



## Community Acquired Spondylodiscitis Caused by Escherichia Coli; Case Report and Literature Review

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### ABSTRACT

Vertebral osteomyelitis, or spondylodiscitis, is a rare disease with increasing prevalence in recent years due to a greater number of spinal surgical procedures, nosocomial bacteraemia, an aging population and intravenous drug addiction. Haematogenous infection is the most common cause of spondylodiscitis. We report a 47-year-old man diagnosed with *Escherichia coli* spondylodiscitis. The patient initially presented with a 4-day history of inflammatory, mechanical pain in the lower back suggesting sciatica. Treatment included NSAIDs and opioids. Two days after discharge from hospital following an admission due to an upper GI bleeding, the back pain intensified, precipitating a new attendance to the emergency department; during which lumbosacral radiography showed marked reduction of L2/L3 intervertebral space. After a new admission to the rheumatology unit due to worsening of symptoms and raised inflammatory markers, an expedited MRI showed loss of intervertebral disc space at L2/L3, with an irregular high intensity area at L2; suggesting a fluid collection extending to adjacent soft tissues. Fluoroscopy-guided core needle bone biopsies were reported positive for *Escherichia coli* sensitive to ceftriaxone. The patient was treated (received treatment) with a three week course of ceftriaxone following a formal diagnosis of *E. coli* spondylodiscitis. Follow-up MRI demonstrated complete recovery with the patient able to return (has returned) to normal activity. In this case we highlight the importance of correct and timely diagnosis of spondylodiscitis. Diagnosis of spondylodiscitis is often difficult, delayed or even missed due to the rarity of the disease but can lead to devastating consequences. Therefore a high index of suspicion is needed for prompt diagnosis to ensure improved long-term outcomes.

**Keywords:** Spondylodiscitis; *Escheria coli*; Back pain.

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## Introduction

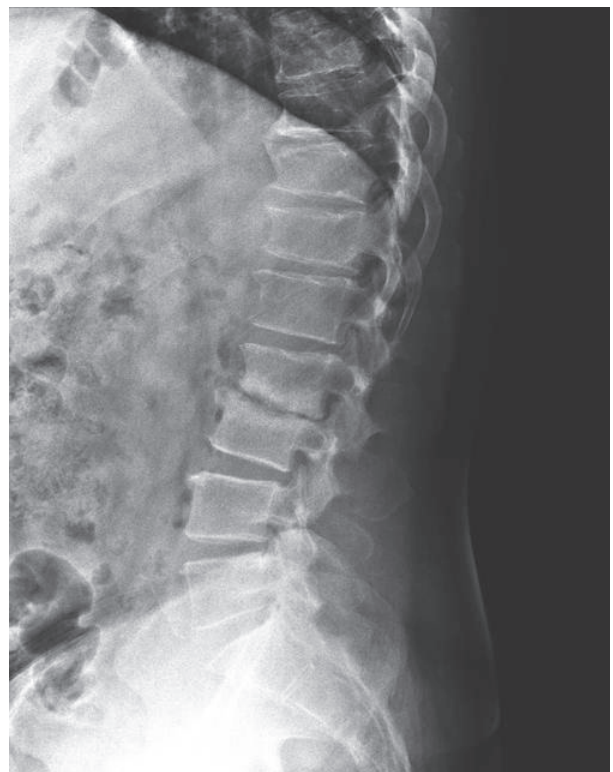
Spondylodiscitis is a diagnosis rarely made in the community and in emergency medicine. Although neck pain and back pain are common presentations in both these settings, osteomyelitis is usually not amongst the forefront of differential diagnoses. The incidence of spondylodiscitis has been estimated at 2.4 cases per 100,000 population, with this incidence increasing with age (from 0.3 per 100,000 in the <20 years age group to 6.5 per 100,000 in the over 70 years group) [1]. As such, the condition often begins insidiously, with progression occurring over weeks to months; thus generally delaying diagnosis [2]. The frequency of diagnosing spondylodiscitis has increased due to a rise in nosocomial bacteraemia causing it (usually related to intravascular access or instrumentation, amongst others), increases in the immunocompromised adult population and also better access to highly sensitive imaging, such as magnetic resonance imaging (MRI). Most cases are caused by haematogenous spread of infection from distant sites rather than by propagation from adjacent tissues and due to the invasive pathogens [3]. In most patients diagnosed with spondylodiscitis, appropriate antimicrobial therapy leads to complete recovery and good prognosis. Ideally, antimicrobial therapy should be guided by Microscopy, Culture & Sensitivity of the isolated pathogen. Surgery however is required in a minority of patients [4]. The aim of this article is to highlight how a diagnosis of spondylodiscitis can be delayed even with the input of multiple appropriate specialties.

