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Delayed diagnosis of osteodiscitis in an adolescent athlete: a case report

Dominique Harmath, DC[1]
Erin Boynton, MD, FRCS(C)
Peter Lejkowski, DC, FRCCSS(C)

Background: *Vertebral osteomyelitis (OM) is an infectious condition of bone caused by an infecting organism, most commonly Staphylococcus aureus (S. aureus). Though rare in adolescents, it is important to remember that this population has vascularized intervertebral discs prior to skeletal maturity and, therefore, is more susceptible to an osteodiscitis infection.*

Purpose: *To determine the possible factors that lead to a delayed diagnosis of osteodiscitis compared to an early diagnosis in an adolescent athlete.*

Summary: *This case provides a unique example of osteodiscitis in an adolescent rowing athlete where an infected heel blister was the only indication toward a diagnosis. Early diagnosis and successful management of osteodiscitis are dependent on recognizing constitutional and non-constitutional signs and symptoms of infection.*

Clinical relevance: *In sport, when skin barriers may be compromised more readily, the risk of infection*

Diagnostic tardif d'une ostéodiscite chez un athlète adolescent: compte rendu de cas

Contexte : L'*ostéomyélite vertébrale (OM) est une affection des os causée par un microorganisme infectieux, le plus souvent Staphylococcus aureus (S. aureus). Bien que cette affection soit rare chez les adolescents, il est important de retenir que cette population a des disques intervertébraux vascularisés avant d'atteindre la maturité squelettique et qu'elle est donc plus exposée à l'ostéodiscite.*

Objectif : *déterminer les facteurs possibles qui conduisent à un diagnostic tardif de l'ostéodiscite par rapport à un diagnostic précoce chez un athlète adolescent.*

Résumé : *Ce cas fournit un exemple unique d'ostéodiscite chez un athlète adolescent de l'aviron présentant une ampoule infectée au talon qui était l'unique signe permettant de poser un diagnostic. Le diagnostic précoce et la prise en charge réussie de l'ostéodiscite dépendent de la reconnaissance des signes et symptômes constitutionnels et non constitutionnels de l'infection.*

Pertinence clinique : *Chez les sportifs, les barrières cutanées peuvent être compromises plus facilement*

[1] Graduate Studies, Sports Sciences, Canadian Memorial Chiropractic College

Corresponding author: Dominique Harmath, Canadian Memorial Chiropractic College, 6100 Leslie Street, Toronto, Ontario M2H 3J1
E-mail: harmath.dominique@gmail.com

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should be considered in the differential diagnosis of unprovoked back pain.

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KEY WORDS: osteodiscitis, osteomyelitis, adolescent, athlete, rowing, chiropractic

et le risque d'infection doit être pris en compte dans le diagnostic différentiel des douleurs dorsales non provoquées.

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mots clés : ostéodiscite, ostéomyélite, adolescent, athlète, aviron, chiropratique

