We present a case of L2/3 interspinous bursitis treated with extraspinal injections. No previous investigations have used fluoroscopically guided spinal injections to confirm the clinical relevance of the MRI features of this type of bursae. Autopsy studies have revealed an increased incidence of interspinous lumbar bursal cavities with advancing age. Afflicted patients present with localised, midline lower lumbar pain exacerbated by extension. In young athletes these symptoms can mimic spondylolysis. MRI is useful in detecting soft-tissue injury of the posterior elements.

Fluoroscopically guided diagnostic and therapeutic extraspinal injections can be used for confirmation and treatment of pain from such bursae.

Persistent lumbosacral pain in the adolescent athlete typically arises from injury to the posterior elements. Spondylolysis is a common cause. Patients usually present with localised lumbosacral pain with or without referred pain to the lower limbs, tight hamstrings, a normal neurological examination, and pain on extension. CT can help identify and stage a defect of the pars interarticularis. MRI may be useful to identify patterns of oedema in early pars lesions, and to differentiate from pathology such as disc herniation, infection or tumour.

Repetitive lumbar spinal movements in the sagittal plane precipitate injury to various posterior elements. Strain of the posterior soft tissues can lead to structural changes in the lumbar interspinous ligaments. Rupture of the supraspinous and interspinous ligaments may occur; this is most common in women aged between 15 and 35 years. Bursae filled with synovial fluid between lumbar spinous processes secondary to trauma have been described. MRI of the lumbosacral spine allows detection of interspinous abnormalities. Fluoroscopically guided, diagnostic extraspinal injections can be used to confirm that pain arises from the interspinous soft tissue. Therapeutic injections have been used with benefit. We present a case of an athlete with a symptomatic lumbar interspinous bursitis, successfully managed with therapeutic injections into an interspinous bursa.

Case Report

An 18-year-old woman, a collegiate basketball player, presented with an acute exacerbation of chronic, episodic lumbosacral pain. She had had a week of low back pain following a day of intensive exercise. She developed symptoms after basketball having undertaken sprinting and weight lifting workouts earlier in the day. There was no history of...
trauma, but there was a history of similar intermittent pain for five years. The pain was stabbing in character, confined to the midline upper lumbar spine, and of an intensity rated six out of ten on a visual analogue scale (VAS). Standing, walking, and extension of the spine aggravated the pain, while sitting or lying on her left or right lateral side relieved it. There was no referred pain or disturbance of bowel or bladder function. She had a past medical history of asthma.

Physical examination revealed a well-developed, well-nourished athletic woman. Her lumbar spine movements were full, but mid to terminal extension was painful. Returning to the full upright position from forward flexion did not evoke pain. She was slightly tender over the spinous processes of L2/4. There were no abnormal neurological signs in the upper or lower limbs.

Plain radiographs of the lumbosacral spine including flexion and extension views, showed normal bony alignment without evidence of instability, tumour, or fracture. Single photon electron emission tomography (SPECT) imaging of the lumbosacral spine was unremarkable. MRI revealed some mild disc desiccation at L3 to S1 and a focal increase in soft-tissue signal intensity on T2-weighted images between the spinous processes of L2 and L3 in the midline sagittal view (Fig. 1). Previous reports have indicated that fluorine-18-deoxyglucose (FDG) positron emission tomography (PET) scanning can detect a stress reaction despite a negative MRI and bone scan, and this showed increased uptake in the inferior aspect of the spinous process of L2 and the superior aspect of the spinous process of L3 without evidence of a pars fracture. Thus, these investigations suggested a diagnosis of interspinous bursitis at L2/3.

The symptoms were not relieved by a four-week trial of a non-steroidal anti-inflammatory medication and stabilisation exercises for the lumbar spine. In order to determine if there was a bursa present and if the symptoms were relieved following the instillation of a local anaesthetic, Ominipaque dye (1.5 cc) was injected into the L2/3 interspinous oedema before reaching a firm endpoint. The pattern did not display a typical fascial spread. The dye collected in a cotton-ball appearance. A similar amount of dye was injected into the L1/2 intraspinous soft tissue demonstrating fascial spread and a soft end-point. An injection of 1.5 cc of 4% xylocaine into the L1/2 interspinous soft-tissue did not produce any change in the symptoms but an injection into the L2/3 space relieved the symptoms. We concluded the diagnosis of L2/3 bursitis was accurate. Two subsequent injections of 1 cc of Celestone Soluspan (betamethasone) and 0.25 cc of 4% xylocaine were performed at the L2/3 level over a 14-day interval. There was a marked reduction in pain. The VAS score changed from 6 to 2, and there was a decrease in the Oswestry disability index score following physiotherapy from 20 to 8. She was able to complete a conditioning programme and return to full activity. At follow-up two months after the second further injection, she remained symptom-free which continued over the ensuing year.

