in this area. Treatment was diet and probanthine for six weeks with complete remission. Since he has used occasional medication when symptoms warrant.

Case No. 2

A 16-year-old boy with no siblings but was an adopted child. Had a very heavy schedule and was a star basketball player. Had farm chores to do early in the morning. For several months would develop epigastric pain before breakfast which was usually relieved by breakfast. Did not like farm work. X-ray showed an ulcer in the distal pre-pyloric region. Symptoms were relieved by probanthine and diet.

Case No. 3

An 8-year-old boy with no siblings who had a chronic belly-ache for 3 mo. Report includes the fact that his father was a heavy drinker which provoked many family quarrels. X-ray showed a tender distorted cap with a very irritable duodenum. A questionable crater was demonstrated in the bulb. Was treated with phenobarbitol, amphogel, and atropine with good results.

Case No. 4

A 7-year-old girl whose family history is not available. She had a stomach-ache at school about two hours after meals which was relieved by food. The symptoms had lasted about 2-3 months. X-ray showed increased gastric peristalsis, intense spasm of the pyloroduodenal segment with localized tenderness over the cap. Showed a "dumping" type behavior of the stomach with the

opaque meal in the descending colon in 3 hours. She showed excellent response to ulcer management and 10 weeks later showed some residual spasm although asymptomatic.

Case No. 5

A 9-year-old boy with two older siblings who was regarded as bright and seemed to have no home or school problems. One year ago had an episode of fairly low abdominal pain of about 1 month duration which was thought to be mesenteric adenitis. Had a 1-month recurrence before present admission. Upper G.I. study showed a small crater at the base of the cap, intense spasm of the duodenum and severe motor disturbance with the head of the column of barium reaching the descending colon in 1½ hours. Was treated with probanthine, alkalis, and frequent feedings, and was asymptomatic in 24 hours with no relapse.

Case No. 6

A 5-year-old boy with two younger siblings in what was described as a happy family. Had a 6-week history of stomach-ache relieved by food and antacids. X-ray showed a 2x3 mm. crater in the duodenum and marked duodenitis. Was treated with amphogel, phenobarbitol, atropine, and diet. The following year, in the first grade, this boy had no symptoms, although he was on no diet or medication. There was apparently a conflict with his kindergarten teacher.

Case No. 7

A 15-year-old boy with two siblings, one of whom was married to an alcoholic, over which a family quarrel was raging. He had had indigestion 3 months previously which had been relieved by antacids but never followed. On date of admission developed excruciating epigastric pain during the noon meal which soon radiated to the right shoulder. An upright film showed gas beneath the right leaf of the diaphragm and at operation was found to have a perforated ulcer of the lesser curvature of the stomach. Postoperative course was uneventful.

Case No. 8

A 9-year-old girl with no significant family history who had pain in the epigastrium in the morning about time to go to school. It was not present on week ends or during vacations and had about a 3-week duration. Upper G.I. study showed pyloro-duodenal spasm, motor disturbance of the small bowel and tenderness over the ulcer crater. She had a good response with diet and probanthine.

Case No. 9

A 4-year-old boy with no siblings. Both parents were working and the child was left to a succession of baby sitters. The kindergarten teacher noted a stomachache late in the forenoon which was relieved by a glass of milk. Symptoms of 10-week duration. X-ray: During the first 45 minutes of the examination none of the barium passed through the stomach due to spasm. Two hours later the stomach was partially empty and a film at this time showed a 4 mm. crater. The duodenal cap was never seen in a normal form during the whole examination. He showed a clinical recovery with diet and Pamine.

Case No. 10

A 9-year-old boy who is the fourth of six children. The father cannot hold a job and the mother runs a cafe. Apparently no home or school problems, but the mother is described as very dominant. On the first admission had had abdominal pain for two months and had to miss 2-3 days of school per week. Pain was relieved by rest. X-ray showed a very small ulcer at the center of the duodenal cap. Was put on a low residue diet with alludrox and donnatol. He became asymptomatic, but 8 months later returned with more epigastric pain and had missed one month of school. Repeat Upper G.I. examination showed a small ulcer at the apex of the cap. He was again put on a low residue diet and probanthine. Has been asymptomatic since except for the first day of school when the mother took him off his medication.

