

An ultrasound study of the long posterior sacroiliac ligament in healthy volunteers and in patients with noninflammatory sacroiliac joint pain

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Abstract

Aim: To describe the sonoanatomy of the long posterior sacroiliac ligament (LPSL) in healthy volunteers and to assess by ultrasound the LPSL in patients with noninflammatory sacroiliac joint pain (SIP). **Material and methods:** We assessed 64 LPSLs of 32 healthy controls and 40 LPSLs of 40 patients with unilateral noninflammatory SIP and a positive Fortin finger test. LPSLs in both groups were assessed for the presence of alterations in their structure, continuity and echogenicity and their thickness was measured in three predefined points. All patients were examined in prone position following a strict scanning protocol. **Results:** Detailed sonoanatomy description and measurement of the LPSL in healthy volunteers are provided (length: 31.32±4.79 mm, width: 8.14±1.28 mm, thickness: 2.05±0.55 mm; 1.64±0.41 mm and 1.51±0.42 mm at the iliac and sacral entheses and in its middle part, respectively). The LPSLs were found to be significantly thicker in the SIP group, with an optimum criterion value of >2.0 mm in its middle part to identify pathologically thickened ligaments. In addition, LPSLs in the SIP group presented significantly more often hypoechogenicity/altered fibrillar structure (57.5% vs.16%) and/or periligamentous edema (72.8% vs 28%). The combination of either altered structure or periligamentous edema, with thickening of the ligament's body showed the best diagnostic accuracy (sensitivity and specificity 83.9% and 94.7% for the first combination and 100% and 84.6% for the second combination) to identify LPSL pathology in noninflammatory SIP. **Conclusions:** LPSL could be assessed by ultrasound and sonopathological lesions could be identified in patients with SIP.

Keywords: ultrasonography; long posterior sacroiliac ligament; sacroiliac joint

Introduction

The long posterior sacroiliac ligament (LPSL) is the most superficial ligament that supports the sacroiliac joint (SIJ) [1]. It spans between the inferior pole of the posterior superior iliac spine (PSIS) and the lateral crest and transverse tubercula of the third and fourth sacral segments. [2]. The main function of this ligament is to stabilize the SIJ, resisting the counternutation of the sa-

crum during walking and standing [3]. It also serves as an important linking point between the myofascial systems of the lower back and the gluteal regions, as both the erector spinae aponeurosis (ESA) and the gluteus maximus aponeurosis (GMA) have attachments on LPSL [4].

It was suggested that, in addition to the SIJ, the LPSL could, by itself, represent a discrete and frequent source of pain in the sacroiliac region [1,3,5]. Anatomical and biomechanical studies reveal several potential mechanisms for this situation: 1. as LPSL resists counternutation in the SIJ, the increased laxity and instability of this joint in a condition known as SIJ dysfunction, can lead to an increased tension and eventual damage of the ligament [3]; 2. the LPSL is pierced by the lateral branches of the dorsal rami of S2 and S3 spinal nerves, which could be compressed in the thickened, slugged or inflamed ligament giving rise to pain due to entrapment neurop-

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athy [6,7]; and 3. histological data shows that both ESA and GMA aponeuroses are in fact an integral part of the LPSL, forming its layered structure (fig 1) [4]. Thus, an unproper or discordant pull from these two big muscles could potentially lead to internal PSIL injury or dehiscence of its layers.

Clinical assessment of the LPSL can be challenging. The ligament can be palpated just caudal to PSIS but is difficult to differentiate due to its bone-like texture [3]. On the other hand, pain in the same area (as pointed out by the patient) is considered a good predictor of SIJ pathology and was named Fortin finger test [8,9].

Being a relatively superficial structure, the LPSL should be well suited for ultrasound (US) examination. Indeed, Moore et al and Le Goff et al were able to visualize the ligament with sufficient details in healthy volunteers [10,11]. These authors described a scanning protocol and the basic normal sonoanatomy of the LPSL, thus providing the background for the assessment of this ligament in patients.

While such an application of US seems logical and practical, to the best of our knowledge, there are no published studies on the subject. The aims of our study were to describe in detail the sonoanatomy of the LPSL in healthy subjects, to assess the LPSL in patients suffering from subacute or chronic noninflammatory sacroiliac joint pain (SIP) and to elaborate diagnostic models to identify accurately LPSL pathology in the clinical practice.

