## Title

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# Health-Related Quality of Life in Sacroiliac Syndrome:

# A Comparison to Lumbosacral Radiculopathy

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Header: Health-Related Quality of Life in SI Syndrome

## Introduction

Sacroiliac joint syndrome (SI syndrome) refers to the phenomenon of pain emanating from the sacroiliac (SI) joint without a readily demonstrable pathology such as spondyloarthropathy or crystal or pyogenic arthropathy.[1] The etiology of the pain is believed to be mechanical in origin.[2] This diarthrodial joint has been implicated as a primary source of pain (i.e., independent of other conditions) as early as 1905 by Goldthwaite and Osgood.[3] To be defined as having SI syndrome by the International Association for the Study of Pain,[4] patients must possess all of the following characteristics: (1) pain in the region of the SI joint with possible radiation to the groin, medial buttocks, and posterior thigh; (2) reproduction of pain by physical examination techniques that stress the joint; (3) complete elimination of pain with intra-articular injection of local anesthetic, and (4) an ostensibly morphologically normal joint without demonstrable pathognomonic radiographic abnormalities. The incidence of SI syndrome is estimated to be as large as 22%-30% in centers specializing in the treatment of low back pain.[5,6] Etiologic factors implicated in the genesis of SI syndrome include trauma, cumulative injury, previous back surgery, or idiopathic causes.[7,8]

Over 90 instruments are available to assess health-related quality of life (HRQoL) in low back pain.[9] However, to our knowledge, there is no study examining HRQoL in patients with SI syndrome. The 36-Item Short-Form Health Survey (SF-36) is a well-validated HRQoL instrument in wide use.[10] The McGill Pain Questionnaire is a widely employed, well-validated instrument used to examine the intensity ("worst", "best" and "average") and quality (e.g., sensory, neuropathic, affective) of pain.[11] This study attempts to describe the intensity and quality of pain and to assess the HRQoL of a population of patients with SI syndrome and to

compare those constructs to patients with lumbar spine-derived leg pain of a non-mechanical nature (i.e. lumbar radiculopathy).

## Methods

After approval from the Institutional Review Board, patient records from the UCLA Pain Management Center were retrospectively examined from December 2001 through August 2005. Upon initial presentation to the practice, all patients had completed the following instruments irrespective of diagnosis: (1) McGill Pain Questionnaire,[11] (2) visual numerical pain scores ("worst", "best" and "average") over the 4 weeks preceding presentation, (3) SF-36 health-related quality of life (HRQoL) measure (version 2). Complete peripheral neurologic and musculoskeletal physical examinations (including SI joint provocation tests and range of motion of the lumbar spine) were performed on all patients.

Patients were defined as having SI syndrome by the criteria of the International Association for the Study of Pain (see Introduction).[4] The Patrick's and Gaenslen's tests were used for SI joint pain provocation. Diagnostic SI injections were performed with 2 ml of 0.25% bupivacaine and 80 mg of triamcinolone acetonide under fluoroscopy.[1] Before injection, contrast joint arthrography was performed. Patients with 100% pain relief and normalization of SI tests 2 weeks postinjection were analyzed. Patients were excluded if they had a dual diagnosis of SI syndrome and lumbar radiculopathy. Demographic data obtained on SI syndrome patients included: age, gender, duration and location of pain, and the inciting event (trauma, cumulative injury, previous back surgery, or "idiopathic" etiology).

Patients were age- and gender-matched to patients with lumbar radiculopathy. Patients were defined as having lumbar radiculopathy if they met all of the following criteria: (1) radicular pain in a defined dermatomal pattern; (2) a positive physical finding (hypesthesia, hyporeflexia, or motor weakness) or positive electromyography and nerve conduction studies (EMG) corresponding to the nerve root responsible for the radicular pain, and (3) a

radiographically defined disk herniation or disc protrusion correlating with the nerve root responsible for the radicular pain and positive physical examination sign or EMG.

