A 64-Year-Old Man with Low Back Pain Due to Clostridium perfringens Lumbar Discitis

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Patient: Male, 64-year-old
Final Diagnosis: Clostridium perfringens infection
Symptoms: Lower back pain
Medication: —
Clinical Procedure: —
Specialty: Infectious Diseases • General and Internal Medicine

Objective: Unknown etiology
Background: Lumbar discitis caused by Clostridium perfringens is extremely rare. There have only been 7 published cases of confirmed discitis caused by Clostridium perfringens. We write this report to underscore this unusual relationship by discussing an additional case and providing a review of the previously published cases so clinicians can adequately evaluate and treat patients presenting with discitis.

Case Report: A 64-year-old morbidly obese man presented with an acute onset of worsening back pain and generalized weakness after incurring physical trauma related to falling. Additionally, he also developed fever and chills before the presentation. Based on the clinical presentation and elevated serum levels of inflammatory markers, magnetic resonance imaging was ordered, which showed L5–S1 discitis with extension of infection into the epidural space. Fluoroscopy-guided aspiration of the L5–S1 epidural space facilitated the detection of Clostridium perfringens as the involved pathogen. Based on the antibiotic susceptibility report, the patient was treated with intravenous ampicillin for 8 weeks, after which his symptoms resolved.

Conclusions: Diagnosis of discitis can be very challenging due to its ambiguous clinical presentation, especially in the elderly population due to the presence of underlying degenerative changes. Even though Clostridium perfringens remains a rare cause of lumbar discitis, it should be considered as a pathogen capable of causing infection of the vertebrae and intervertebral discs, thus allowing clinicians to make necessary diagnostic evaluations to provide appropriate targeted treatment to patients presenting with discitis.

MeSH Keywords: Clostridium perfringens • Discitis • Intervertebral Disc • Spine

Abbreviations: C. perfringens – Clostridium perfringens; ER – Emergency Room; ESR – erythrocyte sedimentation rate; CDC – Centers for Disease Control and Prevention; CT – computed tomography; IV – intravenous; MRI – magnetic resonance imaging

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/928014
Background

Clostridium perfringens is a spore-forming, anaerobic, gram-positive bacillus that is part of the normal intestinal flora. It can cause gas gangrene, cellulitis, and gastroenteritis. Anaerobic causes of discitis involving Clostridium perfringens bacteria are rarely seen. There are only 7 confirmed cases of discitis caused by C. perfringens published to date [1–7]. We report an unusual case of a 67-year-old man who presented with an acute onset of fever and worsening back pain and was discovered to have infectious L5–S1 discitis caused by C. perfringens. Along with our case report, we also provide a literature review with a comparison of the published cases of discitis caused by C. perfringens for a better understanding of this rare presentation.

Case Report

A 67-year-old man presented to the Emergency Department (ED) with complaints of generalized weakness for 4 days. He had repeatedly fallen at home 3 days prior to the arrival in the ED and he had a dull lower-back pain since then. The pain was controlled for a couple of days, but prior to his presentation to the ED, he noted that his pain was worsening. In addition, he noted generalized weakness without any lower-extremity weakness. He developed fever and chills at home but did not take his temperature. He did not report any productive cough, dysuria, hematuria, bowel/bladder incontinence, nausea, vomiting, diarrhea, headache, abdominal pain, or leg swelling. His past medical history was significant for hypothyroidism, diabetes mellitus type 2, morbid obesity (weight 140 kg), and gout. His medication list included levothyroxine, metformin, and allopurinol. His surgical history included bilateral rotator cuff surgeries, right hammertoes, bilateral cataracts status after extraction, and colonoscopy. Family history was significant for hypertension in his father. He had never smoked cigarettes and denied alcohol use or use of illicit substances. He was allergic to ciprofloxacin (reaction unknown).

