

Anatomic Variability of 120 L5 Spondylolytic Defects

Michael R. Chen¹ Timothy A. Moore² Daniel R. Cooperman³ Michael J. Lee⁴

¹Department of Orthopaedic Surgery, Mercy Health Physicians, Fairfield, Ohio, United States

²Department of Orthopaedic Surgery, Case Western Reserve University, Cleveland, Ohio, United States

³Department of Orthopaedics and Rehabilitation, Yale University, New Haven, Connecticut, United States

⁴Department of Orthopaedic Surgery and Sports Medicine, University of Washington Medical Center, Seattle, Washington, United States

Address for correspondence Michael J. Lee, MD, University of Washington Medical Center, 1959 Pacific Avenue NE, Box 356500, Seattle, WA 98195, United States (e-mail: mjl3000@u.washington.edu).

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Abstract

Study Design Adult human osteologic specimens were assessed for spondylolytic defects and characterized.

Objectives To characterize and determine the prevalence of spondylolytic defects in an osteological collection.

Methods Lumbar vertebrae from the Hamann-Todd Osteological Collection at the Cleveland Museum of Natural History were examined. Digital images of specimens with L5 isthmic spondylolytic defects were analyzed, examining the distance of the pars defect in the sagittal plane in relation to the caudal aspect of the pedicle.

Results There were 95 bilateral complete (BC), 16 unilateral incomplete (UI), 5 unilateral complete (UC), and 4 unilateral complete defects with an incomplete defect on the contralateral side. The mean distance of BC defects from the pedicle and inferior vertebral end plate was 4.03 mm and 4.88 mm, respectively. The mean distance of the defect from the inferior end plate on the left and right sides were 5.31 mm and 4.44 mm, respectively ($p = 0.001$, correlation coefficient = 0.56). The mean distance of UI and UC defects from the inferior end plate was 6.38 mm and 2.6 mm, respectively.

Conclusion L5 spondylolytic defects were found in 3.87% of the sample. This large-scale description of isthmic spondylolytic defects reveals that significant variability exists in the location of the defect. The anatomic location of the pars defect likely plays a role in the development of L5 nerve root compression and radiculopathy in this clinical scenario. Classifying these defects might allow surgeons to better identify those patients who might benefit from fusion alone without posterior decompression.

Keywords

- ▶ spondylolysis
- ▶ spondylolisthesis
- ▶ cadaver
- ▶ lumbar radiculopathy

Introduction

Spondylolysis is a condition characterized by a defect or abnormality in the pars interarticularis of the vertebrae. It is a common condition that occurs in ~3 to 6% of Americans and at higher frequencies in certain athletes and ethnic

populations as noted by Buetler et al,¹ Frederickson et al,² Lagroscino et al,³ and Standaert and Herring.⁴ There also appears to be a genetic predisposition for spondylolysis as relatives of index cases have a greater than fourfold increased incidence. Buteler et al¹ and Wiltse et al⁵ have reported that the lytic defects of the pars interarticularis are most common

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at the L5 vertebrae, with decreasing incidence at the more cranial lumbar levels.

The etiology of spondylolysis is poorly understood. Spondylolysis is a defect unique to humans. Studies by Frederickson et al,² Wiltse,⁶ and Wiltse⁷ have suggested that it appears to be acquired, as the fetal incidence has conclusively been shown to be zero. The primary lesion in spondylolysis is believed to be a stress fracture of the pars interarticularis that remains unhealed. Rosenberg et al⁸ supported this theory by observing that spondylolysis also has not been demonstrated in patients who have never ambulated. Other factors that have also been implicated in the etiology or susceptibility to spondylolysis include mechanical stress, anatomic variation, or repeated trauma that leads to a gradual dissolution of the pars as previously suggested by Wiltse,⁵ Cryon and Hutton,⁹ Cryon and Hutton,¹⁰ Cryon et al,¹¹ Dietrich and Korowaski,¹² Farfan et al,¹³ Nathan,¹⁴ and Troup.¹⁵ The result of hyperextension on the development of spondylolysis is also suggested by the high incidence of defects in gymnasts, swimmers, and other young athletes who undergo frequent lumbar hyperextension.¹⁶⁻¹⁹

