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# Spondylodiscitis due to Klebsiella Pneumoniae: Case Report, Review of the Literature, and Diagnostic Reference

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**Background:** Diagnosing spondylodiscitis is challenging and associated with delay. The purpose of this publication is to reduce diagnostic latency of discitis and to provide a reference for diagnostic modalities to assist pain physicians when evaluating patients with nonspecific back pain.

**Case Report:** A 75-year-old man was admitted to the hospital 2 months post total hip arthroplasty owing to severe back and chest pain. Urine and blood cultures were positive for gram-negative bacteria. The patient was treated with antibiotics and referred to an outpatient pain clinic for the treatment of post laminectomy pain syndrome. Diagnostic images showed concern for discitis. Treatment methods led to the successful diagnosis and treatment of Klebsiella pneumoniae spondylodiscitis.

**Conclusion:** A limited number of K. pneumoniae discitis cases have been reported. Pain physicians must be weary of signs and symptoms associated with spondylodiscitis and implement the diagnostic tools when necessary. Earlier diagnosis and appropriate treatment will result in improved outcomes.

**Key words:** Spondylodiscitis, discitis, *Klebsiella pneumoniae*, spinal infection

## BACKGROUND

Spondylodiscitis is rare and occurs in 0.4 to 2.4/100,000 inhabitants per year and represents 3% to 5% of all cases of osteomyelitis (1,2). The condition mainly affects the lumbar spine (i.e., > 50% of all cases) by a microorganism infection that is transmitted via 1 of 3 pathways: (1) hematogenous spread from a distant infection focus, (2) direct external inoculation (e.g., invasive procedure), or (3) spread from adjacent tissues (3).

Hematogenous spread from a distant infection focus is the most common pathway for the development of spondylodiscitis (3). Distant focus sources include urinary tract, respiratory, and soft tissue infections. Monomicrobial infections (i.e., > 90% of cases) are routine with the most common bacterial pathogen being Staphylococcus aureus, which represents more than 50% of all nontuberculosis causes (2). Other bacteria associated with a lower incidence include Enterobacteriaceae (e.g., Enterobacter, Escherichia coli, Klebsiella, and Proteus), coagulase-negative staphylococci (i.e., Staphylococcus epidermidis) and streptococci (2,3). Spontaneous spondylodiscitis caused by K. pneumoniae is rare with only 10 cases reported in the literature, to our knowledge (4-13).

The diagnosis of spondylodiscitis is challenging (3,14). Nonspecific back pain (present in 90%–100% of cases) is the most common symptom; however, fever, neurologic deficits, and weight loss may also be present (1-3,14,15).

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Because of the nonspecific signs and symptoms, diagnosis is often delayed 1 to 6 months (1,14-16). This delay in diagnosis may lead to the progression of the infection (e.g., epidural abscess in 10%–20% of cases) (1,3,15,16).

We report a case of spontaneous K. pneumoniae spondylodiscitis diagnosed in a pain management practice. The patient was referred for interventional care for presumed worsening nonspecific low back pain associated with post laminectomy pain syndrome. The purpose of this publication was to urge pain physicians to keep spondylodiscitis in the differential diagnosis for nonspecific low back pain and to serve as a reference for the most effective diagnostic modalities. The patient consented and provided authorization to the publication of this report.

## CASE REPORT

A 75-year-old man presented to the hospital with increasing abdominal, back, and chest pain, episodes of vomiting, and anorexia. He lost over 35 pounds in the preceding 3 months. Past medical history was significant for chronic back pain, diabetes mellitus, and renal dysfunction. He denied fevers or chills and was afebrile on admission. Notable past surgeries included a lumbar laminectomy with fusion (40 years prior) and a left total hip arthroplasty (THA) performed 2 months prior to hospitalization. Laboratory evaluation demonstrated a normal white blood cell (WBC) count. Computed tomography (CT) of the abdomen and pelvis demonstrated no indication for abdominal pain; therefore cardiovascular causes were excluded. X-rays of the left hip, pelvis, and lumbar spine demonstrated advanced degenerative changes of the lumbar spine with no evidence of complication for the left THA. Blood and urine cultures were obtained. Gram-negative rods were found in the blood and urine cultures. The urine was positive for E. coli, and the patient was started on intravenous (IV) piperacillin/tazobactam. Blood cultures grew K. pneumoniae indicating concurrent bacteremia. The patient remained in the hospital for 5 days and improved. On discharge, he was started on cefuroxime for 10 days. The axial low back pain was assumed to be associated with chronic post laminectomy pain syndrome and advanced degenerative changes demonstrated on x-rays. The patient's primary care physician prescribed oxycodone and prednisone and referred the patient to a pain physician for interventional care.