Material and methods

The study was approved by the Ethical Commission of the Medical University of Plovdiv and all participants signed an informed consent form at enrollment.

In the first stage of the study, 32 healthy individuals with no current or over the last year pain in the lower back, pelvic girdle or hips were recruited. In this group, the normal sonoanatomy of LPSL was assessed and described.

For the second stage of the study, participants were selected from patients referred to a tertiary rheumatology center due to unilateral subacute or chronic SIP. Patients were offered to participate if their SIP did not have the character of inflammatory back pain according to the Assessment of Spondyloarthritis International Society (ASAS) criteria [12], had a recent X-ray of the pelvic girdle with no radiographic signs of sacroiliitis and clinically had a positive Fortin finger test [8]. In total, 40 patients were enrolled according to the outlined inclusion criteria.

Exclusion criteria were a diagnosis of Spondyloarthritis (SpA), age under 18, BMI >30, radiculitis or neuro-

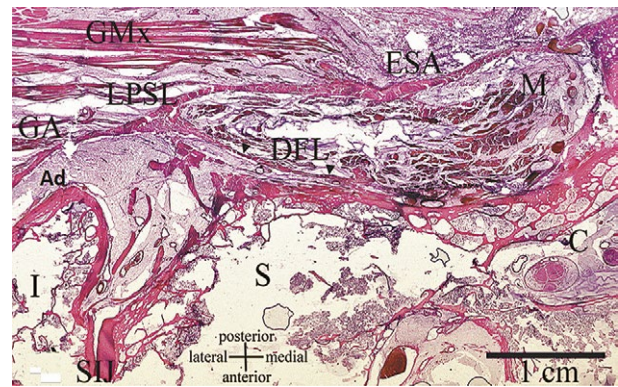


Fig 1. The normal long posterior sacroiliac ligament (LPSL). Transverse section across the posterior sacroiliac region (H&E): The erector spinae aponeurosis (ESA) medially, the deep fascial layer (DFL), inferio-medially and the gluteal aponeurosis (GA) laterally, all attached to the long posterior sacroiliac ligament (LPSL). Deep to the LPSL and posterior to the sacroiliac joint (SIJ), a region of adipose and loose connective tissue (Ad) extended laterally, deep to the GA over the ilium and medially, deep to the DFL over the posterior sacrum. I: Ilium; S: Sacrum; GMx: Gluteus maximus, M: multifidus, C: sacral canal, SF: sacral foramina (Reprinted with permission from: McGrath et al. *Joint Bone Spine* 2009;76:57-62).

pathic pain, fibromyalgia or myofascial pain syndromes, recent osteoporotic fractures, alloplastic hip joint(s), surgical procedures in the lumbar spine, SIJ cortisone injections in the last six months.

Ultrasound examination

Esaote My Lab 7 machine with a linear multifrequency transducer (4-12 MHz) was used. All subjects were examined in prone position by the same sonographer (PT) with 10 years of experience in musculoskeletal US. A strict scanning protocol was followed for all subjects. First, the probe was placed in the transverse plane over the midline of the sacrum. After the characteristic contour of the sacral spinous processes was identified, the probe was moved laterally, first to the right side. When the SIJ was reached, the probe, kept in the transverse plane, was moved upwards following the contour of the iliac bone until the PSIS was identified. At this point the medial part of the transducer was rotated counterclockwise, while the lateral part was kept at the PSIS, until a well-defined fibrillar structure, spanning between the inferior pole of the PSIS and a tuberculum at the lateral border of the sacrum, was identified (fig 2a). This structure, visible in the described oblique position, is the LPSL. To eliminate the anisotropy, a slight pressure was applied with the inferior pole of the probe. Then the transducer was slowly rotated 90 degrees to stand perpendicular to the LPSL, examining it in the transverse axis. The same protocol was carried out for the left sided ligament (here the rotation of the transducer, once PSIS is identified, should be clockwise).