Discussion
Baastrup's disease or ‘kissing spines’ was first described in 1933. It is caused by chronic contact between adjacent spinous processes, and characterised by sclerosis and flattening of the opposing superior and inferior surfaces of the spinous processes. However, in 1825 Mayer had discovered lumbar interspinous synovial fluid filled cavities in ‘well exercised soldiers’ most commonly between the overgrown and facetted L3/4 spinous processes. Subsequently Rissanen and Hazlett observed interspinous bursal cavities between L4/5 and L3/4 in autopsy studies occurring with increasing frequency with advancing age, 85% of subjects between the age of 61 and 70 years having had bursal cavities in the midline L4/5 interspinous ligament at autopsy. The healthy interspinous ligament has a thin central portion, which tends to undergo fatty degeneration with cavitation in patients in their sixth decade. Structurally, these bursae had a cellular lining with microscopic variations of chronic and acute bursitis. No such cavities have been found in autopsy studies of the spines of patients under the age of ten years, but perhaps explained by the development of adult lumbar lordotic curvature after this age.

The incidence of Baastrup’s syndrome in collegiate athletes is 6.3% and most commonly affects gymnasts. Repetitive flexion and extension strain the interspinous ligaments and cause adjacent spinous processes to abut each other.

During flexion the interspinous ligaments are maximally stretched to limit further flexion. Subsequent extension allows adjacent spinous processes to approximate subjecting the intervertebral disc and capsular ligaments. Thus, the posterior ligaments are sprained first following extreme forward flexion. Repetitive or excessive forward flexion strains the attachments of the interspinous ligaments resulting in tenderness at the junction of bone and ligament and the formation of a spur. Repetitive extension compresses these inflamed tissues disrupting healing and producing impingement of painful structures.

There is an abundant nerve supply to the interspinous ligaments. The junction of the interspinous ligament and spinous process has a very active metabolism which may be related to the development of highly vascularised granulation tissue in response to injury, including reconstruction in the bone and cartilaginous zone. Bone SPECT can detect increased osteoblast activity in true Baasstrup’s disease in which reactive sclerosis can be identified on radiography. In the absence of bony eburnation and sclerosis, SPECT may not detect any abnormality. Although the assessment of Baastrup’s disease by PET imaging has not been validated, increased uptake of FDG might occur at the interspinous ligament due to the strain-induced inflammatory reaction. Our case presentation supports this theory by the demonstration of an abnormality on PET imaging, but not on SPECT imaging. Although conclusive evidence is lacking, it is reasonable to presume that lumbar interspinous bursitis precedes the development of Baastrup’s disease. Conclusive evidence does exist of the presence of bursal cavities in lumbar interspinous ligaments.

Relating the MRI finding of interspinous bursitis to the clinical presentation of lumbar pain can be difficult. Various studies have recorded the efficacy of therapeutic extraspinal injections in the treatment of lumbar interspinous bursitis. Yet, none have examined the use of diagnostic injections employed at non-involved segments to confirm the clinical response at the involved segment, or have described the fluoroscopic contrast pattern supportive of the existence of a bursal cavity. In our case, we observed a firm end-point after the injection of a finite volume of contrast into a fixed cavity. Real-time imaging revealed a concise and localised collection of contrast consistent with a cavity of fixed dimensions. In contrast, injection of a similar amount of contrast at a non-involved level demonstrated a fascial spread of contrast with-
out a firm end-point. Subsequent injection of anaesthetic into this uninvolved level did not alleviate the patient's symptoms. Yet, injection of anaesthetic into the bursal cavity relieved the symptoms. Complete resolution of symptoms ensued after two therapeutic injections arrested the local inflammatory cascade allowing the athlete to move freely.

This case report is the first published presentation to characterise the fluoroscopic features of lumbar interspinous bursitis, and to use diagnostic injections to confirm this as the cause of pain.

References