Case No. 11

A 9-year-old boy with no siblings who had headaches and occasional nausea and vomiting over the past year. No family history is available but the child seemed well oriented. X-ray showed a persistent duodenal deformity with an ulcer niche. There was no retention noted. Was dismissed with Alludrox and diet and progressed well.

Case No. 12

A 10-year-old boy who had been followed by the rheumatic fever program for 5-6 years and had been on large doses of aspirin occasionally and cortisone. During the past year had some epigastric pain in the morning which was relieved by milk. X-ray showed a 2-3 mm. ulcer crater and duodenal spasm. Was put on Amphogel and diet and has had no further complaints. Note: His father and mother are divorced and the father remarried.

Case No. 13

A 6-year-old boy with one older sibling who complained of abdominal pain for six weeks which seemed to have no relation to meals. Symptoms apparently started with the "flu." No significant family history. A small ulcer crater was seen in the mid part of the duodenal cap measuring 3 mm. Became asymptomatic on Calogel and Meulengracht diet. Had no recurrences.

Case No. 14

A 9-year-old boy who is the oldest of three children and who had almost constant epigastric burning and gnawing for about four weeks. There is a family history of ulcer in a 6-mo. old sister and on the mother's side of the family. He had a history of a vague nature of rheumatic fever at the age of 18 months. The father also has had some vague G.I. complaints but has not been followed. After the ulcer and spasticity of the duodenum were demonstrated he was put on frequent milk feedings and probanthine and showed marked clinical and subjective signs of improvement. There is no follow-up as yet.

Case No. 15

A 6-year-old girl with no significant family history on whom was made a diagnosis of ulcer elsewhere less than one year ago. Her present illness included slight anorexia with generalized abdominal pain present before meals and relieved by meals. No actual crater was seen by x-ray but there was irritability and spasm of the duodenal cap and pyloris. She was treated with diet alone and progressed well.

Case No. 16

A 12-year-old boy who was the oldest of five children all described as more "nervous" than ordinary. Father is known to have ulcers. He had mild stomachaches for six months but more severe stabbing pain in the last three months which seem to have no relation to meals but which are prevented by frequent glasses of milk. Patient also wets the bed every night. X-ray shows an active fairly large size duodenal ulcer situated mid-way through the cap. He was dismissed with a bland diet with milk between meals, Alludrox and probanthine. No follow-up as yet but clinically shows improvement.

Case No. 17

A 7-year-old boy who has a family history full of ulcers and other G.I. complaints. He is the oldest of three children in a home which is not broken but badly bent. When first seen complained of a stomachache for over three weeks described as periumbilical and not related to meals. No actual ulcer was seen with x-ray examination but he was placed on multiple feedings and Kolantyl gel. He had no more complaints. Several months later during a routine examination he was found to have some epigastric tenderness and reexamined with a barium meal. The films showed an active ulcer in the cap but he denied any symptoms. He was given a bland diet, Gelusil and Donnatol. There has been no further check.

Case No. 18

A 9-year-old boy who is the second of three children. First symptoms started three years previously when his parents were separating. They later remarried and separated again four months before admission. At the time of the present illness he had been having stomach cramps for 2-3 weeks. Upper G.I. showed duodenal deformity and an area of barium retention compatible with active duodenal ulcer. Became asymptomatic with probanthine, Amphogel, diet, and milk between meals.

Case No. 19

This is an 8-year-old boy who is the third of four children. He had a 4-week history of sharp intermittent right-sided pain. He also had vomiting and anorexia. He had been followed for two years on the rheumatic fever program. He had a history of vague abdominal discomfort of a mild nature for several weeks prior to the present illness. His vomiting progressed till the time of admission. X-ray showed an ulcer at the base of the pyloris and marked hyperperistalsis. He was put on Amphogel and diet and returned one month later asymptomatic.

Case No. 20

A 14-year-old boy who is the oldest of four living children. He comes from a very low class home life. The history showed that he had episodes of RUQ pain, epigastric distress and back pain since the age of 4. Had vomited coffee ground material about four months before admission. Each episode of distress seemed to be relieved by vomiting. X-ray examination on admission gave the impression of a perforated viscus. He was operated and found to have a perforated gastric ulcer near the pyloris toward the lesser curvature. It was closed, he progressed well, and was sent home on diet and Amphogel. He was asymptomatic till two years later when he began to have epigastric pain radiating to the right flank. Had one dark stool. X-ray indicated probable duodenal ulceration and he was treated with diet, Amphogel and Banthine. Has been asymptomatic on all follow-ups.

