The Types and Treatments of Spondylolisthesis

ABSTRACT

Spondylolisthesis is a common finding in the adult patient but seldom requires surgical intervention. Up to 18% of the population show spondylolisthesis on spinal imaging with the vast majority requiring little or no treatment. This review explores the aetiology of spondylolisthesis, alongside key findings in the history and examination that should prompt referral, as well as presenting the evidence supporting surgical treatment. Spondylolisthesis affects patients at nearly every stage of life and understanding why and how to manage this common problem will aid in counselling patients and making the right referrals.

KEYWORDS: Spondylolisthesis, spondylosis, back pain, radicular pain, neurogenic claudication, spinal stenosis

CME Pre-test Quiz

Introduction

Spondylolisthesis is the pathologic translation forward or backward of one vertebra over another. It is a common condition with up to 18% of adults showing some degree of slippage on MRI of the lumbar spine.[1] In this review, the aetiology and classification of spondylolisthesis in the adult patient will be described, along with treatment options and clinical considerations for surgery.

Although spondylolisthesis can affect two vertebrae in any section of the back, it usually affects the lumbar spine. The lordotic nature of the lumbar spine in combination with the longitudinal pull of the paraspinal muscles and gravity work to draw the cranial vertebra anteriorly over the caudal vertebra, making antero-listhesis the most common deformity. Due to the slope of the facet joints, minimal, asymptomatic retro-listhesis is an inevitable result of disc narrowing and is not discussed in this article.

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The consequences of anterior translation are varied. In many cases there are no symptoms so the finding is incidental while in other patients back pain and symptoms of neural compression are evident.

Spondylolisthesis has a range of causes with varying frequencies. In 1976 Leon Wiltse et al. created the most referenced classification (Table 1).[2,3]

Type III, degenerative spondylolisthesis, is the most common. The forward slip results from a degenerative failure of the structures responsible for preventing translation. Classically it is found most often in middle-aged women at the L4-5 level.

Degenerative spondylolisthesis is generally thought to be the result of a cascade described by Kirkaldy-Willis.[4] In this sequence of events, disc degeneration leads to a loss of disc height and stability, decreasing the intervertebral space and increasing the stress on the facet joints. As a result, the joints remodel, altering the alignment of the articular surfaces from a transverse to a more sagittal plane and allowing the joint to sublux. The superior vertebra slides forward. The adjacent ligamentous structures, the joint capsules and ligamentum flavum, hypertrophy. Abnormal movement and facet degeneration cause back pain. The malalignment of the vertebrae narrows the spinal canal producing focal spinal stenosis and giving rise to neurogenic claudication.

Depending upon the degree of degeneration, amount of instability and the extent of the canal narrowing, patients present with a disparate set of symptoms. In many cases, spondylolisthesis remains asymptomatic but as the condition progresses, mechanical back pain predominates. In more advanced cases the deformity caused by the

Table 1: The Wiltse classification of spondylolisthesis

Type I	Dysplastic—a congenital abnormality of the posterior elements
Type II	Isthmic—a deficient, fractured, or elongated pars interarticularis
Type III	Degenerative—degeneration of disc and facets causing instability of the motion segment
Type IV	Traumatic—a fracture of pedicle, facet or lamina (but not the pars, which would be Type II)
Type V	Pathologic—neoplasia or bone pathology causing failure of the facet complex
Type VI	Iatrogenic—excessive removal of posterior elements or disc

forward slip of the superior vertebra constricts the spinal canal enough to compress the neural elements producing radicular pain or neurogenic claudication.

The physical examination may show only mechanical back pain, that is typical back dominant pain with specific movements or positions. Even in the later stages, when neural compression is present, as with other causes of neurogenic claudication the neurological examination after the patient has been sitting may be normal.

