Hemorrhage from Prolapse of the Ileocecal Valve:

Report of a Case*

KRISHNA ALANKAR, M.D.,† CARYE-BELLE HENLE, M.D.,‡ ROBERT R. RICKERT, M.D.,§ ERIC J. LAZARO, M.D.,¶

From the Departments of Surgery, Radiology and Pathology, College of Medicine and Dentistry of New Jersey, New Jersey Medical School, and the Harrison S. Martland Hospital, Newark, New Jersey

In 1959, Shepard and Godwin? called attention to the possibility of massive hemorrhage from prolapse of the ileocecal valve. They reported three cases of this condition which were proven by surgical exploration and histopathologic examination. In this communication we report an additional case.

Report of a Case

A 56-year-old Caucasian man was admitted for the first time on June 29, 1969, with the chief complaints of progressive weakness, lightheadedness, increasing episodes of syncope, and rectal bleeding. He admitted to heavy alcoholic intake.

About nine months prior to admission, the patient had been admitted to another institution for the same complaints. Upper and lower gastrointestinal x-ray series had revealed only a spastic duodenum. Hematologic studies had showed iron-deficiency anemia. The patient had been discharged with a diagnosis of iron-deficiency anemia and duodenitis, with no specific therapy recommended. Since his discharge from that institution, his symptoms had recurred.

He denied any history of hematemesis or any change in bowel habits. He complained of non-specific lower abdominal discomfort but had no nausea or vomiting. He was anorexic and had lost 20 pounds in weight in three months. Hematocrit was 11 per cent and hemoglobin 4 g/100 ml. stools were positive for occult blood. Complete gastrointestinal barium studies were not remarkable. Hematologic tests were suggestive of iron and folic acid deficiency. The patient was treated conservatively with blood transfusions, iron, and folic acid, and discharged after hematocrit was restored to normal.

He was admitted for the second time on November 13, 1970, with essentially the same complaints as during the previous admission. Hematocrit was 10 per cent and hemoglobin 4 g/100 ml. The only pertinent findings on complete gastrointestinal x-ray studies were a deformed duodenal bulb and pancreatic calcification. The patient became asymptomatic after blood transfusions, and went home, against medical advice.

The patient was admitted for the third time on January 17, 1971, in an intoxicated condition, with a history of a fall resulting in abrasions of the face and head. Since his last discharge, he had been drinking heavily and had also experienced vague pains in the lower abdomen. He mentioned that his previous symptoms of weakness, lightheadedness and intermittent dark and fresh rectal bleeding had continued. The pertinent radiologic finding at this time was a filling defect in the cecum, found on barium-enema studies (Fig. 1). In addition, the deformity of the duodenal bulb and calcification of the pancreas were again confirmed.

The patient underwent celiotomy on February 18, 1971. The operative findings were minimal scarring of the first portion of the duodenum, an enlarged nodular pancreas grossly consistent with chronic pancreatitis, and a smooth polyposid lesion palpable in the ileocecal area. A cecotomy was then performed. The ileocecal valve was hypertrophied and invaginated into the cecum, creating a blind pouch. The inner wall of the pouch was studded with several punctate hemorrhagic ulcerations (Fig. 2). There was a small pool of residual blood in this pouch, but there was no blood in the small intestine. The redundant fold of ileocecal mucosa was excised, the defect repaired, and the cecotomy closed. The ileocecal orifice was found to be adequately patent on digital palpation.
Fig. 1. Barium-enema study showing the caput coli turned upward and backward and the appendix extending upward from it in a fixed position. A rounded defect, 2 × 2 cm, measured on the film, is present at the location of the ileocecal valve. Its margins are discrete but show lobulation suggestive of mucosa.

Gross examination of the resected portion of ileocecal valve revealed a 4 × 3 × 1-cm pouch-shaped segment of intestinal mucosa and submucosa. There were numerous 1–2-mm punctate hemorrhages on the mucosal surface. Histologically, the specimen consisted of ileocecal mucosa and submucosa with scattered hemorrhages overlying minute superficial mucosal ulcers. There was no histologic evidence of intrinsic inflammatory or neoplastic disease in the specimen.

The postoperative course was complicated by clinical and radiologic findings consistent with distal small intestinal obstruction. Exploration on the fifth postoperative day revealed no site of obstruction, but small discrete, cyanotic patches were seen on the entire antimesenteric border of the small bowel. This was interpreted as nonocclusive ischemia of the intestine. The cyanotic lesions were alleviated by oxygen inhalation alone during the course of the procedure. Nothing further was done and the abdomen was closed.

The patient recovered and was discharged on March 16, 1971. He was seen in the surgical clinic at regular intervals. His appetite improved, he gained 20 pounds in weight, and he had no recurrence of abdominal pain, melena, weakness, or lightheadedness.

Comment

Recent in vivo studies have demonstrated that the ileocecal junction is a conical projection of the terminal ileum into the cecum. Older concepts about the terminal ileum resembling a valve were derived from studies of cadavers and were therefore subject to considerable distortion. Grossly, it resembles the uterine cervix and is appropriately referred to as a papilla. It pro-