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Massive Gastric Dilatation of Uncertain Etiology

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MASSIVE gastric dilatation in the absence of organic obstruction is uncommon, particularly when it does not arise as a postoperative complication. It is typically described as an acute disorder, but the following is an unusual case with an apparently subacute course.

A 25-year-old male university graduate who had spent the preceding seven months doing voluntary social work among the local "hippie" community was admitted to hospital on September 10, 1967, following an acute episode of bilateral carpopedal spasm and tingling sensation in the extremities. These were considered to be manifestations of tetany. Before this, he had complained of nausea, vomiting unassociated with pain, epigastric fullness and "gurgling" in the abdomen for two weeks. These symptoms had appeared after three days of fever, sore throat and painful cervical glands. He had lost over 10 lbs. in weight during the two and one-half weeks before admission to hospital.

He had had "emotional problems" from the age of 5 to 15 years, sometimes manifested by vomiting after meals. He also gave a 10-year history of ankylosing spondylitis, for which he had been treated with two "courses" of x-ray therapy five years previously. Since then the disease had been relatively quiescent, but the patient had been taking from 15 to 30 grains of acetylsalicylic acid daily for the six months before his admission.

He was over 6 feet in height and weighed 148 lbs. He was fully conscious but weak and dehydrated and his hands were tremulous. His oral temperature was 99° F., the pulse 92 per minute, and the blood pressure 120/70 mm. Hg.; his respiratory rate was 20 per minute. Muscular spasm in the face was elicited by tapping over the facial nerve in the parotid region (Chvostek's sign) and characteristic carpal spasm followed the inflation of the sphygmomanometer cuff to a level higher than the systolic blood pressure (Trousseau's sign). The reflexes were brisk. A succussion splash was present in the abdomen. There was no obvious distension of the abdomen.

Laboratory investigations yielded the following results: hemoglobin 17.0 g. per 100 ml.; leukocytes 4000 per c.mm. with 60% neutrophils, 1% staff cells, 27% lymphocytes, 4% monocytes and 2% eosinophils; the erythrocyte sedimentation rate (Westergren) was 90 mm. in one hour. The blood urea nitrogen was 20 mg. per 100 ml.; serum sodium was 141 mEq., serum potassium 2.7 mEq., serum chloride 90 mEq. and serum bicarbonate 34 mEq. per litre. The serum calcium was 10 mg. per 100 ml., phosphate 3.4 mg. per 100 ml.; total bilirubin was 0.6 mg. per 100 ml.; thymol turbidity was 2 units and flocculation, 1 unit. The heterophil agglutination (Paul-Bunnell) test was negative. Urinalysis showed a specific gravity of 1.027, protein +1, white blood cells +1, red blood cells +3, granular casts +1, and an occasional epithelial cell. The electrocardiogram was within normal limits. Radiological examination of the spine revealed an upper dorsal scoliosis with maximal convexity at the level of the fourth and fifth thoracic vertebrae; obliteration of both sacroiliac joints was reported. A barium swallow showed that the contrast medium dropped to the level of the symphysis pubis, outlining an enormously distended stomach (Fig. 1).

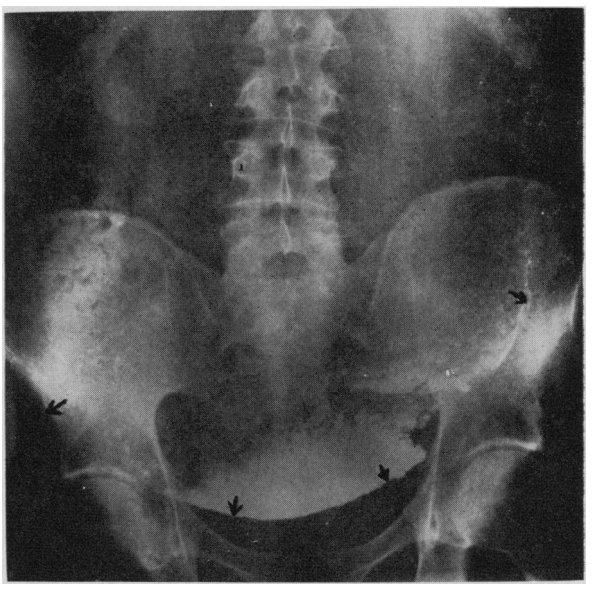

Fig. 1.—Barium meal study showing distended stomach.

