Australia antigen helpful in detecting patients who may have both diseases, as the clinical manifestations of one might easily mask those of the other. Malaria among narcotic addicts appears to be a renewed clinical problem of which physicians should be aware, especially in addicts who might present with bizarre symptoms or with fever of undertermined cause.

Summary

Vivax malaria was observed in ten heroin addicts who admitted to the sharing of needles. None had traveled outside the United States. It was suspected that these cases are related to a Vietnam returnee, although this was not definitely established. All cases were diagnosed by routine peripheral smears. In view of our military involvement in Vietnam and the prevalent use of drugs in the civilian population being joined by returning servicemen, clinicians should be alerted to the problem.

REFERENCES

1. Gerhardt C, Klin Z: Med 1:372, 1887, Cited by Most H: Malignant malaria among drug addicts. Trans R Soc Trop Med Hyg 34: 139-172, Aug 1940

2. Chojnacki, RE, et al: Transfusion induced falciparum malaria. N Engl J Med 279:984-985, Oct 31, 1968

3. Tanowitz H: Transfusion malaria. N Engl J Med 280:275-276, Jan 1969

4. McQuoy RM, et al: Congenital malaria in Chicago: A case report and a review of published reports. Am J Trop Med Hyg 16:258-266, May 1967

5. Biggam AG: Malignant malaria associated with administration of heroin intravenously. Trans Roy Soc Trop Med Hgy 23:147-155, Aug 1929

6. Appelbaum E, Galfand BB: The artificial transmission of malaria among intravenous diacetylmorphine addicts. JAMA 102:1664-1670, May 1934

7. Boyd LJ, Schlackman M: Malaria in drug addicts. NY State Med J 38:974-976, Jul 1938

8. Bradley JA: Transmission of malaria in drug addicts by intravenous use of narcotics. Am J Trop Med 14:319-324, Jul 1934

9. Bradley JA: Intravenous transmission of malaria in drug addicts. J Trop Med Hyg 37:241-244, Aug 1934

10. Eaton LM, Feinberg SM: Accidental hypodermic transmission of malaria in drug addicts. Am J Med Sci 186:679-683, Nov 1933

11. Himmelsbach CK: Malaria in narcotic addicts at the U. S. Penitentiary Annex, Fort Leavenworth, Kansas. Public Health Rep 48:1465-1472, Dec 1933

12. Most H: Malignant malaria among drug addicts. Trans R Soc Trop Med Hyg 34:139-172, Aug 1940

13. Nickum OC: Malaria transmitted by hypodermic syringe. JAMA 100:1401-1412, May 1933

14. Nickum OC: Malaria in Nebraska from a contaminated hypodermic syringe. Nebr State Med J 18:104, Jun 1933

15. Volini IF, Shapiro WW: Malaria with special reference to narcotism. Ill Med J 72:458-463, Nov 1937

16. Neva FA, et al: Malaria: Host-defense mechanisms and complications. Ann Intern Med 73:295-306, Aug 1970

17. Panel on malaria. Ann Intern Med 70:127-154, Jan 1969

18. Malaria surveillance—Annual Summary, 1969. Atlanta Center for Disease Control, 1970

Duodenocolic Fistula

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A NON-MALIGNANT CONNECTION between the duodenum and the colon is a rare condition of diverse causes. The anatomic proximity of the two structures allows disease or injury of either to result in fistula formation, occasionally in combination with a third structure, the appendix or the gallbladder. The unique and the rather distinguishing clinical features of this condition have led to the report of the present case and the review of other reports.

Incidence

Despite the anatomic nearness of the duodenum and the colon, fistula formation is quite rare. In most of the cases in which it does occur it is due to malignant disease (Medhurst, 1956).[1] Non-malignant fistula was reported only 27 times between 1863, when Sanderson reported the first such case, and 1966 when Brindle and Kane[2] added two cases and surveyed the literature to that time. In the same year, another case was reported, by Trickey and Dorling.[3] The present case is the only one reported since. Undoubtedly there are many cases not reported, yet the condition is so rare that few surgeons have seen one.