## Case Report

A 47-year-old male furniture factory worker presented to the emergency department on 1 July, 2014 with a two-month history of back pain. He had been taking regular paracetamol and non-steroidal anti-inflammatory drugs (NSAIDs), which provided only suboptimal relief. The only past medical history of note was (included a history) of mild sciatica. The patient lives in a rural area and has no pets. There is no history of contact with cattle, intake of unpasteurised milk and cheese nor consumption of water from a public supply. The patient described back pain of mixed character (inflammatory-mechanical) with pain and tenderness over the sacroiliac joint and lumbar spine, radiating to the left lower extremities and associated with a reduction in the range of movement in the lumbar spine. Also alternating buttock pain which worsens with movement and improves with rest. There was no loss of peripheral or perianal sensation, weakness nor urinary incontinence. The patient described his current symptoms as typical of his usual sciatica, however with persistent pain despite taking his usual analgesia. The patient was admitted to the observation ward for pain control that was

satisfactorily achieved with administration of opioids, before discharge home the next day with community follow-up.

Two days after discharge from Hospital (following an admission due to NSAID related upper GI bleeding and cannula-induced phlebitis in his left arm), his back pain recurred with similar features to his initial presentation. Patient was commenced on tramadol, paracetamol, and corticosteroids by his general practitioner. Unfortunately, his back pain did not improve and two weeks later he re-attended the emergency department. On examination, the patient again had no saddle/perineal hypoesthesia or anaesthesia, neither any bowel or bladder disturbances nor symptoms suggestive of cauda equina. During this admission, plain film lateral lumbar spine X-rays (Figure 1) showed marked reduction of L2/L3 intervertebral space associated with significant irregularity of the endplates and also vertebral body sclerosis. The on-call orthopaedic surgeon recommended outpatient follow-up with MRI and to continue a decreasing steroid regimen, and the patient was discharged once again. One week later, the patient developed moderate to severe back pain, which was now only temporarily relieved by opioids and muscle relaxants. The general practitioner requested blood tests and a bone scan. Blood tests showed raised inflammatory markers including CRP. Subsequently, the patient was admitted once again to the rheumatology unit three weeks later, where initial investigations revealed a normal abdominal ultrasound scan and otherwise normal bloods. A



**Fig. 1.** Lateral radiograph of the lumbosacral spine with marked reduction of L2/L3 intervertebral space associated with marked irregularity of the endplates and vertebral body sclerosis.

three-phase bone scan showed suspicious findings, with increased uptake of radiotracer in the vertebral bodies of L2 and L3 adjacent to the disc. The early phase of the scan showed increased vascularity indicative of inflammation. These findings are consistent with a diagnosis of spondylodiscitis in L2/L3. CT (Figure 2) of the lumbar spine showed irregularity of the disc space and vertebral endplates in the L2/L3 interspace but no definitive diagnosis. MRI (Figure 3 and 4) showed irregular high intensity area at L2 suggesting a fluid collection extending to adjacent soft tissues. Fluoroscopy-guided core needle bone biopsies were reported positive for *Escherichia coli* sensitive to ceftriaxone. Appropriate Treatment was started immediately with a dramatic improvement in patient symptoms. The patient was discharged with 2 g of ceftriaxone IM for 3 weeks, before switch to oral ciprofloxacin 750 mg 12 hourly for 3 weeks. After 6 weeks the patient was asymptomatic with normalisation of CRP and ESR. Follow-up MRI at 6 weeks showed improvement of the intervertebral disc signal and almost complete resolution of the fluid collection. A further MRI at 12 months indeed showed complete resolution of the infection. In conclusion, a final diagnosis of *E. coli* spondylodiscitis in L2 and L3 was made and successfully treated, and the patient remains well and asymptomatic at the last visit.

