Five Cases of *Haemophilus segnis* Appendicitis

WILLIAM D. WELCH,1* PAUL M. SOUTHERN, JR.,2,3 AND NANCY R. SCHNEIDER2,3

Division of Microbiology and Immunology, Endocrine Sciences, Tarzana, California 91356,1 and Department of Pathology, The University of Texas Health Science Center at Dallas,2 and Parkland Memorial Hospital,3 Dallas, Texas 75235

Received 18 February 1986/Accepted 21 July 1986

The clinical, histological, and bacteriological findings in five cases of acute appendicitis caused by *Haemophilus segnis* are reported. This is the first documentation of appendicitis associated with this organism.

The bacteria most often associated with acute appendicitis include *Escherichia coli*, streptococci, *Bacteroides melaninogenicus*, and *Bacteroides fragilis* (4). Of the *Hae- mophilus* species, only *H. influenzae* has been documented as an infrequent cause of appendicitis (2, 11). We report here, for the first time, the isolation of *Haemophilus segnis* from the peritoneal fluid of five patients admitted to the hospital emergency room (ER) with a clinical diagnosis of acute appendicitis.

**Patient 1.** Patient 1 was a 24-year-old Latin American male admitted to Parkland Memorial Hospital (PMH) in Dallas, Tex., on 2 May 1984 with right lower quadrant (RLQ) abdominal pain for the previous 5 h accompanied by nausea and the urge to defecate. In the ER, the patient had a temperature of 38.6°C and a leukocyte count of 13,000/μl. On physical examination, patient 1 had tenderness and rebound tenderness in the RLQ of the abdomen. He was taken to surgery, where gross purulence was observed. The appendix was adherent to the cecum, and there was perforation near the base of the appendix. A culture of the peritoneal fluid from the day of surgery grew only *H. segnis*. Microscopic examination of the appendix showed a normal mucosa with large reactive lymphoid follicles, focal mild scatterings of eosinophils, and rare polymorphonuclear leukocytes in the muscularis. Fibrinopurulent serositis was seen (with both acute and chronic inflammatory cells). No histological perforation was noted. Patient 1 was placed on 2 g of imipenem per day and discharged without complications on 9 May 1984.

**Patient 2.** Patient 2 was a 27-year-old Latin American male admitted to the PMH ER on 5 May 1984 with RLQ abdominal pain, nausea, vomiting, a temperature of 36.4°C, and a leukocyte count of 19,000/μl. The patient was taken to surgery, where a small perforation was noted at the base of the anterior mesenteric edge of the appendix. A culture of the peritoneal fluid grew only *H. segnis*. Microscopic examination of the appendix revealed a normal mucosa without inflammation, an edematous submucosa, and muscularis with a mild infiltrate of eosinophils and polymorphonuclear neutrophils in the muscularis. A severe fibrinopurulent serositis with extension into the peripancreatic fat was also observed. Patient 2 was placed on 4 g of cefoxitin per day and discharged without complications on 13 May 1984.

**Patient 3.** Patient 3 was an 18-year-old Latin American male admitted to the PMH ER on 12 February 1985 with a 2-day history of RLQ abdominal pain and an elevated leukocyte count. His RLQ pain increased during the day of admission, and he was taken to surgery the same day. At surgery, the appendix was noted to be perforated 1 cm proximal to the base, with seropurulent peritoneal fluid present. A culture of the peritoneal fluid revealed *Bacteroides multiacidicus* and *H. segnis*. Microscopic examination of the appendix revealed a mucosa with mild-to-moderate acute inflammation, which was also seen in the muscularis. An intense and dense fibrinopurulent exudate was observed in the serosal surface. The patient was placed on 4 g of cefoxitin per day and discharged without complications on 20 February 1985.

**Patient 4.** Patient 4 was a 22-year-old Latin American male who came to the PMH ER on 22 April 1986 with RLQ abdominal pain of 1 day's duration. He also had fever but no nausea, vomiting, or diarrhea. His previous history was unremarkable. Physical examination showed a temperature of 39.2°C and blood pressure of 124/86. He had signs of diffuse peritonitis, with RLQ rebound tenderness. Laboratory work included normal electrolytes, blood urea nitrogen, creatinine, and urinalysis. The leukocyte count was 30,700/μl, the hematocrit was 49.7%, and the platelet count was 403,000/μl. The differential was 85% neutrophils, 5% bands, 3% lymphocytes, and 7% monocytes. A chest X ray was normal. The patient received cefoxitin (2 g intravenously) and then was taken to surgery, where a grossly inflamed, edematous, exudative appendix, with a perforation 8 mm from the base, was found. There was 60 ml of purulent fluid in the peritoneal cavity. An appendectomy was done. On 21 April 1986, the antibiotics were changed to ampicillin, clindamycin, and tobramycin. These were continued through 28 April 1986. Cefoxitin was given on 28 and 29 April. Dates and maximum temperatures were: 23 April, 39.1°C; 24 April, 38.4°C; 25 April, 38.6°C; 26 April, 38.5°C; 27 April, 37.6°C; 28 April, 37.4°C; 29 April, 37.4°C; 30 April, 37.0°C. The patient was discharged on 30 April 1986. He was seen in the outpatient clinic on 20 May 1986 and was doing well. The surgical pathology report revealed acute inflammation, transmurally, with acute serositis as well. Two sets of blood cultures were negative. Cultures of the peritoneal fluid at surgery were revealed *E. coli*, *Klebsiella pneumoniae*, and *H. segnis*.

**Patient 5.** Patient 5 was a 32-year-old male who came to the PMH ER on 26 April 1986 with vomiting and RLQ abdominal pain and tenderness. His previous history was negative except for an anal abscess several years earlier. Physical examination showed a temperature of 36.3°C. Chest and abdominal X rays were normal. Laboratory work included normal electrolytes, blood urea nitrogen, and creatine; glucose, 138 mg/dl; leukocyte count, 16,200/μl; hema-

---

* Corresponding author.
NOTES

H. segnis was previously isolated from a pancreatic abscess in a 29-year-old alcoholic male (3). Four of the five patients in the present study were young (18 to 27 years old) previously healthy Latin American males. Although physical examination and surgery revealed classical appendicitis, histological examination of the appendices was remarkable for the lack of any mucosal inflammation in patients 1 and 2. Additionally, a severe fibrinopurulent septum was present in all of the appendices. This unusual histological appearance of the appendix from patients presenting with symptoms of classical acute appendicitis may be characteristic of infection of the appendix caused by H. segnis. The route of infection remains unknown in these patients, but the origin of the organism is most likely the oropharynx. Because this is the second report describing the isolation of H. segnis from severe human infections, H. segnis may indeed have the potential to act as a pathogen.

We thank Sharon Mugleston for preparation of this manuscript.

LITERATURE CITED