What patient parameters influence lumbar stiffness in patients with hip pathology?

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Title page

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Running title

Parameters influencing lumbar stiffness

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Statement and declarations

This project has been approved by the institutional review board of the respective institutions.

All patients have signed an informed consent for participation and publication

Author contributions

- *J. Verhaegen, hip arthroplasty fellow: Data collection, statistical analysis, writing the paper*
- *Nuno Alves Batista, fellowship-trained spine surgeon: Data collection & assessment spinal factors*
- *Ryan foster, fellowship-trained MSK radiologist: Methodology, conceptualization, data collection & assessment MRI factors, interpretation of results, revising the manuscript*
- *Ryan Graham, professor biomechanics: Methodology, conceptualization, interpretation of results, revising the manuscript*
- *Phillipe Phan, fellowship-trained spine surgeon: Methodology, conceptualization, interpretation of results, revising the manuscript*
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Abstract

Background: Lumbar stiffness leads to greater hip dependance to achieve sagittal motion and increases instability after total hip arthroplasty (THA). We aimed to determine parameters that influence lumbar stiffness amongst patients with hip pathology.

Methods: In this retrospective, consecutive case series from a tertiary referral center, patients presenting at a hip specialist clinic underwent standing and deep-seated radiographic assessment to measure lumbar lordosis (ΔLL) (stiffness: ΔLL<20°), hip flexion (ΔPFA: pelvic-femoral angle) and degree of degenerative-disc-disease (DDD) (facet osteoarthritis, disc height, endplate proliferative changes). Of these, 65 patients were selected with previous lumbar spine Magnetic Resonance Imaging (MRI), allowing to determine lumbar facet orientation, spinal canal stenosis (Schizas classification) and flexor- and extensor- muscle atrophy (Goutallier classification).

Results: Mean ΔLL was 45° (range: 11°-72°) and 4 patients (6%) exhibited spine stiffness. Patients with multilevel DDD (n=22) had less ΔLL than those with no/single level (n=43) DDD [34° (range: 11°-53°) vs. 51° (21°-72°); p<0.001]. Number of DDD levels correlated strongly with ΔLL (rho=-0.642; p<0.001). Spinal stiffness was only seen in patients with ≥4 DDD-levels. There was no correlation between ΔLL and facet orientation (p>0.05). ΔLL correlated strongly with extensor atrophy at L3-L4 (rho=-0.473), L4-L5 (rho=-0.520) and L5-S1 (rho=-0.473), and poorly with flexors at L4-L5 (rho=-0.134) and L5-S1 (rho=-0.227).

Conclusion: Lumbar stiffness is dependent on modifiable- (muscle atrophy) and non-modifiable- (extend of DDD) factors. This can guide non-operative management of hip pathology,

emphasizing relevance of core muscle rehabilitation to improve posture and stiffness. Identification ≥4 DDD-levels should alert surgeons of increased THA instability-risk.

Key words: hip-spine syndrome, THA, spinal stiffness, degenerate-disk disease, instability

Level of evidence: IV, cohort series

Introduction

Femur, pelvis and lumbar spine form a kinetic chain that works harmoniously during daily tasks[1-3]. The importance of this interaction was emphasized in studies spanning a wide range of patient-age and conditions[4;5]. Spinopelvic characteristics are associated with the development of symptoms in patients with acetabular dysplasia[6], femoro-acetabular impingement[4;7] and hip osteoarthritis[8]. Similarly, abnormal spinopelvic characteristics and spinal stiffness (due to degenerative disease or lumbar spine fusion) is associated with inferior outcome following hip arthroplasty[9]. In the presence of lumbar stiffness, the hip must flex more for a given task, placing the replaced hip at increased risk of impingement and dislocation[10-13].

All studies that describe the dynamic interaction of femur-, pelvis-, and spine have shown a wide range of lumbar lordosis (LL) and sagittal lumbar range-of-motion (ΔLL), both in studies reporting on symptomatic and asymptomatic cohorts[7;8;14]. The degree of $LL_{standing}$ is directly proportional to the ability of the lumbar spine to flex[14]. The only other factor that has been shown to be associated with LL and ΔLL to-date has been age (increasing age leads to a reduced LL and ΔLL)[14-18].