Seven days following discharge the patient consulted with the pain physician. At the initial visit, previous radiographs of the lumbar spine, pelvis, and left hip were reviewed. The images showed good placement of the left hip, moderate arthritis of the right hip, and no indications for his increasing axial symptoms. A magnetic resonance imaging (MRI) scan of the lumbar region from one year prior was reviewed. These images showed no indication for his current increasing severity of low back pain. Due to a recent history of urinary tract infection and bacteremia with persistent and increasing low back pain, a new MRI study was ordered prior to prescribing a low back care pathway possibly including interventional procedures. The MRI with contrast medium (2 weeks after antibiotics were discontinued) demonstrated extensive marrow edema within the vertebral bodies of T12 and L1 with associated intervertebral disc abnormalities concerning for discitis (Fig. 1). Enhancement was seen in the paravertebral soft tissues and the T12-L1 extradural tissues with associated thecal sac impingement. Ordered blood work demonstrated a normal WBC (5.4 k/L), and an elevated erythrocyte sedimentation rate (ESR; 40 mm/h) and C-reactive protein (CRP; 60.4 mg/dL). Based on the findings of the MRI and blood tests there was concern for spondylodiscitis.

The patient was admitted to the hospital. Laboratory work displayed similar findings to the outpatient laboratory work; normal WBC count (6.4 k/L) remained with continued elevation of the ESR (50 mm/h) and CRP (60 mg/dL). A CT-guided disc biopsy was performed and identified K. pneumoniae. IV cefazolin antibiotic therapy was initiated. The patient was discharged following a 5-day hospital stay. On discharge, the patient was placed on IV ceftriaxone, which was intended to be continued at home for a minimum of 42 days with appropriate outpatient follow-up.

Eleven days after discharge, the patient returned to the hospital with worsening back pain. He remained in the hospital for 4 days. Updated blood and urine cultures showed no growth after 14 days. A new MRI scan with contrast medium was ordered demonstrating worsening spondylodiscitis and bilateral psoas abscesses. The patient's WBC count was normal; ESR and CRP levels remained elevated at 46 mm/h and 52 mg/dL, respectively. The psoas muscle abscesses were aspirated and cultured. The culture was negative for growth. The patient was discharged and remained in a skilled nursing home for 3 months and received oral ertapenem.

One year following diagnosis, the patient's condition improved with appropriate weight gain and he graduated from a wheeled walker to a cane for ambulation assistance. The patient weaned off opioid therapy.

## DISCUSSION

Spontaneous spondylodiscitis has an insidious onset. K. pneumoniae is infrequently the causative agent. The case presented here highlights the importance of maintaining spondylodiscitis in the differential diagnosis for individuals with chronic progressive low back pain with associated clinical risk factors for infection. Understanding the diagnostic spondylodiscitis workup and the interpretation of laboratory and radiologic findings is critical. The case presented here highlights the signs, symptoms, and diagnostic findings associated with K. pneumoniae spondylodiscitis.