In still images the LPSL thickness was measured in its long axis at three reference points: at its iliac enthesis (at a point where the deep margin of the ligament meets the iliac bone), at its sacral enthesis (at a point where the deep margin of the ligament meets the sacrum) and at its middle part (ligament body). In addition, the ligament body thickness and width were measured in the short axis. As a final value at a given point, the mean of three consecutive measurements was recorded, as studies show that repetitive measurements provide better reliability [13]. In addition, LPSL was assessed for possible sonopathological features: 1. hypoechogenicity or altered fibrillar structure; 2. presence of well-defined anechoic zones within the ligament (partial-thickness tears); 3. an anechoic rim along the entire or at least one third of the ligament length (periligamentous edema). All of these sono-markers were recorded as dichotomous variables, i.e. present/absent.

Reference images of the LPSL were saved in a JPG format. They were used to test the intra-reader reliability of the ultrasonographer (PT) and an inter-reader agreement with a novice sonographer with only a several months experience (LM). Before testing, the novice sonographer received a special training in LPSL anatomy and US appearance. For the reliability testing, 10 randomly selected sets of images, each set consisting of two reference images of a single ligament, were used. The intra-reader testing was performed about 6 months after the initial scan, blinded to the previous results and the identity of patients/controls. The sonopathological lesions reported are relative to the first analysis.

Statistical analysis

The statistical software used to perform the analysis included IBM SPSS version 26 (2018), Minitab version 19 (2019) and MedCalc version 19.4.1 (2020). We examined the data for normality through the Shapiro-Wilk's test and took into consideration the values of skewness. Continuously measured and normally distributed variables were described through the mean values and standard deviations (\pm SD). Categorical data were processed in frequencies and percentages. An independent-samples t-test was used for two-group comparisons on normally distributed continuous variables. When the assumption of equal variances was not observed (Levene's statistic $p < 0.05$), we reported the results for equal variances not observed (uv). For multiple comparisons, Bonferroni correction was applied to control for Type I error. In such cases, statistical significance was accepted if $p < 0.0125$.

ROC curve analysis was used to establish the diagnostic potential, optimal criterion values and the associated sensitivity and specificity of LPSL thickness.

Associations between categorical variables (individual sono-lesions) were examined through Chi-square test and/or Fisher's exact test. Odds ratios were calculated to establish the odds for the occurrence of a certain sono-lesion in patients with SIP. Pearson correlation r was employed to examine the relationship between continuously measured and normally distributed variables and Spearman rank-order correlation was performed when those conditions were not observed. The intra-rater and inter-rater agreement were established through Cohen's kappa (95% confidence intervals).

Results

LPSL in healthy individuals

The LPSL was assessed bilaterally in 32 healthy individuals (64 ligaments), 22 females, 10 males, mean age of 41.97 ± 12.87 years and mean BMI of 23.21 ± 3.52 . We were able to visualize the ligament with the above-described protocol in all subjects.

In the longitudinal axis, the LPSL was seen as a hyperechoic, slightly concave fibrillar structure, with well-defined margins and predominantly uniform thickness that spans over the SIJ and attaches to the ilium (just inferior to the PSIS) and the lateral sacrum (to the sacral tubercle, a structure of variable size in different subjects) (fig 2b). In the short axis, the LPSL exhibited a flat to oval shape, layered structure, well-defined upper margin, and less conspicuous inferior margin (fig 2c). Medially, the ligament merged with the lateral portion of ESA, a finding also shown previously [14]. The gluteus maximus muscle was seen to override the ligament from a lateral direction, while the inferior gluteal aponeurosis attached to the LPSL's lateral part. Thus, the LPSL appeared as a link between the thicker and more stretched ESA medially and the thinner and looser GMA laterally. Beneath the ligament, a fibro-adipose tissue of variable amount and echogenicity was seen. This tissue occupied the posterior part of the SIJ and expanded to the sacral foramina and over the ilium.

The mean length of the LPSL was 32.32 ± 4.29 mm, and its thickness at the iliac entheses, the middle part (ligament body) and the sacral entheses was 2.05 ± 0.55 mm, 1.51 ± 0.42 mm and 1.64 ± 0.41 mm, respectively. The width of the LPSL body measured in the transverse plane was 8.14 ± 1.28 mm.

No significant associations were observed between the subjects' age, sex, the side of the body and LPSL measurements.

