LearnSportsMed

Back Injuries

BAsem British Association of Sport & Exercise Medicine

Contributors

Creator: Dr Frank O' Leary, GP with a special interest in sports medicine, Sport Ireland Institute, National Sports Campus, Dublin 15, Ireland

Overview

Acute low back pain is a common cause of loss of competition time in athletes. Causes can vary from nerve root, sacroiliac joint, disc disease, bone, ligament or muscular injury. This resource will focus on acute nerve compression, pars interarticularis injuries and sacroiliac (SI) joint disorders.

Rarer, more sinister causes like tumour and infection should also be considered in the young population.[1]

Anatomy

The Lumbosacral spine includes 5 lumbar vertebrae and 5 sacral vertebrae (fused). Each of the bones of the lumbar spine include a vertebral body, two pedicles, two laminae, two transverse processes and a spinous process. There are three joints which articulate with the lumbar vertebrae. These include the intervertebral joint ('disc' joint located anteriorly) and two facet joints (posterolaterally).[2] The lumbo-sacral spine moves in the following planes;

- flexion/extension (sagittal plane – of note, 80 - 90% of lumbar flexion & extension occurs at L4/5 and L5/S1 level)
- torsion (axial plane)
- lateral flexion (coronal plane)

SI Joint

The sacrum articulates with the ileum of the pelvis to form the sacroiliac joint. It is a diarthrodial joint that contains both hyaline cartilage and fibrocartilage. It is a complicated joint being both a synovial joint anteriorly and a fibrous joint posteriorly. The SI joint is responsible for transferring forces from the upper body to the lower limbs. Its movements involve some translation and rotation but overall it aims to restrict moving in any direction.[3,4]

Nerve roots

The spinal nerve roots exit at the neural foramina which lie posterior to the intervertebral discs and anterior to the facet joints. Narrowing of this space can occur from disc protrusion or from bony spurs (osteophytes) arising from the facet joints. The spinal cord ends at the level of L1/2 vertebrae. Distal to this point the spinal canal contains spinal nerves which travel inferiorly and then exit through their relevant neural foramen. At each disc level there is a traversing nerve root and an exiting nerve root. This is important when considering the location of disc herniations and associated symptoms. A paracentral disc protrusion is more likely to impinge the traversing nerve root where a postero-lateral disc protrusion is more likely to impinge an exiting nerve root.[5-7]

Aetiology

Acute Nerve Compression

Acute nerve compression occurs when the spinal nerve root becomes irritated either directly or indirectly. It tends to occur by acute disc prolapse in a previously injured spinal nerve. Previous injury can occur from a degenerative process of osteophytes irritating the nerve root or from a previous disc protrusion compressing the nerve root. This is why a seemingly innocuous move, like bending down, can cause sudden severe low back pain. The most common location for disk prolapse is L5/S1, followed by L4/5. Disk prolapse is most common between the ages of 20 & 50 years. It is more common in males.[8]

Pars Interarticularis Stress Fracture

The Pars Interarticularis is a bridge of bone between the inferior and superior articular processes incorporating the facets joints of the spinal vertebrae. It is an area vulnerable to fracture especially from repetitive hyperextension +/- rotation of the back.[9] A pars injury is generally as a result of repetitive fatigue /overload rather than an acute event. Sports such as gymnastics,[10] tennis,[11] cricket (fast bowling),[12] high jump and throwing (javelin, hammer etc.) are high risk for this injury. Pars injuries affect about 4 - 6% of the general population,[13 – 16] however, they are a particular issue in adolescence, accounting for up to 50% of back pain cases in the adolescent athlete.[17] Males seem to be more affected than females. The L5 vertebra seems to be the most affected vertebra followed by L4.[18]