Developing a greater understanding on what contributes to lumbar stiffness is of importance to hip and spine surgeons. Spine stiffness is associated with adverse outcomes of all types of hip pathology/surgery[5;9;10;19;20]. If spinal stiffness is due to modifiable factors (e.g., spinal muscle weakness), non-operative measures might be able to help patients with hip pathology by increasing overall contribution of the lumbar spine to the sagittal movement arc. However, if spinal stiffness reflects non-modifiable factors (such as innate facet orientation, degree of facet or disc degeneration, spinal canal stenosis), the ability to improve stiffness is limited, and only measures to prevent progression can be put in place at time of presentation.

The aim of this study was to assess whether lumbar stiffness is associated with anatomical parameters that reflect innate lumbar spine morphology, degeneration, and muscle conditioning (using Magnetic Resonance Imaging).

Methods

Study design

This is a retrospective case-cohort study using prospectively collected data at a single, tertiary referral centre [redacted] and approved by the institutional review board. After informed consent, patients who presented to our hip specialty clinic between January 1st, 2020 and 30th June 2022 were recruited.

An a priori sample size calculation was performed in SPSS Statistics v28 (IBM, New York, United States). Based on a mean ΔLL of 30±12° among controls, versus 19±10° among patients with lumbar degeneration[13], one would need minimum 19 patients per group to achieve sufficient power ($1-\beta=0.80$, $\alpha=0.05$).

A total of 725 patients underwent spinopelvic imaging. Of these, 110 patients had also undergone spinal-MRI. Forty-five patients were excluded due to lumbar fusion (n=10), ankylosing spondylitis (n=1), MRI not within 2 years of spinopelvic imaging (n=17), or insufficient imaging quality: absence of deep-seated x-rays (n=12), x-rays that did not include L1-L2 level (n=2), or MRI without axial cuts (n=3), leaving 65 patients for the definitive analysis (**Figure 1**). There were 40 women (62%) and 25 men patients (39%). Mean age was 56 years (range: 21-86 years) and mean BMI was 39 kg/m^2 (range: 17-49 kg/m^2).

Radiographic assessment

Spinopelvic measurements

Patients underwent radiographic assessment including standing and supine anteroposterior (AP) X-ray of the pelvis, and lateral views of lumbar spine, pelvis and femur in standing and "deep-flexed seated" positions. The "deep-flexed seated" position is performed with the femurs parallel to the floor on a height adjustable chair and with the trunk leaning maximally forward as per patient comfort, without abducting or rotating the femurs[8;13;21]. This position was chosen because it is associated with maximal sagittal flexion of the kinetic chain; it is the position at greatest risk of femoro-acetabular impingement[22], and has been shown to better identify spinal compensatory mechanisms[13;23;24]. The following measurements were performed: Lumbar Lordosis (LL), Sacral Slope (SS), Pelvic Incidence (PI), Pelvic Tilt (PT), Pelvic Femoral Angle (PFA)[11-13;21;25] (**Figure 2**).

Spinopelvic movements were calculated as the difference between standing and "deep-flexed seated" position ($\Delta X = \Delta X_{deep\text{-}seated} - \Delta X_{standing}$) for each spinopelvic parameter[8]. Sagittal Flexion Arc (SFA) is the movement performed by the whole kinetic chain and calculated as the sum of ΔLL and ΔPFA[8]. Spine stiffness was defined as $\Delta LL \leq 20°$[20].

Hip user index quantifies the percentage of sagittal femoroacetabular flexion (ΔPFA) with respect to overall SFA. A high hip user index means that the hip contributes more to sagittal movement, whereas in a low hip user index, the movement takes place primarily in the lumbar spine[8;23;26;27]. Patients being hip users were defined as having a hip user index $\geq 80\%$[23].

Assessment was performed by two reviewers, one fellowship-trained hip arthroplasty surgeon ([redacted]) & one fellowship-trained spine surgeon ([redacted]). Radiographic measurements were repeated for 20% of randomly selected datasets in a blinded fashion. Interobserver reliability (IORs) was calculated using the correlation coefficient with a two-way mixed model, showing excellent agreement: 0.890 (95% C.I. [Confidence Interval] 0.595 to 0.966) to 0.975 (95% C.I.; 0.925 to 0.992).

