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Pyogenic spondylitis due to *Streptococcus agalactiae* with paraspinal abscess and vertebral destruction in a diabetic patient: time course of imagings

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Summary

A 76-year-old female with type 2 diabetes mellitus presented with hematuria, low back pain, and intermittent fever for 7 days. She was admitted to our hospital and diagnosed with *Streptococcus agalactiae* (GBS) bacteremia. CT showed an air density within the right iliopsoas muscle, and an MRI of the spine revealed hyperintensity in the right half of the L1–L2 intervertebral disk, leading to the diagnosis of a paraspinal abscess and L1–L2 pyogenic spondylitis. Antibiotic therapy was started and the clinical symptoms, as well as serologic biomarkers and radiologic images of the paraspinal abscess, were improved. The therapy was stopped on day 72 despite vertebral destruction progression. Vertebral endplate ossification was observed on day 108, and further bone formation was noted on day 177. Our case study with radiologic findings over 6 months demonstrated how bone destruction with pyogenic spondylitis, which had been treated with antibiotic therapy, improved after cessation of antibiotics.

Learning points

- Although GBS is a rare cause of spondylitis, diabetic mellitus is a risk factor for the development of invasive GBS infections, especially under poor glycemic control.
- Bone destruction of pyogenic spondylitis can improve after discontinuation of antibiotic therapy.
- It may be important to decide the period of antibiotic therapy based on clinical conditions, serologic biomarkers, and soft tissue findings rather than bone findings.
- When elderly diabetic patients present with back pain and fever, spondylitis should be considered in the differential diagnosis to avoid potential diagnostic delays or misdiagnosis.

Background

Pyogenic spondylitis (PS) is an uncommon clinical entity and is difficult to diagnose; however, its prevalence has been increasing, most likely due to the increase in the number of elderly and MRI-facilitated diagnoses (1). Diabetes mellitus is known to be a risk factor for PS (2). It is also a risk factor for *Streptococcus agalactiae* (GBS) infection (3), which has been conventionally considered to be an uncommon pathogen of spondylitis.

The clinical features of PS are not fully understood because there are few reports on the long-term course of PS. In addition, the appropriate treatment approaches, including the duration of antibiotics, are still controversial. Herein, we present a case of PS due to GBS complicated by a paraspinal abscess and vertebral destruction with detailed longitudinal follow-up imaging findings over time.

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Case presentation

The patient was a 76-year-old female who had a 25-year history of type 2 diabetes mellitus. She developed diabetic chorea 10 years before. She was prescribed medications, including linagliptin, 15 (6–6–3) units of insulin aspart and 18 units of insulin glargine but had poor glycemic control with an HbA1c level of approximately 9%. She was admitted to our hospital with complaints of hematuria, lower back pain, and intermittent episodes of fever for the preceding 7 days. On admission, the vital signs were as follows: body temperature, 38.2°C; heart rate, 94 bpm; blood pressure, 148/92 mmHg; and respiratory rate, 24 b.p.m. Severe tenderness of the lower back, including the costovertebral angle, left sternoclavicular joint, and right ilium, was present and made walking difficult. Although she had a left paresis due to a lacunar cerebral infarction that developed 7 years prior, no novel neurologic impairment was observed.

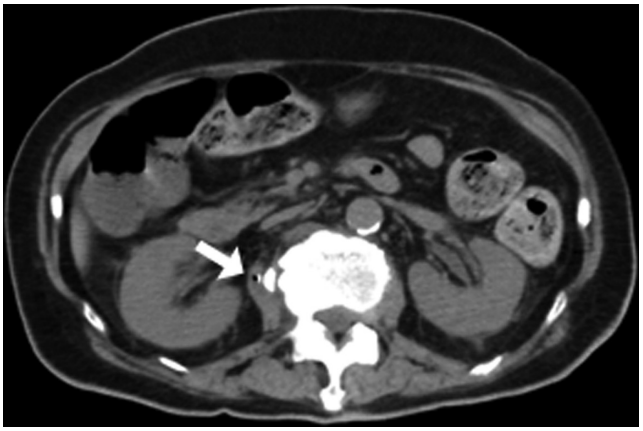

Figure 1
CT scan of the abdomen on admission showing air density (white arrow) and an increased concentration of adipose tissue inside the right iliopsoas muscle, suggestive of an abscess.