Introduction

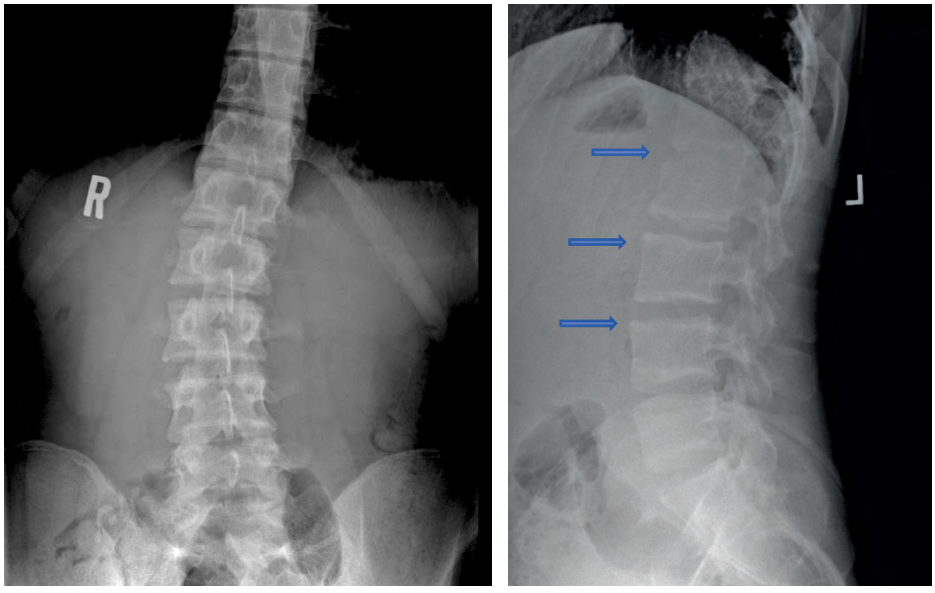
Vertebral Osteomyelitis (OM) is an infectious condition of bone caused by a pathogenic organism, most commonly *Staphylococcus aureus* (*S. aureus*).[1] It is more common in men than in women, and its incidence increases with age with a reported 21.8 cases per 100,000 person-years.[2] Though rare in adolescents, it is essential to keep in mind that unlike adults with avascular intervertebral discs, this population has vascularized intervertebral discs prior to epiphysial growth plate closure and therefore are more susceptible to an osteodiscitis infection.[1] Unfortunately, the diagnosis of OM is difficult and often delayed when constitutional signs such as fever, fatigue and malaise are not present.[3] Delays in diagnosis vary between 2-12 weeks with a 20% mortality rate.[4] To our knowledge, there have been no reported cases of an osteodiscitis infection developing from a heel blister in the sport of rowing in an adolescent individual. The clinical case presented had a delayed diagnosis of two months. Such a delay may leave the individual with long term complications or debilitating symptoms from chronic OM. High clinical suspicion is necessary in the early detection and treatment of the infection in order to prevent further tissue destruction and improve the prognosis.

Case summary

An 18-year-old competitive male rower presented with an acute onset of left-sided lower back pain of two days duration with no previous history of back pain. He woke up with pain and stiffness in his back but could not recall a mechanism of injury. The pain was described as dull at rest and sharp with walking, forward bending and active left hip flexion. The pain was localized to the left lumbar region from L2 to the lumbopelvic region bilaterally. No signs of illness were noted.

Upon examination, the patient was apprehensive to move with a restricted and painful range of motion in all directions, primarily flexion. Bilateral Kemp's, posterior to anterior shear at L5, active single leg raise (ASLR) and FABER's test on the left side were positive tests for left-sided back pain in the L2-L3 and sacroiliac (SI) joint areas. Negative tests included Valsalva, SLR, cross SLR, SI compression and Braggard's. The left quadratus lumborum muscle was in spasm; however, there was no evidence of swelling or deformity. Neurological exam was within normal limits. When asked about a fever, the patient denied having one, and there was no mention of the blister during the initial visit. The treating chiropractor recorded a clinical diagnosis of acute left, flexion-intolerant lower back pain with suspicion of discogenic origin. The day after the initial visit, the patient's parent took the patient to a physician to investigate a heel blister of one-week duration that became red and presented with a fever. The patient was placed on antibiotics (Cephalex, CT, Ger-Germany), no signs of cellulitis were noted. The fever resolved that day and did not return.

Radiographs of the lumbar spine were ordered due to the fact that lower back pain was present with no direct cause, a possible discogenic origin was suspected, the patient was skeletally immature, and that a possibly infected blister with a slight fever was present. The radiograph revealed a dextroscoliosis of the lumbar curve, ill-defined superior and inferior endplates of L2 and a mild loss of L1, L2 disc space was noted (see Figure 1). A bone scan/CT/MRI examination of the lumbar spine was advised by the chiropractor to rule out osteodiscitis. Due to a gap in interprofessional communication, a second set of lumbar radiographs were ordered that confirmed the unremarkable findings of the first radiograph. The patient was recommended to continue with conservative therapy. After



1A. 1B.