The patient was treated with continuous gastric aspiration by means of a Levin tube; 7500 ml. was aspirated on the first day and the daily average was almost 4000 ml. The gastric return was dark brownish-green, bile-stained, acidic fluid with a pH of 3.5. Normal saline and dextrose-saline solutions, an average of 5000 ml. per day, were given intravenously. Daily supplements of between 40 and 240 mEq. of potassium chloride, 2 to 6 ml. of vitamin B complex and 1000 mg. of ascorbic acid were added to the infusions. On this regimen he was adequately maintained in fluid balance, and the serum electrolytes returned to normal values. After 48 hours, he improved considerably, and the gastric output began to diminish on the fifth day. By the

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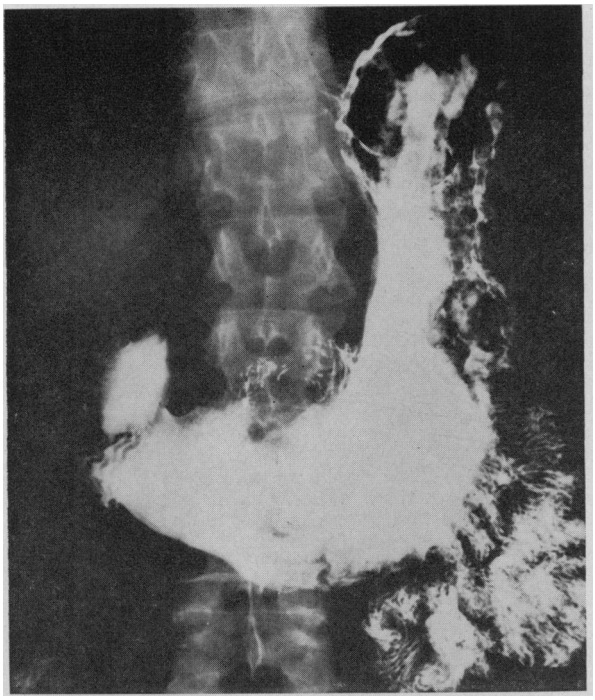

Fig. 2.—Repeat barium meal study six days later shows normal-sized stomach.

sixth day, hourly milk drinks were tolerated. A repeat barium meal study, taken six days after the first one, demonstrated a normal-sized stomach without abnormalities (Fig. 2). An ulcer-type diet was prescribed, followed by an advanced bland diet a week later. At this time, gastric aspiration using a wide-bore stomach tube, four hours after the last oral intake, produced a return of less than 20 ml. He was discharged from hospital on October 1, 1967.

DISCUSSION

Most cases of acute gastric dilatation have occurred as a postoperative complication,[15] and almost every common intra- and extra-abdominal operative procedure has been implicated.[12] Other predisposing causes which have been documented include debilitating chronic illnesses,[3] acute infections,[12] emotional stress,[8] physical injuries,[6] "the cast syndrome",[5] typhoid,[12] pneumonia,[12] diabetes mellitus,[9] progressive muscular atrophy,[13] spinal deformities such as lumbodorsal scoliosis and spondylolisthesis,[12] anorexia nervosa,[14] excessive food intake following starvation in prisoners of war[11] and childbirth.[12] Certain features associated with the present case may therefore be of etiological significance. These are: the deformity of the spine, the preceding febrile illness and the possible mental stress related to his occupational environment in the "hippie" community.

