Laboratory tests revealed the following values: hemoglobin, 10.8 g/dL (6.4 mmol/L), with an RBC smear showing several Howell-Jolly bodies and Burr cells; WBCs, 2.7 X 10^9/L; thrombocytes, 36 X 10^9/L, with coagulation tests indicating severe disseminated intravascular coagulation; glucose, 14.3 mg/dL (0.8 mmol/L); creatinine, 3.9 mg/dL (345 μmol/L), and serum lactate, 99.2 mg/dL (11.9 mmol/L). A chest roentgenogram showed no infiltrates. Blood cultures became positive for S. pneumoniae.

Despite aggressive treatment including high-dose penicillin, the patient died 10 hours later with irreversible shock. Postmortem examination showed a small spleen weighing only 20 g (normal weight, ≥200 g). Microscopic examination revealed a complete absence of red pulp, with a general increase in fibrous tissue. Although clinical signs of a relapse had been absent, the entire colon showed signs of moderate to severe inflammation.

This rapidly fatal infection with S. pneumoniae in a patient with ulcerative colitis is suggestive of an asplenic spleen. This diagnosis is supported by the presence of Howell-Jolly bodies and Burr cells in the peripheral blood smear. At postmortem examination the spleen was extremely small (20 g) and contained no red pulp. The increase in fibrosis points to an acquired form of hyposplenism.

The association between functional hyposplenism and ulcerative colitis was first described in 1974 [1-3]. In 1978 Ryan and co-workers presented the results of a prospective study on splenic function in cases of inflammatory bowel disease [4]. Thirteen (37%) of the patients with ulcerative colitis had hyposplenism, as compared with only one patient (5%) with Crohn's disease. The presence of Howell-Jolly bodies in the blood film was associated with severe and extensive colitis. Pneumococcal septicemia developed in one patient on day 3 after a panproctocolectomy.

In a recent study by Muller and co-workers, six of 29 patients (21%) with ulcerative colitis showed signs of functional hyposplenism [5]. All but one had pancolitis in acute relapse. Patients experiencing a severe relapse had significantly smaller spleens than those of patients whose disease was quiescent or controlled. In contrast, Pereira and co-workers found no relation between spleen size and the site or extent of the inflammation in 116 patients with inflammatory bowel disease [6]. However, there was a relation between small spleen size and infectious complications after surgery. The etiology of hyposplenism remains uncertain. The hypothesis that circulating immune complexes may block splenic reticuloendothelial function has not been proved [7].

Besides the patient described by Ryan and coworkers [4], we could find only one other patient with fulminant pneumococcal sepsis and ulcerative colitis described in the literature [8]. In this case the spleen weighed only 32 g and showed extensive atrophy of both T and B cell areas.

Considering the prevalence of ulcerative colitis, we feel that fulminant pneumococcal sepsis must be a relatively rare complication. Therefore, we do not think that all patients with ulcerative colitis should undergo prophylactic pneumococcal vaccination, although it is reasonable to consider vaccination of a patient when persistent signs of hyposplenism are evident in the peripheral blood smear. However, every patient with ulcerative colitis who presents with circulatory collapse and signs of sepsis should immediately be treated for a presumed fulminant pneumococcal infection.

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References

Pyomyositis as the Sole Manifestation of Disseminated Gonococcal Infection: Case Report and Review

Disseminated gonococcal infection (DGI) occurs in ~0.5%–3.0% of all cases of gonorrhea [1]. Reports of extrapelvic skeletal muscle abscess caused by Neisseria gonorrhoeae are rare. We report a case of DGI presenting as a mass in the right biceps brachii.

