Case Report

Paralytic Ileus in Myxedema

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REVERSIBLE hypotonia of the visceral musculature and myxedema are known to be associated.[1-4] The intestinal atony may be of any degree, and, along with hypochlorhydria and mucosal atrophy, probably accounts for the common gastrointestinal symptoms of thyroid deficiency. Although any of the hollow intraabdominal viscera may be affected, the most striking derangement in motility often occurs in the colon.[5-8] If the impairment in motility reaches an exaggerated degree, a severe ileus results which may dominate the clinical picture and lead to ill-advised surgical intervention. The intestinal hypomotility associated with hypothyroidism may be potentiated by the use of drugs with an atropine-like effect. It is the purpose of this paper to report the occurrence of a severe paralytic ileus in an athyroid patient who was withdrawn from thyroid hormone replacement while receiving phenothiazine and iminodibenzyl derivatives.

Report of a Case

A 41-year-old white woman was admitted to the Hospital of the University of Pennsylvania for the first time in January 1965 with a 48-hour history of increasing abdominal distension.

At age 15 she sustained a 40% burn requiring multiple grafting procedures. Eight years prior to admission she underwent total thyroidectomy and bilateral radical neck dissection for carcinoma of the thyroid. She was then maintained on 3 grains of dessicated thyroid per day. Three years prior to admission she had a right radical mastectomy for primary carcinoma of the breast.

During the previous six years she had had multiple admissions to psychiatric hospitals and had been treated with a variety of drugs and electric shock therapy. She was rehospitalized eight months prior to admission and maintained on large doses of dextroamphetamine sulfate and amobarbital (Dexamyl), prochlorperazine (Compazine), imipramine hydrochloride (Tofranil), and pentobarbital (Nembutal). Her thyroid dose had been gradually reduced to ½ grain daily during the two months prior to admission because of her extreme agitation.

During the week prior to admission she noted increasing constipation and the gradual development of progressive abdominal distension. On the day of admission, she developed occasional nausea and vomiting of undigested food. She denied abdominal pain.

The patient was short, obese, and disheveled, and appeared pale and chronically ill. There was periorbital edema and puffiness of the hands and feet. Her general appearance suggested profound myxedema. Rectal temperature was 98 F; radial pulse, 85 per minute and regular; blood pressure, 120/80 mm Hg. The skin and hair were coarse and dry. There was a marked delay in the relaxation phase of her deep tendon reflexes. Chvostek and Trousseau tests were negative. There were skin graft scars over the upper arms and shoulders and well-healed bilateral radical neck dissection scars without evidence of recurrence of carcinoma. Examination of the chest and heart was unremarkable, except for the well-healed right radical mastectomy wound. The abdomen was nontender, grossly distended, and tympanitic with hypoactive and high pitched bowel sounds. No organs or masses were felt. Pelvic and rectal examinations were unremarkable. Sigmoidoscopic examination to 25 cm was normal. No other abnormalities were noted.

The hemoglobin was 12.0 gm/100 ml; white blood cell count (WBC), 7,100 (72% polymorphonuclear, 14% band forms, 1% monocytes, 13% lymphocytes). The urine was unremarkable. Blood urea nitrogen fell from 26 mg/100 ml to normal after hydration. The following laboratory values were normal: serum electrolytes, fasting blood sugar, serum amylase, lipase, SGOT, SGPT, and alkaline phosphatase. Serum proteins were 4.8 gm/100 ml, with an albumin of 3.1 gm/100 ml and globulin 1.7

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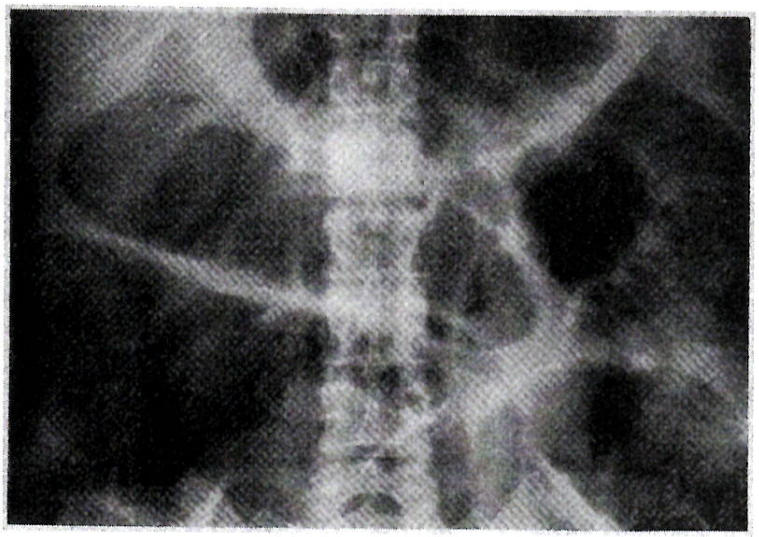

