Spondylolisthesis: a pictorial review

Spondylolisthesis is often poorly understood by clinicians and radiologists. Incorrectly perceived as being chiefly related to pars defects, in reality spondylolisthesis has multiple aetiologies with differing clinical implications. This article addresses the salient radiological findings in the assessment of spondylolisthesis.

Spondylolisthesis, derived from the Greek (spondylos – vertebra, olisthesis – slippage), is often poorly understood by clinicians. Misperceived as being chiefly related to pars defects, in reality spondylolisthesis has multiple aetiologies with differing clinical implications (Wiltse et al, 1976; Wiltse and Winter, 1983; Butt and Saifuddin, 2005). It is important to be familiar with the different types of spondylolisthesis and their imaging appearances as they may have important different clinical implications, e.g. neural compromise, and therefore different management and follow-up regimens. This article defines spondylolisthesis and explains the six main aetiological sub-types: dysplastic, isthmic, degenerative, traumatic, pathologic and iatrogenic (Wiltse et al, 1976; Wiltse and Winter, 1983). It then discusses the use of imaging in the management of spondylolisthesis, the methods used to grade spondylolisthesis on lateral lumbar radiographs and the rationale for performing additional imaging with use of relevant supportive imaging.

Anterolisthesis and retrolisthesis

Anterolisthesis is the anterior slippage of one vertebra over the one directly below it. In order for this to occur, there must be a failure of a specific part or functional unit of the vertebral posterior elements. If a vertebra slips backwards relative to another it is termed retrolisthesis.

Measuring the severity of spondylolisthesis

Spondylolisthesis can be objectively assessed on a lateral lumbar radiograph by either quantifying the magnitude of the slip or calculating the slip angle (*Figures 1* and *2*). The magnitude of the slip is calculated by one of two methods: as a percentage of the anterior displacement of the inferior endplate of the superior vertebral body over the superior endplate of the inferior vertebral body (Taillard method), or by dividing the inferior vertebral body into quarters and assigning a grade I–V for slips of 0–25% (I), 25–50% (II), 50–75% (III), 75–100% (IV) or greater than 100% (V) (Meyerding method). A slip of greater than 100% is also termed spondyloptosis (Danielson et al, 1988; Butt and Saifuddin, 2005).

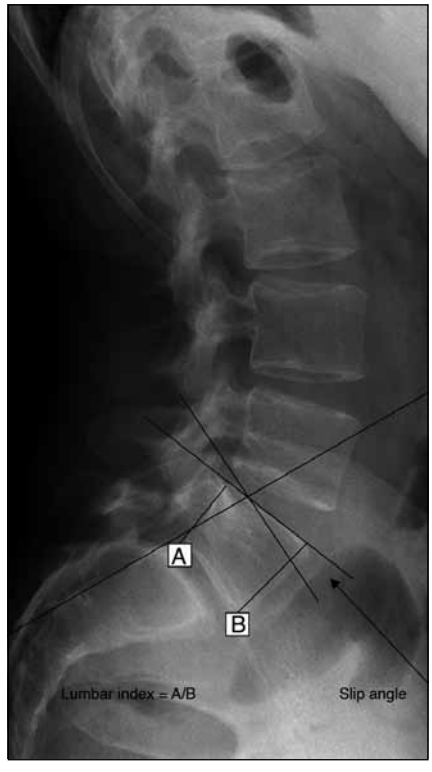
Measuring the slip angle (angle of kyphosis) is the most sensitive indicator of potential instability and clinical symptoms (Boxall et al, 1979; Danielson et al, 1989). It is defined as the angle between the superior endplate of L5 and a line perpendicular to the posterior border of the sacrum (Jinkins and Rauch, 1994; Ohmori et al, 1995; Butt and Saifuddin, 2005) (*Figure 2*). A high slip angle is associated with an increased incidence of instability, progression of the slip before and after fusion, and pseudoarthrosis.

A further marker of spondylolisthesis is the lumbar index, which is expressed as the quotient of posterior height divided by anterior height of the slipped vertebra. This measure is most valuable when used to assess progression of a slip in children.