Figure 1.
Radiographic imaging. 1A. Initial radiographs with no evidence of infection. 1B. The anterior superior vertebral endplates of L3 and L4, as well as the ill-defined superior and inferior vertebral endplates of L2 with a mild loss of L1, L2 disc space coincidentally, mimic Romanus and Andersson lesions (blue arrows).

four weeks of treatment and only 50% improvement, it was acknowledged that the patient's current condition did not match up with the natural history of the diagnosis. For this reason, the treating provider acted on their clinical suspicion and once again referred the patient for special imaging and/or a second opinion. The patient went for a second opinion to a sports medicine doctor two weeks later, where they were clinically diagnosed with a disc herniation. The chiropractor explained that due to the lack of related findings in the patient's presentation, including the absence of neurological signs (soft and hard), the patient was unlikely to be suffering from a frank disc herniation. The patient was subsequently encouraged to seek further medical work-up, including blood tests.

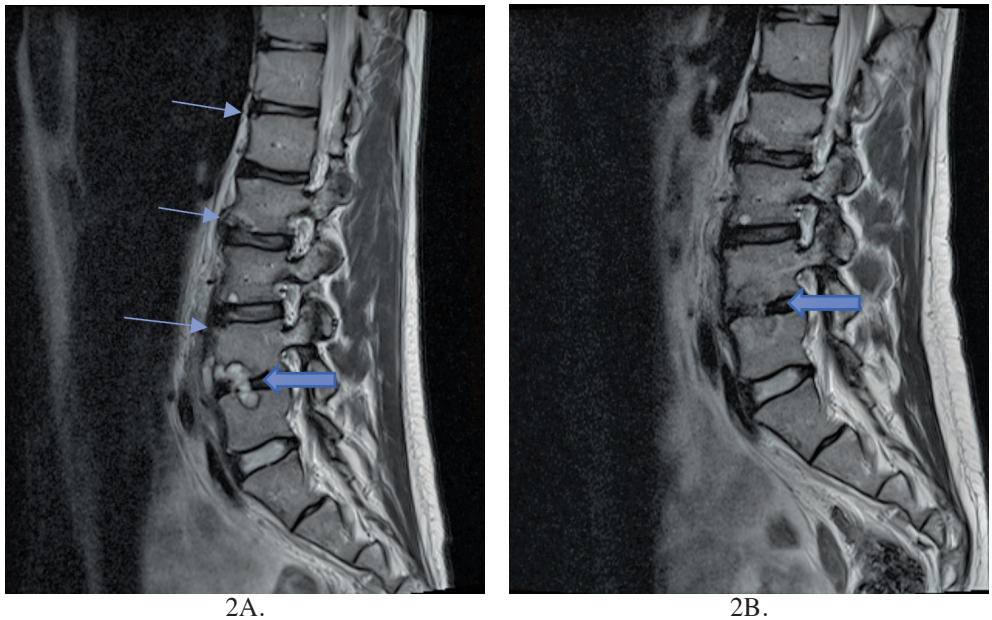
One week later, the blood tests came back positive for inflammatory markers with an elevated erythrocyte sedimentation rate (ESR) and c-reactive protein (CRP); however, HLA B-27 was negative. Three days later, an MRI was taken of the lumbar spine, where a diagnosis of L4-L5 osteodiscitis was confirmed (see Figure 2).

Three days later, a biopsy identified *S. aureus* as the bacterial culprit. Three days following the diagnosis, intravenous (IV) and oral antibiotics were administered for eight weeks. A follow-up MRI was conducted at the 6-week mark of the 8-week treatment plan, where the infection was shown to still be present. The patient saw improvement with treatment; however, there was no planned follow-up. Four months later, the infection was confirmed to still be present on MRI and biopsy, and the individual underwent a second course of IV antibiotic treatment (cefazolin) for suspected chronic osteodiscitis. Upon completing the second round of intravenous antibiotics (ten months post initial presentation), the patient felt he was back to normal and had returned to exercising regularly. The patient's current condition is stable, with a resolution of the infection as indicated with negative blood tests for inflammatory markers.