Investigation

The initial hematologic work-up showed a total white blood cell (WBC) count of 23 900/μL, with 84.0% neutrophils, a C-reactive protein (CRP) of 18.49 mg/dL, and a procalcitonin level of 0.8 ng/mL. The urine dipstick was positive for protein, blood, and leucocytes. Furthermore, the HbA1c of 8.4% and a random blood glucose level of 347 mg/dL suggested poor glycemic control over the preceding months. The rheumatoid factor and T-spot were negative. Blood cultures grew GBS and urine cultures were negative. She underwent CT to localize the source of infection, which showed air density and increased concentration of adipose tissue inside the right iliopsoas muscle, suggesting the presence of an abscess around the right iliopsoas muscle (Fig. 1). There were no abnormal findings in the lumbar area on the CT at that time (Fig. 2A). Thereafter, she underwent an MRI of the spine, which revealed hyperintensity in the right half of the L1–L2 intervertebral disk and right paravertebral muscle on the short tau inversion recovery (STIR) sequence, suggesting discitis and a paraspinal abscess (Fig. 3A). Furthermore, swelling of the kidneys and an area of hyperintensity around the kidneys were observed, suggesting the presence of pyelonephritis (Fig. 3A). There was no evidence of valvular vegetations on transthoracic echocardiogram.

Treatment

Initial treatment with i.v. cefazolin (3 g/day) was started for bacteremia of unknown origin. The patient also received an analgesic and s.c. insulin infusion according to the blood glucose level. Because blood cultures grew GBS, cefazolin was changed to i.v. ampicillin (8 g/day) on day 2 based on the sensitivity report and continued for 8 weeks. Ampicillin was subsequently changed to oral clindamycin (0.6 g/day) due to eosinophilia. Percutaneous abscess drainage was not

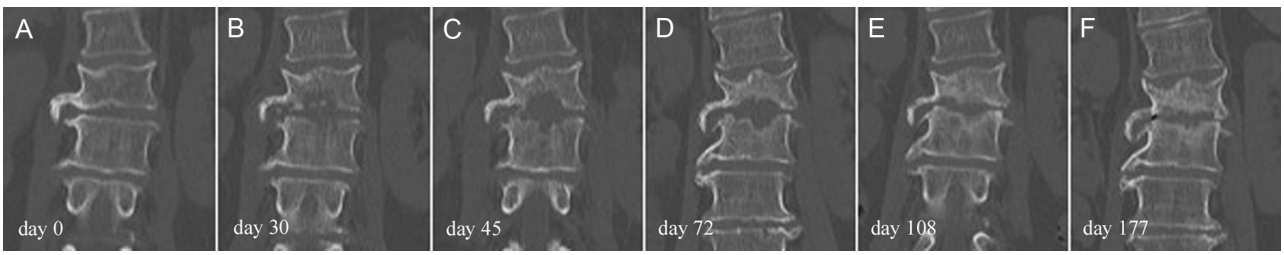

Figure 2
CT scan of lumbosacral spine showed no abnormal findings on admission (A). The destruction of L1–L2 endplates and subsequent severe bone destruction of L1–L2 vertebral body were observed on days 30 (B) and 45 (C), respectively. No further bone destruction of the L1 vertebral body was observed, while L2 bone destruction was slightly exacerbated on day 72 (D). The ossification of the L1–L2 endplates was demonstrated on day 108 (E). Further bone formation was recognized on day 177 (F).

