CASE REPORT

Spondylodiscitis in a 54-year-old female scuba diver

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SUMMARY

A 54-year-old woman presented to a Sports Physician with a 4-year history of haemochromatosis, and she had a medical history that included a congenital spondylolisthesis resulting in a fusion of L4-S1 at age 16 years, episodic mechanical low back pain and an absence of other significant musculoskeletal symptoms. On presentation, she reported 18 months of severe low back pain that started after a scuba diving trip. After the onset of this low back pain, she developed gastrointestinal symptoms from *Salmonella*. The gastrointestinal symptoms improved with a course of antibiotics, but the back pain persisted in spite of analgesics, non-steroidal anti-inflammatories and several attempts at different conservative management. CT imaging ordered by the Sports Physician demonstrated an erosive spondylodiscitis of L2/3 that was not present on initial investigations. However, even in the presence of significant bony changes, the patient was successfully treated with targeted conservative therapy.

BACKGROUND

This case highlights diagnostic difficulties for a patient with a complex presentation of low back pain. Second, the case illustrates that despite having had chronic pain, severe spinal bony changes and a number of other comorbidities, an individual with severe spondylodiscitis can recover and return to premorbid functional activity with appropriate conservative intervention. Finally, the case highlights the importance of a multidisciplinary approach to treatment in patients with complex presentations.

CASE PRESENTATION

A 54-year-old female scuba diving instructor presented to a Sports Physician with an 18-month history of severe persistent lower back and right buttock pain. She reported that her current symptoms had started after returning from an overseas diving trip although she could not recall a specific mechanism. At that time, her symptoms were unrelated to specific positions or activities. When the pain continued to worsen over a 2-week period, she presented to the emergency department of a local hospital. She was discharged with analgesics and non-steroidal anti-inflammatories. When she developed a fever, loose stools and abdominal cramping a week later, she re-presented to the emergency department and was admitted for investigation.

The patient reported to the hospital staff that she had been diagnosed with haemochromatosis 4 years earlier and had spinal surgery at the age

of 16 years for congenital spondylolisthesis. She reported previous episodes of persistent back and buttock pain that was similar to but much less severe than her current low back symptoms. Given the severity of symptoms, CT scans were performed that showed multilevel degenerative changes consistent with the patient's age and a non-instrumented L4-S1 spinal fusion with a full overlap of S1 on S2 (figure 1). The patient reported that while in hospital, several other tests were performed and she gave the Sports Physician the hospital discharge summary. The discharge summary included abdominal CT results that demonstrated 'gallbladder wall and periportal oedema raising the differential of cholecystitis and cholangitis'. Biliary ultrasound was suggestive of cholecystitis with sonographic Murphy's sign and gall bladder wall thickening of up to 4 mm with non-mobile foci. Urea and electrolytes were unremarkable. Haemoglobin and red cells were slightly below normal range at 114 g/L and $3.68 \times 10^{12}/L$, respectively. C reactive protein was elevated at 155 mg/L. Liver function tests indicated low albumin at 30 g/L and high alanine transaminase at 73 U/L. Microbiology returned with *Salmonella*, described as 'acting like' either *S. typhi* or *S. paratyphi* serotype. The patient's discharge medications included baclofen, ciprofloxacin,

Figure 1 Lumbar CT 3 weeks postonset of symptoms.

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Figure 2 Lumbar CT 18 months postonset of symptoms.

diazepam, meloxicam, omeprazole, paracetamol and tramadol hydrochloride.

The patient reported resolution of her systemic symptoms within a month of discharge but persistence of the severe back pain to the extent of being wheelchair user for periods of time. She was taking tramadol (50 mg five times a day and 100 mg nocte), pregabalin (75 mg two times a day), Valium (2 mg nocte) and baclofen (10 mg nocte) but did not feel she was improving. She had been seen by several medical and allied health practitioners but had not been given a specific structural or clinical diagnosis nor had any significant relief in symptoms over the 18-month period. She rated her average pain over the preceding week as an average of 9/10 on an 11-point Likert Pain scale anchored at no pain and worst pain imaginable. Although she had constant pain, which was not the case prior to her hospitalisation, her symptom behaviour otherwise was largely mechanical being particularly worse with activities including walking or being upright. Due to difficulty in interpretation of the concept of a pain scale and in order to not focus on pain, future assessments focused on levels of medication and indicators of function rather than pain.

INVESTIGATIONS

On examination, the patient stood in a hyperlordotic posture. Range of active movements were Flexion, fingertips to mid-thigh, Extension 15° short of neutral, Lateral Flexion L 8°, R 5°. There was marked tenderness at L2/3. There were no neurological deficits. Given the duration and severity of symptoms, the Sports

Physician ordered plain X-rays and a repeat CT that showed severe erosion and endplate deterioration at L2/3 in addition to the findings from 18 months earlier (figure 2). On referral to a specialist infective diseases physician, the infective organism was confirmed to be *S. corvallis* (rather than the originally suspected *S. typhi* or *S. paratyphi*) but was considered to be permanently quiescent. The patient was referred back to the Sports Physician.

DIFFERENTIAL DIAGNOSIS

The erosive changes at L2/3 were consistent with either dysbaric osteonecrosis or infectious pathology. In the current case, the pathology was considered more likely to have been infectious in aetiology, but it is unclear as to what extent dysbaric events may have predisposed her to developing these symptoms. It is possible that the symptoms could have been an acute on chronic episode of back pain or a manifestation of haemochromatosis. Although potential contributing factors, both of these diagnoses are considered less likely due to the timing of both the symptoms and radiographic changes after her infection.