## Discussion

Spondylodiscitis, or vertebral osteomyelitis, is

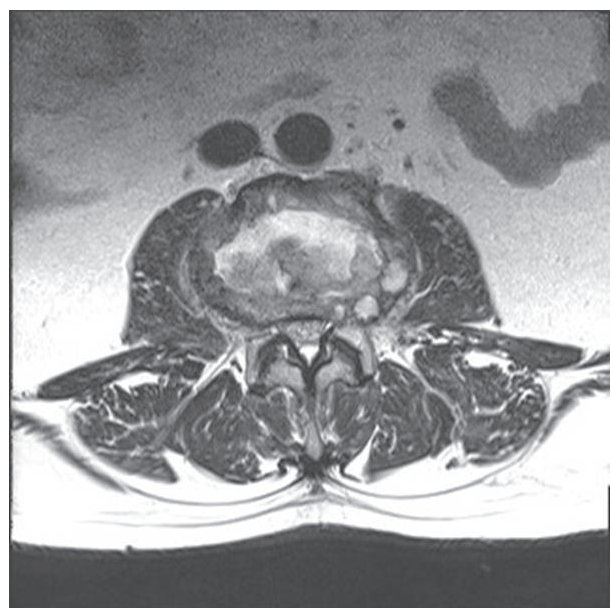


**Fig. 2.** Sagittal lumbosacral contrast enhanced Computed Tomography (CT) Scan showing marked irregularity of the disc space and vertebral endplates in the L2/L3 interspace. Discrete posterior disc protrusion in L2/L3 interspace. Degenerative signs in the rest of the spine with osteophytes on the anterior margin of vertebral bodies in the last dorsal vertebrae.

an uncommon diagnosis in the developed world, representing 2-4% of all cases of osteomyelitis [3] and affects mainly adults. One of the most recent case series outlines its appearance in older patients (mean age 50-60 years) with a clear predominance in the male sex [3,4]. Common predisposing factors include diabetes, immunosuppression, and active neoplasia. Postoperative forms may present in patients with a history of spinal surgery or trauma. Risk factors are shown in Table 1. The annual incidence ranges from



**Fig. 3.** Sagittal Magnetic resonance imaging (MRI) showed loss of intervertebral disc space at L2/L3, with an irregular high intensity area at L2 suggesting a fluid collection extending to adjacent soft tissues. With slight predominance on the left, leaving adjacent to the psoas, the collection has a foraminal component that compresses the left root. Marked bone irregularity of adjacent joint plates, signs of sclerosis associated with extensive bone oedema in L2/L3 vertebral bodies, and soft tissue



**Fig. 4.** Axial Magnetic resonance imaging (MRI) of the lumbar spine at L3 level demonstrating extensive bone edema in adjacent vertebrae and soft tissues.

**Table 1.** The risk factors of spondylodiscitis

Remote infection (present in 25%)
Ascending infection, e.g. from urogenital tract instrumentation or spinal instrumentation or trauma
Intravenous drug use
Immunosuppression
Long-term systemic administration of steroids
Advanced age
Diabetes mellitus
Organ transplantation
Malnutrition
Cancer

0.5 to 2.5 cases per 100,000 population and appears to be rising in incidence as a result of the ageing population, an increasing number of intravenous drugs users (IVDUs), a greater number of both spinal surgical procedures and catheter-related bacteraemia. Comparatively, studies in Spain have shown a similar incidence (0.7 to 2.4 cases per 100,000 inhabitants). For example, the most recent case series in the UK [5] has recorded a 150% increase over the past fifteen years. Patients diagnosed with spondylodiscitis have increased long-term mortality rates, mainly due to a generally poorer pre-morbid state.