A 16-year-old woman presented to the emergency department complaining of right shoulder pain that had been preceded by symptoms of respiratory tract infection, generalized arthralgia, and myalgia. She denied any fever, chills, dysuria, or vaginal discharge. Physical examination revealed tenderness over the right bicipital groove. A firm, indurated, tender mass was noted in the right biceps. Extension lag was present at the elbow. Axillary adenopathy was absent. Laboratory studies revealed a WBC count of 16,800/mm³. A urethral culture was negative for N. gonorrhoeae. A bone scan and plain films of the shoulder did not show any abnormalities. MRI showed a fluid collection in the biceps and marked inflammation of surrounding tissues (figure 1).
Figure 1. Proton-weighted (spin echo = 3,000/18) coronal MRI view of the right arm of a woman with disseminated gonococcal infection; an inhomogeneous mass with increased signal intensity is seen within the biceps muscle. Adjacent muscle shows less increase in signal intensity.

A gram stain of the fluid obtained by needle aspiration showed many WBCs and RBCs but no organisms. Culture yielded a few colonies of *N. gonorrhoeae*. Treatment with oral ciprofloxacin (500 mg b.i.d.) was started empirically before the culture results were known. Three days later the swelling had resolved, and full range of motion of the elbow joint was restored. Oral doxycycline (100 mg b.i.d.) was added to the therapeutic regimen. The patient did not return for a follow-up examination.

Abscess formation in gonococcal disease may result either from direct extension of the primary infection or from hematogenous dissemination. *N. gonorrhoeae* has been reported as a cause of intrapelvic abscesses. Abscess of the obturator internus muscle has been reported, probably as a result of local extension from the uterine adnexa [2].

Primary cutaneous abscesses have occurred when breaks in epithelial integrity facilitate transmission of *N. gonorrhoeae* via digital-genital contact [3]. In the present case, there was no break in the skin overlying the muscle and no history of brachio-genital contact or biting.

Muscle abscesses associated with *N. gonorrhoeae* infection were occasionally reported before the antibiotic era. Bacteriologic data were often incomplete or rudimentary. In 1904, a case of gonococcal abscess of the calf muscle, with no evidence of tenosynovitis, was reported [4]. More recently, Swarts et al. [5] reported a case of gonococcal muscle abscess. Although gram stain of abscess fluid showed no organisms and routine cultures were negative, gonococci were recovered when the fluid was cultured on Thayer-Martin medium. Owino et al. [6] reported a case in which *N. gonorrhoeae* recovered from a discharging subcutaneous abscess was the initial manifestation of DGI. It is unclear whether the underlying muscle was involved. In both cases, the upper arm was the site of involvement.

The occurrence of pyomyositis in temperate zones is unusual: a review of the North American literature over a 20-year period revealed only \~100 cases [7]. Aerobic gram-negative organisms accounted for 10% of the cases, a higher proportion than is described in reports of “tropical” pyomyositis, in which *Staphylococcus aureus* accounted for \~99% of isolates.

We hypothesize that the symptoms initially reported by our patient represented an episode of DGI. The tenderness noted over the bicipital groove on physical examination was probably due to gonococcal tenosynovitis, which had not been treated and had progressed to involve the body of the muscle.

Consideration should be given to *N. gonorrhoeae* as a causative organism when evaluating any patient who is sexually active and presents with pyomyositis, especially in an upper-extremity site, even in the absence of genitourinary symptoms. We advocate the use of chocolate agar medium for culture of abscess fluid or biopsy material. Use of Thayer-Martin medium may diminish the diagnostic yield, as some strains of *N. gonorrhoeae* are susceptible to vancomycin.

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ties. He had been in good health until 2 1/2 months before admission, when he had developed back pain and leg numbness. Two weeks later, an MRI study performed elsewhere demonstrated an infection due to Shigella boydii. A comprehensive search of the English-language literature for the period 1970 to 1994 (with the use of MEDLINE and a review of secondary references), we found only three published reports of human bone and/or joint infection due to Shigella species [1–3]. We report, to our knowledge, the first case of vertebral osteomyelitis due to Shigella species.