Case No. 21

A 12-year-old boy, the oldest of three. A history for over one year of grand mal type seizures with epigastric pain following. X-ray showed duodenal deformity and irritability but no niche. Epilepsy medication was adjusted and he was sent home. Repeat upper G.I. one month later showed no change. He was again seen 8 months later with no abdominal complaints but an ulcer niche was now seen on the films. Treated with diet and Amphogel. No further repeat films as yet.

Case No. 22

A 9-year-old boy with a family history of ulcers. Admitted with complaint of periumbilical pain ever since birth, much nausea and some vomiting, and occasional night vomiting. The radiologist reported no crater could be visualized. He was operated and much scarring was seen at the inferior portion of the duodenum which appeared like healing or healed ulcer. There was also much scarring in the gastro-hepatic and duodenal-hepatic structures which the surgeon said if seen in an adult would have been explained as an old perforated ulcer. There was also found mesenteric adenitis and chronic appendicitis. The postoperative course was uneventful and there was no follow-up.

Case No. 23

A 10-year-old boy who is described as being a B plus student in school and having many friends and enjoying sports. He had a history of about 4 years of epigastric and periumbilical distress frequently in the morning and relieved by breakfast. He questionably had polio at the age of 11 months at which time he was semi-comatose for 20-25 days. X-ray examination showed a 5 mm. crater in the center of the duodenal cap. Was put on a diet with Alludrox and Sebella. Six weeks later the cap showed some deformity but no actual crater. Still had symptoms. Five months later had had only two episodes of distress and films showed still a spastic cap. He was asymptomatic for five more months and x-ray showed only a hypertonic stomach. Another film five months later showed only minimal scarring.

Case No. 24

A 14-year-old female who has been hospitalized previously for "low blood count" and later for splenectomy for congenital hemolytic anemia. There is reported to be much strife with the parents, especially the mother. Her abdominal symptoms started while on a vacation three months ago with the parents and consisted of vomiting at bed-time and occasionally shortly after meals. She had lost 18 pounds in the last year. Barium swallow revealed extensive scarring with almost complete obstruction due to duodenal cap ulcer. A previous examination about 20 months earlier revealed a normal bulb. She was operated and a gastric resection done. Her postoperative course was uneventful. No follow-up reported.

Case No. 25

A 16-year-old girl who has one older sibling. She had a history of severe family difficulty and at one time went into an hysterical paralysis of both legs after an incident at home. She was a very good student and worried considerably about her grades. When first seen was living at a foster home which she did not like at all. She had some vague epigastric distress with slight nausea for about four months. X-ray showed an ulcer niche in the duodenal bulb with spasticity and tenderness. Was treated with a bland diet, Alludrox and milk between meals. Had repeated episodes of symptoms till moved from the foster home to her Grandmother's. Follow up film showed healing of the ulcer.

X. CONCLUSIONS

This series of 25 case reports represents only the proven cases by x-ray. There were many more cases seen and on whom upper G.I. studies were done and reported as either negative or reported as duodenitis. Even in this series was one child who had a normal film

with symptoms only to have a follow-up film show an ulcer crater when he was asymptomatic. Many of these cases were treated as if they had ulcer and became asymptomatic which proves only that the diagnosis of ulcer or other vague abdominal complaints in children is very difficult.

The ages of the children in the series range from the one boy who was four years to the two who were sixteen. The bulk of the cases were nine years old, there being eight of them. There were eight younger and nine older than the 9-year-old group.

The sex ratio which has been reported as about equal in children does not hold true in this instance. Actually, the ratio is the same as that reported for adults, 4:1 males over females. The ages of the girls are 6, 7, 9, 14, and 16 years. Even those cases which were investigated but showed no actual ulcer had a higher male ratio than the 3:2 mentioned earlier.

The duration of symptoms ranged from a little over 3 weeks to 10 years in one case. The majority range from 4 weeks to one year. They are usually intermittent over the longer time span and constant or daily occurrences with the shorter duration. Many times the symptoms have become severe or other diagnoses have been made before a final diagnosis or adequate testing is done. Many times further questioning reveals a much longer

history and lowers the age incidence of onset of the ulcer considerably.