In all cases, lateral radiographs should be obtained with the patient standing. Boxall et al (1979) showed an increased slip in 40% of patients and an increase in slip angle in 87% of patients when standing compared to sitting.

Figure 1. Sagittal radiograph showing pars defects at L5 producing a grade 1 isthmic (lytic) spondylolisthesis at L5/S1.

Figure 2. The slip angle and lumbar index are labelled.



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Although plain lateral lumbar radiographs can be used for assessment, multiplanar computed tomography and magnetic resonance imaging now play a central role in the assessment of spondylolisthesis. Some authorities advocate the use of dynamic flexion and extension views or axial-loaded magnetic resonance imaging (Saifuddin et al, 2003) before a slip can be considered 'stable'. Full assessment includes measuring the slip angle and lumbar index, in addition to measuring the magnitude of the slip with the Taillard or Meyerding method.

Classification of spondylolisthesis

Wiltse described six causes of spondylolisthesis on the basis of aetiology: dysplastic, isthmic, degenerative, traumatic, pathologic and iatrogenic (Wiltse et al, 1976; Wiltse and Winter, 1983).

Dysplastic (or congenital)

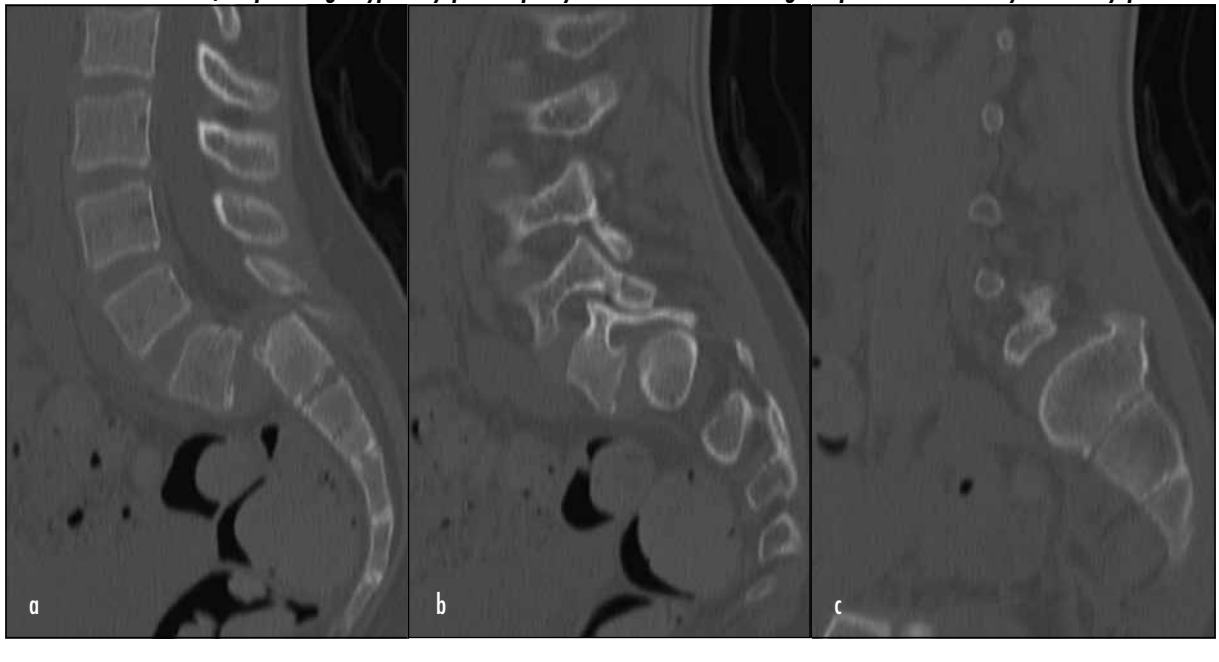
Congenital spondylolisthesis is usually the result of dysplastic facet joints, and is further subdivided into three groups:

1. Horizontal malorientation of facet joints as a result of dysplastic articular processes of L5 and S1. There is an association with spina bifida occulta, and the pars may be elongated and intact or fragmented. The status of the pars is best evaluated using multiplanar computed tomography (*Figures 3a–c*) or magnetic resonance imaging (*Figure 4*).
2. Sagittal malorientation of the facet joints, which tends to present in adulthood. The presence of an intact neural arch increases the likelihood of neural compression significantly in this subtype.
3. Other congenital anomalies, e.g. failed vertebral body formation, lumbosacral angular deformities.