Haematogenous and post-operative spondylodiscitis are most often caused by *Staphylococcus spp* [6], whereas the most common aetiological agent of spontaneous Gram-negative spondylodiscitis is *E. coli* [7]. The most frequent pathogenic organisms isolated in anaerobic spondylodiscitis include *Bacteroides* species, *Propionibacterium acnes*, and *Peptococcus* species. The primary site of infection is the lumbar spine (in which 50% of cases occur), whilst only 10% of infection are cervical. Patients with *E. Coli* spondylodiscitis reported in the literature are listed in Table 2 [8-13].

The symptoms of spondylodiscitis are non-specific. Back or neck pain is very common, but up to 15% of patients may be pain-free. The onset of pain is usually insidious and a 'red flag' feature includes constant pain that worsens at night. On average, there is a three-month interval until diagnosis is reached [1-3]. Radicular pain radiating to the chest or abdomen is not uncommon and may lead to misdiagnosis or even unnecessary surgery. Fever occurs in approximately 50% of cases. Neurological deficits, including leg weakness, paralysis, sensory deficit, radiculopathy, or loss of anal sphincter control, are present in a third of cases. These deficits are more likely to be associated with delayed diagnosis, patients with an epidural abscess, presence of cervical abscess, or a primary infecting microorganism of tuberculosis. Paralysis occurs more commonly in those with diabetes mellitus, advanced age and with steroid use. Other potential complications include vertebral crush fractures or the formation of epidural abscesses [2-3].

Diagnosis requires a structured sequence: clinical suspicion, laboratory tests, appropriate imaging, and isolation of the pathogen for identification

and sensitivity testing [1-3]. Significant laboratory findings include leucocytosis (present in 13-60% of cases), elevated erythrocyte sedimentation rate (ESR) and C-reaction protein (CRP) levels. Although ESR is an inflammatory marker lacking specificity, many studies have shown that it is a sensitive marker for infection and is elevated in 90% of patients with spondylodiscitis. Meanwhile, CRP remains the most sensitive and specific marker and may even be used as a reliable marker of response to treatment.

Initial diagnostic imaging includes plain radiograph, which is insensitive to early spinal changes, with generally normal appearances being maintained for up to 4 weeks. Thereafter disc space narrowing and irregularity or ill-defined vertebral endplates may be seen. In untreated cases, bony sclerosis may begin to appear in 10-12 weeks. CT findings are similar to that of plain X-rays, but may be more sensitive in detecting earlier changes. Additionally, other manifestations including surrounding soft tissue swelling, fluid collections, and epidural abscesses may be evident. MRI is the imaging modality of choice due to its high sensitivity and specificity: it has a reported sensitivity of 96% and specificity of 93%. It is also useful in differentiating between pyogenic infections and neoplasms.

Because antibiotic treatment is lengthy and difficult, it is imperative to initiate appropriate antibiotic treatment as early as possible; although blood cultures should always be taken prior to commencing therapy. CT-guided or surgical biopsies should also be performed as imaging alone is not definitive, and other differential diagnoses such as malignancy should be fully excluded. Different bacteria in blood cultures and biopsies can occur, but is nevertheless very rare. If blood cultures and biopsies provide negative or inconclusive results, the lesion should be re-biopsied. Should the patient be stable; antibiotic treatment can be awaited until the results of the biopsy or blood cultures are available. Selection of antibiotic therapy should be based on local policy. A recent [14] randomised controlled trial enrolled patients who were aged 18 years or older who had microbiologically confirmed pyogenic vertebral osteomyelitis and typical radiological features, from 71 medical care centres across France. Patients