TREATMENT

The Sports Physician referred the patient to a Specialist Musculoskeletal Physiotherapist. Initial treatment was very gentle oscillatory movements of approximately 10–20N targeted at L2/3 with a slow decrease in discomfort during the passive mobilisation.¹ Within three treatments, the patient reported a significant reduction in pain and both passive and active lumbar extension had improved. However, there was no change in standing posture or functional ability. Treatment then focused on enabling the patient to find more efficient ways of moving within the structural limitations. Rather than teaching her to 'correct' the movement pattern, an exploratory method of movement education (Feldenkrais method) was used to assist the patient to find her own movement solutions that were more comfortable and efficient.²

OUTCOME AND FOLLOW-UP

After a total of seven sessions of physiotherapy over 9 weeks, treatment was discontinued and the patient stated that her symptoms and functional abilities approximated her preinfection level. Range of active movements were Flexion, fingertips to upper 1/3 shin, Extension 10°, Lateral Flexion L 20°, R 30°. She was reviewed a year later and reported that she was able to maintain an acceptable level of symptoms and functioning with regular independent hydrotherapy and had commenced a Master's degree. She had, however, reduced her diving workload. Her decrease in pain level is indicated by the change from her previous polypharmacy to only taking Tramadol hydrochloride (50 mg A.M and 25 mg nocte).

DISCUSSION

Salmonella are gram-negative bacilli infecting both human and animal populations.³ Transmission is most commonly through contaminated food, but can also occur from a variety of animals including poultry and reptiles.⁴ Most *Salmonella* infections present as a gastroenteritis which is usually self-limiting, but can be persistent or life-threatening. Non-gastrointestinal, focal manifestations occur in 5%–10% of cases which may only present weeks or months after the infection and can occur in the absence of gastroenteritis or fever.⁵ Such focal manifestations include infections of the viscera, skin, nervous system, muscle, joints and bone. In less than 1% of people, the focal infections affect bones of the periphery or spine.^{5,6} Infections of the spine

can be described as discitis or osteomyelitis, but they are probably better described as spondylodiscitis due to overlap and difficulty in differentiating between infections affecting the two structures.

Factors that impact on whether a given individual with *Salmonella* develops osseous foci include concurrent medical conditions, the serovar of *Salmonella* and bony structural integrity. The outcomes of *Salmonella* infections are clearly different for different serovars.⁷ The impact of serovar on osteomyelitis or spondylodiscitis, however, is less clear. In immunocompetent individuals, some authors suggest osteomyelitis appears to be more commonly found with *S. enterica* ser. Typhi and *S. enterica* ser. Paratyphi,^{8,9} whereas others suggest this is not the case.¹⁰ *S. enterica* ser. Corvallis is a lesser known serovar which accounts for between 1%¹¹ and 3%¹² of *Salmonella* infections and has previously been associated with osteomyelitis in children.⁹

When predisposing factors, such as a depressed immune system, are present, the chances of developing extraintestinal infection increase.⁶ As a result, the incidence of osseous foci from *Salmonella* is much higher in individuals with compromised immune systems such as HIV⁶ or with sickle cell disease with one series finding that 90% of children with osteomyelitis from *Salmonella* had sickle cell disease.¹³ It may be thought that the high levels of iron that occur in people with haemochromatosis might predispose them to infection by or focal manifestations from *Salmonella*, this does not appear to be the case. The iron in haemochromatosis is not accessible to *Salmonella* and it has even been suggested that the high frequency of haemochromatosis gene may be accounted for by its protective ability against infections such as *Salmonella*.¹⁴

Bloodborne infections are more likely to lodge in an injured tissue. In this case, there are two potential predisposing injuries.

Patient's perspective

- For 18 months after being discharged from hospital, the patient was taking multiple medications which she believed were not progressing her mobility, functionality or her level of pain. She stated 'I couldn't walk more than a few steps so I was wheelchair user for a period of time, which made me feel really old and really short all of a sudden'.
- Following the physiotherapy treatment, the patient stated 'I felt that all of my muscles that were holding me in place had relaxed and my flexibility had improved.' She explained that her symptoms had been fairly stable over the year since finishing treatment as long she continues with pool exercises. She stated 'I am still at work, I'm still diving... I just make sure I don't lift any of the heavy tanks'.

Learning points

- Given the identification of *Salmonella* on discharge from hospital and the duration of unremitting severe lower back pain, bone infection should have been considered earlier as a cause of the patient's lower back pain.
- MRI may have been more effective at detecting infectious spondylodiscitis during the acute stage.
- Conservative management can be effective in patients with severe, chronic low back pain even in the presence of significant pathology.

First, the lumbar spine had been subjected to surgical intervention more than 30 years previously and was the source of persistent pain since this time. Second, although dysbaric events from scuba diving had previously been thought to result from a single event of rapid decompression, the effects of pressure changes now appear to be cumulative with factors including the rate of compression¹⁵ and flying after diving¹⁶ contributing to the onset of symptoms. Furthermore, onset of symptoms may only occur days or weeks after the dysbaric events. Perhaps one explanation for this delayed response is that the osteonecrosis from dysbaric events may result from ischaemia produced by fat emboli rather than nitrogen bubbles.¹⁷ It is unclear how or if the presence of haemochromatosis might interact with either of these factors related to predisposing tissue injury.

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