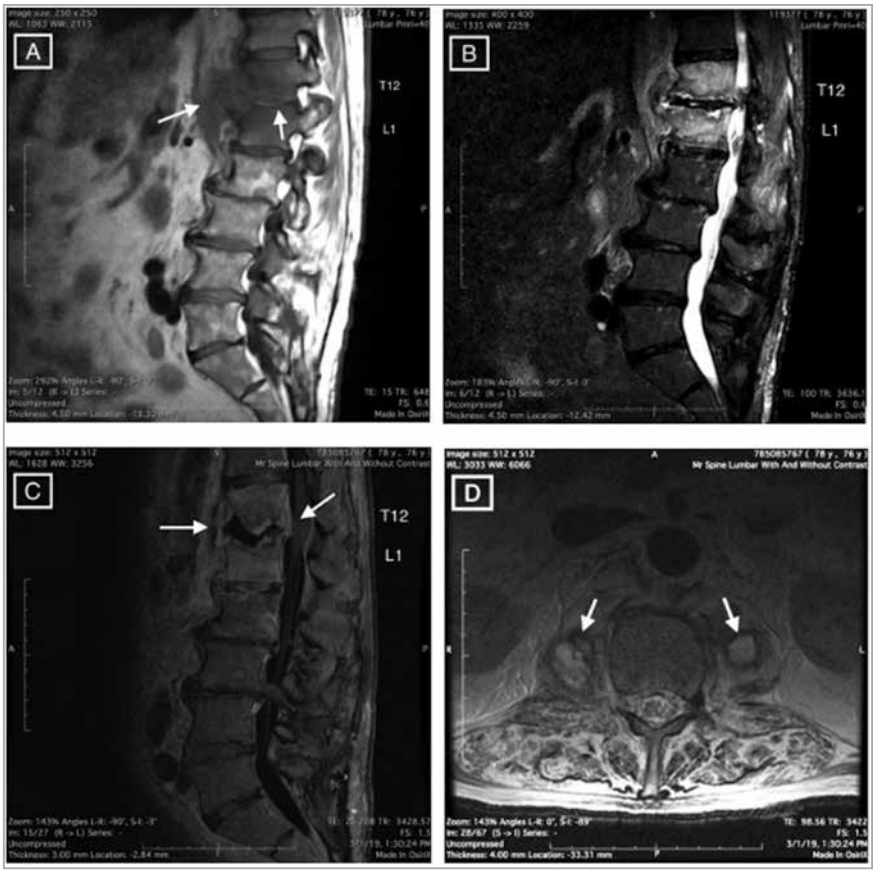


Fig. 1. Magnetic resonance imaging showing perpetuation of spondylodiscitis. Top images (A and B) are the initial outpatient images ordered 2 months prior to bottom images (C and D) obtained during the third hospital admission. (A) T1-weighted sagittal view: edema in soft tissue anterior to T12-L1 (left arrow) and edema in vertebral bodies; loss of intranuclear cleft (right arrow). (B) STIR sagittal view: high signal in disc; bright T12 and L1 vertebral bodies indicating edema; fluid accumulation visible in endplates. (C) T1-weighted sagittal view: endplates demineralized; enhancement of T12 and L1 vertebral bodies; disc completely filled with purulent fluid (left arrow); small epidural abscess (right arrow). (D) T2-weighted axial view: bilateral psoas abscesses (arrows); extensive paraspinous phlegmon.

Diagnosing spondylodiscitis relies on 3 clinical elements: clinical signs and symptoms, radiologic imaging, and laboratory tests (Table 1). An in-depth understanding of these 3 investigative components is essential to reduce the reported diagnostic latency of 1 to 6 months (3). In this case, the diagnosis was delayed by approximately 2 months. Clinical signs and symptoms with varying degrees of prevalence include back pain, fever, neurologic deficits, anorexia and associated weight loss, nausea, and vomiting (2,3,14,15). Fouquet et al (16) demonstrated that fever was most commonly present in cases of spondylodiscitis associated with septicemia (78%), followed by previous invasive spine procedures (33%), and spontaneous infectious causes (35%). Documented spondylodiscitis risk factors (excluding surgical procedures) include diabetes mellitus, renal dysfunction, inflammatory joint disease, alcohol abuse, and immunosuppression (1,3,16).

