Relationship between spine range of motion, pain, disease activity and muscle activation in axial spondyloarthritis patients

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Summary

Patients with axial spondyloarthritis had motion capture, electromyography and patient surveys collected three times over one year (6 monthly). Pain, disease activity and muscle activation do not appear to be individually related to changes in sagittal plane spine range of motion.

Introduction

Axial spondyloarthritis (axSpA) is an inflammatory disease that mainly affects the spine, resulting in joint stiffness, entheseal inflammation and bone growth that can lead to vertebral fusion [1]. While changes in patient mobility and posture over time are common, likely due to pain, disease activity or muscle stiffness (among other factors such as formation of syndesmophytes), no longitudinal study has examined if these measures are correlated to range of motion (RoM) over time [2]. It is hypothesised that patients who experience a reduction in range of motion during forward flexion would have increased pain, disease activity, and muscle activation, suggesting increased muscle loading.

Methods

In this ongoing multi-year study, 21 of 27 patients have completed three biomechanical assessments of spine flexion over a 1-year period (6-month intervals). Prior to each session, patients completed the Bath Ankylosing Spondylitis Disease Activity Index (BASDAI), the McGill pain questionnaire and a pain visual analogue scale (VAS) during spine flexion.

3D motion capture (Qualisys) marker clusters were placed on spine segments; head/neck, upper thoracic, lower thoracic, lumbar and pelvis (Figure 1), to examine an inverse kinematic segment-segment range of motion (RoM) in OpenSim [3]. RoM for each segment was calculated with respect to the inferior segment (e.g., lumbar RoM in respect to the pelvis). Maximal range of motion was calculated from full flexion to full extension, performed three times at the participants preferred speed and then averaged.

Figure 1: Marker and EMG sensor placement on segments.

Electromyography (EMG) of the left and right erector spinae longissimus muscle at the lumbar (L3) lower thoracic (T9/10) and upper thoracic (T3/4), as well as the erector spinae iliocostalis (L3) were measured (Figure 1). EMG data was bandpass filtered, rectified, and a moving window (250 ms) was used to extract mean EMG amplitude during concentric spine extension. EMG data were normalised by pulling on a force transducer to 50% of the participants maximal force in a seated position.

A linear mixed-effects model was used to examine the relationship between RoM and pain, disease activity and muscle activation, accounting for individual differences (independent intercept). P values were corrected using the Benjamini and Hochberg false discovery rate approach.

Results and Discussion

Relative to the baseline RoM, there was no significant improvement or decline in overall average patient RoM at the 6 month or 1 year mark, despite individual patient changes. There were no significant results within the linear mixed models used to examine the relationship between spine RoM and disease activity, pain, general pain, or muscle activation, once p value correction was performed ($P > 0.15$).

Thus, individual patient RoM changes over a one-year period do not appear to be well correlated with pain, disease activity or muscle activation on their own. There was a trend towards significance for standing EMG and spine flexion, suggesting those with reduced ROM may have increased standing muscle activation.

Conclusions

Alternative factors, or a combination of factors, may need to be explored to better understand what may be impacting patient RoM longitudinally. Additionally, expansion of this study to include all recruited participants, as well as extension to include additional time points are likely needed to detect potential significant relationships.

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References

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