Assessment of lumbar spine degeneration

Spinal degeneration was classified based on facet osteoarthritis, disc height narrowing, and endplate proliferative changes (**Table 1**)[13]. Patients were categorized with none/single level degenerative disc degeneration (DDD) if ≤1 degenerative disc, or with multilevel DDD in case of ≥2 degenerative discs[13]. Assessments were made by a fellowship-trained spinal surgeon ([redacted]).

MRI assessments

Patients underwent 1.5- or 3.0-Tesla lumbar spine MRI for clinical purposes. A 16-channel posterior array matrix coil was utilized for signal reception. All underwent routine MRI sequences: sagittal T1-weighted spin echo (field of view 30cm; slice thickness 3mm; repetition time 400-850; and flip angle 90-180º), sagittal T2-weighted spin echo (field of view 30cm; slice thickness 3mm; repetition time 2500-11000; and flip angle 130-180º), and axial T2-weighted spin echo (field of view 18-22cm; slice thickness 3-4mm; repetition time 2500-11000; and flip angle 142-180º). Images were reviewed on institutional Picture Archiving and Communications System (PACS).

Degree of lumbar spinal stenosis

Using Schizas classification[28], spinal canal stenosis (SCS) was graded at each lumbar spine level; from grade A to D, based on presence of cerebrospinal fluid (CSF) and visibility of the rootlets inside the dural sac on T2-weighted axial slices (**Figure 3**). Patients were then identified as having SCS in case of one or more levels with grade ≥B stenosis. Assessment was performed by a fellowship-trained spine surgeon ([redacted]).

Facet orientation

Facet orientation was measured using T2-weighted axial slices. A line was drawn between the margins of each articular facet. Facet angle was measured between the facet line and midsagittal line (**Figure 4**)[29], and the mean between left and right was calculated at each level between L1 and S1. Assessment was performed by a fellowship-trained spine surgeon ([redacted]).

Assessment of muscle fatty infiltration

Qualitative assessment of muscle fatty infiltration was performed using Goutallier classification system[30;31] by a fellowship-trained senior musculoskeletal radiologist ([redacted]). Axial T2-weighted sequences were used to evaluate muscle composition, which was graded into 5 different grades based on the visually assessed fat/muscle ratio of the flexors at the levels L3-L4 and L4-5 (psoas), and of the extensors (multifidus and longissimus) at the levels L3-L4, L4-5 and L5-S1. Spinal muscular fatty infiltration generally worsens from cranial to caudal[31-33], thereby most significant in the lower lumber spine, dictating that these levels were selected for assessment. The grades ranged from grade 0, where there was no fatty infiltration, to grade 4 where there was more than 50% fat within the muscle (**Figure 5**).

Statistics

Non-parametric statistical analyses were performed after testing normal distribution of data with the Kolmogorov-Smirnov test and Q-Q plots, showing no normal distribution of data. Mann-Whitney-U was used to compare demographics, spinopelvic measurements and facet orientation between patients with and without a stiff spine. Spearman correlation coefficient was calculated. Agreement was graded as poor (*rho* ≤0.3), moderate (*rho* 0.31-0.5), strong (*rho* 0.51-0.6), very strong (*rho* >0.61)[34]. Predictors for lumbar stiffness were determined using a multiple regression analysis with stepwise data entry method. To exclude collinearity a tolerance level of >0.20 was required. Statistical analysis was performed using SPSS v28 (IBM). A p-value of <0.05 was considered significant.

Results

Demographics and spinopelvic measurements

Patients with a stiff spine were older than those without a stiff spine [79 years (range: 71-84) vs. 55 years (range: 21-86); p=0.002] (**Table 2**). Mean ΔLL was 45° (range: 11°-72°) and 4 patients (6%) had spine stiffness. Patients with a stiff spine had less $LL_{standing}$ [44° (range: 28°-67°) vs. 57° (range: 24°-80°); p=0.096) and a higher hip user index [85% (range: 83-87%) vs. 68% (51-83%); p<0.001] (**Table 2**). There was no correlation between ΔLL and ΔPFA (p=0.514).