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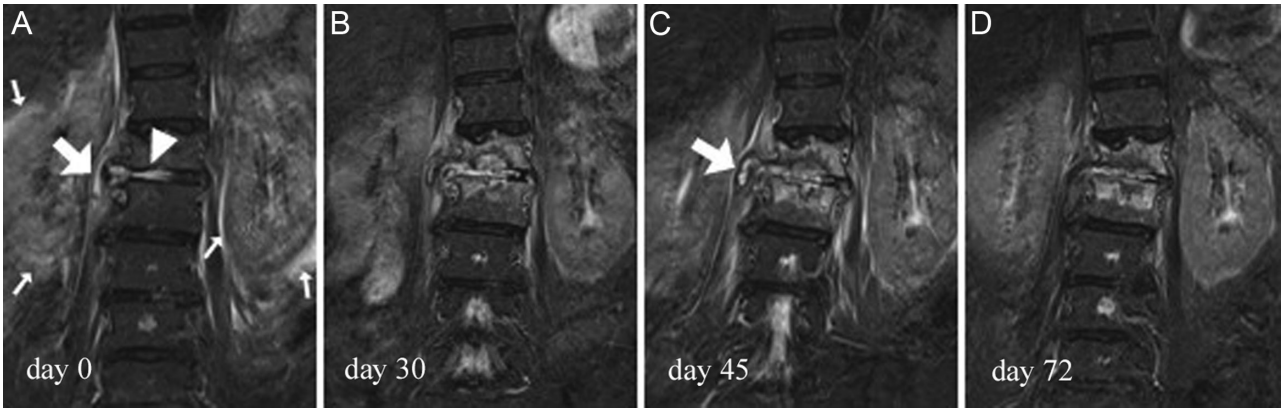

Figure 3
MRI of lumbosacral spine in short tau inversion recovery (STIR) sequence showed hyperintensity in the right half of the L1–L2 intervertebral disk (white arrow head) and right paravertebral muscle (white thick arrow), suggesting discitis and paraspinal abscess on admission (A). A hyperintensity area around the bilateral kidneys was also observed (white thin arrows), suggesting the presence of pyelonephritis (A). Expansion of the L1–L2 vertebral bodies hyperintensity area was demonstrated on day 30, suggesting progression to spondylodiscitis (B). The L1–L2 vertebral bodies hyperintensity area and paraspinal abscess (white thick arrow) were further exacerbated on day 45 (C). Improvement in the size of the paraspinal abscess and no further expansion of the L1–L2 vertebral bodies hyperintensity area were observed on day 72 (D).

undertaken because the paraspinal abscess in this case was too small.

Outcome and follow-up

The patient defervesced on day 3 and her blood cultures were negative on day 8. The WBC count normalized and the serum CRP decreased to 7.34 mg/dL on day 20 (Fig. 4). While the back pain and serologic tests improved, a repeat CT and MRI on day 30 showed destruction of the L1–L2 endplates (Fig. 2B) and expansion of the hyperintensity area on the STIR sequence of the L1–L2 vertebral bodies (Fig. 3B), suggesting exacerbation from discitis to spondylodiscitis. The serum CRP further improved to 3–4 mg/dL with conservative treatment of antibiotics and bed rest (Fig. 4); however, destruction of the L2 vertebral body was exacerbated on day 45 (Figs. 2C and 3C). External fixation with a corset was prescribed. On day 72, when destruction of the L2 vertebral body was slightly exacerbated (Fig. 2D), antibiotic administration was discontinued based on the clinical conditions and improvement in the serum CRP level (Fig. 4) and the paraspinal abscess (Fig. 3D). During the course of treatment, her diabetic status was under control, requiring a total of approximately 30 units of insulin per day. On day 84, i.v. cefmetazole ((CMZ), 6 g/day) was started because the serum CRP and hepatobiliary enzyme levels were elevated. Infectious cholangitis was excluded by endoscopic retrograde cholangiopancreatography findings, and CMZ was discontinued on day 90 (Fig. 4). The serum CRP level decreased to 0.78 mg/dL and a CT revealed L1–L2 endplate ossification on day 108 (Figs. 2E and 4). She was transferred to a rehabilitation hospital on day 113. On day 177, a CT revealed further vertebral bone formation (Fig. 2F). As of day 300, she had no relapses or sequelae.