LPSL in patients with SIP

For the second part of the study, 40 SIP patients were enrolled according to the above-pointed criteria and com-

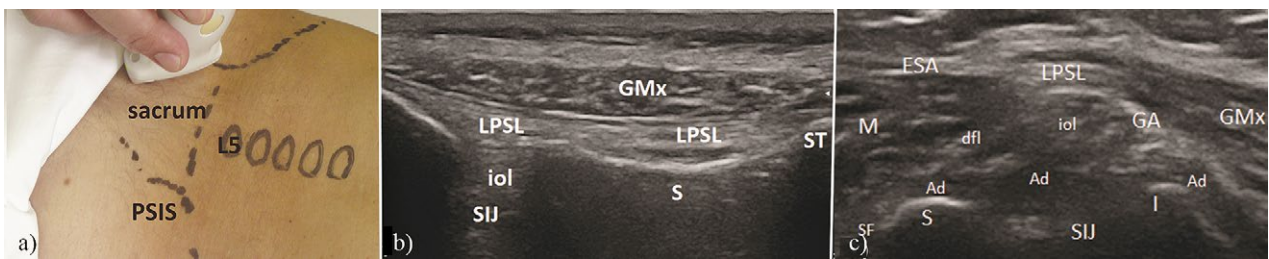


Fig 2. a) Position of the transducer to assess the left LPSL in its longitudinal axis; b) ultrasound image of the normal LPSL in the longitudinal plane. The ligament spans from the posterior superior iliac spine (PSIL) of the ilium (I) to the sacral tuberculum (ST) of the lateral sacrum (S). Deep to it are laying the thinner and less conspicuous interosseous ligaments (iol) that occupy the posterior part of the sacroiliac joint (SIJ) cleft; c) ultrasound image of the middle part (body) of a normal LPSL in the transverse plane, the left side of the image is medial. The erector spinae aponeurosis (ESA) and the deep fascial layer (dfl) attach to the LPSL from the medial side, while the inferior gluteal aponeurosis (GA) attaches to the lateral side. Deep to LPSL, the thinner and less conspicuous interosseous ligaments (iol) in cross section might be seen. Posterior to the sacroiliac joint (SIJ), a region of adipose tissue is seen (Ad). GMx: Gluteus maximus, M: multifidus, SF: sacral foramina.

pared to the healthy controls. The demographic data is detailed in Table I.

The thickness of the LPSL in SIP patients measured at the three reference points, i.e. the iliac entheses, the middle part (ligament body) and the sacral entheses was 3.23 ± 1.00 mm, 2.55 ± 0.70 mm and 2.52 ± 0.66 mm, respectively. These measurements were compared through independent-samples t-tests with an adjusted *alpha* level of 0.0125 to the thickness of the 64 ligaments (32 right and 32 left sided) of the 32 control subjects described in the first part of the study.

A strong to very strong significant trend associated with higher LPSL thickness values in the SIP patients group as compared to the healthy controls was established at all three measurement points (<0.001) (fig 3)

A ROC curve analysis revealed that among the three measurements, the ligament body had the highest level of diagnostic potential in distinguishing LPSL in SIP from those in controls (AUC = 0.910, $p < 0.001$) with optimum criterion value of >2.0 mm (79.49% sensitivity and 91.53% specificity).

Table I. Demographic data of the sacroiliac pain (SIP) patients and healthy controls

Variables	Groups		p
	SIP patients (N = 40)	Healthy controls (N = 32)	
Age	42.90±11.35 (20-70)	41.97±12.87 (18-69)	0.708 ^t
Female gender	28(70)	22 (69)	1.00 ^f
Height	169.78±6.62	167.50±9.07	0.143 ^{t(uv)}
BMI	24.56±3.26	23.21±3.52	0.054 ^t

The results are expressed as mean±standard deviation, (minim-maxim) or number(%). t - independent-samples t-test; f - Fisher's exact test; t(uv) - independent-samples t-test with unequal variances

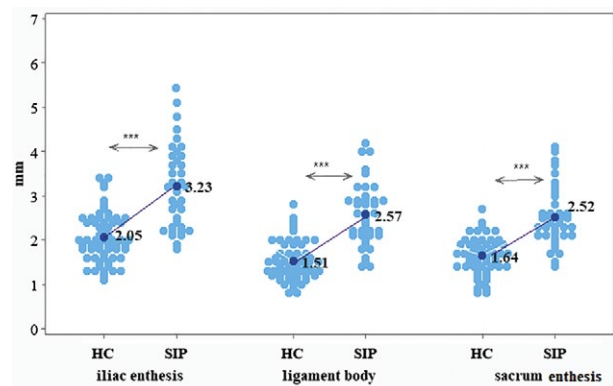


Fig 3. Individual value plot of the iliac entheses, ligament body and sacrum entheses thickness measurements. ***: $p < 0.001$.