Sacroiliac Joint (SIJ) disorder

SIJ disorder accounts for about 15% of lower back and buttock pain in the general population.[19] However, there appears to be little consensus on the best way to assess or treat SIJ disorder due to the complexity of its anatomy and biomechanics. Imaging is unable to determine differences between a symptomatic and asymptomatic patient. Traditionally, if there was ambiguity whether a patient's pain originated from the SIJ or not, a physician would inject the affected area with local anaesthetic. However, recent evidence suggests that the anaesthetic can diffuse out of the joint thus affecting other areas like nerve roots and local ligaments.[20]

It is worth noting that sacroiliitis is associated with seronegative spondyloarthropathies such as ankylosing spondylitis and Reiter's syndrome.
These conditions should always be considered in a young person presenting with SI Joint pain.

Clinical Features

Acute Nerve Compression

Acute nerve compression can present with sudden onset of low back pain which may or may not travel in a certain dermatomal distribution down the ipsilateral leg (radicular pain). Symptoms depend on the level of nerve where the compression is occurring as well as the direction of disk prolapse e.g. paracentral prolapse, posterolateral prolapse etc. Other symptoms would include paraesthesia in a dermatomal distribution and leg weakness of a particular myotome. Sometimes there is no back pain but pain in a particular dermatomal distribution. Symptoms can be aggravated by sitting, lifting, bending or coughing. Symptoms can be eased by lying flat. Examination can be difficult in the acute phase due to pain severity. On inspection, the patient may be leaning to one side. Restriction in all lumbo-sacral movements may be evident with a positive slump test and straight leg raise test on the affected side.[21] Knee (L3/4) and ankle (L5/S1) reflexes should be tested and compared to the contralateral side as should the plantar response. Myotome power should be assessed and compared to the opposite side. If there is a question/concern of cauda equina then additional examinations involving assessment of anal tone and saddle anaesthesia need to occur. It is important to establish if there are any clinical signs of spinal cord compression. This would be displayed by upper motor neurone signs like hyperreflexia and an upward plantar response (Babinski's).

Pars Interarticularis Injury

Symptoms include unilateral lower back pain +/- buttock pain which is aggravated by lumbar extension. Pars stress fractures may be asymptomatic. Examination findings may include localised tenderness on palpation of the affected side/area. Pain can be reproduced with rotation and extension while standing on the leg of the affected side (fitch test). The patient's posture may reveal hyperlordosis of the lumbar spine with very tight hamstrings. Neurological examination should be normal.

SI joint disorder

There appears to be no specific symptoms or examination techniques that is sensitive or specific for SIJ disorder. Classically SIJ pain is below L5 and is unilateral but it can on occasion be bilateral. SIJ pain can refer to the buttock, posterior thigh, groin and genital area. Pain can be aggravated by rolling in bed, going up & down stairs and long drives. Examination involves observing for a Trendelenburg gait and assessing leg length.[22] There can be localised tenderness over the Posterior Superior Iliac Spine (PSIS). There can also be asymmetrical movement of the PSIS on active S1 flexion. Positive findings can be seen on FABER (Flexion, Abduction and External Rotation), stork test, axial femoral compression test (highest specificity)[23] and thigh thrust test.[24] Joint degeneration of the SI joint can begin in a person as early as their twenties.[25]

Investigations

Acute Nerve Compression

In the absence of red flag signs on clinical history and examination then no investigations are required. MRI may be considered if the physician is considering referral for intervention e.g. injection or surgery or if symptoms persist.[26] Disc herniations are a common incidental finding in asymptomatic individuals.[27 - 29]

Pars Interarticularis Injury

Investigations may include x-ray, however in the acute stage an x-ray may not reveal the fracture. Chronic cases may reveal a "Scottie dog" appearance to the affected vertebrae on a 45-degree oblique x-ray view. If a pars injury is suspected despite a normal x-ray, further investigations like SPECT CT or MRI are warranted.