Dysplastic spondylolisthesis represents up to 20% of all cases, and has a 2:1 female:male ratio. Facet dysplasia results in elongation and possible subsequent fracture of the pars (Hession and Butt, 1996), although this is a secondary effect of the dysplasia, rather than a primary pars defect as seen in isthmic spondylolisthesis (Leone and Lamont, 1999). As the neural arch is intact there is increased incidence of neural compression of either the cauda equina or exiting nerve roots with even low grade

Figure 4. Sagittal T2 weighted magnetic resonance shows the features of dysplastic spondylolisthesis with severe central canal stenosis below the slip and widening of anteroposterior canal dimensions above.

Figure 3. a–c. Sequential sagittal computed tomography images demonstrating the features of dysplastic spondylolisthesis with horizontally maloriented facets at L5/S1 producing a type A dysplastic spondylolisthesis. Note the elongated pars of L5 secondary to facet dysplasia.



slips. On an anteroposterior radiograph, forward slip of the L5 vertebra results in an axial view of the vertebra and a characteristic 'Napoleon's hat' sign. Owing to the high incidence of neural compromise, even patients with slips which are considered stable should be followed up every 4–6 months with serial imaging until skeletal maturity is reached.

Isthmic

The isthmic form is the most common type of spondylolisthesis, present in 8% of the population in adults under 50 years of age and is caused by pars interarticularis defects (Newman, 1975; Wiltse et al, 1975; Wiltse and Winter, 1983; Wiltse and Rothman, 1989). By definition, only bilateral pars defects can cause a vertebra to slip forward (*Figures 5a* and *b*). The basic pathophysiology is non-union of a stress fracture (Ulmer et al, 1994; O'Brien, 2003) and these slips generally do not progress post adolescence (Newman, 1975; Wiltse et al, 1975; Wiltse and Winter, 1983; Wiltse and Rothman, 1989; Tsirikos and Garrido, 2010). Radiologically there is characteristic widening of the canal (*Figures 6a* and *b*).

These defects are not present at birth. It is likely that an erect posture, combined with the normal 40–60° of lumbar lordosis, exerts constant mechanical stress on the pars. In support of this theory, the condition is not present in quadripeds, and usually manifests itself by adolescence. The Wiltse classification further subdivides isthmic causes into three subtypes, caused by:

1. An early stress fracture in a congenitally weakened pars
2. Repeated microfractures which heal leading to elongation of the pars
3. An acute fracture of the pars interarticularis which is exceedingly rare.

Isthmic disease caused by an early stress fracture in a congenitally weakened pars can be a combination of an abnormal pars and repeated mechanical stress. Hence, there is an increased incidence in ballet dancers, runners and sports involving repetitive lumbar extension.

Both disease caused by an early stress fracture and that resulting from repeated microfractures which heals leading to elongation of the pars have richly innervated abnormal fibrous tissue, accounting for the painful nature of each condition. The elongated pars is a consequence of repeated healing microfractures and not a congenital abnormality.

Isthmic spondylolisthesis has a strong genetic component with an incidence of up to 35% in families where one member has spondylolysis or spondylolisthesis, compared to 5% in the general population (Newman, 1975; Wiltse et al, 1975; Wiltse and Winter, 1983; Wiltse and Rothman, 1989). It has a particularly high incidence in certain ethnic populations, e.g. Inuit Indians and Alaskan natives. Spondylolysis has a male to female ratio of 2:1, but females are more likely to progress to high-grade slips than males.

Most patients with isthmic defects are asymptomatic, but if they do present then this is usually with back pain (related to the pars itself or the disc) and leg pain (related to the L5/S1 disc) or a combination of the two.