On examination, the patient had mild tachypnea and was mildly febrile. His vital signs examination revealed blood pressure of 126/76 mmHg, heart rate of 104 beats per minute, temperature of 39°C (102.3°F), respiratory rate of 18 breaths per minute, and oxygen saturation of 94% on nasal cannula at 2 liters. His mucus membranes were slightly dry. There were no cracks or rhonchi on respiratory examination. He did not have any hepatosplenomegaly or abdominal tenderness on examination. An examination of his back revealed mild tenderness on palpation of the lumbosacral spine. He did not have tenderness on palpation of cervical or thoracic spines. He had no palpable paraspinous muscle spasm. Neurologically, sensation was intact to light touch in all lower extremities, but subjectively decreased in the feet. He had 5/5 strength in his bilateral extensor hallucis longus, peroneals, gastroc soleus complex, hamstrings, quadriceps, and iliopsoas. He had no pain with range of motion of the hips. He had negative straight leg raise bilaterally. He had bilateral down-going toes with Babinski testing. He had a slow, steady gait. He was alert, awake, and oriented to time, place, and person. A skin exam showed no lesions or rash. Results of the systemic examination were within normal limits otherwise.

Laboratory studies on presentation showed leukocytosis (17.2 k/ul with bands (17%), mild thrombocytopenia (158 k/ul), hyperglycemia (235 mg/dl), lactic acidosis (3.5 mmol/L), elevated D-dimer (635 ng/ml), elevated ESR (erythrocyte sedimentation rate – 50 mm/h), elevated CRP (C-reactive protein – 194 mg/L), and normal renal function (Table 1). Urine analysis did not suggest urinary tract infection. A chest X-ray did not show any abnormal findings. A computed tomography (CT) angiogram of the chest was done to rule out pulmonary embolism and it did not show any signs of pulmonary embolism or pneumonia, but showed bibasilar atelectasis. A lumbar spine X-ray showed lumbar spondylosis but no evidence of fracture.

Due to back pain and elevated inflammatory markers, a magnetic resonance imaging (MRI) of the lumbosacral spine with and without contrast was obtained. It showed abnormal thin enhancement within the L5–S1 disc contiguous with infiltrative enhancement in the epidural space of the lumbosacral junction, concerning for discitis with extension of infection into the epidural space. No large focal fluid collection was identified to suggest a drainable abscess formation. Degenerative disc disease was present (Figure 1). Blood cultures were obtained as well. He was started on IV ceftriaxone 2 g and IV vancomycin empirically based on the MRI findings.

The patient also underwent fluoroscopy-guided L5–S1 aspiration of the epidural space, and 2 mL of brown blood-tinged fluid was removed and sent for culture. The procedure was performed after starting the empiric antibiotics. An initial gram stain was negative. Meanwhile, the patient was continued on antibiotic and other supportive care. His back pain improved, and his fever also resolved. White cell counts gradually improved. Inflammatory markers also improved (Table 1). He slowly started working with physical therapy.

After 4 days, his spinal fluid cultures grew light growth of slow-growing anaerobic Clostridium perfringens (identified by a Microscan rapid anaerobic panel using biochemical properties). Based on the sensitivity report (Table 2), antibiotics were switched to IV ampicillin. His blood cultures showed no growth. Due to the identification of C. perfringens, we did a CT scan of the abdomen and pelvis with contrast to look for gastrointestinal causes, and it showed mild sigmoid diverticulosis but no masses, bowel thickening, or inflammation. On
Table 1. Laboratory data.

| Laboratory test               | Reference range | Day 1      | Day 8 (discharge day) |
|-------------------------------|-----------------|------------|-----------------------|
| White blood cell count        | 3.9–10.5 k/uL   | 17.2 k/uL | 6.3 k/uL              |
| Red blood cell count          | 4.33–5.73 M/uL  | 4.9 M/uL  | 4.5 M/uL              |
| Hemoglobin                    | 13.6–17.0 gm/dl | 14.3 gm/dl| 13.8 gm/dl            |
| Hematocrit                    | 40.0–54.0%      | 43.6%     | 40.6%                 |
| Platelet count                | 150–450 k/uL    | 158 k/uL  | 185 k/uL              |
| Prothrombin                   | 10.68–13.72 seconds | 13.1 seconds | 13.2 seconds         |
| INR                           | 1.1             | 1.1       | 1.1                   |
| D-dimer                       | 0–253.5 mg/ml   | 635 ng/ml | –                     |
| Sodium                        | 135–145 mmol/L  | 135 mmol/L| 137 mmol/L            |
| Potassium                     | 3.4–5.1 mmol/L  | 4.5 mmol/L| 4.3 mmol/L            |
| Chloride                      | 98–107 mmol/L   | 103 mmol/L| 102 mmol/L            |
| Carbon dioxide                | 22–30 mmol/L    | 21 mmol/L | 26 mmol/L             |
| Blood urea nitrogen           | 7–17 mg/dL      | 10 mg/dL  | 12 mg/dL              |
| Creatinine                    | 0.7–1.2 mg/dL   | 0.7 mg/dL | 0.8 mg/dL             |
| Glucose                       | 60–110 mg/dL    | 235 mg/dL | 120 mg/dL             |
| Lactic acid                   | 0.7–1.9 mmol/L  | 3.5 mmol/L| –                     |
| C-reactive protein            | 0.0–9.9 mg/L    | 194 mg/L  | 62 mg/L               |
| ESR                           | 0–25 mm/hr      | 50 mm/hr  | 19 mm/hr              |