Radiologic imaging analysis consisting of x-ray, MRI, and CT performs a dynamic role in diagnosing spondylodiscitis. MRI has the highest level of sensitivity (93%–96%) and specificity (92.5%–97%) (1-3,14,15,17). Gadolinium contrast medium is recommended, particularly in the early stages of the disease (2). Repeat imaging may be required because only 70% of cases have positive MRI findings during the first 4 weeks of an infection (Table 1) (1). Although CT has lower specificity in comparison to MRI, it allows for observing bone destruction and is used when MRI is contraindicated (3,15). X-ray imaging has the lowest sensitivity. Later in the course of disease, plain radiography may demonstrate disc space narrowing with bony irregularities and erosion of adjacent vertebrae

Table 1. Diagnosing discitis.

| | Diagnostic Tool | Findings | Sensitivity | Specificity | Comments | References |
|---|---|---|---|---|---|---|
| **Radiographic Imaging** | X-ray | • Loss of definition of endplates<br>• Loss of disc height<br>• Loss of vertebral height<br>• Loss of bone density in later course of disease. | 82% | 57% | • Often an early screening test.<br>• False positives due to degenerative changes.<br>• In later stages non-specific reduction of the disc space occurs. | 2,3,15 |
| | MRI | • Loss of intervertebral disc cleft.<br>• With contrast, vertebral body, endplates, and disc space are enhanced.<br>• T1-weighted images show low signal in affected vertebral body and intervertebral disc.<br>• T2-weighted images show high signal in affected vertebral body and disc. | 93-96% | 92.5-97% | • Most accurate for imaging neural tissues and abscesses.<br>• Gadolinium contrast improves accuracy of MRI particularly in early stages of infection when other changes are subtle.<br>• Repeat imaging maybe needed. For 50% of cases findings are present within the first two weeks. At 4 weeks an additional 20% of cases have positive findings. | 1-3,14,15,17 |
| | CT With Contrast Media | • Disc changes appear as hypodense areas.<br>• Endplate demineralization or destruction visible with or without contrast. | 90% | No quantitative data found | • Used when MRI is contraindicated.<br>• Details of bone destruction as compared to other modalities.<br>• Epidural and paravertebral abscesses more easily visualized with contrast medium. | 1,3,15 |
| **Laboratory Tests** | CT-Guided Disc Biopsy / Aspiration | • Cultures may reveal specific causative agent. | 43-90% | 94% | • A repeat disc biopsy improves sensitivity; from 50% to 79%, respectively. | 2,14-16 |
| | Inflammatory Markers Elevated | | | | | |
| | WBC | • Increased | 36-65% | No quantitative data found | • WBC considered least sensitive inflammatory marker for discitis. | 2,3,14,15 |
| | ESR | • Increased | 72-100% | | • ESR may not return to baseline. | |
| | CRP | • Increased | 72-100% | | • CRP normalizes faster than ESR indicating improvement of condition with treatment. | |
| | Alkaline Phosphatase | • Increased | 50-62% | | • Elevated alkaline phosphatase levels associated with bone destruction | 2,3 |
| | Blood Cultures | • Blood and urine cultures may reveal specific causative agent. | 33-70% | No quantitative data found | • Blood cultures may be negative even when CT-guided disc biopsy is positive. | 2,3,14-16 |
| | Urine Cultures | | - | | • No quantitative data was found relating urine cultures to proper diagnosis of discitis. | - |
| **Clinical Signs and Symptoms** | Back Pain | • Localized back pain associated with the affected area. | 90-100% | No quantitative data found | • Non-specific signs and symptoms necessitate further methods of analysis by physician such as imaging and laboratory studies. | 1-3,14,15 |
| | Fever | • Temperature >37.5°C | 12.5-70% | | | |
| | Neurological Deficit | • Neurological deficits reported: Leg weakness, paralysis, sensory deficit, radiculopathy, and sphincter loss. | 10-50% | | | |
| | Weight Loss | • Significant loss of weight associated with loss of appetite. | 5-50% | | | |
| | Nausea and Vomiting | • Nausea, vomiting, and anorexia | 4.2-50% | | | |

[a]Abbreviations: MRI - Magnetic resonance imaging; CT = Computed tomography; WBC = White blood cells; ESR = Erythrocyte sedimentation rate; CRP = C-reactive proteins; "-" = Not determined

(16). As spondylodiscitis progresses, bone density may decrease with loss of trabeculation of adjacent vertebrae.