Discussion

GBS has been recognized mainly as a pathogen in neonates and peripartum females; however, the incidence of invasive infections caused by GBS has increased in recent years in non-pregnant adults, especially patients with diabetes or immunosuppressive agents (4). It has been reported that

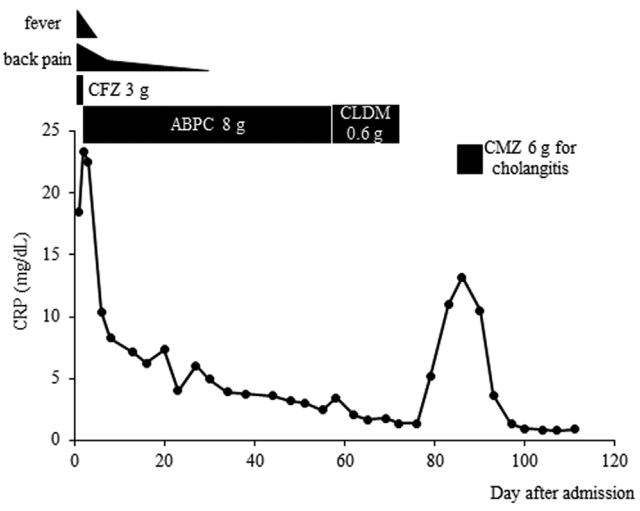

Figure 4
Time course changes of clinical symptoms, antibiotic therapy, and serum CRP after admission. CFZ, cefazolin; ABPC, ampicillin; CLDM, clindamycin; CMZ, cefmetazole

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diabetes of longer duration and poor glycemic control are risk factors for GBS bacteremia (5). The source of primary infection is difficult to identify in many cases of GBS infections. In the present case, although urine cultures were negative, we reasoned that the primary infection lesion was pyelonephritis for the following reasons: specific symptoms such as hematuria were observed before admission; GBS is a possible pathogen in genitourinary tract infections; and MRI showed findings suggestive of pyelonephritis.

For the treatment of PS, the Infectious Diseases Society of America guidelines recommend 6 weeks of antibiotics to be adequate (6); however, the duration of antibiotics differs from patient to patient. Roblot *et al.* (7) reported that short-duration (≦ 6 weeks) antibiotics did not enhance the risk of spinal infection relapse. In contrast, the risk of relapse was higher when antibiotics were <12 weeks in another study (8). In real-world practice, the duration of antibiotics should be determined based on clinical findings, as well as laboratory data and radiologic images. In the present case, the duration of antibiotics was approximately 10 weeks, which was longer than the one suggested by the above guideline. When we decided to discontinue the antibiotics, the L2 vertebral destruction still exacerbated; however, it has been suggested that bone findings take more time to be normalized than soft tissue findings (9), and soft tissue findings may correlate better with therapeutic response (6). A prospective study involving 29 patients with bacterial spondylodiscitis reported that vertebral destruction persisted at 6 months despite a favorable response to antibiotics (10), indicating the discrepancy in the time course between infection and vertebral destruction. Thus, it may be important to determine the period of antibiotics based on clinical conditions, serologic biomarkers, and soft tissue findings, but not on bone findings, to avoid unnecessary treatment.

To the best of our knowledge, this is a very unique case report in which detailed radiologic findings were followed over time, leading to an understanding of the clinical features of PS. Spondylitis is a rare but life-threatening disease. However, spondylitis is often diagnosed late or misdiagnosed because the symptoms are non-specific. Indeed, it is sometimes difficult to distinguish between early PS and modic type 1 degenerative endplate changes, which are commonly associated with low back pain, because they both show similar MRI findings (11). Moreover, modic changes are recently reported to be associated with diabetes mellitus (12).

In conclusion, when diabetic patients present with back pain and fever, PS should be considered in the differential diagnosis.

Declaration of interest
The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of the research reported.

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Patient consent
Written informed consent for publication of their clinical details and/or clinical images was obtained from the patient.

Author contribution statement
TK and HA wrote the manuscript. All authors treated patients, discussed the data, and were involved in revising the manuscript.

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