Lumbar spine degeneration

There were 43 patients (66%) with no/single level DDD [0-levels: 34/655 (52%); 1-level: 9/65 (14%)] and 22 patients (34%) with multiple DDD [2-levels: 6/65 (9%); 3-levels: 5/65 (8%); 4-levels: 5/65 (8%); 5-levels: 6/65 (9%)]. Patients with multilevel DDD had less ΔLL than those with no/single level DDD [34° (range: 11°-53°) vs. 51° (21°-72°); p<0.001] and a higher hip user index [76% (range: 66-87%) vs. 65% (range: 51-79%); p<0.001]. The number of DDD levels showed a very strong correlation with ΔLL (rho=-0.642; p<0.001) (**Figure 6**), with 6° decrease in ΔLL per additional DDD level. A stiff spine was only found among patients with either 4- (n=1) or 5- (n=3) degenerative levels.

There were 15 patients with SCS (23.1%). Patients with multi-level DDD were more likely to have SCS (**Table 2**). ΔLL was lower in SCS patients [35.2° (range: 11.0°-53.0°) vs. 48.0° (18.0°-72.0°); p=0.002].

Facet orientation

Mean facet orientation was 39° (range: 26°-54°) and facet angles increased from proximal to distal. There was no difference in facet orientation between patients with or without stiff spine (**Table 2**). There was no correlation between ΔLL and facet orientation in any of the lumbar spine levels (rho between 0.059 and 0.292; $p>0.05$).

Muscle fat infiltration

A higher degree of muscle atrophy was associated with decreased ΔLL (**Figure 7**). This correlation was strong for the extensors at the levels L3-L4 (rho=-0.473), L4-L5 (rho=-0.520) and L5-S1 (rho=-0.473). Correlation between ΔLL and flexor muscle atrophy was poor at L4-L5 (rho=-0.134) and L5-S1 (rho=-0.227). Presence of SCS was more common in patients with muscle atrophy of extensors (p=0.009-0.029) and flexors (p=0.013-0.052).

Multivariate regression analysis

Multiple regression analysis adjusted for age, $LL_{standing}$, number of DDD levels, SCS, and spinal muscle atrophy could explain 58% of the variation ($R^2=0.578$) of ΔLL (**Table 3**). This analysis demonstrated that low ΔLL is associated with older age, higher number of DDD levels and low $LL_{standing}$.

Discussion

Hip osteoarthritis patients often present with coexisting lumbar spine degeneration[35;36]. Spine stiffness increases hip dependency during sagittal range of motion. In a seated position, a stiff spine places the hip at risk for impingement, due to decreased posterior pelvic tilt, which can be a source of pain in the native hip[7], and a risk factor for dislocation in the replaced hip[10-12]. This study showed that spine stiffness is primarily determined by non-modifiable factors such as age and DDD, defined by facet osteoarthritis, disc height narrowing and endplate proliferative changes[13]. Identification of more than 4 DDD levels should alert hip surgeons of increased risk of spinal stiffness and THA instability. Other non-modifiable factors such as facet orientation were not associated with spinal stiffness. Modifiable factors such as lumbar lordosis and spinal muscle atrophy were associated with spinal stiffness. However, their association is subordinate to age and degeneration. In younger patients with less degenerative changes, non-operative management (core muscle rehabilitation) may help to improve posture and range-of-motion. Extensors (multifidus/erector spinae) had a higher correlation with maintenance of lumbar spine curvature and motion than flexors (psoas).

In standing position, patients with a stiff spine have a decreased lumbar lordosis, relatively to those with no stiff spine, which is accompanied by increased posterior pelvic tilt. Whereas when seated, patients with a stiff spine show increased lumbar lordosis [8;12-14]. Thus, the reduction of the sagittal arc occurs on both in flexion and extension. Age and $LL_{standing}$ are important predictors of spine stiffness. With aging, the lumbar spine loses its LL and ΔLL to a greater extent that then the hip and resultantly, the hip's relative contribution to overall sagittal movement increases[14]. ΔLL can be expected to decrease with 4.5° per decade[14]. In addition, this study showed that patients lose 6°

ΔLL per degenerative level. Esposito et al found that DDD patients had 10° less ΔLL. However, this study used the relaxed-seated position, instead of the deep-flexed seated position, which represents the maximal sagittal flexion of the kinetic chain, and is the position at risk for impingement[22] or dislocation[9], allowing to better identify spinal compensatory mechanisms[23;24].