Table 2. Antimicrobial susceptibilities of *C. perfringens*.

| Antibiotic                     | MIC (μg/ml) | Interpretation |
|--------------------------------|-------------|----------------|
| Amoxicillin/Clavulanate        | ≤0.5        | S              |
| Ampicillin                     | ≤0.5        | S              |
| Ampicillin/Sulbactam           | ≤0.5        | S              |
| Cefotetan                      | ≤1          | S              |
| Cefoxitin                      | ≤1          | S              |
| Chloramphenicol                | 4           | S              |
| Clindamycin                    | 1           | S              |
| Meropenem                      | ≤0.5        | S              |
| Metronidazole                  | 16          | R              |
| Penicillin                     | ≤0.06       | S              |
| Tetracycline                   | 1           | S              |
| Piperacillin/Tazobactam        | 0.5         | S              |

S – sensitive; I – intermediate; R – resistant; MIC – minimum inhibitory concentration; Drug susceptibility testing as per Clinical and Laboratory Standards Institute guidelines (CLSI M11).

Figure 1. MRI of lower lumbar spine demonstrating abnormal enhancement within the L5-S1 disc, consistent with discitis (blue arrow).
day 7, he underwent colonoscopy and had 3 sessile polyps of 3–5 mm removed from the transverse colon and the descending colon (Figure 2). A biopsy of these polyps identified them to be tubular adenomas. On recommendation of the infectious disease specialist and back surgeon, on the 8th day of the hospitalization, the patient was discharged to a skilled nursing facility for IV ampicillin therapy for 8 weeks, which resulted in clinical improvement.

Discussion

Clostridium perfringens is a spore-forming, anaerobic, gram-positive bacillus that is part of the normal intestinal flora of humans and animals [1]. According to the Centers for Disease Control and Prevention (CDC), C. perfringens is a leading cause of foodborne illness in the United States, causing nearly 1 million cases every year [8]. Ingesting a significant amount of this bacteria can generate sufficient toxin to cause illness, leading to an imbalance in the normal intestinal flora, which is a common pathological mechanism behind its foodborne illness presentation. However, endogenous organisms can also lead to endogenous infections in general when normal bacterial flora disseminate to a sterile part of the human body. This can occur when there is a break in the natural barrier between sterile and nonsterile environments of the body. C. perfringens is no exception to this method of pathogenesis. Gas gangrene, cellulitis, and gastroenteritis are common manifestations of its endogenous infections. However, C. perfringens causing discitis is a very rare phenomenon.

Only 7 confirmed cases of C. perfringens discitis have been published [1–7]. We present an additional case in this report, underscoring its potential to cause infection of the vertebrae and intervertebral discs. The main features of 6 of these published cases are outlined in Table 3. One commonality present among all the published cases is patient presentation with low back pain, whether acute or chronic, accompanied with elevated ESR and CRP levels.