The configuration of the neural foramen changes from oval to bilobed, resulting in exiting nerve root compression in the superior recess of the foramen. This occurs as a result of caudal translation of L5 relative to S1 (Jinkins et al, 1992). In contrast to dysplastic and degenerative spondylolisthesis, there is no central canal or lateral recess stenosis as the neural canal is effectively decompressed as a result of the isthmic defect. A similar phenomenon in the cervical spine accounts for the relatively low incidence of cord compromise in cases of traumatic C2 anterolisthesis (hangman's fracture).

Although spondylolisthesis can be evaluated in terms of the grade of slip, slip angle, lumbar index and sagittal plane deformity, in usual clinical practice, management depends entirely on patient symptoms. The majority of cases of truly asymptomatic pars defect with no slip

Figure 5. a. Sagittal radiograph demonstrating isthmic spondylolisthesis and pars defect at L5/S1. b. Sagittal computed tomography in the same patient following surgical fusion.

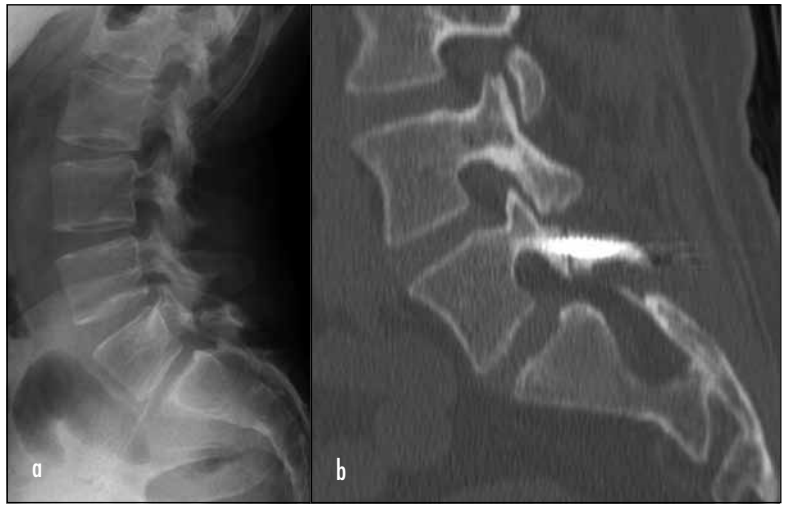


Figure 6. (a) and (b) Sequential sagittal T2 magnetic resonance sequences demonstrating isthmic anterolisthesis with characteristic widening of the spinal canal. There is an isthmic spondylolisthesis of L5/S1 associated with transitional vertebra at S1, which is lumbarised.

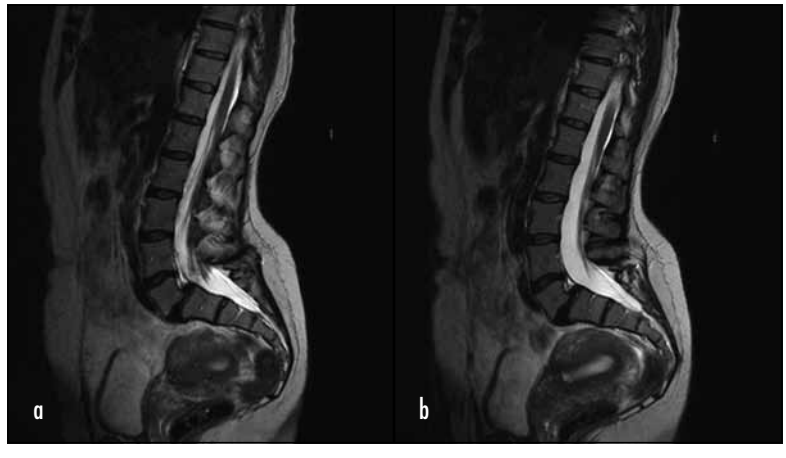


Figure 7. a. Axial and (b) sagittal T2 magnetic resonance images of L3/4 degenerative spondylolisthesis showing sagittal malorientation of facet joints and severe lateral recess stenosis where there is impingement of L4 nerve root. This may progress to central canal and foraminal stenosis. Degenerate facets are additional source of significant pain and morbidity.