Although the orthopedic literature regarding spondylolysis and spondylolisthesis is extensive, there is a paucity of information describing the nature of spondylolytic defects. Edelson and Nathan described the area of the spondylolytic defect as varying from 2 to 9 mm below the caudal aspect of the pedicle in a sample of 34 bony specimens.²⁰ They also described the consistent finding of a hooklike projection of the proximal lamina at the inlet to the intervertebral foramen. Merbs has also characterized and described the asymmetry of spondylolytic defects.²¹ We believe that the location of the pars defect may affect the development of radiculopathy in isthmic spondylolisthesis. The purpose of this study was to describe the anatomic variability of the lytic L5 pars defect in a large sample population to better understand the spectrum of these defects and how they may potentially contribute to neurocompressive pathology.

Materials and Methods

The Haman-Todd Osteological Collection is a collection of human and primate skeletons housed at the Cleveland Museum of Natural History (Cleveland, Ohio, United States). This collection has 3,100 human skeletons, composed of white and black males and females born between the years of 1825 and 1910, obtained from the city morgues of Cleveland. One hundred twenty human vertebral columns were identified as having a spondylolytic defect at the L5 level. Spondylolytic specimens were identified as having bilateral complete (BC) defects (separate neural arch), unilateral incomplete (UI) defects (neural arch remains attached to the vertebra), unilateral complete (UC) defects (loss of ipsilateral half of neural arch), and UC defect with contralateral incomplete defect.

The age, sex, and race of all the specimens were recorded. Images were made of each L5 vertebra from a direct lateral view from both the left and right sides using a digital camera (Cybershot DSC-W7, Sony Corp, Tokyo, Japan). An image processing program (ImageJ, National Institutes of Health,

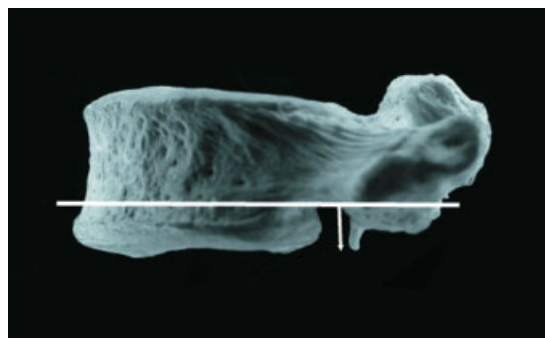


Fig. 1 Absolute size of the spondylolytic defect, measured from the pedicle.

Bethesda, Maryland, United States), was used to measure distances. Images of each vertebra were made next to a ruler to allow the program to standardize for magnification. A single individual made all measurements. A consistent hook-like projection of the proximal lamina was seen in the spondylolytic specimens and the caudal tip is referred to as the level of the spondylolytic defect. The distance of the level of the spondylolytic defect below the caudal aspect of the pedicle in the sagittal plane was measured to the nearest millimeter. This was performed by drawing a horizontal line at the caudal aspect of the pedicle (most superior aspect of the neural foramen). The distance from this line inferiorly to the level of the spondylolytic defect was then measured (► **Fig. 1**). As a control for vertebral size, the distance of the level of the spondylolytic defect to the inferior vertebral end plate in the sagittal plane was also measured to the nearest millimeter. A horizontal line representing the posterior continuation of the inferior vertebral end plate was drawn. In cases where the inferior vertebral end plate was asymmetrical, the line was based off the most posterior point of the end plate. The distance from this line to the most caudal part of the proximal lamina was then measured (► **Fig. 2**).

Statistical Analysis

Histograms were made of all groups of specimens to determine whether there was a normal distribution. A paired *t* test was performed to compare side-to-side differences in the BC defect specimens. A two-sample *t* test was used to compare BC samples with the UI samples. A Wilcoxon rank sum test

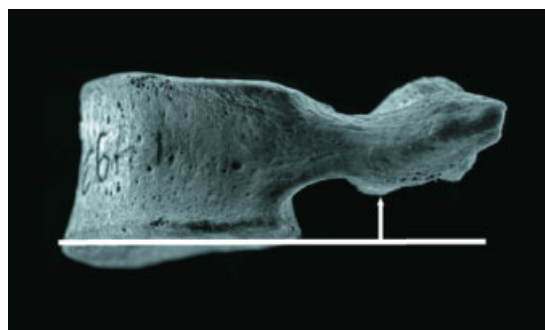


Fig. 2 Standardized size of the spondylolytic defect, measured from the inferior vertebral end plate.

was used to compare all other groups because of non-normal distributions or small number of specimens. Correlation coefficients were determined to compare side-to-side differences. Analysis was performed using statistical software (Minitab 13, Minitab Inc., State College, Pennsylvania, United States).