Among the 7 reported cases, 2 had diverticular disease as a potential risk factor, and hematogenous dissemination of C.

| Authors            | Sex | Age (years) | Disc level | Risk factors | Presentation | Blood culture | Needle biopsy |
|--------------------|-----|-------------|------------|--------------|--------------|---------------|---------------|
| Lotte et al. [1]   | F   | 83          | L4/L5      | Nil gastrointestinal pathology on CT | Chronic lumbar back pain over 6 months | Negative | Positive |
| Caudron et al. [2] | F   | 79          | L4/L5      | Colonic diverticulosis | Low back pain over 2 months | Negative | Positive |
| Bednar [3]         | M   | 68          | L4/L5      | Laminotomy, discotomy | Acute back pain over 6 weeks | Positive | Positive |
| Pate and Katz [4]  | F   | 62          | L3/L4      | Diverticular disease | Low back pain over 6 weeks | Positive | Negative after 3 days of antibiotics |
| Beguiristain et al. [5] | M   | 33          | L1/L2      | No obvious risk factors | Low back pain over 7 days | Not completed | Positive |
| Seller et al. [6]  | M   | 64          | L5         | No obvious risk factors | Low back pain, urinary retention, and an inability to walk | Positive | Unknown |
| Yong and Lam [7]   | M   | 80          | T10/T11    | C. perfringens bacteremia | Back pain over 5 weeks | Positive | Negative |
perfringens from gastrointestinal tract to the lumbar spine was postulated as the probable cause of their discitis [2,4]. Yang et al. carried out a retrospective cohort study showing that the intraabdominal (52.7%) and lower respiratory tract (19.4%) were the leading sources of C. perfringens bacteremia [9]. The present case was an elderly patient without any significant immunocompromise and no concomitant infections. Diverticulosis was also discovered in this patient, with diagnostic workup completed after identification of C. perfringens on L5–S1 aspiration. Additionally, the 3 polyps removed were identified to be tubular adenomas, for which the patient was recommended to follow up with a gastroenterologist after discharge.

We postulate that our patient’s discitis likely originated from the gastrointestinal tract after infiltrating the vascular submucosal tissue, which led to its dissemination to the lumbar intervertebral disc area.

Our patient was on empiric IV ceftriaxone and IV vancomycin for 4 days until identification of C. perfringens, and the antibiotic regimen was then switched to targeted IV ampicillin. It is important to note, however, that antibiotic treatment was empirically started prior to the use of fine-needle biopsy to identify the involved pathogen. Additionally, there exists a small possibility of site contamination resulting from the process of needle biopsy itself, but this is very unlikely due to the sterile technique and substantial measures taken during the procedure to minimize the chances of contamination. It is possible that the initial broad empiric antibiotic treatment the patient received could have lowered the presence of C. perfringens at the site of infection, but IV ampicillin alone should be used in patients presenting with discitis due to C. perfringens [10]. A randomized controlled trial carried out by Bernard et al. provides evidence for continuing antibiotic treatment for 6 weeks for treating intervertebral osteomyelitis. This trial suggested that reduction of antibiotic treatment duration from 12 weeks to 6 weeks is not substandard when compared to 12 weeks [10]. Nonetheless, our patient continued to receive outpatient IV ampicillin therapy for 8 weeks, which resulted in resolution of the symptoms.

Additionally, while the presented case shares some similarities with the previously reported cases, it does contain several differences. For example, the time between onset of symptoms and adequate diagnosis was several weeks to months in the previous cases. Our case resulted in an adequate diagnosis in only 4 days. This shows the need for prompt diagnosis allowing for the use of targeted antibiotic therapy early in the course of the disease to improve morbidity. Not all previously reported cases identified gas in the lesions. Beguiristain’s and Yong’s case reports did not report lesions with gas. We believe that in our case the time between the onset and diagnosis was the key, and the diagnosis was made rather quickly before gas could form within the lesions.

Conclusions

Although C. perfringens is a rare cause of discitis, it should be considered as a pathogen capable of causing infection of the vertebrae and intervertebral discs. Only 7 confirmed cases have been previously published. Patients often present with complaints of back pain and may or may not have associated fever. Diagnosis of discitis can be very challenging because of vague clinical presentation, especially in the elderly due to pre-existing back pain related to degenerative changes of the spine. Laboratory workup can be significant for elevated ESR and CRP levels. MRI of the spine is the best imaging method, followed by the image-guided aspiration and culture. CT of the abdomen/pelvis along with colonoscopy should also be performed to identify any risk factors and/or potential portals of entry.

Treatment with IV ampicillin for 6 weeks appears to be the appropriate treatment. Through comparison of our case with previously published cases, we highlight the importance of thorough evaluation and prompt initiation of diagnostic measures in patients with back pain and discitis in order to start the necessary antibiotic therapy and limit the morbidity and mortality.

Conflict of interests

Non.

References:

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