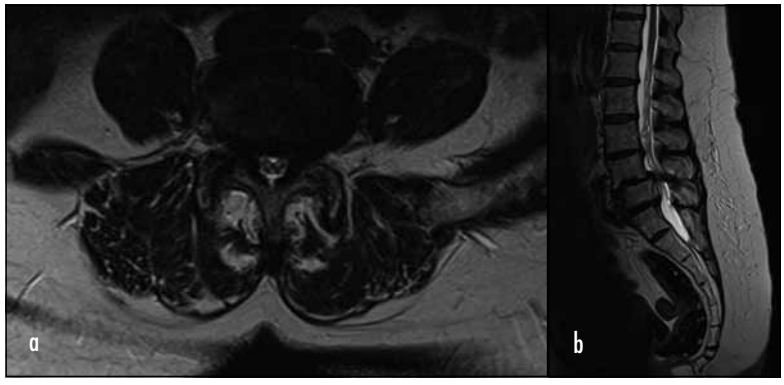


Figure 8 (a) and (b) Sequential sagittal computed tomography images demonstrating a traumatic spondylolisthesis in polytrauma with multi-level posterior element fracture-dislocations.

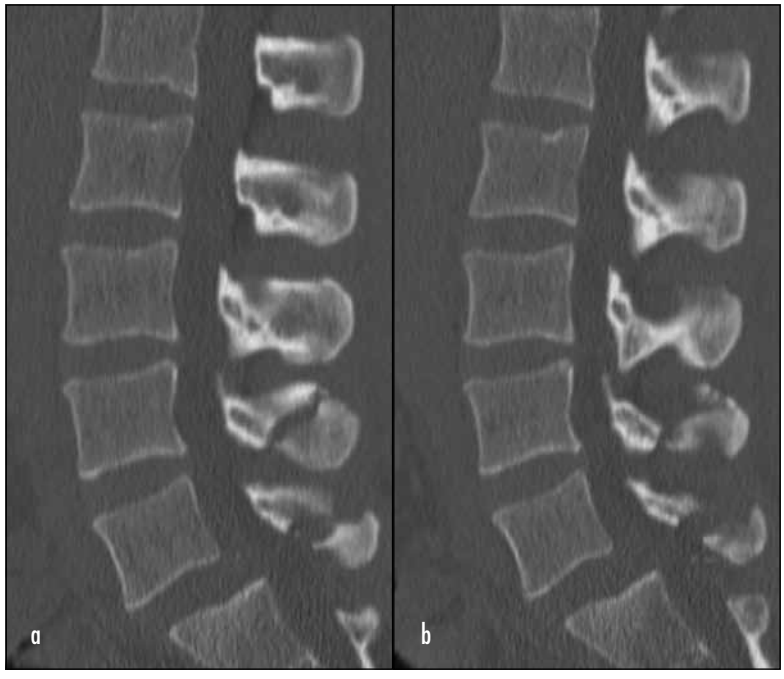
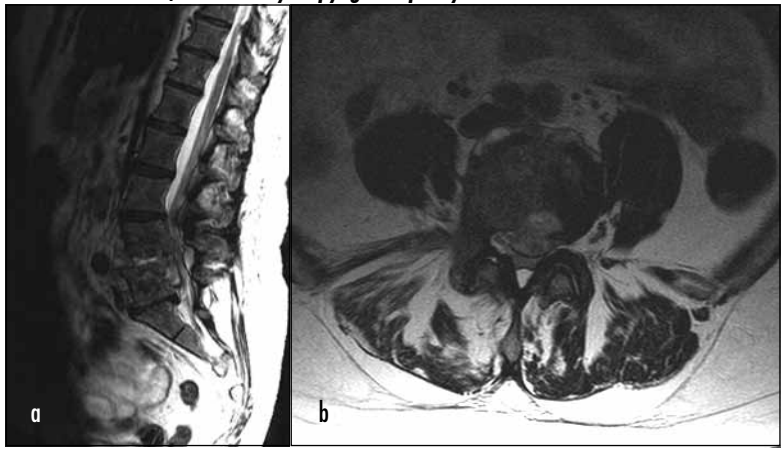


Figure 9. a. Sagittal and (b) axial T2 weighted images demonstrating a pathological retrolisthesis at L4/5 secondary to pyogenic spondylodiscitis.



require no treatment, although 3–6-monthly follow-up is recommended to determine stability of the slip until skeletal maturity. If no progression is present, the patient can be safely discharged.