The concomitant presence of facet arthropathy, spinal stenosis, spondylosis, spondylolisthesis, spondylolysis and previous lumbar surgery were recorded for both diagnostic groups. Facet arthropathy was diagnosed by the presence of: (1) positive physical findings of pain with lumbar extension and lateral flexion with rotation and, (2) elimination of pain with medial branch nerve blocks. Spinal stenosis, spondylosis, spondylolisthesis, and spondylolysis were diagnosed using radiography.

*Statistics:* Mean ± standard error was reported as the measure of central tendency for parametric data. The median with range was reported as the measure of central tendency for ordinal data. Nonparametric data were analyzed using chi-square analysis with Fisher's exact test. Student's t-test and analysis of variance were used to analyze parametric data. A $p$ value of $< 0.05$ was chosen to indicate statistical significance.

## Results

A total of 872 charts were reviewed to obtain 86 age- and gender-matched (74F:M12) subjects in each study group. The mean age was 58.0 ± 1.8 years with a range of 30-89 years. Of 364 potential subjects with SI syndrome, 165 patients did not meet inclusion criteria for SI syndrome, and 113 subjects were removed from analysis because of insufficient/incomplete data. Of 518 potential subjects with lumbar radiculopathy, 267 patients did not meet inclusion criteria, and 165 subjects were removed from analysis because of insufficient or incomplete data.

The demographic characteristics of the patients with SI syndrome as a function of the inciting event for the genesis of pain are described in Table 1. As a group, patients with SI syndrome experienced pain for 4.8 ± 0.7 years prior to diagnosis (range = 0.08 to 34 years). The mean duration of relief after diagnostic injection was 35.2 ± 5.0 days (range = 1 to 210 days). With respect to the four etiologies of SI syndrome,[7] patients with idiopathic SI syndrome endured a shorter duration of pain of 2.6 ± 0.4 years before seeking medical attention in comparison to patients with previous back surgery (7.7 ± 2.2 years; $p < 0.005$). The age of patients with SI syndrome caused by trauma was lower (49.4 ± 3.3 years) than the age of patients with previous lumbar surgery (63.9 ± 3.1 years) or idiopathic (62.5 ± 2.5 years) etiologies ($p < 0.02$). No statistical difference was found with respect to gender or postinjection duration of analgesia among the established etiologies for SI syndrome.

There was no difference between patients with SI syndrome and lumbar radiculopathy with respect to the concomitant presence of spinal stenosis, spondylolisthesis, spondylolysis and previous lumbar surgery. However, patients with SI syndrome had a higher incidence of concomitant facet arthropathy ($p < 0.0009$), while patients with lumbar radiculopathy had a higher incidence of concomitant spondylosis ($p < 0.03$).

There was no statistical difference between patients with SI syndrome and lumbar radiculopathy with respect to the McGill Pain Questionnaire (Table 2), visual numerical pain scores (Table 3), and SF-36 HRQoL scores (Table 4).

## Discussion

SI syndrome is a disease of mechanical origins.[2,12-14] Fortin et al.[13] demonstrated the mechanical pathogenesis of SI syndrome by provocation of pain in asymptomatic volunteers using fluoroscopically guided contrast injections into the SI joint. By physically disrupting the joint, pain referral maps were generated caudal to the posterior inferior iliac spine.[13] The joint is susceptible to mechanical disruption from direct axial or sagittal loads. Compared to the lumbar motion segments, the SI joint is exposed to a threefold increase in sacral translation and as much as an eightfold increase in rotation when pressure is applied to one ilium and not the other.[15] Mechanical shear forces, torsion, and ligamentous disruption cause traumatic injury and are common causes of SI syndrome.[2,7,12]

In contrast, lumbar radiculopathy is caused by compression and/or irritation of the sensory root or dorsal root ganglion, which is perceived as pain in the distribution of the respective spinal nerve.[16] Disc protrusions or herniations cause a release of chemical mediators that induce a localized inflammatory reaction.[17]

It was hypothesized that significant differences in HRQoL might be found between patients with SI syndrome and lumbar radiculopathy based upon disparity in their pathogenesis. The SF-36 has discerned differences in HRQoL among diagnostic categories in other disease states.[18]

