Appendicitis with Typical Symptoms
But Ectopic Appendix Due to Malrotation
Of Colon

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WHEN ACUTE APPENDICITIS occurs in the presence of malrotated colon, the examining physician and the operating surgeon may be confronted with unusual findings, depending on the location of the cecum and appendix. In the case reported herewith, the clinical findings suggested right lower quadrant disease and yet the cecum and acutely inflamed appendix were in the left upper quadrant.

REPORT OF A CASE

A 15-year-old white boy was in good health until November 27, 1961, when abdominal pain developed. He was admitted to Mount Zion Hospital on November 29 with complaint of generalized abdominal pain, fever, diarrhea and vomiting of two days' duration. On physical examination, generalized abdominal tenderness was noted, most pronounced in the right lower quadrant, with definite rebound tenderness in this area. Hemoglobin content was 14.6 gm. per 100 cc. of blood. Leukocytes numbered 15,200 per cu. mm.—89 per cent polymorphonuclear cells, 5 per cent lymphocytes and 6 per cent monocytes. The specific gravity of the urine was 1.027. There were trace reactions for protein and acetone. Three to five leukocytes and 1 to 2 erythrocytes per high-power field were noted. On November 30, leukocytes numbered 16,500 per cu. mm. with 88 per cent polymorphonuclear forms, 4 per cent monocytes and 8 per cent lymphocytes. The rebound tenderness had become localized in the right lower quadrant and a diagnosis of appendicitis was made. At operation a McBurney incision was made and free fluid and fibrin were observed to be present in the right lower quadrant. The small bowel was distended. Neither the appendix nor any of the large bowel was visible or palpable through the incision. The McBurney incision was then closed and a right rectus incision was made. The distended small bowel was followed until the cecum and suppurative appendix were located in the left upper quadrant of the abdomen. As the liver and gall-bladder were in the usual location, the anomaly was that of failure of rotation of the bowel. No organisms grew in 72 hours on a culture of peritoneal fluid taken at the time of operation. Appendectomy was carried out and the wound was closed in the usual manner. There were no complications and the patient was discharged on the ninth post-operative day.

DISCUSSION

During the sixth to tenth week of embryonic life the alimentary tube grows at a faster rate than does the coelomic cavity; therefore, a portion of the mid-gut normally protrudes into the umbilical cord. At about the tenth week the peritoneal cavity grows at an accelerated rate and the mid-gut is withdrawn into it. As it recedes into the abdomen it rotates in a counter-clockwise direction, the lower portion of the mid-gut lying wholly within the left side of the abdomen. By the eleventh week the cecum and the first portion of the colon are in the epigastrium. As the rotation subsequently progresses the cecum passes into the right upper quadrant and finally ends its migration in the right lower quadrant. After this rotation is completed the ascending mesocolon and the descending mesocolon both fuse to the back wall of the abdomen, which anchors the base of the small bowel mesentery posteriorly from the ligament of Treitz obliquely downward toward the cecal area. In malrotation, arrest of development can be characterized by an incompletely rotated cecum, and there may be a lack of attachment of any of the mesenteries to the posterior abdominal wall.1

A subhepatic cecum is an abnormality of the third stage of rotation of the mid-gut due to failure of the cecum to elongate and descend into the right iliac fossa, with the result that it remains in a subhepatic position.1 If, with the cecum in this position, there is also failure of fixation of the small bowel mesentery, with a band attachment to the posterior abdominal wall, volvulus of all the small intestine may occur. The subhepatic cecum may be of surgical significance (because of the appendix's occupying a high position).2 If appendicitis occurs in a person with this abnormality, diagnosis may be difficult. In the case here reported the findings were

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localized to the right lower quadrant of the abdomen, yet the appendix and cecum were found in the left upper quadrant. Its presence there was owing to congenital malrotation of the bowel.

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REFERENCES


An Intestinal Complication of Anticoagulant Therapy

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HEMORRHAGE AS A COMPLICATION OF HYPOPROMO-thrombinemia due to anticoagulant therapy has been frequently reported. There have been few references to gastrointestinal complications other than hematemesis and melena. Reports of eight cases were found in the literature (Table 1). In each instance while the patient was receiving anticoagulant drugs an acute abdominal syndrome occurred, due to hemorrhage into the bowel wall. Under similar therapeutic circumstances, Beamish and McCreath reported upon seven patients with intestinal obstruction that was thought to be due to intramural hematoma formation. Non-operative management led to recovery in this group of cases. Intramural bowel hematomata as a cause of intestinal obstruction was cited by Spencer and co-workers. Reports of 34 such cases were collected from the literature, and in none of them had anticoagulant drugs been given. Many hematomata were of traumatic origin while a few were spontaneous or due to a hemorrhagic diathesis.

The case here reported is an example of intestinal obstruction associated with hemorrhage into the bowel wall due to anticoagulant therapy.

REPORT OF A CASE

A 54-year-old Caucasian man was admitted to the Mount Sinai Hospital on November 23, 1961, following 24 hours of generalized abdominal pain, nausea and anorexia. About eight hours before admission, profuse, foul hematemesis began. The abdominal pain became progressively worse and was most severe in the epigastrium. Weakness and dizziness developed approximately two hours before admission. There was no oral intake and no defecation during the entire episode.

Several surgical procedures had been carried out in 1959, because of right lower extremity pain due to advanced obliterative arteriosclerosis. These included right lumbar sympathectomy, iliofemoral endarterectomy, and insertion of an iliofemoral bypass graft. The graft did not function and was removed on August 8, 1961. For approximately three months, the patient used 5 mg. of an anticoagulant, Coumadin®, twice daily. The most recent prothrombin determination, one month before the present episode, was 15.7 seconds, 58 per cent (the control, 13.5 seconds).

On admission the patient was in great distress. His skin was pale and warm. The blood pressure was 130 mm. of mercury systolic and 90 diastolic. The pulse was 120 and respirations 16 per minute. The abdomen was flat and firm. Pronounced tenderness on pressure and rebound was noted in all quadrants. No peristaltic sounds were audible. The rectal ampulla contained brown feces. The dorsalis pedis and posterior tibial artery pulsations were strong on the left and absent on the right.

Leukocytes numbered 30,100 per cu. mm. with 87 per cent neutrophiles. The blood urea nitrogen was 37.2 mg. per 100 cc. and serum amylase was 43 units. The hematocrit was 36 per cent. Hemoglobin content was 12.3 gm. per 100 cc. Prothrombin time was 97.9 seconds or less than 10 per cent (the control was 13.5 seconds). Microscopically the urine contained many red blood cells, a few white blood cells and numerous hyaline and granular casts.

Roentgenographic films of the abdomen in both the supine and standing positions revealed no free peritoneal air. The colon contained gas and was not dilated. The stomach contained a considerable amount of gas and fluid. One strikingly dilated loop of small bowel, thought to be proximal jejunum, was seen in the mid-abdomen. It appeared to contain fluid. (See Figure 1.)

A diagnostic abdominal tap produced several drops of thick, dark red, grossly bloody fluid.

During the passage of a nasogastric tube, the patient vomited approximately 2,000 cc. of foul, dark reddish-brown and black material. Similar gastric contents were aspirated through the tube with gentle suction.

The patient was given 100 mg. of vitamin K1