Etiology

The most common cause of this fistula is duodenal ulcer disease (Table 1). The fistula may develop insidiously from direct extension of the ulcer crater, or it may occur secondary to a perforation and abscess formation, as reported by Starzl (1959).[4] Foreign body penetration was reported by Rosenqvist (1955)[5] and Trickey (1966).[3] Tuberculosis involving abdominal lymph nodes with caseous necrosis and fistula formation was a factor in five instances. Fistula secondary to ulcerative colitis has been reported twice, and to regional enteritis twice—somewhat surprising since fistulas elsewhere in the gastro-

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TABLE 1.—*Causes of Benign Duodenocolic Fistula*

Duodenal Ulcer
Duodenal Diverticulitis
Tuberculosis
Typhoid Fever
Appendicitis
Cholecystitis
Foreign Body
Trauma
Ulcerative Colitis
Regional Enteritis

intestinal tract are not uncommon with these diseases. Cholecystitis caused a fistula in a case reported by Neville (1954).[6] Other case reports indict typhoid ulcer, duodenal diverticulitis and trauma.

Appendicitis as the cause of duodenoappendicocolic fistula has been reported twice previously. Clayton and Thornton described a patient with chronic symptoms of acute appendicitis who was treated with aspirin and hot packs.[7] After six days of stormy illness, he recovered but had continuing diarrhea. Later, at operation, he was found to have a fistula between the duodenum and the cecum, adjacent to the appendix base, and an open appendix. A similar case was reported by Marinaccio et al in 1953 and cited by Starzl in 1959.[4]

Report of a Case

A 40-year-old Filipino with bronchial asthma had onset of diarrhea which was difficult to control about four months before admission to hospital. He noted postprandial cramping in the upper abdomen and postprandial diarrhea. Stool studies for ova and parasites, fat, undigested protein and enteropathogens were negative. Barium enema demonstrated filling of the small intestine, but the presence of a fistula was not determined. The diarrhea and abdominal pain ceased spontaneously. Then the asthma worsened so much that the patient was admitted to hospital for that reason. Steroids were given with satisfactory response and he was discharged. Three days later he was readmitted with nausea, vomiting, abdominal pain, abdominal distension and recurrence of diarrhea. Abdominal x-ray studies showed abnormalities interpreted as partial small bowel obstruction or severe ileus.

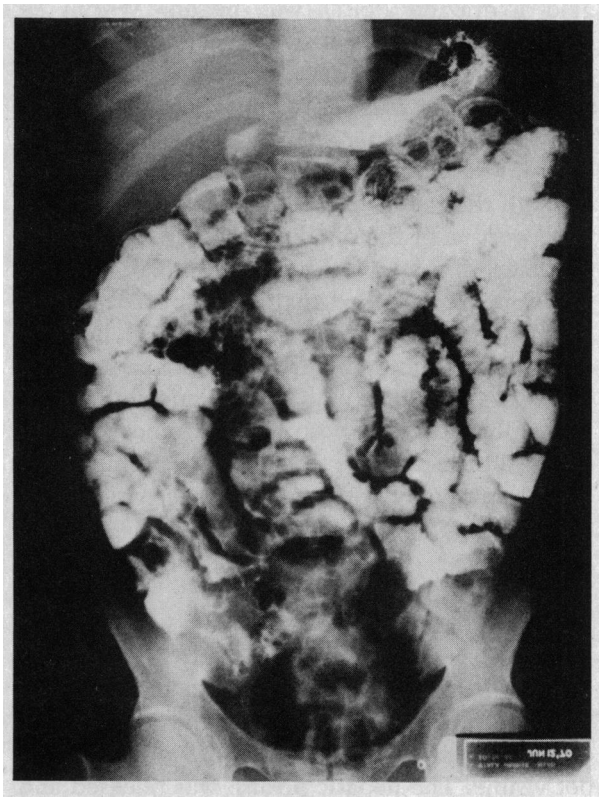

Figure 1.—Barium enema study shows distal small intestine is edematous and somewhat amorphous. It was noted to be rigid at fluoroscopy.