Fig 1.—Supine film of abdomen on admission showing massive dilatation of colon with 13 cm cecum and distension of stomach and a few small bowel loops.

gm/100 ml. Serum calcium was 6.2 mg/100 ml, and serum phosphate was 1.7 mg/100 ml. The protein-bound iodine drawn on admission was 1.4μg/100 ml. A prothrombin time was 26% rising to 90% after intramuscular phytonadione (Aqua Mephyton). Findings of the chest roentgenogram and the electrocardiogram were normal. X-ray films of the abdomen obtained on admission showed a massive dilatation of the colon with air fluid levels, more prominent on the right side. The cecum measured 13 cm in diameter. With the patient placed in the right lateral decubitus position, the air in the right colon shifted to the left colon. There was also distension of the stomach and of a few small bowel loops (Fig 1 and 2). A bone survey showed no evidence of metastatic disease; the sella turcica was normal.

During the first five days following admission she was treated with parenteral thyroid hormone (Levoid) (100μg every eight hours), gastric intubation and suction, enemas, colloid, and fluid and electrolyte replacement. She slowly improved and never developed signs of peritoneal irritation although her differential WBC continued to show a marked shift to the left until the tenth hospital day. By the sixth hospital day she was able to take fluids orally and was no longer distended. She was then maintained on oral thyroid hormone (sodium liothyronine [Cytomel], 25-25-12.5μg every 8 hours) with a gradual disappearance of her myxedematous features and return of her deep tendon reflexes to normal. On the seventh hospital day she developed a transient right pleural effusion for which no cause could be determined.

Fig 2.—Erect film of abdomen on admission showing colonic dilatation with air fluid levels.

After 12 days of thyroid hormone therapy, her gastrointestinal tract was functioning adequately. The following studies were performed: the 5-hour urinary excretion of d-xylose (25 gm orally) was 3.48 gm (normal, 5 gm or more), and the serum carotene was 34μg/100 ml (normal 60μg to 100μg/100 ml). After 45 days of thyroid therapy, the 5-hour urinary excretion of d-xylose was 5.3 gm and the serum carotene was 46μg/100 ml. An oral cholecystogram showed a poorly visualized gallbladder containing multiple calculi. A barium enema showed a dilated and redundant colon with decreased prominence of the haustral pattern (Fig 3). Radiographic examination of the upper gastrointestinal tract and small bowel on the 14th hospital day showed normal esophageal motility, and a slightly dilated small bowel, with a normal total transit time. There appeared to be a persistent area of marked narrowing in the midileum with both proximal and distal dilatation. A Miller-Abbott tube was passed to a point just proximal to the narrowed area and a small bowel enema was performed by the injection of barium into the tube. This study confirmed the impression of a narrowed segment of ileum, for which operation seemed indicated.

Preoperatively parenteral calcium was administered. Her central venous pressure was zero, rising

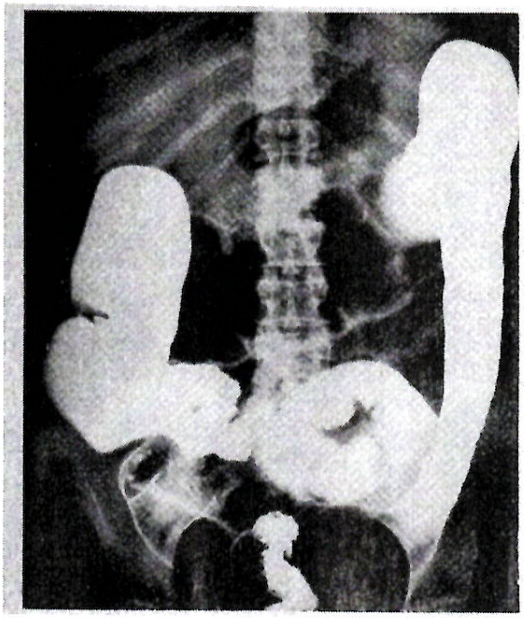

Fig 3.—Barium enema obtained after 11 days of thyroid therapy showing dilated and redundant colon with decreased prominence of haustral pattern.