Image-guided disc biopsy is needed for definitive identification of the microorganism and for antibiotic selection. Repeating an image-guided disc biopsy should be considered if the first disc biopsy culture is negative. A repeat disc biopsy improves sensitivity, from 50% to 79%, respectively (2). Blood cultures and urinalysis may be effective for isolating the pathogen; however, positive blood cultures are only present in 33% to 70% of cases (2,3,14-16). In this case, blood cultures were positive for K. pneumoniae. Elevated inflammatory markers are common. Elevated WBC count has the lowest level of sensitivity and an elevation is only present in 36% to 58% of cases (2,3,14,15). ESR is elevated in 72% to 100% of cases and may not return to baseline following treatment (2,3,14,15). Importantly, it has been indicated that there is no relation between severity of infection and level of ESR increase (2). CRP levels are elevated in 72% to 100% of cases and normalize quickly during treatment (2,3,14,15). CRP levels are the most sensitive to determining treatment efficacy because they return to baseline more rapidly than other inflammatory markers (3). In this case, the patient's WBC was normal; however, other inflammatory markers were elevated at the time of diagnosis. For the presented case, the MRI scan ordered with the addition of the inflammatory markers assisted in the diagnosis of spondylodiscitis, which was confirmed by a CT-guided biopsy.

Treatment regimens for discitis range from nonoperative medical management to surgical intervention. Nonoperative methods include antibiotics with analgesics and bed rest or immobilization. Appropriate antibiotic choice is based on microorganism sensitivity (11). Reportedly, 33% to 77% of patients respond to conservative methods, although surgical options are enlisted in 10% to 27% of cases (11,14,15,17). Recurrence of infection within the first 6 months after antibiotic treatment may occur in 0% to 16% of patients (1). Recurrence is more common in immunocompromised individuals (1). The patient presented responded well to conservative antibiotic treatment coupled with analgesics, bed rest, and physiotherapy.

Controversy remains on the optimal duration of antibiotic usage, although a minimum duration of 6 weeks is recommended with confirmation of treatment success through normalization of CRP levels (18,19). Evidence suggests that antibiotic treatment lasting a minimum of 12 weeks is associated with the lowest recurrence rate (19,20). Because of the possibility of recurrence, interventional pain physicians should be cautious and confirm successful treatment prior to performing invasive procedures (e.g., epidural steroid injections, facet injections, spinal cord stimulator procedures). A review of 253 patients with spondylodiscitis demonstrated a 1-year cumulative relapse rate of 11.8% ± 2.1% (95% confidence interval [CI], 7.6–16.0) with 75% of these recurrences occurring within the first 12 months following the cessation of antibiotic treatment (21). Prior to progressing with interventional treatments, consultation with an infectious disease specialist and normalization of the CRP levels should occur. A follow-up MRI is also helpful, but it must be remembered that signal abnormalities may exist several months after successful recovery (20).

K. pneumoniae uncommonly infects the spine. Only 10 cases have been previously documented to our knowledge (Table 2), 2 of which were not in English and thus not included in the table (4-13). Identified risk factors for K. pneumoniae infections are similar to those of spondylodiscitis: diabetes mellitus, renal dysfunction, and immunosuppression. Specifically, the relative risk for K. pneumoniae bacteremia associated with underlying diabetes mellitus is 8.3 (95% CI, 6.8–10.2) (22). The patient presented here had diabetes mellitus, as did 5 out of the 8 (62.5%) other reported Klebsiella cases. K. pneumoniae is the second most common cause of gram-negative bloodstream infections within overall annual population incidence of 7.1 per 100,000 and an annual population mortality rate of 1.3 per 100,000 (22). Elderly patients are most commonly infected with a median patient age of 68.9 years (interquartile range, 53.0–79.3 years) (22). Discitis due to K. pneumoniae results from hematologic spread from a distant infection focus. Some 55% of the K. pneumoniae spondylodiscitis cases experienced anorexia, weight loss, and/or abdominal pain (Table 2) (1-3,14,15). The patient presented here demonstrated all of these symptoms.

## CONCLUSIONS

We describe a patient who developed a rare spontaneous K. pneumoniae pyogenic spondylodiscitis. Although spondylodiscitis occurs infrequently, the associated consequences are substantial and if left untreated result in significant morbidity. Discitis is accurately diagnosed using MRI, CT-guided disc biopsy, and laboratory tests. Pain physicians must be weary of common signs and symptoms associated with spondylodiscitis and implement the diagnostic tools when necessary, to aid in detection. Earlier treatment and appropriate therapy will result in improved outcomes.