The results of this study suggest that there is no true difference in the HRQoL between patients with SI syndrome or lumbar radiculopathy. One must examine the possibility that the SF-36 could be too insensitive an instrument to detect differences in HRQoL for patients with differing etiologies of low back pain. The SF-36 is a generic instrument containing 8 subscales that have been shown to be responsive to change in a patient during the course of

treatment.[10,19] The SF-36 has undergone rigorous testing for data quality and reliability across diverse patient groups.[20] For most scales a difference of 5 points is considered to be clinically significant.[19] Published data define the threshold for detection of significance in the Physical Functioning scale as 16-22 points, the Role Limitations-Physical scale as 62-66 points, and the Bodily Pain scale as 33-41 points.[21] Such large changes suggest these particular scales are unable to detect small changes in most subjects.[21] However, the mean values between the two groups of patients in our study do not differ by more than 3 points for any scale, which is significantly below the minimum detectable change for these 3 particular scales as well as below the minimum detectable change for the instrument in general. This suggests that there is no true difference in the HRQoL between patients with SI syndrome or lumbar radiculopathy.

In the literature, over 90 different instruments have been published in approximately 600 clinical evaluations of low back pain.[9] Would a condition-specific instrument (i.e., an instrument specifically designed for use in low back pain alone) be able to detect differences in HRQoL? For instance, the Roland-Morris and the Oswestry Low Back Pain Disability Questionnaire have been used in a great number of studies. Using the Oswestry, Caragee found different functional profiles among patients with discogenic pain, spondylolisthesis and chronic vertebral osteomyelitis despite similar pain levels and duration of pain.[22] We did not utilize a condition-specific instrument to assess HRQoL in the present study and cannot make a direct comparison to the results with the SF-36. However, the literature suggests that the general SF-36 is a sufficient measure of HRQoL for studies of patients with any type of low back pain, without the need for condition-specific measures.[9,23]

In the present study, rigorous selection criteria were employed, attempting to stratify subjects into two distinct homogenous pathophysiologic groups representing isolated spinal

phenomena. However, the different pathophysiologies of low back pain do not occur in isolation, and there is commonly greater than one pathophysiology in any individual patient.[24,25] All patients in the present study carried radiographic evidence of some other degenerative process. The two groups differed with respect to the concomitant presence of facet arthropathy (greater in patients with SI syndrome) and spondylosis (greater in patients with lumbar radiculopathy). These results are not surprising as the etiology of facet arthropathy is believed to be mechanical in origin, and foraminal stenosis (spondylosis) causes radiculopathy.

The presence of concomitant spinal comorbidities may make it impossible to obtain truly distinct homogenous pathophysiologic groupings. The customary presence of multiple pathophysiologic conditions in any patient with low back pain may confound the ability of the SF-36 to detect differences in HRQoL among different spinal diagnostic categories.

On the other hand, factors other than the etiology of low back pain may determine HRQoL. Previous studies have emphasized functional capability and psychological stress as prime determinants of HRQoL in low back pain.[26-29] Kovacs et al.[30] have suggested that HRQoL is correlated with pain and disability rather than etiology of low back pain. Biomechanical factors determine pain, but psychosocial factors influence the development and duration of disability. The results of the present study would suggest that the construct of diagnostic categories of low back pain may not be a determinant of HRQoL.

The results of this study also suggest that there is no difference in pain scores between patients with SI syndrome or radiculopathy. With respect to pain descriptors (McGill), SI syndrome and lumbar radiculopathy patients experienced the same “quality” of pain as represented by their sensory, affective, neuropathic and non-neuropathic scale sums. The “intensity” of pain was also statistically similar. If HRQoL is correlated with pain as suggested

by Kovacs et al.[30], then the similarities in pain scores would suggest that there is no difference in HRQoL between patients with SI syndrome or lumbar radiculopathy when age and gender are controlled.