SI Joint pathology

Osteophytes can develop in the SI joint and impinge on the sciatic nerve. Traditionally X-ray has been the investigation of choice to assess the SI joint.[30] However, the complexity of the joint as well as the overlying structures mean that visualising the entire joint can be extremely difficult. In fact, there is no imaging modality available that can reliably identify the SI Joint as a source of a patient's pain.[19]

Management Options

Acute Nerve Compression

Generally, acute nerve root injuries can be managed conservatively as symptoms tend to resolve within a few weeks to months of symptoms.[31] Initial treatment involves managing pain and restoring function. Movement is encouraged from the outset unless the pain is very severe, in which case a few days of bed rest may be warranted. Oral analgesia can be considered during conservative management as a short-term adjunct. These may include paracetamol, NSAIDS, opiates or neuropathic analgesics. These should be prescribed for as short a time as possible and their side effect profile needs to be considered before prescribing.[32] Time should be spent educating the patient on the natural history of this condition. A management plan needs to be agreed and follow up is advised. Epidural injections are considered in those refractory to conservative management.[33] Surgery is considered if neurological symptoms/signs progress or don't resolve. At one year, outcomes of surgery versus conservative management are similar in those with lumbar disc herniation.[34,35]

Pars Interarticularis Injury

There is a lack of consensus on the treatment of pars interarticularis stress fractures. A period of relative rest is generally accepted. The use of antilordic bracing is controversial.[36] Management involves avoiding the aggravating activity, hamstring & gluteal stretching, strengthening of abdominal muscles (especially transversus abdominus and multifidus)[37] and back extensors. Unilateral defects are more likely to heal than bilateral ones.

In the absence of early detection or if the injury is inappropriately managed, then pars stress fractures run the risk of progressing to an open fracture (spondylolysis) or anterior translation of that particular vertebra (spondylolisthesis) if bilateral pars fractures have occurred. In order to prevent recurrence, techniques in athletes should be reviewed in order to reduce the amount of hyperextension.

SI Joint Disorder

Treatment options include managing initial pain, addressing biomechanical factors, strength training and addressing any muscle imbalances. An example of addressing the biomechanical factors may be the use of appropriate footwear for leg length discrepancy so that equal force can be distributed throughout the SI joint. Core muscle strengthening should be part of the treatment protocol. The focus of the strengthening programs should be not only on the SI joint but also on the pelvis and lumbar spine.[38]

Injecting the SIJ with a combination of steroid and local anaesthetic under guidance is a treatment option if conservative measures have failed. There have been numerous studies that have shown the analgesic benefit of such an intervention which can last between 6 and 12 months.[39 – 47] Prolotherapy involves injecting a sclerosant into the dorsal interosseus ligament, which is extra-articular. This is indicated with a hypermobile SIJ.[48]

