Case Reports

Bilateral Isthmic L3 Spondylolisthesis in an Adult Female

Ali Nourbakhsh, MD,[a] Rudiger H. Von Ritschl, MD,[b] and Kim J. Garges, MD[c]

Abstract

Objective: The purpose of this study is to describe an incidental finding of bilateral isthmic L3 spondylolysis in an adult female.

Clinical Features: A 26-year-old woman with sickle cell anemia was involved in a motor vehicle accident. Lumbar radiographs were reported normal. Computed tomography scan showed bilateral L3 spondylolysis of the pars interarticularis.

Interventions and Outcomes: On the basis of the normal results of physical and neurologic examinations, the spondylolysis was considered to be an incidental finding.

Conclusions: L3 spondylolysis is described very rarely in the literature. According to the unique features of L3 in the lumbar spine, which include its relatively horizontal position and its equal anterior and posterior diameters, we suggest that mechanical shearing forces may be less effective in causing spondylolysis in this area. This case is more suggestive of congenital and genetic causes as the contributing factors of spondylolysis. (J Manipulative Physiol Ther 2008;31:160-163)

Key Indexing Terms: *Spondylolysis; Spine; Lumbar Vertebrae*

Spondylolysis is defined as a break of the isthmic part of the pars interarticularis of a vertebra, without forward displacement of that vertebra on the next adjacent vertebra.[1] Spondylolysis occurs with a prevalence of 4% to 6% in the general population.[2] It is a common cause of low back pain in children and adolescents. The incidence of spondylolysis at the age of 6 years is 4.4% and increases to 6% in adulthood.[3] Spondylolysis may stay asymptomatic especially if spondylolisthesis is not present.[4]

Spondylolysis can be classified into 2 types, acute and chronic, depending on the severity and duration of symptoms. It is considered acute when either significant trauma or repeated microstresses produce a fracture of the isthmus, causing pain and decreased mobility in the lumbar area. Spondylolysis is considered chronic when it has been discovered late, often after the major symptoms have resolved, and sometimes discovered incidentally on radiographs.[1]

To our knowledge, this is the first case of isolated bilateral L3 spondylolysis in the literature. The etiology and the reasons for low prevalence are being discussed. The authors' institutional review board approved this retrospective chart review study. The patient gave consent to have her personal information published without divulging personal identifiers.

Case Report

A 26-year-old woman with sickle cell anemia was involved in a motor vehicle accident. She was the passenger in a car traveling at a speed of 50 mph. At presentation, she complained of low back pain, but the result of physical and neurologic examination was normal. Lumbar radiographs showed the L3 spondylolysis (Fig 1). A Computed tomography scan showed a bilateral L3 spondylolysis (Fig 2). On the basis of the normal physical and neurologic findings, the spondylolysis was considered to be an incidental finding. During the patient's 3-day stay in the hospital, her back pain improved, and she was safely discharged.

Discussion

Most spondylolysis defects occur at L5 (85%-95%), followed by L4 (5%-15%). The more proximal lumbar levels

[a] Research Fellow, Division of Spine Surgery, Department of Orthopedic Surgery and Rehabilitation, The University of Texas Medical Branch, Galveston, Tex.

[b] Assistant Professor, Department of Radiology, The University of Texas Medical Branch, Galveston, Tex.

[c] Associate Professor and Chief, Division of Spine Surgery, Department of Orthopaedic Surgery and Rehabilitation, The University of Texas Medical Branch, Galveston, Tex.

Submit requests for reprints to: Kim Garges, MD, Associate Professor and Chief, Division of Spine Surgery, Department of Orthopaedic Surgery and Rehabilitation, The University of Texas Medical Branch, Galveston, TX 77555-0165
(e-mail: *kjgarges@utmb.edu*).

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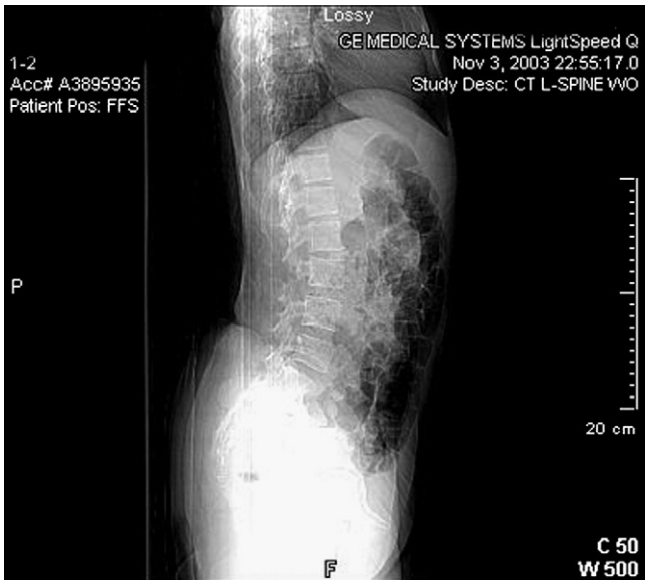