Degenerative

This is the most common cause of spondylolisthesis in patients over the age of 50 years. It is most common at the L4/5 level and in women. The basic pathophysiology is a sagittal malorientation of facet joints in association with degenerative disc disease, flaval hypertrophy and facet joint osteoarthritis (*Figure 7a* and *b*). In contradistinction to lytic causes, the neural arch is intact and the anteroposterior diameter of the neural canal is narrowed (Rosenberg, 1975; Weinstein et al, 2009; Herkowitz, 2010). This leads to a much higher incidence of cauda equina and nerve root compression in the lateral recess (Huang et al, 2009). The degree of slip in degenerative causes is rarely greater than grade I. However, neural compression is relatively common. When compared to elderly patients without spondylolisthesis, those with the condition have a significantly greater incidence of neurogenic symptoms and functional limitations (Ulmer et al, 1994).

Traumatic

This refers to spondylolisthesis secondary to traumatic fracture or dislocation of the posterior elements at any site other than the pars interarticularis, e.g. laminae, pedicles, facet joints (*Figure 8*). These injuries are extremely uncommon because of the high energy required to disrupt an otherwise normal neural arch (Fabris et al, 1999; Miyamoto et al, 2004). It is worth remembering that, even in the setting of acute trauma, spondylolisthesis is far more likely to be the result of incidental pars defects than a true traumatic fracture-dislocation. Owing to the high energy required, full spinal imaging is mandatory because of the high association of additional injuries at other levels.

Pathologic

Pathologic spondylolisthesis is further classified into two types: type A refers to causes relating to generalized bony disease, e.g. osteoporosis, osteomalacia (where microfractures and remodelling result in segmental instability), Paget's disease or osteogenesis imperfecta (caused by elongation of the pedicle). Type B refers to focal pathology affecting the pars directly leading to pathological fracture, e.g. infection (*Figures 9a* and *b*) or neoplasm. Management is of the underlying disease process and complications.

Iatrogenic

Also called postoperative spondylolisthesis, this is usually the result of surgical intervention, in particular excessive removal of the posterior elements of the pars or other spinal supporting structures after laminectomy performed to correct disc herniation or nerve compression caused by spinal stenosis. The key causes are decompressive laminectomy, medial facetectomy without fusion and proximal

Figures 10. a. Sagittal, (b) axial computed tomography images and (c) sagittal T2 weighted magnetic resonance demonstrating laminectomy without fusion resulting in retrolisthesis.

junction failure above an instrumented fusion (*Figures 10a–c*). The incidence of iatrogenic spondylolisthesis is approximately 3–5% of cases of treated spondylolisthesis.

Conclusions

Of the six types of spondylolisthesis, the most common causes are isthmic, degenerative and dysplastic. Dysplastic or degenerative spondylolisthesis is associated with a higher incidence of neural compromise because the patient has an intact neural arch. This allows differentiation from isthmic spondylolisthesis in which the neural canal is widened, and consequently central canal stenosis is not a usual feature. Undiagnosed dysplastic and high-grade isthmic spondylolisthesis needs referral and follow up until skeletal maturity to prevent unnecessary morbidity. BJHM

Conflict of interest: none.

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KEY POINTS

- Spondylolisthesis is a displacement or 'slippage' of the vertebral column (usually anteriorly) as the result of a dissolution of continuity or defect of the pars interarticularis of the neural arch of a vertebra.
- Spondylolisthesis may occur as the result of multiple aetiologies, all of which lead to loss of the stability offered by the locking mechanism of the articular processes that permit the superior vertebrae to slide anteriorly over the inferior vertebrae.
- Causes may be classified as dysplastic (or congenital), isthmic (or spondylolytic), degenerative, traumatic, pathologic, or iatrogenic (e.g. postoperative).