Bacterial Meningitis and Sigmoid Diverticulitis Caused by *Listeria Monocytogenes*

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This case report describes a previously healthy woman who suffered from both bacterial meningitis and sigmoid diverticulitis. The meningitis was caused by the pathogen L*isteria monocytogenes*, which can normally be cultured from human gastrointestinal bacterial flora. It is well known that hematogenous spread of this bacterium causes infection of the central nervous system. However, a search of the English-language medical literature did not reveal any reports of a patient with perforated colonic diverticulitis causing the spread of L*isteria monocytogenes* into the blood and finally into the CNS.

Patient Description

A 79 year old woman was admitted to the emergency department because of abdominal pain, fever and headaches over the previous 3 days. Her past medical history included hospitalization 6 years prior to the current admission because of uncomplicated sigmoid diverticulitis. On examination she appeared ill. She was conscious. Blood pressure was 130/70 mmHg, pulse 98 regular and fever 38ºC. Examination of the head and neck revealed nuchal rigidity. The abdomen was soft without any tenderness. The rest of the examination was unremarkable. Her white blood count was 13,400 cells/µl. Blood biochemistry and urine tests were within normal limits. Chest X-ray showed no signs of pneumonia. Because of the nuchal rigidity and fever, lumbar puncture was performed. Examination of the cerebrospinal fluid demonstrated 600 neutrophils/µl, total protein 98 mg/L and glucose 4.7 mMol. Gram staining of the CSF was negative for any bacteria.

The patient was treated empirically with intravenous ceftriaxone and ampicilin. Two days after hospitalization, one set of blood culture tubes had grown L*isteria monocytogenes*. A day later, CSF cultures with the same pathogen were positive. The patient continued to deteriorate during 6 days of hospitalization and her mental status declined. On the seventh day of hospitalization, abdominal and pelvic computed tomography was performed owing to abdominal tenderness on physical examination. The CT demonstrated a large amount of free peritoneal air and several left colon diverticules with suspected extra-colonic air around them. The patient was taken to the operating room for exploratory laparotomy. During exploration of the abdomen we found pneumoperitoneum without any fluid or feces contaminating the peritoneum. A perforation at the sigmoid colon was demonstrated. Both the descending and the sigmoid colon contained many diverticula. We performed descending and sigmoid colectomy with end-transverse colostomy and Hartman's rectal pouch. The patient was transferred to the intensive care unit for further medical management. Antibiotics (sulphamethoxazole and trimpethoprin) against the bacteria were continued for 6 weeks. The postoperative course was surgically unremarkable. However, neurological improvement was not achieved.

Comment

L*isteria monocytogenes* is a Gram-positive rod-shaped bacterium that usually causes diseases in neonates, pregnant women and immunocompromised patients [1]. L. *monocytogenes* can normally be cultured from human gastrointestinal bacterial flora. The transmission of the bacteria to humans is food-borne [2]. There are well-known groups of patients in whom disease can develop from infection with L. *monocytogenes*. Pregnant women, usually in the third trimester, comprise one group. The presentation is sepsis with a tendency towards preterm labor and with no signs of meningitis. The second group constitutes neonates and infants under the age of 3 months. The presentation is sepsis with or without involvement of the meninges. The third group of patients comprises immunocompromised patients and the elderly. It has been reported that in the population of patients over the age of 50, meningitis caused by this bacterium is secondary to infection with S*treptococcus pneumoniae*.

Diseases caused by infection with L. *monocytogenes* may develop as sepsis without a source of infection, or as specific involvement of organs such as the CNS [3], heart, eye, gastrointestinal system, bones and lungs. The most common presentation of CNS infection with L. *monocytogenes* is meningitis. Other presentations of CNS involvement are encephalitis, cerebritis, brainstem infection and intracranial abscess. Hematogenous spread is thought to be the means whereby the CNS is infected. The presenting symptoms and physical examination signs are the same as for any other bacterial meningitis. Examination of the CSF reveals pleocytosis, elevated protein level and

CNS = central nervous system
CSF = cerebrospinal fluid

usually normal glucose concentration. In only 30% of patients does the CSF stain positively for L. *monocytogenes* and usually the diagnosis is approved only after L. *monocytogenes* has been isolated in CSF or blood cultures. The treatment of choice is intravenous penicillin for 2 weeks in immunocompetent patients and 6 weeks for immunocompromised patients. An alternative treatment for patients who cannot tolerate penicillin is sulfamethoxazole combined with trimethoprim. The role of corticosteroid as an adjuvant therapy for meningitis is not known in meningitis caused by L. *monocytogenes*. It is worth mentioning that L. *monocytogenes* utilizes iron as a virulent factor. It is recommended that iron supplement treatment in patients with iron deficiency be postponed for the duration of infection with L. *monocytogenes*.

The patient described here suffered from meningitis caused by the pathogen L. *monocytogenes*, whose isolation from blood cultures has been noted. This laboratory finding is probably evidence for hematogenous spread of the bacterium. The patient was finally diagnosed to also have perforation of sigmoid colon diverticula. Despite attempting to connect our clinical findings to a single disease, the fact that the patient presented with abdominal pain on admission, CNS infection, L. *monocytogenes* bacteremia and perforation of the colonic diverticula indicated a primary infection of sigmoid colon diverticulitis initiated by the bacteremia, meningitis and finally perforation of the colon. A search of the English medical literature for reports of colonic diverticulitis that progressed to meninigitis was fruitless. We assume that the patient was a host of L. *monocytogenes* in the gastrointestinal bacterial flora and that the hollow viscous perforation allowed contamination of the peritoneum [4] and spread of the bacteria to the blood and the CSF. However, we did find several reports of patients with colitis such as inflammatory bowel disease who developed L. *monocytogenes* bacteremia [5]. Observations from the current patient supported the fact that when L. *monocytogenes* bacteremia is found the physician must intensively seek out the origin of the infection. However, the source of infection may be difficult to find, especially in cases of debilitated patients with impaired mental status who have difficulties expressing their complaints. Early and wide-ranging use of imaging modalities may help in finding the origin of infection. Ultimately though, there is probably no substitute for repeated and meticulous physical examinations of the patient.

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Capsule

Fibromyalgia and IVIG

Fibromyalgia (FM) is a chronic, distressing disorder that affects mainly women in the prime of life. The diagnosis relies on subjective criteria of ongoing pain, tender points, fatigue, mild cognitive impairment and other manifestations. The etiology of the disorder is unclear, but a subgroup of FM patients exhibited clinical and serological features of immune deregulation. Recently Caro et al. evaluated the presence of immune-mediated demyelinating polyneuropathy and response to therapy with intravenous immunoglobulin G (IVIG) in 58 FM patients compared with 26 rheumatic and 52 non-rheumatic subjects. Paresthesias was found in 76% of FM patients compared to 20% in controls ($P < 0.0001$), and stocking distribution hypoesthesia in 88% of FM patients vs. none of the controls ($P < 0.0001$). Weakness and muscle strength were also very common and significantly elevated in FM patients ($P < 0.0001$). Chronic inflammatory demyelinating polyneuropathy (CIDP) including stocking hypoesthesia, proximal muscle weakness and electrodiagnostic abnormalities were documented in 16 FM patients (33%) vs. only 2 controls (5%) ($P = 0.005$). After treatment with IVIG (400 mg/kg/day for 5 days) in 15 patients with CIDP and FM, a significant improvement in pain, tenderness and muscle strength as well as a non-significant decrease in fatigue and stiffness were noted. Thus, it might be concluded that a substantial subgroup of FM patients have clinical findings suggestive of CIDP for which IVIG therapy might be beneficial.

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