**Table 2.** Reported cases of *Escheria Coli* spondylodiscitis

Author References	No. cases	sex	Age (years)	Risk factor	Outcome
<b>Rivero MG et al., [9], 1999</b>	30 cases of Spontaneous Infectious Spondylodiscitis, Escherichia coli 6.6% of cases.	56.7% were males	The mean age of the patients was 68.8 years.	The identifiable causes were infectious endocarditis 13 (43.3%); tuberculosis 7 (23.3%); urinary tract infection 4 (13.3%); bacteremia with focus 2 (6.7%) and without focus 2 (6.7%)	All patients received antibiotic treatment with a median duration of 6 weeks for pyogenic SIS and one year for tuberculous SIS. Eighty three percent required immobilizing brace and 10% surgery for stabilization. Thirty six percent of patients presented complications, most of them related to the causal disease.
<b>Nolla JM et al., [10], 2002</b>	Sixty-four patients, Staphylococcus aureus and gram-negative bacilli, mainly Escherichia coli, were the predominant etiologic agents		Mean age of 59 ± 17 years	In 29 (45%) patients, 1 or more underlying medical illnesses were found	Two patients died in relation to the infectious process and 3 relapsed; functional sequelae often were found.
<b>Eftichia Kapsalaki et al., [11], 2009</b>	Eight consecutive patients one case of Enterococcus faecalis and Escherichia coli	4 men, 4 women	age range 53–82 years	Diabetes mellitus was identified in 6 (75%)	None of the patients underwent surgical intervention. Seven patients (87.5%) recovered to full activity
<b>Safak Kaya [12], 2014</b>	A total of 107 patients who underwent treatment for spondylodiscitis, Escherichia coli 5 cases(15.2)	64 (59.8%) were male.	Ranging between 17 to 83 years of age,	Twenty-seven (25.2%) patients had diabetes	Surgical treatment was performed in 44 (41.1%) patients. Ninety-four (87.8%) patients had full recoveries, and 13 (12.1%) recovered with minimal neurologic abnormalities. No patients died due to the complications of SD and/or the treatment modalities.
<b>Garkowski A et al., [13], 2014</b>	11 cases (Escherichia coli 1 case).	7 men and 4 women	Age ranged from 21 to 74 years	Risk factors of spondylodiscitis were observed in 7 patients	After therapy, all patients had rapid regression of symptoms and no permanent neurological impairments and recurrence of infection were observed
<b>Dobson G et al., [14], 2015</b>	1	Man	69	Transrectal ultrasonography guided prostate biopsy	First case of spondylodiscitis secondary to fluoroquinolone resistant Escherichia coli, complete recovery

were randomly assigned to either 6 weeks or 12 weeks of antibiotic treatment. The study concluded that 6 weeks of antibiotic treatment was not inferior to 12 weeks of antibiotic treatment with respect to the proportion of patients with pyogenic vertebral osteomyelitis cured at 1 year. This suggests that the standard antibiotic treatment duration for patients with this disease could be reduced to 6 weeks. The rate of sequelae in pyogenic spondylodiscitis varies from 25 to 45% of cases. Jiminez-Mejias, *et al.*, [15] reported that only 45% of patients returned to work and daily normal activities. Most patients with pyogenic spondylodiscitis, even those with associated epidural abscess, can be successfully treated using intravenous antibiotics, bed rest, and external immobilization. Nonetheless, a small subset of patients (25%) will require surgery.

In conclusion, we present a case of *E. coli* spondylodiscitis which unfortunately due to vague

presentation had a delayed diagnosis. There were flaws in both diagnosis and mismanagement of the case that are useful as learning points. Worsening back pain developing in the patient should have prompted further investigation, however due to his previous history of back pain the diagnosis of spondylodiscitis was delayed until a blood test showed raised inflammatory markers. Clinical suspicion is important because diagnosis is often delayed by a very nonspecific presenting symptomatology. It is important to recognise red flag symptoms such as non-mechanical back pain, fever, night sweats and weight loss in those patients with recognised risk factors that produce a high index of suspicion of spondylodiscitis. MRI is warranted to look for changes consistent with vertebral osteomyelitis as well as epidural or other abscesses. Treatment should be guided by both the results of the culture and the stability of the patient's condition. Although RCT

controlled trials are lacking, a treatment duration of 6 weeks is generally recommended, with longer courses recommended for persons with complicated

infections and for those with spinal implants.

**Conflict of Interest:** None declared.

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