VASTUS LATERALIS: VASTUS MEDIALIS ACTIVATION RATIO CORRELATES WITH VASTUS MEDIALIS ACTIVATION DELAY AND PATELLAR MALTRACKING IN PATELLOFEMORAL PAIN PATIENTS

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INTRODUCTION

Vasti muscle activation imbalance is considered a common cause for patellar maltracking in patellofemoral pain (PFP) subjects. Vasti activation imbalance can be evaluated by measuring vastus medialis (VM) activation delay compared to vastus lateralis (VL) activation [1], and normalized VL:VM activation ratio [2]. Measurement of VM activation delay during functional tasks such as walking or running requires synchronization of electromyography (EMG) data with joint kinematics and ground reaction forces (GRF) [1]. These measurements are impractical in most clinical settings. However, VL:VM activation ratio can be obtained by simply placing surface electrodes on a patient while performing the functional tasks during clinical evaluation, and may be a surrogate measure for VM activation delay. The first aim of this study was to investigate the relationship between VL:VM activation ratio and VM activation delay in PFP subjects and pain-free controls. Next, evidence relating VL:VM activation ratio to patellar maltracking is sparse. The second aim was to investigate the relationship between VL:VM activation ratio and patellar maltracking measured under upright, weightbearing conditions.

METHODS

We recruited 54 subjects; 39 (18M, 21F) had chronic PFP (> 3 months) but no prior surgery or knee injuries and 15 (7M, 8F) served as pain-free controls. All subjects were between 18-42 years of age, and there were no statistical differences in age, height or weight between gender-specific controls and PFP subjects. The PFP subjects were diagnosed by an experienced clinician.

We measured VL:VM activation ratio and VM activation delay in all subjects during walking at self-selected speeds in a gait laboratory (Fig. 1A).

All subjects performed a minimum of three trials. Acquired EMG activity, lower-limb kinematics, and GRF data were synchronized. Vasti activation onsets during the swing phase prior to heel strike were determined using a threshold function. A muscle was determined to be “on” if its EMG signal exceeded the greater of 3 standard deviations of its resting EMG value or 2% of the larger peak activation between the VM and VL muscles [1]. Vastus medialis activation delay was calculated as the difference between the VL and VM activation onset times; VL:VM activation ratio was determined from peak normalized activity over the entire gait cycle.

All subjects were classified into normal tracking and maltracking groups based on their patellar tilt and bisect offset measures obtained from weightbearing magnetic resonance imaging (MRI) (Fig. 1B) [1]. Images were obtained using a 0.5T Signa SP open-MRI scanner (GE Healthcare) fit with a backrest to stabilize a subject in an upright position (knee flexed ~5°). An oblique-axial plane image intersecting the center of the patella and the most posterior points of the femoral condyles was

Figure 1: Measurement of (A) VM activation delay relative to VL activation, and VL:VM activation ratio, & (B) patellar tracking measures, tilt & bisect offset (BO), during an upright, weightbearing squat.
identified from the MRI volume. Patellar tilt is a measure of the angle between the patella and the posterior femoral condyles. Bisect offset is a measure of the medial-lateral position of the patella relative to the femur. A gender-specific classification using a non-Gaussian Weibull distribution with 75% confidence intervals was used to determine thresholds for patellar maltracking. A subject was classified as a maltracker if his/her patellar tilt or bisect offset value was in the highest quartile of measured population data [1].

RESULTS

VL:VM activation ratio displayed a significant relationship with VM activation delay in PFP subjects classified as maltrackers (Fig. 2). There were no correlations between VL:VM activation ratio and VM activation delay in the pain-free controls, all PFP subjects grouped together, and PFP subjects classified as normal trackers.

VL:VM activation ratio displayed a significant relationship with patellar tilt in PFP subjects classified as maltrackers (Fig. 3). There were no correlations between VL:VM activation ratio and patellar tracking measures in the pain-free controls, all PFP subjects grouped together, and PFP subjects classified as normal trackers.

CONCLUSIONS

The results of this study suggest that VL:VM activation ratio may be a surrogate measure for VM activation delay in PFP subjects classified as maltrackers. This study demonstrates a significant relationship between VL:VM activation ratio and patellar tilt in maltracking PFP subjects, providing evidence in support of the role of vasti activation imbalance on patellar maltracking. In PFP subjects classified as normal trackers, there were no clear relationships between VL:VM activation ratio and VM activation delay, or patellar tracking measures. A possible explanation is that vasti activation imbalance is one of several factors affecting patellar tracking. It is plausible that in normal tracking subjects, increased joint conformity [3] and well-functioning PF ligaments minimize the effects of vasti activation imbalance on patellar tracking.

Although we have identified a cost-effective method for diagnosing vasti activation imbalance, the classification of subjects is based on open-MRI, which is expensive and available in few clinics. We are evaluating alternate imaging modalities that are both accurate and cost-effective to classify subjects as part of standard clinical assessment.

This study has the potential for broad clinical impact. Accurate diagnosis of vasti activation imbalance and patellar maltracking during clinical assessment will help identify patients who will most benefit from interventions treating these mechanisms of PFP, such as VM strengthening and EMG biofeedback training.

REFERENCES


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