Table 2. Preexisting case reports denoting discitis caused by klebsiella pneumoniae.

| Author/ Year | Causes | Age/ Gender | Diagnostic Latency | Presenting Symptoms | Spinal Region | WBC (x10^9/L) | ESR (mm/h) | CRP (mg/ dL) | Radiographical Findings | Treatment | Complications During Treatment | Patient Outcome | Notable Medical History |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Sugawa et al (4) 1989 | Not determined | 67/F | 1 month | • Fever (37.8°C)<br>• Productive cough<br>• Pain in adbomen and lumbar region | L3-L4 | 9.0 | 90 | - | • Radiograph revealed narrowing of intervertebtral space between L3-L4 and an irregular disc edge.<br>• CT showed destructive changes of the L3-L4 disc. | • Drainage of abscess<br>• Antibiotics: gentamicin<br>• Surgical intervention: Right axillobifemoral graft, and debridement of L3 and L4 lesions with bone transplantation. | • Unclear if the infectious spondylitis extended to the aorta or whether an infectious pseudoaneurysm secondarily infected the spine. | • The patient's postoperative course was uncomplicated, but no follow-up information is indicated. | • DM |
| Honan et al (5) 1996 | Most likely resulted from hemicolectomy. | 68/M | 2 months | • Progressive back pain<br>• Lower extremity weakness and paresthesias<br>• No fever or chills | L1-L2 | 13.8 | 96 | 12 | • Bone scan showed increased uptake at L1-L2.<br>• MRI showed narrowing of the L1-L2 disc space with endplate destruction. | • IV ceftriaxone for 6 weeks | None | • At follow-up exmination the patient had resolution of symptoms. | • Hypertension<br>• Stage D colon carcinoma<br>• Post right hemicolectomy |
| Kouroussis et al (6) 1999 | Unknown; Periodontitis speculated as causative agent. | 55/M | 6 months | •Thoracolumbar pain<br>• Paravertebral mass<br>• Anorexia<br>• Weight loss<br>• No fever or chills | T10-T11 | - | 80 | 5.5 | • CT revealed destruction of T10-T11 vertebrae and intervertebral disc. Paravertebral soft tissue mass extended from T9 to T11.<br>• CT-guided disc biopsy positive for K. pneumoniae. | • IV amikacin for 10 days<br>• Oral Ciprofloxacin for 8 weeks | None | • 10 days after initial treatment, significant back pain relief and ESR/ CRP levels decreased.<br>• Patient fully recovered 2 months after treatment. | • No underlying disease<br>• Oral hygiene was poor |
| Barton et al (7) 2008 | Not specifically determined but history of sepsis, multiple operations, Hickman line, and UTI's. | 63/F | 3 weeks or more | • 2 week of increasing neck pain, dysuria, and increasing urinary frequency. | Cervical; Level not specified | 4.6 | 95 | 4.4 | • MRI showed cervical discitis/ osteomyelitis with anterior paraspinal soft tissue mass and epidural collection. | • IV ceftriaxone; IV metronidazole<br>• Changed to IV imipenem<br>• Changed to IV meropenem<br>• Changed back to IV imipenem<br>• Changed to amikacin for two weeks until temocillin was available. | • First diagnosed as UTI and neck spasms<br>• Developed neutropenia twice | • After 7 weeks of temocillin patient had a complete resolution of symptoms.<br>• Patient remained asymptomatic 6 months later. | • Multiple episodes of sepsis.<br>• Hickman line<br>• Short bowel syndrome<br>• History of recurring UTI's<br>• Multiple GI surgeries<br>• Rectal stump abscess |

Table 2 con't. Preexisting case reports denoting discitis caused by klebsiella pneumoniae.