On physical examination the patient appeared chronically ill and there was evidence of loss of weight. The abdomen was distended and doughy to palpation, with tenderness in the right upper quadrant. The temperature was 38.9°C (102.0°F). The blood count was bizarre: Leukocytes numbered 6,000 per cu mm with a differential of 6 percent neutrophiles, 45 percent non-segmented neutrophiles, 25 percent lymphocytes and 24 percent monocytes. The hematocrit was 45. Three days later leukocytes were 6,200 but the monocytosis had disappeared and the neutrophiles had risen to 30 percent and the non-segmented neutrophiles had dropped to 34 percent. Acid-fast sputum stains were negative. Results of stool examinations were within normal limits.

The patient was treated with intravenous fluids only for three days while investigation was pursued. He remained febrile during that time. A presumptive diagnosis of intestinal tuberculosis was made, and treatment with streptomycin, para-aminosalicylic acid (PAS) and isonicotinic

acid hydrazide (INH) was begun. Within 24 hours fever abated and it did not recur.

Barium enema studies again showed diffuse rapid filling of the entire small intestine, and a diagnosis of a fistula was entertained. An upper gastrointestinal series and small bowel follow-through then demonstrated a fistula between the third portion of the duodenum and the right side of the colon. The distal half of the small intestine was smudged and edematous, appeared chalky and amorphous, and the wall of the ileum was of rigid configuration. (Figure 1). The interpretation was regional enteritis. Alimentation was gradually increased with no recurrence of diarrhea. Antituberculosis drugs were continued and after ten days of rest and food the patient was operated upon.

Operative Findings

Except for white coloration and thickening of the inferior mesenteric surface, no abnormalities were noted in the free peritoneal cavity. The cecum was normal except that the appendix was not visible. The hepatic flexure was released and the colon was freed and moved to the left. The appendix, which lay behind the cecum, was connected by a fistula to both the duodenum and the cecum. Immediately adjacent to that fistula was a second, separate duodenocecal fistula. There were no signs of inflammation in the retroperitoneal tissues. Operative repair consisted of simple division of the fistulas and two-layer closure of the duodenum and colon. The appendix was removed. Healing and recovery were uneventful. The patient gained eight pounds in the first three weeks after operation and remained well, without diarrhea, thereafter.

Clinical Presentation

The symptoms of duodenocolic fistula (Table 2) are similar in all cases regardless of etiologic factors, although a primary disease may add other signs and symptoms to those of this condition. All patients lose weight and nearly all will have diarrhea. Pain—vague upper quadrant aching or severe upper quadrant or central pain with radiation to the back—is present in 60 percent of cases, and in 50 percent there are foul eructations and vomiting. Barium contrast studies will almost always demonstrate the fistula. Although the barium enema is said to be the better test, in the present case the upper gastrointestinal series was superior. Both studies should be done as part of the preoperative investigation.

TABLE 2.—*Clinical Symptoms of Duodenocolic Fistula*

Weight Loss
Diarrhea
Abdominal Pain
Vomiting (Feculent)
Foul Eructations

Pathophysiology

Diarrhea and weight loss may be secondary to the mechanical short-circuiting of the small intestine. However, the diarrhea is seldom profuse, or "total," and frequently is intermittent, suggesting that some food travels through the intestine in normal fashion. There may be impaired function of this intestine due to "contamination" by colon bacteria, causing jejunitis with resulting decrease in absorption of vitamins and proteins and other nutrients. Indeed, most of the fistulas described are 1 cm or less in diameter. In the present case diarrhea ceased completely when streptomycin was given as a preoperative measure (because of the possibility of tuberculosis). In addition, the x-ray studies of the small intestine showed edema and flocculation (Figure 1) that were interpreted by the radiologist as diffuse inflammation. It was for this reason that our preoperative diagnosis was regional ileitis. If the symptoms are caused by this fecal contamination, then antibiotic treatment should abate the malnutrition and help overcome some of the nutritional deficiencies. In a poor risk patient, long-term nonabsorbable antibiotics might even be selected as preferential treatment.