Reliability and Validity

For intraobserver reliability, the same individual made three sets of measurements on the left and right sides from seven different vertebrae at 1-week intervals. For interobserver reliability, two different individuals who had experience with the ImageJ software made measurements from the same seven different vertebrae. The validity of the ImageJ software was tested by comparing values calculated by the program with those obtained from a more orthodox measuring tool (digital caliper, Mitutoyo Co., Kanagawa, Japan, 0.01 mm).

Results

Reliability and Validity

Intraobserver reliability was found to be 97%. Interobserver reliability was found to be 96%. The validity of the ImageJ software was found to be 99%.

Study Population

The study population consisted of 120 of the 3,100 human skeletal specimens that were found to have a L5 spondylolytic defect, representing an incidence of 3.87%. Ninety-five of these specimens were found to have BC spondylolytic defects in the L5 lumbar vertebrae (►Fig. 3a). Sixteen specimens had a UI defect (►Fig. 3b). Five specimens had a UC defect (►Fig. 3c). Four specimens had a complete defect on one side and an incomplete defect on the contralateral side (►Fig. 3d). One hundred fourteen specimens (95%) were male and six were female. Ninety-six specimens (80%) were white and 24 were black. The mean age was 49.43 years (range 18 to 105 years) at the time of death.

Bilateral Complete Defects

The mean distance (\pm standard deviation) of the defect from the pedicle was 4.05 ± 1.88 mm (range 0 to 10 mm). The mean distances for the left and right sides were 3.92 ± 2.05 mm and 4.18 ± 1.70 mm, respectively, which were not significantly different ($p = 0.197$) but demonstrated moderate correlation (correlation coefficient 0.46) within

each vertebra. The mean distance of the defect from the inferior vertebral end plate was 4.88 ± 2.51 mm (range -2 to 13 mm). The mean distances for the left and right sides were 5.31 ± 2.57 mm and 4.44 ± 2.41 mm, respectively, which was significantly different ($p = 0.0005$) but also demonstrated a high correlation (correlation coefficient 0.56) within each vertebra.

Unilateral Incomplete Defects

UI defects occurred on the right side in 10 specimens and on the left in 6. No significant differences were found between the level of the defect on the left and right sides in relation to the pedicle or the inferior vertebral end plate ($p = 0.25$ for both).

The mean distance of the defect from the pedicle was 3.88 ± 0.89 mm (range 2 to 5 mm). No significant difference could be detected when all UI defects were compared with all BC defects ($p = 0.55$). Because of a possible association between the left and right defects in each vertebra of BC defects as determined by the correlation coefficients, single sides from the BC group were compared with the other groups. However, there was no significant difference when only left BC defects were compared with the UI defects ($p = 0.89$) or when only right BC defects were compared with the UI defects ($p = 0.29$).

The mean distance of the defect from the inferior vertebral end plate was 6.38 ± 2.13 mm (range 2 to 10 mm). This was significantly greater (the defect was located more cranially) when all UI defects were compared with all BC defects ($p = 0.016$). The majority of this significance appears to be contributed from the right side of BC defects ($p = 0.01$ when compared with all UI defects) than from the left side of BC defects ($p = 0.08$ when compared with all UI defects).

Unilateral Complete Defects

All five UC defects occurred on the right side. The mean distance of the defect from the pedicle was 6 ± 3.16 mm (range 2 to 10 mm). The mean distance of the defect from the inferior vertebral end plate was 2.6 ± 3.97 mm (range -3 to 6 mm). No difference was found when compared with right sides of BC defects ($p = 0.17$ and $p = 0.52$, respectively). No difference was found when UC defects were compared with the UI defects in relation to the pedicle ($p = 0.16$). With the number of samples available, no difference relative to the inferior vertebral end plate was found between UC and UI defects, although the values did approach statistical significance ($p = 0.0546$).