| Author/ Year | Causes | Age/ Gender | Diagnostic Latency | Presenting Symptoms | Spinal Region | WBC (x10^9/L) | ESR (mm/h) | CRP (mg/ dL) | Radiographical Findings | Treatment | Complications During Treatment | Patient Outcome | Notable Medical History |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Kosai et al (8) 2008 | Not determined | 72/F | 1 month | • Low back pain<br>• Fever (38.0°C) and chills<br>• Diabetic reticulopathy | L2-L3 | 2.9 | - | 28.9 | • Lumbar spine x-ray showed signs of degenerative changes.<br>• MRI revealed spondylitis lesions and narrowing of the L2-L3 disc space. | • IV meropenem for 4 weeks<br>• Oral levofloxacin | • None | • Discharged on 29th day | • DM2 |
| de Leon-Borras et al (9) 2018 | Possibly due to injuries sustained from car accident. | 36/M | Not specified | • Nausea and vomiting<br>• Abdominal pain<br>• Fever (39.9°C) | T12-L2 | - | - | - | • CT showed L1 Chance fracture with severe retropulsion and 50% decrease in vertebral body height.<br>• MRI confirmed L1-L2 discitis and pre-vertebral abscess causing spinal canal stenosis. | • Amikacin<br>• Changed to carbapenem and meropenem<br>• Changed to ceftazidime/ avibactam for 6 weeks | • Drug eruption secondary to carbapenem<br>• Bacteremia persisted for 6 weeks | • Full recovery after antibiotic treatment | • DM2<br>• Obesity<br>• Recent major car accident |
| Inagaki et al (10) 2019 | Possibly due to inflammation around fecalith in colon. | 90/M | 2 weeks | • Fever (39.4°C) and chills<br>• Stomach pain | T8-T9 | 10.1 | - | 0.63 | • CT showed preexisting fecalith and vertebral destruction at T8 and T9.<br>• MRI showed signal intensity at T8-T9 disc space and vertebrae. | • Piperacillin/ tazobactam<br>• Changed to ceftriaxone<br>• Changed to cefmetazole<br>• Changed to moxifloxacin | • Fever returned at day 9 of treatment. | • Patient transferred to rehabilitation hospital on day 64.<br>• No further information provided. | • DM |
| Wakabayashi et al (11) 2020 | Sepsis due to invasive liver abscess. | 65/M | Not specified | • Fever (40.5°C)<br>• Back pain<br>• Loss of appetite<br>• Tachycardia (115/min) | L3-L4 | 6.95 | - | 23.33 | • CT revealed worsening spondylodiscitis and bilateral iliopsoas abscesses.<br>• MRI showed L3 and L4 vertebral destruction, L3-L4 canal stenosis | • Piperacillin/ tazobactam<br>• Changed to amoxicillin/ clavulanate<br>• L3-L5 laminectomy | • Discitis worsened despite improvement of inflammatory response and liver abscess. | • Back pain was relieved after laminectomy and patient discharged on the 85th day.<br>• No recurring infection afterward | • DM<br>• Liver dysfunction |

[a]Note: All lab work listed is from time of admittance and prior to treatment

[b]Abbreviations: WBC = white blood cells, ESR = Erythrocyte sedimentation rate, CRP = C-reactive proteins, DM = Diabetes mellitus, UTI = Urinary tract infection, "-" = Not determined

## Author Contributions

DP was involved in the drafting and review of all aspects of this manuscript; JH was involved in the drafting and review of all aspects of this manuscript; LD aided in the revision of the manuscript, interpretation of diagnostic images, and creation of the figures. All authors have read, critically revised, and approved the entirety of the manuscript.

