Spondylolysis and spondylolisthesis in children and adolescents: current concepts and treatment

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Abstract

Spondylolysis and spondylolisthesis usually affect the lumbosacral junction and are the most common causes of back pain in children and adolescents. In severe forms the deformity may be progressive and produce neurological deficits, sagittal imbalance and physical disability. The mainstay of treatment in early stages is conservative, while surgery is offered in persistently symptomatic patients or progressive deformities. Severe forms of spondylolisthesis require spinal stabilization and deformity correction; however there is lack of consensus regarding the optimal operative management. The aim of this article is to present the current concepts on the development, progression and classification of this condition, to illustrate the clinical and imaging diagnostic approaches and describe the principles of surgical treatment in all stages of the disease.

Keywords lumbosacral fusion; pars defect; pars repair; sagittal imbalance; spinopelvic parameters; spondylolisthesis; spondylolysis; spondyloptosis

Introduction

Spondylolysis refers to an acquired defect of the pars interarticularis.[1] Spondylolisthesis is the forward translation of one vertebral segment over the one beneath it. In approximately 90% of patients, spondylolysis occurs at the junction of the sacrum and the mobile lower lumbar spine.[1,2] Spondylolysis is never present at birth.[1,3,4] Genetic predisposition among certain ethnic groups (20–50% in Northern Canada Inuits), increased familial incidence and a parallel occurrence of spondylolysis and spina bifida occulta have been reported.[2,5]

Lumbosacral spondylolisthesis can have significant impact on quality of life, causing low back pain, hamstring tightness and neurological abnormalities.[6] In severe forms it may be progressive and produce global sagittal and spinopelvic imbalance with consequent disability. Conservative management is the mainstay of treatment, however in the presence of persistently symptomatic or progressive deformities surgical treatment is recommended.[1,2]

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Pathophysiology

Spondylolysis

The lumbosacral junction is the commonest area to develop spondylolysis due to its unique anatomy and biomechanics. The forward-facing inferior L5 facets lock against the backward-facing sacral facets, preventing anterior displacement of L5 vertebra on the sacrum. The pars interarticularis being the link between the mobile spine and the L5 posterior elements resists compressive, tensile and bending forces. Lumbar hyperextension creates a pincer mechanism on the L5 pars between the L4 inferior facets proximally and the S1 superior facets distally. Repetitive flexion/extension movements considerably increase the stresses on the L5 pars leading to fatigue fracture, subsequent spondylolysis and pain.[3] Congenital anatomical variations or small L4-S1 distance may predispose to L5 spondylolysis.

Spondylolisthesis

In bilateral pars defects (isthmic-traumatic spondylolisthesis) the vertebral body loses connection to its posterior elements (Figure 1). The static stability provided by this anatomical restraint is lost and the oblique orientation of the lumbosacral junction predisposes to forward slippage of L5 on S1. Shear forces are transferred to the L5/S1 disc and the adjacent growth plates.[4,7] The stability becomes dynamic relying on interaction of the muscles and the integrity of the disco-ligamentous complex.[3]

The growth plate is the weakest link in the immature spine[4] undergoing distinct maturation phases until its final ossification around the age of 10–15 years; the cartilaginous, apophyseal and epiphyseal stages. The cartilaginous stage provides the least resistance to shear forces when spondylolisthesis is more likely to occur (80%).[4] No slippage occurs along the growth plate after ossification of the apophyseal ring onto the vertebral body (epiphyseal stage).[4]

The stresses exerted on the growth plate during lumbar flexion/extension may also lead to physeal stress fracture and subsequent spondylolisthesis.[4] The disturbance of the growth plate and a fracture of the anterior apophyseal ring cause dysplasia of the superior S1 endplate with rounded shape of the sacral dome, remodelling of L5 vertebra leading to trapezoidal shape, facet insufficiency and disc space narrowing.[4,6,8] This anatomy predisposes to further olisthesis of L5 onto S1[6,8] (Figure 2b).

Adult-type olisthesis or olisthesis after final ossification of the endplate do not cause secondary deformities of L5 and S1 endplates.[4,7] The adult-like olisthesis happens at the level of the intervertebral disc due to its degeneration and dysfunction (Figure 2a).

In case of congenitally deficient S1 facets (dysplastic spondylolisthesis) the locking mechanism preventing the slippage is lost, however the connection with the posterior elements is maintained.[7] Contrary to isthmic type, in dysplastic spondylolisthesis the posterior arch follows the olisthesis of the vertebral

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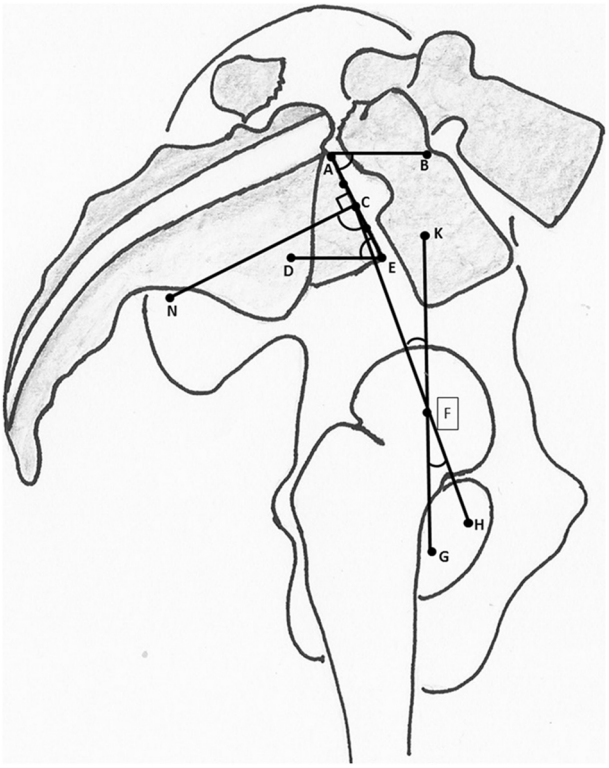

