DO MUSCLE ACTIVITY PATTERNS VARY ACCORDING TO THE SEVERITY OF CARPOMETACARPAL OSTEOARTHRITIS DISEASE?

Tamara Ordonez Diaz[1], Kalyn Kearney[1], Thomas W. Wright[2] and Jennifer A. Nichols [1,2]

[1] J. Crayton Pruitt Family Department of Biomedical Engineering, University of Florida, Gainesville, USA

[2] Department of Orthopaedic Surgery & Sports Medicine, University of Florida, Gainesville, USA.

Email: tordonezdiaz@ufl.edu

INTRODUCTION

The thumb is essential for manipulating objects and performing tasks such as pinching and grasping. Its strength and dexterity stem from the interaction of 9 thumb muscles, particularly at the carpometacarpal (CMC) joint with 3 degrees of freedom. Unfortunately, when CMC osteoarthritis (OA) occurs, it can cause debilitating pain, diminished strength, and limited range of motion. [1]. Despite the importance of the thumb muscles, limited research has been conducted on healthy or pathologic cohorts [2-4] due to the challenges posed by the size and location of these muscles and the constraints of surface electromyography. Currently, there is a gap in our understanding of muscle activity in patients with CMC OA, including how these muscles may (or may not) compensate for pain and its impact on hand biomechanics. The purpose of this study was to investigate differences in muscle activity across the clinical spectrum of CMC OA.

METHODS

Women with clinically diagnosed CMC OA [n = 16; age 65.6 ± 11 years] and healthy, pain-free thumbs [n = 15, age 62.1 ± 12 years] were recruited for this IRB-approved study. Participants completed self-reported questionnaires on pain and function, including the Australian/Canadian Osteoarthritis Hand Index (AUSCAN). Disease severity was classified from radiographs using the Eaton-Littler scale. Muscle activity was recorded during range of motion (ROM) tasks: flexion/extension, ad/abduction, opposition, and circumduction. Each task was performed three times. Muscle activity was recorded from 4 extrinsic and 4 intrinsic muscles using fine-wire electromyography (fEMG) sampled at 3,000 Hz. Data were filtered, rectified, amplitude normalized, and time normalized to percent task completion. Analyses evaluated differences in muscle activity across participants.

RESULTS AND DISCUSSION

No clear differences in muscle activation existed between the two recruited cohorts. However, imaging and self-reported pain data demonstrated an overlap between the recruited cohorts (Fig. 1A), suggesting little correlation between pain and disease severity. To determine if there were any muscle activation patterns that separated participants into meaningful subgroups, various clustering methods were employed, including dynamic time warping (DTW), functional principal component analysis (fPCA), and non-negative matrix factorization (NMF). Results were examined to decide if self-reported pain, disease severity, or other measured characteristics could describe the identified clusters.
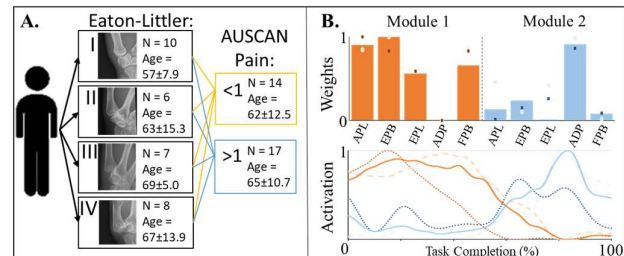

Figure 1A. Patient groups based on disease (Eaton-Littler classification) or pain severity (AUSCAN). **B.** Average muscle synergy using two modules (blue and orange) with examples of individuals with early- (light) versus end-stage (dark) disease.

When presented with 8 muscle activation patterns for each participant, both fPCA and DTW failed to identify meaningful subgroups. This outcome and the variability in muscle activation patterns across and within participants highlight the complexity and redundancy of thumb muscle actions. Clusters could be identified when examining each muscle individually, but valuable information about muscle coordination and co-activation was lost. Importantly, NMF analysis of muscle synergies, revealed that participants with diverse pain and disease severity levels could be grouped together. NMF results also showed agonist and antagonist muscles were activating simultaneously (Fig. 1B), emphasizing the significance of accurately measuring co-activation. To further understand the biomechanical changes in CMC OA, adding more features to clustering analysis, such as kinematic data and co-activation indices, is of immediate interest.

CONCLUSIONS

Our results elucidate the complex presentation of CMC OA and indicate a need for feature extraction techniques capable of analyzing multimodal datasets. Through these efforts, a more comprehensive understanding of CMC OA may be achieved.

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