## REFERENCES


1. Cottle L, Riordan T. Infectious spondylodiscitis. *J Infect* 2008; 56:401-412.
2. Gouliouris T, Aliyu SH, Brown NM. Spondylodiscitis: Update on diagnosis and management. *J Antimicrob Chemother* 2010; 65(Suppl 3):iii11-24.
3. Petkova AS, Zhelyazkov CB, Kitov BD. Spontaneous spondylodiscitis--Epidemiology, clinical features, diagnosis and treatment. *Folia Med (Plovdiv)* 2017; 59:254-260.
4. Sugawa M, Tanaka R, Nakamura M, et al. A case of infectious pseudoaneurysm of the abdominal aorta associated with infectious spondylitis due to Klebsiella pneumoniae. *Jpn J Med* 1989; 28:402-405.
5. Honan M, White GW, Eisenberg GM. Spontaneous infectious discitis in adults. *Am J Med* 1996; 100:85-89.
6. Kouroussis C, Georgoulias V, Souglakos J, Simvoulakis E, Karabekios S, Samonis G. Spontaneous spondylodiscitis caused by Klebsiella pneumoniae. *Infection* 1999; 27:368-369.
7. Barton E, Flanagan P, Hill S. Spinal infection caused by ESBL-producing Klebsiella pneumoniae treated with temocillin. *J Infect* 2008; 57:347-349.
8. Kosai K, Seki M, Yanagihara K, et al. Spontaneous pyogenic spondylitis caused by Klebsiella pneumoniae. *Intern Med* 2008; 47:1121-1124.
9. De León-Borrás R, Álvarez-Cardona J, Vidal JA, Guiot HM. Ceftazidime/avibactam for refractory bacteremia, vertebral diskitis/osteomyelitis with pre-vertebral abscess and bilateral psoas pyomyositis secondary to Klebsiella pneumoniae carbapenemase-producing bacteria (KPC). *P R Health J* 2018; 37:128-131.
10. Inagaki A, Moriyama Y, Morioka S, Wakimoto Y, Shida Y, Ohmagari N. A case of Klebsiella pneumoniae spondylitis and bacteremia potentially due to inflammation around a fecalith. *J Infect Chemother* 2019; 25:470-472.
11. Wakabayashi SI, Kimura T, Tanaka N, et al. Invasive liver abscess syndrome accompanied by spondylodiscitis: A case report and review of the literature. *Clin J Gastroenterol* 2020; 13:927-934.
12. Gómez Rodríguez N, Penelas-Cortés Bellas Y, Ibáñez Ruán J, González Pérez M, Sánchez Lorenzo ML. [Infectious spondylodiscitis in a health area of Galicia (Spain) from 1983-2003]. *An Med Interna* 2004; 21:533-539.
13. Porras JA, Bayona C, Gutiérrez MC, Vidal F. [Vertebral osteomyelitis due to Klebsiella pneumoniae]. *An Med Interna* 1994; 11:154-155.
14. Waheed G, Soliman MAR, Ali AM, Aly MH. Spontaneous spondylodiscitis: Review, incidence, management, and clinical outcome in 44 patients. *Neurosurg Focus* 2019; 46:E10.
15. Butler JS, Shelly MJ, Timlin M, Powderly WG, O'Byrne JM. Nontuberculous pyogenic spinal infection in adults: A 12-year experience from a tertiary referral center. *Spine (Phila Pa 1976)* 2006; 31:2695-2700.
16. Fouquet B, Goupille P, Gobert F, Cotty P, Roulot B, Valat JP. Infectious discitis diagnostic contribution of laboratory tests and percutaneous discovertebral biopsy. *Rev Rhum Engl Ed* 1996; 63:24-29.
17. Cebrián Parra JL, Saez-Arenillas Martín A, Urda Martínez-Aedo AL, Soler Ivañez I, Agreda E, Lopez-Duran Stern L. Management of infectious discitis. Outcome in one hundred and eight patients in a university hospital. *Int Orthop* 2012; 36:239-244.
18. Lener S, Hartmann S, Barbagallo GMV, Certo F, Thomé C, Tschugg A. Management of spinal infection: A review of the literature. *Acta Neurochir (Wien)* 2018; 160:487-496.
19. Aljawadi A, Jahangir N, Jeelani A, et al. Management of pyogenic spinal infection, review of literature. *J Orthop* 2019; 16:508-512.
20. Grados F, Lescure FX, Senneville E, Flipo RM, Schmit JL, Fardellone P. Suggestions for managing pyogenic (non-tuberculous) discitis in adults. *Joint Bone Spine* 2007; 74:133-139.
21. McHenry MC, Easley KA, Locker GA. Vertebral osteomyelitis: Long-term outcome for 253 patients from 7 Cleveland-area hospitals. *Clin Infect Dis* 2002; 34:1342-1350.
22. Meatherall BL, Gregson D, Ross T, Pitout JD, Laupland KB. Incidence, risk factors, and outcomes of Klebsiella pneumoniae bacteremia. *Am J Med* 2009; 122:866-873.