In younger patients with Type I and II spondylolisthesis, the spinal canal increases in size because the defect or deficiency in the pars interarticularis allows the anterior and posterior portions of the vertebra to separate. For this reason, compression producing neurogenic claudication is not part of the symptom complex. But compensation for the altered spinal alignment can cause other problems. To compensate for the spinal malalignment, the pelvis tilts backwards (retroverts). Because of this abnormal posture, patients develop tight hamstrings and, with reduced hip extension, exhibit a waddling gait. Attempting to alter their posture causes back pain. The spondylolisthesis occasionally impinges on an exiting nerve root producing radicular pain.

Diagnosing spondylolisthesis is usually straightforward. The deformity is visible on a standing radiograph and, although is it not routinely required, the deformity can be exacerbated by flexion views. A CT scan, which studies the bony anatomy, aids in classifying the type of spondylolisthesis and therefore directs the treatments offered. An MRI gives the best look at the soft tissues and thus the degree of neural compromise. It is most helpful with the degenerative Type III. Remember that patients with radicular pain or claudication may have an unrelated and asymptomatic spondylolisthesis.

Additional investigations can be useful. Single Photon Emission Computed Tomography (SPECT) is a technique that uses a gamma ray-emitting radioisotope to identify the areas of high metabolic activity, such as active degeneration and remodelling. In the case of Type II isthmic spondylolisthesis, it can demonstrate subtle pars defects not visible on conventional imaging. Nerve conduction studies can isolate compromised nerve roots not clearly identified through the history and examination.

Generally, making the diagnosis is not a problem. The challenge is correlating symptoms with the correct pathology to offer treatment with the best chance of success.

Treatment

The treatment of spondylolisthesis depends on the aetiology and the symptoms. Asymptomatic cases


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require nothing beyond reassurance and education about possible future complaints. When symptoms correlate with the investigations, there are several routes of treatment to consider.

Dysplastic spondylolisthesis

A dysplastic aetiology is when the morphology of the posterior elements is abnormal (but not fractured) and, as a result, spondylolisthesis occurs. Dysplasia is responsible for up to 21% of spondylolistheses, with a predilection for female patients of 2:1.[5] The treatment is generally surgical but the need for an operation depends on the grade of the slip. High grade deformities show a 'trapezoidal' L5 vertebra with a domed S1 endplate and a retroverted pelvis, as the patient tries to compensate for the deformity. High grade slips are much more likely to progress and become symptomatic than low grade deformities which do not exhibit these features and where conservative treatment is the mainstay.

Isthmic spondylolisthesis

This type is twice as common in male patients as in female patients, and has been long recognised as a common aetiology for spondylolisthesis, usually affecting the L5/S1 level in adolescents or young adults.[6] It occurs as a failure of the pars interarticularis. The most widely held theory suggests that this arises from a fatigue failure or stress fracture in response to repetitive injury. This accounts for the higher-than-expected incidence in gymnasts or interior linemen in football who experience repeated forced extension of the lumbar spine.

Healing is possible and isthmic spondylolisthesis can be treated with strict activity modification that limits hyperextension to promote bony union, or asymptomatic fibrous non-union.[7,8] Bracing has a role but the advantage of a rigid versus a soft brace remains debated.[9,10] Up to 84% of patients with isthmic spondylolisthesis will make a good recovery without surgery.[10]

Traumatic, pathologic, and iatrogenic spondylolisthesis

These types are rarely the presenting diagnosis to the primary care provider, as other symptoms predominate. Unless the comorbidities prevent safe treatment, surgery is almost always required.

Degenerative spondylolisthesis

Degenerative spondylolisthesis presents a wide permutation of symptoms. Mechanical back pain precedes and may accompany neurogenic claudication or radicular pain. In contrast to dysplastic or isthmic spondylolisthesis where the pars is deficient and the spinal canal capacious, here it is the facets and disc that fail. This pathology leaves the canal intact and therefore able to constrict the neural elements.

Since degenerative spondylolisthesis is most common between L4 and L5, it usually compresses the exiting L4 and traversing L5 roots, as well as the cauda equina.[11] Because of the residual hold of the facet joints, the spondylolisthesis rarely progresses beyond 30% translation so cauda equina syndrome is rare. Nonetheless, the spondylolisthesis can be the source of significant neurological distress.