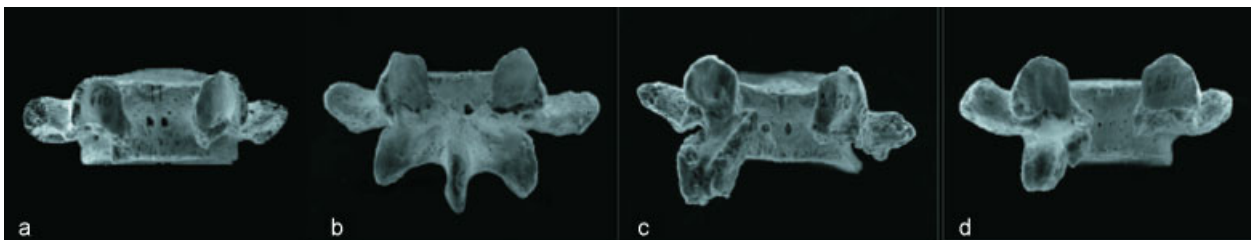


Fig. 3 Types of spondylolytic defects. (a) Bilateral complete. (b) Unilateral incomplete. (c) Unilateral complete. (d) Unilateral complete and incomplete.

Unilateral Complete with Unilateral Incomplete Defects

Of the four specimens with a complete defect on one side and an incomplete defect on the other side, the complete defect was always on the right side and the incomplete on the left. A very large inverse correlation appeared to exist between the complete and incomplete defect (correlation coefficient -0.83) within each vertebra. However, with the limited number of specimens available, this correlation was not significant ($p = 0.165$).

The mean distance of the complete defect from the pedicle was 3.75 ± 2.06 mm (range 2 to 6 mm). No significant differences were found when compared with the right sides of BC defects ($p = 0.68$) or with UC defects ($p = 0.32$). The mean difference of the complete defect from the inferior vertebral end plate was 4.5 ± 2.38 mm (range 2 to 7 mm). No significant differences were found when compared with the right sides of BC defects ($p = 0.89$) or with UC defects ($p = 0.53$).

The incomplete defect was a mean distance of 2 ± 0.82 mm (range 1 to 3 mm) from the pedicle. This distance was significantly less than UI defects ($p = 0.01$). The mean distance of the incomplete defect from the inferior vertebral end plate was 5.25 ± 1.26 mm (range 4 to 7 mm). There was no difference when compared with UI defects ($p = 0.25$).

Discussion

This study is the largest description of L5 isthmic spondylolytic defects. Our 3.87% incidence corresponds to that seen in population studies, even though we only studied L5 vertebrae. Because the Hamann-Todd Collection consists of ~85% male specimens, it is to be expected that a large portion (95%) of our defects occurred in males.

As previously described by Merbs,²¹ several types of spondylolytic defects were found. The only type of defect not observed was a bilateral incomplete defect. As expected, the most common type observed were BC defects (the classic form of spondylolysis). The relative order in frequency of all the defects was also similar to what Merbs observed in a Canadian Inuit population. Unlike Merbs, all UC defects, which included the UC with UI defect group, in our study were observed on the right side. Overall, however, unilateral defects were more frequently observed on the right side, which is similar to previous studies.^{21,22}

Based on the mechanical stress theory where hyperextension causes repeated stress on the pars, leading to dissolution, one might assume that there would be equal stresses on the pars, leading to equal defects bilaterally. Asymmetry appears to be the general trend in spondylolytic defects though, as evidenced by the significant difference in the location of the spondylolytic defect, when standardized for vertebral body size. However, the defects do not appear to be completely independent of each other because a moderate to high correlation was observed between the location of the defects on the left and right sides in BC defects.

Complete defects may lie more caudally compared with incomplete defects. When standardized for vertebral size, BC defects were significantly different from UI defects. There was

also almost a statistically significant difference between UC and UI defects. There was no difference, however, when all types of complete defects were compared with each other.

Although unilateral defects are more common on the right side, no difference existed between left and right UI defects. When present in the same specimen, however, the incomplete defects may be affected by complete defects. The incomplete pars defect was significantly more caudal in specimens with UC and UI defects compared with UI defects. There was also a very large inverse correlation between the level of the incomplete and complete defect in the UC and incomplete group. With our limited sample size, though, this correlation was not significant.