to 10 cm of water after the administration of an additional 2,200 ml of whole blood and plasma. A laparotomy was performed on the 27th hospital day. At the time of operation no ascites was noted, and there was no enlargement of lacteals or mesenteric lymph nodes. The colon was moderately dilated and the small bowel entirely normal. No explanation for the x-ray appearance of a narrowed area of ileum could be found. The gallbladder contained multiple calculi and showed evidence of mild chronic inflammation. A cholecystectomy and appendectomy were performed. A mesenteric lymph node was normal microscopically, and there was no mucoid infiltration in the appendix.

The postoperative course was complicated by atelectasis and bronchopneumonia. Otherwise she made an uneventful recovery. Because of the patient's desire to return to the psychiatric hospital, tests to determine the existence of a latent form of hypoparathyroidism, possibly contributing to her mental symptoms, were not performed. At the time of discharge her serum proteins and serum calcium and phosphorus had returned to normal.

Comment

Deficiency of thyroid hormone leads to a hypotonia of the esophagus, stomach, gallbladder, small intestine, colon, uterus, and bladder.[1-8] The colon seems to be particularly affected, and in some fatal cases there has been a mucoid infiltration and edema of the colonic wall which may have contributed to the functional disturbance.[5,9] In other instances of colonic distension and elongation as a result of hypothyroidism no infiltrations were described.[10] Loss or decreased prominence of haustrations are a result of the hypotonia with inhibition of the annular contractions which normally serve as the chief mixing movements of the colon. Amelioration of abdominal symptoms and improvement in contractility generally follow adequate thyroid hormone replacement.[1-4,6,8]

When the functional disturbance is pronounced, a severe ileus may result and become the major manifestation of the underlying endocrine disturbance. If unrecognized, a debilitated and hypovolemic patient [11] may be subjected to laparotomy for intestinal obstruction.[5,7]

Impaired intestinal absorption has been associated with the hypothyroid state.[12,13] Certain features of this case suggest a malabsorptive state although fecal fat loss was not determined. The patient had hypoproteinemia, hypocalcemia, a phytonadione responsive hypoprothrombinemia, and a low serum carotene, usually elevated in myxedema. The low urinary excretion of d-xylose is probably related to a decreased rate of renal clearance of the pentose, rather than malabsorption.[14] All of these disturbances remained corrected following supportive therapy and thyroid replacement, except the serum carotene which did not reach normal levels.

The present case would appear to represent another example of deficient motor action of the gastrointestinal tract as a result of the hypothyroid state leading to a severe paralytic ileus. The concomitant use of drugs with an atropine-like effect (imipramine hydrochloride and prochlorperazine) while the patient was being withdrawn from thyroid hormone replacement was probably contributory.[15,16] No gastrointestinal difficulties were encountered after the prolonged use of these drugs until the elimination of thyroid hormone replacement led to the development of myxedema. When this endocrinopathy is suspected or a myxedematous patient is being withdrawn from thyroid replacement, drugs that may potentiate the

intestinal hypomotility associated with hypothyroidism probably should not be used.

Summary

A case of hypothyroidism presenting as severe paralytic ileus is presented. The gastrointestinal symptoms were reversed by adequate thyroid hormone replacement and the elimination of drugs that may have been contributory. Phenothiazines, iminodibenzyl derivatives, and other drugs with an atropine-like effect may potentiate the intestinal hypomotility associated with hypothyroidism. They should not be used when the endocrinopathy is suspected or a myxedematous patient is being withdrawn from thyroid hormone replacement. The danger of surgical intervention when the underlying endocrinopathy is unrecognized is stressed.

Generic and Trade Names of Drugs

Prochlorperazine, prochlorperazine edisylate, prochlorperazine maleate—*Compazine, Compazine Edisylate, Compazine Maleate*

Imipramine hydrochloride—*Tofranil*

Pentobarbital—*Nembutal*

Phytonadione—*Aqua Mephyton, Konakion, Mephyton, Mono-Kay*

Sodium liothyronine—*Cytomel*

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