References

1. Daniels, J. M., Pontius, G., El-Amin, S., Gabriel, K. Evaluation of Low Back Pain in Athletes. *Sports Health* 2011;*3*(4):336–345.2. Moore KL, Agur AMR. Essential Clinical Anatomy. (2nd Ed.) USA: Lippincott Williams & Wilkins, 2002. 3. Forst S.L., Wheeler M.T., Fortin J.D., Vilensky J.A. The sacroiliac joint: Anatomy, physiology and clinical significance. Pain Physician 2006;9(1):61-68. 4. Franklyn-Miller F, Falvey E, McCrory P, Brukner P. Clinical Sports Anatomy. Sydney: McGraw-Hill; 2011. 5. Porter RW, Hibbert C, Evans C. The natural history of root entrapment syndrome. Spine 1984;9:418-21. 6. Kunogi J, Hasue M. Diagnosis and operative treatment of intraforaminal and extraforaminal nerve root compression. Spine 1991;16:1312-20. 7. Orita S, Inage K, Eguchi Y, Kubota G, Aoki Y, Nakamura J, Matsuura Y, Furuya T, Koda M, Ohtori S.Lumbar foraminal stenosis, the hidden stenosis including at L5/S1. Eur J Orthop Surg Traumatol 2016;26:685-93. 8. Brukner P, Khan K. Clinical Sports Medicine. Australia: McGraw-Hill; 2012. 9. Vanhoenacker FM, Maas M, Gielen JLMA. Imaging of orthopedic sports injuries. Berlin: Springer-Verlag; 2007.10. Tenforde AS, Carlson JL , Chang A, et al. Association of the female athlete triad risk assessment stratification to the development of bone stress injuries in collegiate athletes. Am J Sports Med 2017;45:302–10. 11. Maquirriain J, Ghisi JP. The incidence and distribution of stress fractures in elite tennis players. Br J Sports Med 2006;40:454–9. 12. Elliott BC. Back injuries and the fast bowler in cricket. J Sports Sci 2000;18:983–91.13. Fredrickson B, Baker D, McHolick WJ, et al. The natural history of spondylolisthesis and spondylolysis. J Bone Joint Surg Am 1984;66(5):699–707.14. McCarrol JR, Miller JM, Ritter MA. Lumbar spondylolysis and spondylolisthesis in college football players. A prospective study. Am J Sports Med 1986;14:404–6.15. Hensinger RN, Arbor A. Spondylolysis and spondylolisthesis in children and adolescents. J Bone Joint Surg Am 1989;71(7):1098–107.16. Harvey CJ, Richenburg JL, Saifuddin A, et al. Pictorial review: the radiological investigation of lumbar spondylolysis. Clin Radiol 1998;53(10):723–8.17. Micheli LJ, Wood R. Back pain in young athletes. Significant differences from adults in causes and patterns. Arch Pediatr Adole*sc Med*. 1995;149(1):15-18. 18. Kurd MF, Patel D, Norton R, et al. Nonoperative treatment of symptomatic spondylolysis. J Spinal Disord Tech 2007;20:560–4.19. Dreyfuss P, Dreyer SJ, Cole A, Mayo K. Sacroiliac Joint Pain, Journal of the American Academy of Orthopaedic Surgeons 2004;12(4):255-265. 20. Schwarzer AC, Aprill CN, Bogduk N. The sacroiliac joint in chronic low back pain. Spine 1995;20:31–7.21. Koes BW, van Tulder MW, Peul WC. Diagnosis and treatment of sciatica. BMJ. 2007;334(7607):1313-1317. 22 .Schuit D, McPoil TG, Mulesa P. Incidence of sacroiliac joint malalignment in leg length discrepancies. J Am Podiatr Med Assoc 1989;79:380–3.23. Laslett M, Williams M. The reliability of selected pain provocation tests for sacroiliac joint pathology. Spine 1994;19:1243–9.24. Malanga G, Nadler S. Musculoskeletal physical examination. An evidence-based approach. 1st ed. Philadelphia: Elsevier Mosby; 2006.25. Shibata Y, Shirai Y, Miyamoto M. The aging process in the sacroiliac joint: helical computed tomography analysis. J Orthop Sci 2002;7:12–18. 26. Jensen RK, Kongsted A, Kjaer P, Koes B. Diagnosis and treatment of sciatica BMJ 2019; 367 :l6273.27. Stadnik TW, Lee RR, Coen HL, et al. Annular teas and disk herniation: prevalence and contrast enhancement on MR images in the absence of low back pain or sciatica. *Neuroradiology* 1998;206:49–55.28. Jensen MC, Brant-Zawadzki MN, Obuchowski N, et al. Magnetic resonance imaging of the lumbar spine in people without back pain. *N Engl J Med* 1994;331:69–73. 