Pathogenesis

The pathogenesis of gastric dilatation remains obscure, but various hypotheses have been proposed. The theory of "vascular compression of the duodenum" was first advanced by Rokitansky in 1842.[1] This postulated that acute gastric dilatation was the result of mechanical compression of the duodenum by the overlying superior mesenteric artery at the region where the duodenum crosses the spine. Anterior displacement of the duodenum as produced by a pronounced lordosis of the lumbar spine is said to increase the likelihood of such a compression.[3, 10] In addition, the syndrome seems to be most commonly seen in thin, asthenic individuals following a period of bed confinement,[2, 10] features which were present in our patient.

However, doubt arose regarding the mechanism of duodenal compression as a primary phenomenon when it was suggested that the duodenum may be secondarily compressed by a distended stomach, either from direct pressure or indirectly by the displaced small intestine dragging on the arteriomesenteric tissue.[4] Moreover, autopsy findings and experimental studies in dogs failed to substantiate mechanical obstruction as an essential etiological factor in gastric dilatation.[6]

Brinton (1859) introduced the atonic theory,[3] which attributed the condition to reflex inhibition of the gastric motor nerves, the atonic stomach becoming progressively distended by the accumulation of static secretions and swallowed air. Gastric atony is recognized as a frequent postoperative occurrence, and although gastric dilatation is extremely rare it does occur following transection of the vagus nerve in humans and in experimental animals.[17]

Morris *et al.*[12] suggested that the superior esophageal sphincter may be relaxed by debilitation or by anesthesia, thereby allowing continuous suction of air into the stomach during respiratory movements, with resultant abnormal distension of the organ. Others considered that electrolyte disturbance could induce an ileus of the proximal part of the gastrointestinal tract.[16] A disorder of the electrolyte balance is, however, more likely to arise secondarily to the vomiting which characterizes the clinical picture of gastric dilatation.

In the absence of conclusive evidence in favour of any one of the hypotheses described, it is probable that the pathogenesis of gastric dilatation is a multifactorial one.

In the case recorded here, the role of acetylsalicylic acid in the pathogenesis of the illness also needs to be considered. Firstly, salicylate ingestion into an empty stomach could have

induced the initial vomiting. Secondly, epigastric discomfort associated with gastric mucosal irritation may have been followed by excessive aerophagia, which is a common manifestation of an instinctive attempt to gain relief from any abdominal distress. Of perhaps greater significance is the fact that salicylate may delay emptying of the stomach by inhibiting gastric motility. Progressive dilatation of the organ may have arisen from a combination of these factors.[16]

Clinical Features

These were classically described by Fagge[7] as vomiting in the presence of tremendous gaseous and fluid distension of the stomach, but they can be minimal in the early stages. The onset may be gradual but is characteristically abrupt. Vomiting occurs as a presenting feature in 90% of the cases;[12] the vomitus usually consists of copious, dark greenish-brown, non-feculent fluid, but can be only an overflow phenomenon. According to Barner and Sherman,[1] Duval attributed the painless bilious nature of the emesis to the associated incompetent or relaxed pylorus. The vomiting is rapidly followed by progressive hypovolemia and a metabolic alkalosis, which may be manifested by carpopedal spasm (tetany). Succussion splash is elicited and a tympanitic note is obtained on percussion over a large area of the abdomen.

Diagnosis

This condition needs to be differentiated from mechanical obstruction of the pylorus and of the small intestine, in both of which pain is usually a prominent symptom, and from postoperative ileus. The diagnosis is readily established by the radiological demonstration of a fluid level in a markedly distended stomach, the outline of which may be delineated by a swallow of barium.

Treatment

Early recognition of this condition is of the utmost importance, since the mortality is high if treatment is delayed or not instituted, as can happen in the occasional "silent" case with minimal symptomatology.[8] Astonishingly rapid relief usually follows evacuation of the stomach contents. Continuous suction, together with careful restoration and maintenance of water and electrolyte balance, constitutes the essence of the therapy.

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