Among the assessed sonopathological lesions, the most frequently detected one was the periligamentous edema (fig 4a), followed by the hypoechoogenicity/altered fibrillar structure of the ligament (fig 4b,c), while partial thickness tears (fig 4d) were rare. The frequency of observation of these lesions in the LPSLs of both study groups are summarized in Table II.

The majority of the LPSLs in the SIP patients (84%) exhibited ≥ 2 of the assessed sonopathological markers (fig 4e-h). No significant association was found between the cumulative number of presented sonopathological features and the age and sex of the participants.

The intra-reader agreement was perfect for hypoechoogenicity (kappa = 1.0, 95 CI:1.0 to 1.0) and tears (kappa = 1.0, 95% CI:1.0 to 1.0) and good for periligamentous edema (kappa = 0.800, 95% CI: 0.43 to 1.0). The inter-reader agreement with a novice sonographer was perfect for periligamentous edema (kappa = 1.0, 95% CI:1.0 to 1.0), acceptable for tears (kappa = 0.782, 95%CI: 0.38 to 1.00), but low for hypoechoogenicity/altered fibrillar structure (kappa = 0.600, 95% CI: 0.14 to 1.00).

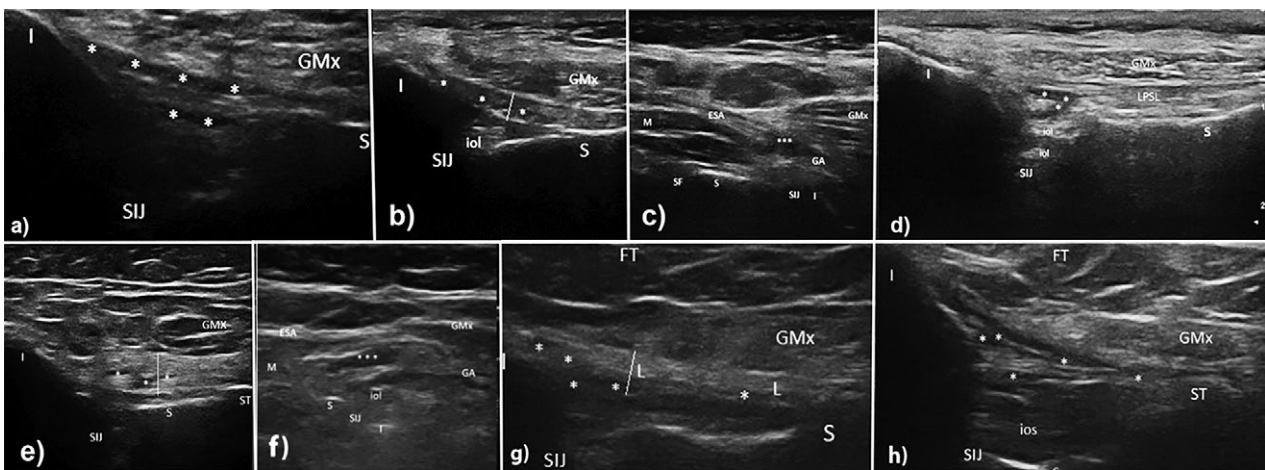


Fig 4. The spectrum of LPSL sonopathological lesions: a) Periligamentous edema (*); b) thickened and hypoechoic LPSL (*) in the longitudinal plane and c) in the transverse plane; d) LPSL with a partial thickness tear (*); e) a thickened LPSL with a partial tear (*) in the longitudinal plane and f) in the transverse plane; g) a thickened LPSL with altered, hypoechoic structure (*); h) LPSL with a pariligamentous edema and an altered fibrillar structure (*). I-ilium, S-sacrum, ST-sacral tuberculum, SIJ-sacroiliac joint; GMx – gluteus maximus. iol – interosseus ligaments, M-multifidus, FT – fatty tissue.