Fig 1. *Lateral spine radiograph showing spondylolysis at L3 with minimal spondylolisthesis at L3 through L4.*

are affected much less often. It is considered to be a fatigue fracture of the pars interarticularis of the neural arch.[5] Spondylolysis in the pediatric and adolescent population is more common in males than females, but it is more likely to progress in females.[6] No cases of spondylolysis are reported in otherwise healthy newborns.[5] Because 4-footed animals do not walk or sit upright, there is no excessive load on the articular processes, which means the articular processes cannot act on the pars interarticularis to result in spondylolysis/spondylolisthesis. Thus, the triggering factors only exist in humans,[7] and many authors agree that most cases probably occur in the early school age years or during early childhood.[5,8]

Both mechanical and genetic factors have been described as possible etiologies.[9]

Upright posture and ambulation[10]; repetitive loading; hyperlordosis, such as that seen in Scheuermann's kyphosis; and participation in sports,[6] especially for athletes who repeatedly hyperextend and rotate their lumbar spine (eg, gymnasts and javelin throwers),[8] have all been described as the contributing factors to the mechanical etiology of spondylolysis.

Several authors have investigated the effects of mechanical loading on the region of the pars interarticularis. In a modeling experiment, Dietrich and Kurowski[11] found that the greatest loads with flexion/extension movements occur at L5/S1, and pars interarticularis receives the highest mechanical stresses. Once the defect has been created, biomechanical and anatomical forces conspire to prevent healing of the fracture, resulting in chronic spondylolysis or spondylolisthesis. Shear forces due to the center of the gravity tend to displace the L5 vertebral body with the pedicles and superior articular processes anteriorly, whereas posterior muscles tend to extend the posterior element, which will distract the fracture site, preventing spontaneous healing.[12] A lower lumbar index, which is the percentage of posterior vertebral body height compared with the anterior height, has also been associated with L5 spondylolysis in Fredrickson's study.[3] A recent study by Roussouly et al[13] on 82 patients with L5 spondylolysis showed increased pelvic incidence and lumbar lordosis, but less segmental extension between L5 and S1 than in the normal population. He stated there is less tension on the pars interarticularis because of shear forces when the L5-S1 junction is oriented more horizontally.

L3 has unique characteristics in the lumbar spine. First, the bodies of L1 and L2 are deeper dorsally, and the bodies of L4 and L5 are deeper ventrally, whereas the L3 vertebra is transitional.[14] Second, the apex of the lumbar lordosis is located at the junction of L3 with L4,[13] and L3 lying relatively horizontally in the lumbar spine. Because of its horizontal position, the weak effect of gravity-induced downward slip, and its equal anterior and posterior shape, mechanical forces seem to be less effective in induction of L3 lysis. This explains the rare incidence of spondylolysis in L3. In Fredrickson's study on 500 unselected children, no L3 lysis was found.[3]

The second proposed cause of spondylolisthesis is genetic influences. More than 25% of members of some families have been reported to show pars interarticularis defects.[15] Fredrickson stated that there is a hereditary predisposition to the defect, and a strong association exists with spina bifida occulta.[3]

Sagi et al[16] performed microscopic analysis of human fetal spines. They noted an uneven distribution of trabeculation and cortication in the region of the pars interarticularis in the lower lumbar vertebrae. They concluded that this may create a potential stress riser in the pars, which makes it more vulnerable to repetitive stress. Their findings also suggest the possibility of a congenital anomaly in this region, which predisposes a person to the development of isthmic spondylolysis.

In 1979, Wynne-Davies and Scott[17] investigated 147 relatives of 47 patients with spondylolysis and spondylolisthesis. The dysplastic type showed a higher prevalence in near relatives (1 in 3 affected) than the isthmic type (1 in 7 affected), and both were more than the estimated frequency for the general population of less than 1% and 5%, retrospectively. They suggested a genetic element to both types (an autosomal dominant with reduced penetrance to multifactorial inheritance). Other authors have suggested an autosomal recessive character for these defects.[18] Some family studies of individuals with isthmic spondylolysis and spondylolisthesis report that more than 20% of first-degree relatives show similar radiographic changes.[18,19] A high frequency of these disorders has also been observed in Alaskan Eskimos.[20]

Multiple levels of spondylolysis have been described in the lumbar spine. Privett et al[21] reported a bilateral L3-4-5 spondylolysis and proposed a genetic predisposition as the