Figure 1 Sagittal pelvic parameters based on standing radiograph. Definitions: AB ⊥ DE (Horizontal line), AB ⊥ KG (vertical line), AE: (sacral endplate, AC = CE), AE ⊥ CN, F: centre of femoral heads. Angles: NCF = pelvic incidence (PI); BAE = AED = sacral slope (SS). AFK = GFH = pelvic tilt (PT). PI is an anatomical parameter for each individual which remains unchanged following skeletal maturity whereas PT and SS are variable and depend on the position of the pelvis (PI = SS+PT).

body causing stenosis of the canal and predisposing to cauda equina compression.[7]

Natural history

The long-term natural course of spondylolisthesis is associated with a) stretching of the anatomical restraints (anterior/posterior longitudinal ligaments, intervertebral disc, epiphyseal plate), b) disc degeneration and c) spontaneous stabilization of the displaced segment occurring between 13 years and 17 years of age without further slippage after skeletal maturity.[3,9] The tendency for segmental displacement progression is greatest around pubertal growth spurt.[4]

Predictors of slip progression are: a) female gender, b) prepubertal age, c) trapezoidal L5, d) dysplastic dome-shaped, vertical sacrum with sagittal rotation, e) slip angle (SA) $\geq 10^{\circ}$ and f) high-grade slip (>50%) (Figure 2).[10]

Spinopelvic and sagittal balance

Regardless of pain symptoms, global sagittal balance and spinopelvic parameters remain within normal limits in low-grade isthmic spondylolisthesis.[3] High-grade spondylolisthesis can cause lumbosacral kyphosis resulting in global sagittal deformity affecting the spine and pelvis[6] (Figure 2). There is always secondary deformity or dysplasia of the lumbosacral junction at the time of diagnosis.[7] Increased lumbar lordosis and marked pelvic tilt by contracting the hamstrings in an attempt of the body to resist forward displacement of the spinal column and re-establish skeletal weight-bearing line.[3] Hamstring contraction drives pelvic retroversion and causes knee flexion, while at the same time it promotes further vertebral displacement (Figure 2).[3] The higher the degree of anterior vertebral displacement the greater the risk of neurological injury to the cauda equina or the exiting nerve roots at the affected levels.[3]

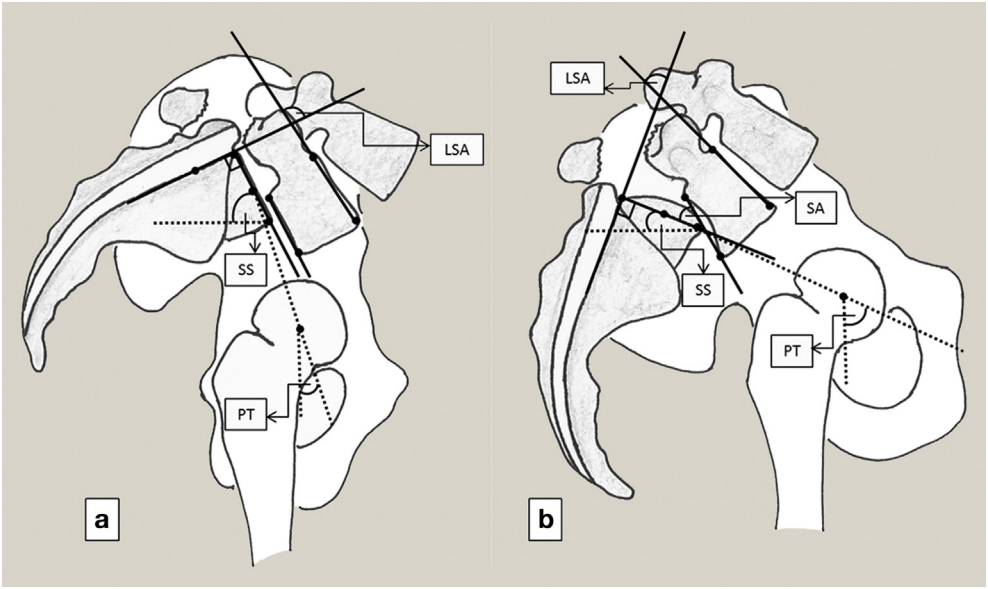

Figure 2 Spinopelvic alignment in spondylolisthesis based on standing radiograph. Definitions: PT: pelvic tilt, SS: sacral slope, SA: slip angle, LSA: lumbosacral angle. (**a**) Grade III lytic spondylolisthesis without lumbosacral kyphosis, as LSA = 100° and SA = 0°. This is classified as 'balanced' pelvis.[11] (**b**) Grade III lytic spondylolisthesis with dysplastic changes (e.g. domed-shaped sacral endplate and trapezoidal L5 vertebra) producing lumbosacral kyphosis, as LSA <90° and SA = 30°. PI is the same as in pelvis (**a**), however SS is decreased and PT is increased in pelvis (**b**) indicating compensatory pelvic retroversion to maintain upright posture. This is classified as 'unbalanced' pelvis and reduction prior to instrumented fusion should be considered.[11]

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