Table II. Sonopathology of long posterior sacroiliac ligament in sacroiliac pain (SIP) patients and healthy controls

Parameters	SIP patients	Healthy controls	OR (95% CI)	p
Hypoechoogenicity				
Positive	23 (57.5)	10 (16)	7.20 (2.80 to 18.50)	<0.001
Negative	15 (37.5)	47 (73)		
Undetermined	2 (5)	7 (11)		
Periligamentous edema				
Positive	29 (72.5)	18 (28)	7.25 (2.94 to 17.82)	<0.001
Negative	11 (27.5)	46 (72)		
Undetermined	0 (0)	0 (0)		
Intraligamentous tear				
Positive	7 (17.5)	0 (0)	26.64 (1.47 to 481.24)	0.026
Negative	33 (82.5)	59 (87.5)		
Undetermined	0 (0)	8 (12.5)		

The results are expressed in absolute numbers(%).

Diagnostic US models for identification of LPSL pathology

Thickening of the ligament body was found to be associated with the largest AUC among all three points of measurement. Periligamentous edema and hypoechoogenicity/altered fibrillar structure were the two most frequently observed sonopathological markers. Accordingly, we tested the cumulative diagnostic accuracy of the ligament body thickening in conjunction with either hypoechoogenicity or periligamentous edema through binary logistic regression and ROC curve analysis.

The first model (ligament body thickening plus hypoechoogenicity/altered structure) was significant (Chi-square = 76.217, p<0.001), with AUC = 0.953 (95%CI: 0.911 to 0.995), sensitivity = 83.78%, specificity =

94.74%, positive predictive value = 91.19%, negative predictive value = 89.98%.

The second model (ligament body thickening plus periligamentous edema) was also significant (Chi-square = 80.49, p<0.001) with AUC = 0.964 (95%CI: 0.931 to 0.996), sensitivity = 100%, specificity = 84.75%, positive predictive value = 81.26%, negative predictive value = 100%.

Discussion

There is growing evidence that soft tissue structures posterior to the SIJ (including the LPSL) could be responsible for a significant proportion of SIP [1,3,5,15,16]. For example, it is frequently reported that there is no major

difference in the efficacy of SIJ injections, regardless of whether they are delivered strictly intraarticularly or the infiltration is done periarticularly [17,18]. Indeed, the US evaluation of the LPSL, performed in our study, revealed ligamentous sonopathological lesions in more than two thirds of the patients with SIP. This is in line with the data of Murakami et al, who advocate that the first intervention in SIP should be an injection in its ligamentous area, as four of five SIP patients would have benefit from this approach [19]. The role of sacroiliac ligaments as pain generators was further confirmed empirically by Saunders et al. The authors evaluated the effect of US guided injections in the LPSLs in patients with SIJ dysfunction and pain [20] and reported significant improvement in all scores and performance indicators that lasted throughout the entire 12-month follow-up period. In addition, a recent study, performed by SPECT-CT, shows increased uptake in the region of LPSL in patients complaining of SIP and a clinical diagnosis of SIJ dysfunction [21]. Furthermore, the intensity of the uptake could be well quantified and possibly used as a follow-up tool. However, this imaging method utilizes an ionizing radiation, which would limit its use in the daily clinical practice.

In the present study we used US, a safe and widely available imaging method. US was first used to assess the LPSL in healthy volunteers by Moore et al and later by LeGoff et al [10,11]. In addition to these studies, we provide data on the normal thickness and width of LPSL as well as more detailed description of its sonoanatomy and relations to the surrounding structures. We have measured the LPSL thickness at three easily reproducible points, namely at its iliac and sacral entheses and at its middle part. These sites were chosen on the basis of the morphology data, showing a different structure of the ligament's body in comparison to its entheses [4].

Thickening is one of the major US features of ligamentous injury [22]. Our results show that thickness was increased at all three sites, the ligament body being most pronounced. Comparing to its entheses, the LPSL body has a more complex structure, consisting of layers and incorporating the ESA and GMA. Thus, ligament body thickening could in fact reflect the thickening of either or both of these aponeuroses – a finding similar to the thickening of the thoracolumbar fascia in patients with chronic low back pain, described by Langevin et al [23]. However, this suggestion should be confirmed by histological proof. This ligament thickening could also be the morphological basis for the compression of the lateral branches of S2 and S3 dorsal rami and entrapment neuropathy which was proposed by different authors as a potential reason for SIP [6,7,11].