Treatment

The above considerations notwithstanding, surgical treatment will generally be selected. The specific operation in each case will be determined by the status of the tissues and the underlying etiologic factors. Obviously, simple division and closure of a fistula is preferable if possible, but if there is inflammatory tissue or an active abscess the surgeon will have to decide whether it is best to use drainage, resection, serosal patching or

some other measure. The surgeon also must consider the cause of the fistula, since the primary process may require definitive surgical treatment. Such treatment may be indicated at the time of fistula division (for example, total colectomy for ulcerative colitis) or at a secondary laparotomy (for example, gastrectomy or vagotomy or both for duodenal ulcer).

Preoperative restoration of vitamins, minerals, electrolytes, blood, protein, and blood volume is essential to a safe operation and an uncomplicated postoperative course. The degree to which this restoration can be accomplished will depend on the severity of the process, the condition of the patient and the response to treatment. As in gastrointestinal fistula of other kinds, no time should be lost in nonhelpful treatment when division of the fistula per se is required to reverse pathophysiologic changes.

Summary

Duodenocolic fistula not associated with cancer is a rare condition of diverse cause. A case secondary to appendicitis with a resulting duodenoappendicocolic fistula is reported. Certain aspects of this case suggest that the cardinal findings of diarrhea and weight loss are more likely due to colon bacterial contamination of the small intestine than to mechanical by-pass through the fistula. Treatment of duodenocolic fistulas is governed by a variety of factors.

REFERENCES

1. Medhurst GA: Duodeno-colic fistula. Br J Radiol 29:381-385, 1956

2. Brindle MJ, Kane JF: Benign duodenocolic fistula. Br J Surg 53: 749-753, 1966

3. Trickey SE, Dorling GC: Benign duodenocolic fistula. Br J Radiol 39:464-465, 1966

4. Starzl TE, Dorr TW, Meyer WH Jr: Benign duodenocolic fistula. AMA Arch Surg 78:611-619, 1959

5. Rosengvist J, Sjoberg SG: Acta Chir Scand 109:293-296, 1955

6. Neville WE: Duodenocolic fistula due to acute cholecystitis. Am J Surg 87:300-302, 1954

7. Clayton RS, Thornton WL: Benign duodeno-colic fistula. Radiology 60: 832-836, 1953

Retrieval of Catheter Fragments

Report of Two Cases

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THE RETRIEVAL OF BROKEN POLYETHYLENE catheters, guide wires and pacemaker catheters from various sites in the cardiovascular system has recently received increasing attention.[1-13] It is clear that many, if not most, of these fragments can be retrieved by non-surgical means.[4,8-13] Several methods have been described in the literature for such retrieval.[8,9,12] During the past year we have had opportunity to remove broken pieces of catheters from two patients, utilizing variations of existing techniques. The purpose of this report is to emphasize that in all such incidents an attempt first should be made to remove the catheter by a non-surgical method, with operation held in reserve.

Case 1. A 20-year-old white woman was admitted to hospital following an overdose of tranquilizers. When first seen, she was semicomatose and responded only to deep pain. A medium-Intracath®* was inserted into the left median basilic vein. Within a half hour the patient became restless and agitated, and after attempts to calm her succeeded it was noted that the intravenous catheter had been dislodged and was several inches shorter than when inserted. An x-ray film of the chest showed the catheter fragment (which was radio-opaque) to be lodged in the apex of the right ventricle and extended to the right lateral wall of the right atrium. The patient was taken to the cardiac catheterization laboratory, where a No. 7 Goodale-Lubin catheter with an 0.035-inch Teflon guide

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