Four of the children in the series showed a family history of ulcer but in many cases this was not specifically asked. Usually the information was volunteered. In those children with a history of family ulcers the symptoms seem to be, on the average, longer than the other children. Many of them are regarded as just plain indigestion or a "weak stomach, it runs in the family."

Concerning ulcer personalities it remains here the same as in other series. There is no accurate study of the personalities of the patients or children of comparable ages. Those reported by the investigators show them to be of the typical ulcer personality. The 16-year-old girl was very bright in school but constantly worried about her grades. One boy was reported as nervous and tense although he had siblings reputedly worse than he was. The other reportedly worried about nothing and made friends easily and fast but there is a suspicion of underlying family trouble.

Although if one digs deeply enough into any family or environmental situation one could possibly come up with different situations of stress or conflict; there are 16 of the above cases in which there is a marked conflict or

period of stress. Four of them show signs that perhaps an organic disease in the past may have been an etiological agent. Two had histories of rheumatic fever; one, epilepsy; and the other, a previous diagnosis of congenital hemolytic anemia. The other 12 showed conflict in the environmental situation. Usually the conflict arises at home with one or both of the parents. Sometimes, however, as in the case of the 4-year-old boy, the conflict is either with school or a particular teacher. Occasionally an undesirable situation not directly pertaining to the patient may be the upsetting agent.

The location of pain is interesting. In eight of the children it was diffuse and generalized over the whole abdomen. These patients on the average had a longer duration of symptoms. The pain in another eight was described as epigastric and relatively localized in comparison. Two had lower abdominal pain simulating appendicitis or early bowel obstruction if it were not for the time element. Four had periumbilical pain. This is reported fairly characteristic in the younger patients. One boy had right-sided pain, rather generalized and sharp, while another had no special abdominal pain, but was bothered with headaches along with slight nausea and some vomiting. Then, of course, there was the boy who had symptoms but no crater, then later no symptoms and a

demonstrable ulcer.

Vomiting as a prominent part of the story was present in four with no particular age predominating. Relief of symptoms by food or milk was common, while only a few could correlate neither time nor meals with the distress.

There were three surgical procedures done: one for perforation with simple closure, another as an exploratory and diagnostic procedure, and the third was a gastrectomy for obstruction of the duodenum.

Laboratory values were not available in nine of the cases, but in the rest of them, unless there was coexisting organic disease, they were not altered enough to make any comparison or draw any conclusions.

XI. SUMMARY

Peptic ulcer, although a relatively common adult ailment, has been until recent years thought to be either nonexistent or extremely rare in children. In recent years there have been appearing in the literature an increasing number of case reports and ideas for treatment.

The etiology of ulcer in adults is still obscure and in children no less so. They are being classified into eroding element, the defense mechanisms, and the still debated constitutional factor. Eroding elements

are both chemical and mechanical. The defense mechanism lies mostly in the living nature of the cells. The constitutional factor includes heredity, hormones, and neurogenic and personality factors.

The symptomatology of ulcer is usually in the adult a characteristic syndrome. However, in children the symptoms may be variable and oftentimes are misdiagnosed and frequently operated on needlessly. The older the child, the more characteristic the symptoms. The younger children will usually bleed profusely or bleed and perforate without warning and go into shock and die. The older children show symptomatology of stenosis and oftentimes perforation after a long period of vague abdominal complaints. Past ten years old the symptoms are usually those of adults.

The diagnosis for the most part lies in visualizing the ulcer crater by the use of barium swallow and plotting the healing phase similarly. In children a differential diagnosis must include hemorrhagic disease of the newborn, syphilis, septicemia, gastroenteritis, bowel obstruction, colitis, polyps, Meckel's diverticulum, parasites, neuroses, intussusception, appendicitis, mesenteric adenitis, and allergy states.

Medical management in the main is the same as adult medications with exception of the dosage. Surgical

indications are uncontrollable bleeding, perforation, and intractable symptomatology. Recently extensive gastric resection has become the operation of choice in extensive cases.

In a series of twenty-five case histories it was shown that children definitely do have ulcer and much more frequently than previously supposed. The symptoms are varied. The sex ratio in this particular series was 4 male to 1 female, far above the expected ratio. It was shown in this series that stress either organic or emotional does play an important part in the etiology. A study is under way to evaluate both parental and patient liability for the stress. Therapy should be directed toward both parties.

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