In the majority of ligaments in the SIP study group (72.5%), we observed a periligamentous edema. It was seen as well-defined anechoic rim superiorly or, less frequently, inferiorly along the LPSL body. This phenomenon has been previously reported in traumatic and degenerative ligamentous pathology [24,25], but its histological nature remains unclear. In the case of LPSL it may represent a pathological process similar to paratenonitis that involves the ESA and/or GMA.

In assessing the fibrillar structure and the echogenicity of the LPSL, we found diffuse or local hypoechoogenicity/altered fibrillar structure significantly more often in ligaments from the SIP patients group. Hypoechoogenicity of ligaments is considered to be caused by the disorganization of the collagen fibrils and edema of the clefts between the fibrils and the ligament matrix [22]. Thus, hypoechoogenicity may indicate a pathological transformation of the ligamentous tissue, especially when this finding is accompanied by a thickening. In addition to this, the altered fibrillar structure may represent focal or more global dehiscence between the ligament's layers.

Less frequently, partial intrasubstance tears of the LPSL were detected. This might be a rare injury in a tight ligament such as the LPSL that supports an intrinsically stable joint as the sacroiliac [1,2], but nevertheless, no tears were detected among the 64 ligaments in the control group.

The analysis of the cumulative occurrence of pathological sono-markers (other than thickening) in the LPSL of patients vs. controls revealed that the majority of the painful ligaments (84%) exhibited two or all three pathological sono-markers. The combination of ligament's body thickening and the presence of hypoechoogenicity/altered fibrillar structure or periligamentous edema yielded the highest values for sensitivity and specificity in identifying ligamentous injury in SIP patients.

In our study we used a single clinical test to diagnose patients as having probable SIP (Fortin finger test [8]), while most of the studies on SIJ pathologies use a composite of tests [9,16,20]. Anyway, our target was the PSIL, which is an extraarticular and relatively superficial structure [3]. Such extraarticular and superficial source of pain would be expected to be associated with a well-described locus of pain [1,6] that the patients would be able to point out – like in the Fortin finger test. In addition, a similar test was used by other authors under the name of a one-finger test resulting in a more accurate identification of the site of pain in the sacroiliac region [26].

Our study has certain limitations. First and foremost, findings were not compared to a “gold standard” or another imaging modality. However, there is no approved

gold standard for the imaging of LPSL and people with SIP would not normally need an operation. Thus, a confirmation of the sonographic findings at surgery as a gold standard is virtually impossible. Two other possible imaging modalities to assess the LPSL would be MRI and SPECT-CT. MRI has certain limitations as ligaments are relatively avascular structures. Besides MRI is still an expensive option. SPECT-CT seems promising for depicting LPSL pathology, but it involves a considerable amount of ionizing radiation, and the images have a lower anatomical resolution. Secondly, patients and control subjects in the study were with normal BMI while in more obese patients, the examination of the LPSL could be more difficult. However, as the ligament presents a well-defined hyperechoic fibrillar structure, it should be possible to assess it even in these cases. Thirdly, throughout the course of data collection, the researcher undertaking the US assessment was not blinded to the group of each participant, and as such, there was a potential for observer bias. However, to minimize subjectivity, a strict scanning protocol was followed in each subject and the sonopathological lesions were well- and predefined and repeated measurements undertaken. Lastly, we did not use diagnostic periarticular SIJ injections to differentiate patients with ligamentous pain. Yet, periarticular SIJ injections are still not that well standardized (as the intraarticular ones) and one problem to their diagnostic use might be the unpredictable spread of the injectate in the periarticular tissues.

Conclusion

Our study confirms that the LPSL could be visualized by US in details and its structure and relations - accurately assessed. In addition, in patients with noninflammatory SIP, the US evaluation of the LPSL could reveal its thickening as well as sonopathological transformation: hypoechogenicity/altered fibrillar structure and periligamentous edema. Further studies are required in order to confirm the value of these findings for the clinical practice.

Conflict of interest: none

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