A 69-year-old man presented to our hospital, for the first time, with back pain and weakness and sensory loss in the lower extremities. He had been in good health until 2 1/2 months before admission, when he had developed back pain and leg numbness. Two weeks later, an MRI study performed elsewhere demonstrated an infection of the ninth thoracic disk space with associated epidural abscess; he underwent decompressive laminectomy. Pathological studies revealed acute inflammation; cultures were reported as yielding "diphtheroids." A CT-guided biopsy of the same area was performed 3 days later to better define the etiology of the infection, but cultures were negative for bacteria. He was treated with iv cefazolin for 6 days, followed by oral cephalexin for 5 weeks. While he was receiving antibiotics, his back pain gradually abated, and he progressed from using a wheelchair to using a walker, and finally to walking with a cane. However, the numbness in his lower extremities persisted.

Shortly after treatment with cephalaxin was discontinued, the patient developed increasing back discomfort and increasing numbness and decreasing strength in the lower extremities. He had been in good health until 2 1/2 months before admission, when he had developed back pain and leg numbness. Two weeks later, an MRI study performed elsewhere demonstrated an infection of the ninth thoracic disk space with associated epidural abscess; he underwent decompressive laminectomy. Pathological studies revealed acute inflammation; cultures were reported as yielding "diphtheroids." A CT-guided biopsy of the same area was performed 3 days later to better define the etiology of the infection, but cultures were negative for bacteria. He was treated with iv cefazolin for 6 days, followed by oral cephalexin for 5 weeks. While he was receiving antibiotics, his back pain gradually abated, and he progressed from using a wheelchair to using a walker, and finally to walking with a cane. However, the numbness in his lower extremities persisted.

Notably, the patient resided in Minnesota. He was a hunter and had traveled to Mexico for vacations every year; his last trip had been 10 months before his admission to our institution. He had glaucoma and hyporeflexia of the lower extremities and sensory loss below the tenth thoracic level. His WBC count was 10.9 × 10⁹, and his erythrocyte sedimentation rate was 85 mm/h. Cultures of blood and urine were negative.

An MRI study demonstrated osteomyelitis of the ninth and tenth thoracic vertebral bodies, diskitis of the ninth thoracic interspace, and an epidural abscess extending from the fifth to the eleventh thoracic vertebral bodies (this had progressed since MRI had been performed 2 months earlier). A myelogram demonstrated compression of the spinal cord at the level of the ninth and tenth thoracic vertebral bodies.

The patient underwent a revision posterior decompressive laminectomy of the eighth through eleventh thoracic vertebrae, with a transpedicular biopsy of the tenth thoracic vertebra body. Anterior decompression and debridement were not performed because of the need for transection of the segmental vascular supply to the spinal cord and the associated risk of further vascular insult. Purulent material was found in the epidural space and in the tenth thoracic vertebral body; pathological studies of specimens from these two areas demonstrated acute inflammatory cells and osteomyelitis, respectively. Cultures of specimens taken from the disk space, epidural space, and vertebral body yielded a gram-negative bacillus, which was identified by the Minnesota State Department of Health as S. boydii. The organism was susceptible to cefazolin, amikacin, gentamicin, mezlocillin, trimethoprim-sulfamethoxazole, imipenem, and ciprofloxacin, and it was resistant to ampicillin and chloramphenicol. Stool cultures were negative, but they were performed after the patient had received antimicrobial treatment.

He was braced in a thoracolumbar sacral orthosis and treated with cefazolin for 6 weeks. He continued to have mild weakness of the left leg postoperatively, with numbness of both lower extremities, and urinary retention; he remained hospitalized for 3 1/2 weeks while undergoing physical rehabilitation. At follow-up 5 months after admission to our institution, sensory and motor function of his lower extremities had improved.

Our patient represents the first reported case of vertebral osteomyelitis due to Shigella species. His only obvious exposure to the organism was his trip to Mexico; however, this trip had preceded his presentation by several months. The reason for the negative cultures (with the exception of "diphtheroids") of material obtained during his prior laminectomy is enigmatic; the possibility that this infection was acquired intraoperatively seems unlikely but cannot be excluded. The reason for his failure to respond to the initial treatment likely consists of a combination of inadequate length of effective intravenous antimicrobial therapy and inadequate debridement. We report, to our knowledge, the first case in which cefazolin was used to treat extraintestinal shigella infection.