Discussion

Epidemiology

OM most commonly affects those exposed to a penetrating injury, surgery, intravenous drug use, diabetes, or a compromised immune system. Such risk factors leave in-



2A. 2B.

Figure 2.
L4-L5 Osteodiscitis, magnetic resonance imaging (MRI). 2A. Initial T-2 weighted MRI of the lumbar spine with an L4-L5 intervertebral disc centered process with intervertebral disc height loss, irregularity, and high signal intensity. There is a small enhancing collection within the left paraspinal soft tissues anterior to the L4/L5 intervertebral disc indicative of early abscess formation. Well demarcated destructive lesions are present at the adjacent vertebral endplates (thick blue arrow). Multilevel inflammatory changes of the lumbar spine are also noted with high signal irregularities and erosions at the anterior corners of the vertebral bodies mimicking Romanus lesions with similar lesions centrally at the disco-vertebral junction of the T12, L1 and L3 vertebral bodies mimicking Andersson lesions (thin blue arrows). These findings are suspected to be caused by the infection. 2B. Follow up T-2 weighted MRI of the lumbar spine at 6-weeks of treatment with L4-L5 osteodiscitis still noted (thick blue arrow).

dividuals vulnerable to contracting OM; specifically, its most common bacterial offender, *S. aureus*. In the paediatric population the incidence is no higher than 0.3 per 100,000.[5]

Pathophysiology

S. aureus, the most common perpetrator of OM, may have a hematogenous or contiguous spread or may be transmitted by direct inoculation.[6] Its virulence can be attributed to both its extracellular and intracellular factors. [6] Its ability to adhere to extracellular matrix proteins allows it to establish itself in the host organism.[7] The evasion capability of the bacteria to survive in host cells and its invasion capabilities via exotoxins or hydrolases give it the extra edge to thrive in the human body.[8] Biofilms, a collection of communicating microbes with altered gene expression, can also form during the infection, making antibiotic treatment's success exceptionally difficult.[9] In this case, it is suspected that the *S. aureus* infection of the intervertebral disc occurred due to hematogenous spread from an infected heel blister.

Diagnosis

The diagnosis of vertebral OM is multifactorial and challenging at times when constitutional symptoms are not present. In general, key history findings would include non-specific lower back pain, fever, malaise, fatigue or a trivial skin infection.[10] It is important to note that fever presents in less than 50% of OM cases.[11] Physical findings may mimic discogenic pain, and in cases such as the one currently presented, ambiguous findings paired with a failed response to care should elicit follow up with a ser-

ies of diagnostic imaging. A definitive diagnosis of OM is a combination of clinical signs of infection, laboratory tests, imaging abnormalities, biopsy and isolation of cultures.[6]

In the case presented, the heel blister was key toward the diagnosis. Discitis should be a top differential diagnosis in youth with discogenic pain.[12] Ultimately, it was the high clinical suspicion for discitis that prompted the chiropractor to continue pushing for a second opinion and refer for special imaging while the patient's pain continued to be dismissed as mechanical. In order to prevent future delayed diagnoses, as in the current case where recommendations for advanced imaging were missed, interprofessional communication should be prioritized.

Diagnostic imaging

The diagnostic process is often broken down into two parts: radiographic imaging and bone scan or blood testing, and the second part; magnetic resonance imaging and biopsy cultures.[13] Part two is necessary to determine the underlying organism responsible for the infection to guide antibiotic treatment.