29. Weishaupt D, Zanetti M, Hodler J, et al. MR Imaging of the Lumbar Spine: Prevalence of Intervertebral Disk Extrusion and Sequestration, Nerve Root Compression, End Plate Abnormalities, and Osteoarthritis of the Facet Joints in Asymptomatic Volunteers. *Radiology* 1998;209:661–6. 30. Vogler JB 3rd, Brown WH, Helms CA, *et al*. he normal sacroiliac joint: a CT study of asymptomatic patients. *Radiology* 1984;151:433–7.31. NICE. *Low back pain and sciatica in over 16s: assessment and management.* 2016. Available from: www.nice.org.uk/guidance/ng59. 32. NICE. *Sciatica* (*Lumbar radiculopathy)* Available from: https://cks.nice.org.uk/sciatica-lumbar-radiculopathy#!scenario.33. Stafford MA *et al*. Sciatica: a review of history, epidemiology, pathogenesis and the role of epidural steroid injection in the management. *British Journal of Anaesthesia* 2007;99(4):461-473 34. Peul WC *et al.* Surgery versus prolonged conservative treatment for sciatica. *N Engl J Med* 2007; 356:2245-56. 35. Peul WC *et al*. Prolonged conservative care versus early surgery in patients with sciatica from lumbar disc herniation: cost utility analysis alongside a randomised controlled trial. *BMJ*. 2008;336(7657):1351-4.36. McCleary MD, Congeni JA. Current concepts in the diagnosis and treatment of 180 spondylolysis in young athletes. *Curr Sports Med Rep* 2007;6:62–6. 37. O' Sullivan PB, Phyty GD, Twomey LT *et al*. Evaluation of specific stabilizing exercises in the treatment of chronic low back pain with radiologic diagnosis of spondylolysis or spondylolisthesis. *Spine* 1997;22(24):2959-67 38. Mooney V, Pozos R, Vleeming A, *et al*. Exercise treatment for sacroiliac pain. *Orthopedics* 2001;24:29–32. 39. Maugars Y, Mathis C, Vilon P, Prost A. Corticosteroid injection of the sacroiliac joint in patients with seronegative spondylar- thropathy. *Arthritis Rheum* 1992;35:564–8. 40. Gunaydin I, Pereira PL, Daikeler T, *et al*. Magnetic resonance imaging guided corticosteroid injection of the sacroiliac joints in patients with therapy resistant spondyloarthropathy: a pilot study. *J Rheumatol* 2000;27:424–8. 41. Hanly JG, Mitchell M, MacMillan L, *et al.* Efficacy of sacroiliac corticosteroid injections in patients with inflammatory spondyloarthropathy: results of a 6 month controlled study. *J Rheumatol* 2000;27:719 –22. 42. Pereira PL, Gunaydin I, Duda SH, *et al.* Corticosteroid injections of the sacroiliac joint during magnetic resonance: preliminary results [in French]. *J Radiol* 2000;81:223–6. 43. Pereira PL, Gunaydin I, Trubenbach J, *et al*. Interventional MR imaging for injection of sacroiliac joints in patients with sacroiliitis. *AJR Am J Roentgenol* 2000;175:265–6. 44. Ojala R, Klemola R, Karppinen J, *et al.* Sacro-iliac joint arthrography in low back pain: feasibility of MRI guidance. *Eur J Radiol* 2001;40:236–9. 45. Karabacakoglu A, Karakose S, Ozerbil OM, Odev K. Fluoroscopy-guided intraarticular corticosteroid injection into the sacroiliac joints in patients with ankylosing spondylitis. *Acta Radiol* 2002;43:425–7. 46. Fischer T, Biedermann T, Hermann KG, *et al*. Sacroiliitis in children with spondyloarthropathy: therapeutic effect of CT- guided intra-articular corticosteroid injection [in German]. *Rofo* 2003;175:814 –21. 47. Boswell MV, Shah RV, Everett CR, *et al*. Interventional techniques in the management of chronic spinal pain: evidence-based practice guidelines. *Pain Physician* 2005;8:1–47.48. Cusi M, Saunders J, Hungerford B, *et al*.The use of prolotherapy in the sacroiliac joint. *British Journal of Sports Medicine* 2010;44:100-104.