Limitations of the present study include its retrospective design and the large number of patients excluded because of insufficient or incomplete data. Moreover, the typical quality of life study analyses multiple assessments over time to evaluate the effect of an intervention on quality of life. Quality of life studies that use only a single assessment (as does the present study) typically examine one disease state (unlike the present study) and then perform uni- and multivariate analyses to determine which factors contribute to promoting or reducing quality of life (unlike the present study).

At the time of this study, no literature has been published assessing the quality of life in SI syndrome patients. This is only the second study to assess the epidemiology of SI syndrome in low back pain patients.[7] Our findings show a higher prevalence of cumulative injury causing SI syndrome in contrast to the previous study.[7]

The results of this study suggest: (1) there is no true difference in the HRQoL or pain scores/descriptors between patients with SI syndrome or lumbar radiculopathy, or (2) the presence of comorbid spinal conditions confounds the ability of the SF-36 to detect disparities in HRQoL among differing etiologies of low back pain despite the use of rigorous diagnostic criteria, and/or (3) other factors besides the etiology of low back pain (e.g, functional capability, psychological stress) are primary determinants of HRQoL. To our knowledge, no other study has attempted to detect differences in HRQoL among different spinal diagnostic categories using the SF-36.

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| **Table 1.** Demographics of SI Syndrome by Etiology | | | | |
|---|---|---|---|---|
| | Age | Gender | Duration of Pain (yr) | Postinjection Analgesia (d) |
| Trauma (N = 23) | 49.4 ± 3.3 | F14:M4 | 3.8 ± 1.0 | 33.1 ± 12.9 |
| Cumulative injury (N=8) | 54.0 ± 6.0 | F6:M2 | 5.7 ± 4.0 | 42.8 ± 24.7 |
| Previous Back Surgery (N = 13) | 63.9 ± 3.1 | F10:M3 | 7.7 ± 2.2 | 21.6 ± 8.8 |
| Idiopathic (N=42) | 62.5 ± 2.5 | F37:M7 | 2.6 ± 0.4 | 45.6 ± 7.7 |

Abbreviations: d, day; F, Female; M, Male; SI, sacroiliac; yr, year.

**Table 2.** McGill Pain Scores

| | Sensory Σ | Affective Σ | Neuropathic Σ | Non-neuro Σ |
|---|---|---|---|---|
| SI Syndrome | 12.8 ± 0.9 | 3.9 ± 0.4 | 6.0 ± 0.4 | 6.8 ± 0.6 |
| Radiculopathy | 12.5 ± 0.9 | 3.8 ± 0.4 | 5.7 ± 0.4 | 6.9 ± 0.6 |

Abbreviations: Σ, sum; Non-neuro, Non-neuropathic; SI, sacroiliac.

**Table 3.** Visual Numerical Pain Scores

| | VNP "Worst" | VNP "Best" | VNP "Average" |
|---|---|---|---|
| SI Syndrome | 8.6 $\pm$ 0.2 | 4.1 $\pm$ 0.3 | 6.0 $\pm$ 0.2 |
| Radiculopathy | 9.9 $\pm$ 0.7 | 4.6 $\pm$ 0.3 | 6.7 $\pm$ 0.2 |

Abbreviations: SI, sacroiliac; VNP, verbal numerical pain score.

**Table 4.** SF-36 Subscale Scores

| | Physical Functioning | Role Physical | Bodily Pain | General Health |
|---|---|---|---|---|
| SI Syndrome | 28.2 ± 1.2 | 28.0 ± 1.3 | 30.7 ± 0.8 | 40.2 ± 1.4 |
| Radiculopathy | 30.0 ± 1.5 | 30.5 ± 1.5 | 30.6 ± 1.1 | 41.0 ± 1,4 |
| | | | | |
| | Vitality | Social Functioning | Role Emotional | Mental Health |
| SI Syndrome | 38.6 ± 1.3 | 31.7 +/- 1.5 | 34.3 +/- 2.1 | 41.1 +/- 1.7 |
| Radiculopathy | 40.9 ± 1.3 | 31.3 +/- 1.7 | 35.4 +/- 2.1 | 40.3 +/- 1.6 |

Abbreviations: SI, sacroiliac.