The treatment of degenerative spondylolisthesis varies widely. Although the SPORT trial showed that surgery produced the best outcomes at 2 and 4 years, if appropriate all patients should have the opportunity for lifestyle modification before more invasive measures.[12,13] Non-steroidal anti-inflammatory medications can be employed to relieve both pain from failing facets and the minute neural swelling that contributes to compression. Neuromodulators can be used to manage neuropathic and radicular pain, however opioids should be avoided as evidence for their efficacy in this condition is poor.[14]

The goal of physical therapy is to improve the biomechanical environment. In isthmic spondylolisthesis this means reducing the extension force across the affected motion segment to heal a lysis. In degenerative spondylolisthesis, the aim is to sustain posture, through flexion-based or stabilisation exercises, which will increase the diameter of the neural canal.[15] Although Pilates has been used for many years to develop core strength in patients with chronic back pain, the evidence for success in spondylolisthesis is poor—equally there is no evidence of it being responsible for harm.[16]

Local steroids have a place in the management of isthmic spondylolisthesis. A steroid injection placed directly into a pars defect may help define the source of pain or relieve enough pain for a non-invasive regime to be aggressively pursued.

In patients with neural compression, steroid and local anaesthesia injections targeted to the sites of the compromise can be beneficial in confirming the source of neurogenic pain and in some cases reliving symptoms through an anti-inflammatory effect.[17] In some cases, epidural steroid injection has been reported to avoid the need for later surgery.[18]

Surgery

The goal of surgery is to stabilise the motion segment or to relieve neural compression or both. Where neural decompression is not required, such as in isthmic defects, stability of the motion segment can be achieved either by repairing the pars defect and restoring normal anatomy or by fusing the motion segment. There are over 50 techniques described for pars repair, all of which combine identification and debridement of the defect, placing bone graft and

SUMMARY OF KEY POINTS

- Spondylolisthesis affects 18% of adults.
- Surgical treatment for spondylolisthesis is rarely required.
- Risk factors depend on the patient's age and include specific athletic activities, trauma and degenerative changes to the posterior elements.
- Examination findings can be normal.
- Surgical options include repair, decompression, and stabilisation of affected segments.

using instrumentation to stabilise and compress across the break.[3,19]

With Types I and II, when pars repair is insufficient and decompression is required, the operative technique usually includes fusion of the two adjacent vertebrae. Typically, this is an instrumented posterolateral approach frequently supplemented with an interbody cage or graft.

The surgery generally attempts only a partial reduction of the spondylolisthesis.

Aggressively pursuing a full reduction is a high-risk manoeuvre.[20-24] Neural elements are distorted, scarred and in many cases poorly protected by the remaining bony elements. Implants are technically difficult to place, leading to improper positioning, failed fusion, and rapid collapse of the construct.[25]

In cases of degenerative spondylolisthesis, Type III, it is unclear whether decompression must necessarily be followed by fusion. A recent controlled trial by Austevoll et al. randomised over 200 patients with neural compression and three millimetres or more of slip to either decompression alone or decompression with fusion.[26] Analysis showed that at two years, decompression alone was not inferior to decompression and fusion.

CLINICAL PEARLS

- Spondylolisthesis is a common incidental finding.
- Not every spondylolisthesis needs treatment.
- Uncontrolled pain is a valid reason for referral.
- Analgesia, physiotherapy, and injection therapy manage most cases successfully.

Conclusion

Lumbar spondylolisthesis has a range of causes. In adolescents and young adults, the forward slip of the vertebral body is usually due to a deficiency or a defect in the pars interarticularis, the bony bridge that connects the body to the posterior elements. In middle-aged adults the slip of an entire vertebra is the generally the result of failure of the interlocking facet joints. Treatment options include con-

servative care, which is successful for most patients, and surgery to remove neural compression and/or stabilise the motion segment. Spondylolisthesis is a common incidental finding so the essential first step is to ensure the patient's symptoms are related to the defect.

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