Radiographic imaging is a cost-effective and quick way of ruling in OM and, for this reason, is part of the first line of the diagnostic process. The challenge, however, is that radiographs will appear normal during the first 21 days of spinal infection.[14] Both radiographic imaging and blood testing have high specificity but low sensitivity in OM diagnosis and are commonly used when wait times for magnetic resonance imaging (MRI) are high.[13] The specificity and sensitivity for MRI are reported as 60-90% and 78-90%, respectively, and MRI also can identify areas of soft tissue involvement that may guide treatment decisions.[12] Lastly, microbiologic cultures via CT-guided biopsy is the gold standard in OM diagnosis with 99.9% specificity.[13]

The diagnosis of the case began with radiographic imaging that came back negative for infection, followed by blood testing and MRI that confirmed the diagnosis of an osteodiscitis infection, while a CT-guided biopsy identified the bacterial culprit.

Treatment

As soon as the underlying organism is identified, intravenous antibiotics can be administered for approximately two weeks, followed by oral antibiotics for an additional 2-4 weeks.[6] Surgery is not usually required; however, it may be considered if signs of neural compression, spinal instability, progressive destruction of bone, limb deterioration or signs of the development of an epidural abscess or paravertebral abscess appear in order to prevent progression to a chronic OM.[6] In such instances, the patient should be monitored during treatment and managed appropriately. Lingering complications may last on average up to two years if an acute case of OM becomes chronic.[6]

In the current patient case, infection was still present after the eight weeks of treatment with intravenous and oral antibiotics. Factors responsible for the lack of healing response may have been attributed to the antibiotics received for the heel blister and the presence of an abscess.[6] An abscess is challenging to treat with antibiotics as the drug cannot diffuse into the middle of the abscess. [6] This was likely the case with the patient as abscesses have no blood supply and are surrounded by very necrotic/fibrotic material making penetration of drugs difficult.[6] If there is no drug reaching the bacteria, they continue to grow.

Prognosis

The level of precision for the prognosis of OM is not well studied. Delays in diagnosis vary between 2-12 weeks with a 20% mortality rate.[4] According to Gupta *et al*.[4], longer duration of symptoms prior to diagnosis with a *S. aureus* pyogenic vertebral OM infection were associated with increased rates of treatment failure. Tuberculous and brucellar vertebral OM, however, remain the leading causes of delayed vertebral OM diagnosis at a rate of 21%.[15]

Generally, if the infection is diagnosed and treated early, possibly prior to the infection becoming evident on radiographic imaging (<21 days), the effects with antibiotics should be rapid.[6] This emphasizes that osteomyelitis cannot be ruled out from radiographic imaging alone as it was in our case. On the other hand, chronic OM is dependent on the patient's physiological status and the duration of the infection.[6] Recovery can vary from 2 months to 2 years.[6]

The time from initial presentation to diagnosis in the patient was two months. The condition should have been further assessed with bloodwork and an MRI when the patient's presentation changed revealing a fever and an infected blister. Further delays in prognosis occurred when the patient was discharged from the hospital after receiv-

ing intravenous and oral antibiotics without a scheduled follow up post-treatment. This was especially a problem when the individual's pain persisted for four additional months post-treatment. Though back pain is often present once the infection has resolved, follow up is necessary to ensure resolution of the infection via blood testing of ESR and CRP inflammatory markers and radiographic imaging for evidence of bony fusion.[6,16]

Future studies should investigate the role of range of motion findings and the absence of muscle spasms in conjunction with blood testing of inflammatory markers to provide more insight on clinical signs of improvement.

Summary

This case provides a unique example of osteodiscitis in an adolescent rowing athlete where an infected heel blister was the only clue toward an early diagnosis. Early diagnosis and successful management of osteodiscitis are dependent on recognizing constitutional and non-constitutional signs and symptoms of infection, in our case, an infected heel blister and a mild fever. Frequent monitoring and scheduled follow-up visits during and after the treatment process is advised to avoid further delays in prognosis.

The overseeing practitioner did his due diligence referring the patient for further studies when his clinical suspicion was high. Early investigation is essential for a promising prognosis of osteodiscitis when the clinical course of a musculoskeletal diagnosis does not meet the expectation.

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