Previous studies have shown a relationship between facet orientation and kinematics of the lumbar spine, suggesting that patients with lower facet angles have greater mobility than those with higher facet angles[29;37], predisposing them to degenerative changes[38]. However, most of these studies have examined the effect of facet joint orientation on anteroposterior motion of one vertebra over the other, in the context of spondylolisthesis[29;37;38]. In this study, we found no correlation between facet orientation and spinal stiffness, nor with any of the other spinopelvic parameters, and hence it would unlikely influence hip pathology or outcome of surgery.

Fat infiltration and lumbar muscle atrophy are related to spinal degenerative disorders and may contribute to changes in posture[39]. Among asymptomatic volunteers, with normal sagittal balance, spinopelvic parameters have been shown to be associated with lumbar muscle volume, but not with muscle fat infiltration[40]. Whilst among symptomatic patients, spinopelvic malalignment, defined as an increased standing posterior pelvic tilt >20° or as a mismatch between PI and LL >10°, was found to be associated with greater fatty infiltration of lumbar spine flexors and extensors[41;42]. In this study, we found that spinal stiffness (ΔLL<20°) was associated with a higher degree of muscle fatty infiltration. This association was the strongest for the extensors (multifidus/erector spinae), but less present for the flexors (psoas). Previous studies in patients with degenerative kyphosis have shown that multifidus and erector spinae are critical to maintain lumbar spine curvature, by increasing anterior pelvic tilt and lumbar lordosis[43], whilst the psoas was not correlated with changes in spinopelvic configuration[44]. These were studies on *static* standing spinopelvic characteristics, whilst this is the first study to describe *quasi-static* characteristics i.e. spine stiffness, associated with risk of THA instability[10-13]. Muscle atrophy can occur for several reasons, including age and disuse[45]. It is plausible that muscle atrophy contributes to development of degenerative changes, but it may also be caused by the degenerative process itself, leading to disuse and muscle atrophy. Spinal stenosis is caused by degenerative changes[46], but could also attribute to muscle atrophy[47]. Based on the multivariate analysis in this study, the association between spine stiffness, stenosis and muscle atrophy was subordinate to age and lumbar spine degeneration. Therefore, strategies to modify and improve muscle atrophy may yield only limited effect on spine stiffness in older patients with multilevel DDD. However, in younger patients with less degenerative changes, but signs of muscle atrophy, future, prospective, research should evaluate whether non-operative management (core muscle rehabilitation exercises) can improve posture and stiffness.

This study is not without limitations. First, whilst spinopelvic assessments are prospectively recorded on patients seen in clinic, assessment of MRI studies was done retrospectively. Therefore, this study suffers of the limitations associated with its retrospective design. Secondly, all patients that underwent a lumbar spine MRI were symptomatic and no asymptomatic comparison group with lumbar spine MRI was available. Thirdly, whilst conventional T2-weighted MRI images are the most commonly used tool to assess for muscle fat infiltration, its accuracy is relatively low[48]. Advanced MRI approaches, such as Magnetic Resonance Spectroscopy (MRS) and chemical-shift MRI[49;50], or advanced imaging parameters, based on area and signal intensity, might provide better accuracy in the assessment of muscle atrophy[48]. Furthermore, whole fatty infiltration describes the fatty tissue within a muscle relative to the muscle cross-sectional area, whilst muscle atrophy describes a decrease in muscle cross-sectional area[41], which was not measured in this study. Lastly, muscle atrophy and fatty infiltration does not necessarily reflect (modifiable) muscle weakness. It is unknown whether fatty infiltration can be improved with an intervention or exercise therapy.

Conclusion

Spine stiffness is primarily determined by non-modifiable factors such as age and DDD. Identification of more than 4 DDD levels should alert hip surgeons of increased risk of spinal stiffness and THA instability. Other non-modifiable factors such as facet orientation were not associated with spinal stiffness. Modifiable factors such as lumbar lordosis and spinal muscle atrophy contribute to spinal stiffness. However, their contribution is subordinate to age and degeneration. In younger patients with less degenerative changes, non-operative management (core muscle rehabilitation) may help to improve posture and range-of-motion. Extensors (multifidus/erector spinae) have a higher correlation with maintenance of lumbar spine curvature than flexors (psoas).

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