Foraminal stenosis may be an important etiology of nerve root compression when it is associated with spondylolysis and spondylolisthesis. We believe that the anatomic location of the pars defect may play a role in the development of L5 nerve root compression and radiculopathy in this clinical scenario. A more cranial or ventral defect may simply unroof and decompress the foramen with progressive spondylolisthesis as suggested in the past by Newman²³ and Léger et al.²⁴ A more caudal or dorsal defect, however, may produce further foraminal stenosis as the remaining part of the proximal lamina causes traction or compression on the nerve root with progression of spondylolisthesis as suggested by Edelson and Nathan.²⁰ Our study demonstrates a normal distribution in the location of the pars defect in the BC group (→ Fig. 4), which are the most likely to progress to isthmic spondylolisthesis as shown in natural history studies.¹ Those defects at the extremes may represent patients who are the most and least likely to experience neurologic symptoms.

The clinical significance of the location of the pars defect may influence surgical strategy and may also be predictive. When a spondylolysis without spondylolisthesis is diagnosed and evaluated in adolescence, the location of the pars defect may predict which patient is at higher risk for developing radiculopathy with aging and progressive slippage. In regard to surgical strategy, fusion alone has been advocated for those patients with mechanical lumbar symptoms with radicular extremity symptoms from nerve root irritation and not compression.²⁵ Classifying the location of these defects preoperatively may allow surgeons to better identify those patients with only nerve root irritation who might benefit from fusion alone, avoiding a larger dissection and longer recovery time.

A notable weakness of this study is that soft tissue pathology cannot be accounted for and symptoms are unknown. Nerve root compression may be resultant from a wide spectrum of pathoanatomy including synovial cyst, ligamentum hypertrophy, and disk pathology. This study examines only the variation among spondylolyses and cannot comment on soft tissue pathology or degree of slippage. In addition, it should be noted these specimens are over 100 years old. Given population changes in health and nutrition between then and now, it is possible that the anatomic variation observed in these specimens may not represent the current population. Although we suggest that the location of the pars defect may play a role in exacerbating foraminal stenosis, the present study does not support this hypothesis in this study population.

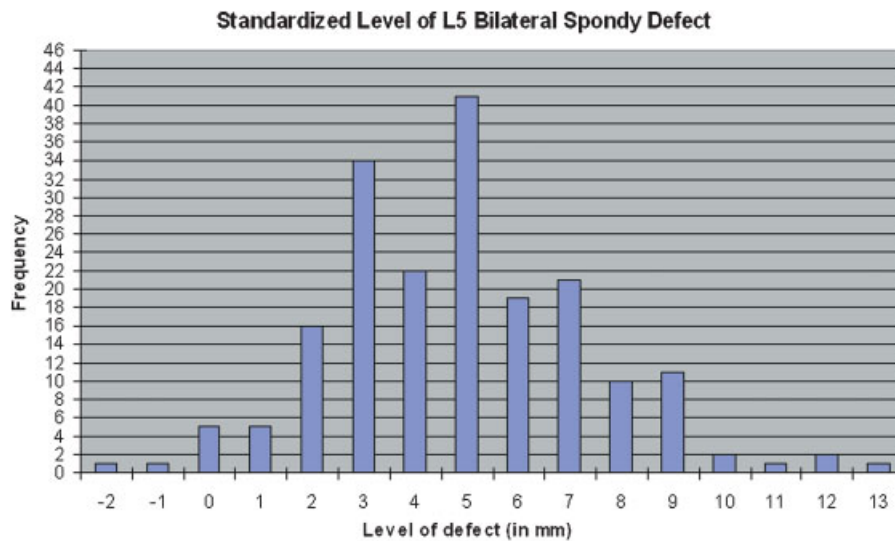


Fig. 4 Distribution of spondylolytic defects from the inferior end plate.

Foraminal stenosis can occur from multiple etiologies and in multiple directions. In the setting of spondylolysis, we postulate that the location of the pars defect may play a role in contributing to nerve root compression in the foramen. Further clinical studies with computed tomography or magnetic resonance imaging in symptomatic and asymptomatic patients are likely to be of benefit when further examining the impact of the pars defect location.

Disclosures

Michael R. Chen, None
 Timothy A. Moore, None
 Daniel R. Cooperman, None
 Michael J. Lee, Consulting; Stryker Spine; Faculty: AOSpine

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