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Improving Vastus Medialis Obliquus Function Reduces Pressure Applied to Lateral Patellofemoral Cartilage

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Abstract

The current study was performed to characterize how improving vastus medialis obliquus (VMO) function influences the pressure applied to patellofemoral cartilage. An additional focus was characterizing how lateral and medial cartilage lesions influence cartilage pressures. Ten knees were flexed to 40°, 60° and 80° in vitro, and forces were applied to represent the VMO and other muscles of the quadriceps group while a thin film sensor measured joint pressures. The knees were loaded with a normal VMO force, with the VMO force decreased by approximately 50%, and with the VMO unloaded. After tests were performed with the cartilage intact, all tests were repeated with a 12 mm diameter lesion created within the lateral cartilage, with the lateral lesion repaired with silicone, and with a medial lesion created. Based on a two-way repeated measures ANOVA and post-hoc tests, increasing the force applied by the VMO significantly ($p < 0.05$) decreased the maximum lateral pressure and significantly increased the maximum medial pressure at each flexion angle. A lateral cartilage lesion significantly increased the maximum lateral pressure, while a medial lesion did not significantly influence the maximum medial pressure. Improving VMO function can reduce the pressure applied to lateral cartilage when lateral lesions are present.

Keywords

patellofemoral joint; cartilage; pressure; vastus medialis obliquus; lesions

INTRODUCTION

Patellofemoral disorders are commonly attributed to an excessive lateral force applied to the patella. Due to the normal valgus orientation of the knee, the quadriceps muscles and the patella tendon act to shift and tilt the patella laterally. Passive resistance provided by the medial retinacular structures and forces due to contact with the trochlear groove resist the forces applied by the quadriceps and the patella tendon [1,2]. An excessive lateral force or inadequate resistance can lead to overloading of the lateral cartilage or lateral instability [3]. Overloading lateral cartilage can lead to areas of cartilage degradation, or lesions [3,4]. Instability episodes damage the medial retinacular restraints and can lead to lesions within the medial cartilage due to the medial facet of the patella contacting the bone on the lateral condyle of the femur [5,6]. Cartilage lesions increase the pressure applied to the surrounding cartilage [7] and can lead to pain due to overloading of the subchondral bone [3,5,8].

Physical therapy regimens prescribed for patellofemoral disorders commonly focus on improving the function of the vastus medialis obliquus (VMO). The force applied by the VMO has a medial component that can help resist the lateral force applied by the other quadriceps muscles. Some studies have indicated that patients with patellofemoral pain generate less force through the VMO than asymptomatic subjects [9,10]. The onset of VMO activity can also be delayed in patients with patellofemoral pain [11–15]. Although studies have indicated that training the VMO improves the strength [16] and activation timing [15] of the VMO, and provides better outcomes than a placebo treatment [15,17], training the VMO is not strongly supported [8,18]. In addition, biomechanical data showing how improving VMO function influences loads applied to cartilage is limited.

The current study was performed to characterize how improving VMO strength and activation timing influences the pressure applied to patellofemoral cartilage. An additional focus was characterizing how lateral and medial cartilage lesions influence the relationship between improved VMO function and cartilage pressures. The hypothesis of the study is that improving VMO function reduces the pressure applied to lateral cartilage of the patellofemoral joint, and that the pressure decrease is magnified when a lesion is present within the lateral cartilage.

METHODS

Ten cadaveric knees from ten separate donors were tested in vitro to address the hypothesis. The average age ($\pm$ standard deviation) was 74 ± 10 years, and two of the knees were from female donors. At full extension, the average angle between the orientation of the patella tendon and the shaft of the femur was $15^\circ \pm 3^\circ$. Each knee was stored at -20°C prior to dissection. The knees were dissected to remove all soft tissues except for the joint retinaculum, the patella tendon, and the quadriceps muscles. The lateral retinaculum was sectioned for insertion of the pressure sensors, and the patellofemoral cartilage was inspected to exclude knees with severe degenerative joint disease. The VMO and the vastus lateralis (VL) were separated from the combination of the vastus intermedius/vastus medialis longus/rectus femoris (VI/VML/RF) [19–21]. The femur and tibia were osteotomized approximately 18 cm from the joint line.

Each knee was secured to a testing frame consisting of two plates connected by a hinge, fixtures for positioning the knee, and fixtures for loading the quadriceps muscles (Fig. 1). Steel threaded rods were inserted into the diaphysis of the femur and tibia. The femur was secured horizontally by passing the femoral rod through a vertical fixture and locking the position with nuts. The posterior condyles were aligned in a horizontal plane before fixing the rotational position of the femur at the diaphysis with set screws through a second fixture. The tibia was secured to the second plate with the tibiofemoral joint line aligned with the hinge. The tibial rod was passed through another fixture and locked in place with nuts, although this fixture was attached to the plate through a slotted groove that allowed medial-lateral adjustment in order to set the varus-valgus alignment determined by the soft tissues. The varus-valgus alignment was set at 60° of flexion and left unchanged for the other flexion angles. A positioning device was secured to both plates that fixed the tibiofemoral flexion angle at 40° , 60° or 80° of flexion. Prior to each test, the flexion angle was checked with a goniometer. Even though the tibial rod was locked in place distally, rotation on the order of a few degrees in each direction could occur between the tibia and the rod in response to the applied quadriceps forces. The knees were kept moist with 0.9% saline solution while on the flexion frame. The VL, VMO and VI/VML/RF were secured to loading cables through clamps with serrated grips that were fixed to the muscles at their insertion sites on the quadriceps tendon. The loading cables passed over pulleys and were connected to weight holders. The VI/VML/RF cable was aligned along the axis of the

femur. The VMO cable was aligned at an angle of 47° medial to the axis of the femur in the coronal plane, and the VL cable was aligned at an angle of 19° lateral to the same axis [22]. The VMO and VL cables progressed posteriorly to the pulleys to represent the anatomical orientation in the sagittal plane.

The quadriceps muscles were loaded to represent a normal quadriceps force distribution, a weak VMO and a VMO with delayed activation. Loads were applied to produce a physiologically realistic extension moment of approximately 30 N-m at each flexion angle [9]. Previously published EMG-derived contributions of each muscle to the extension moment for patients with pain and asymptomatic subjects [9,23] were input into a computational model [24] to determine the force applied by each muscle for each case. The normal quadriceps force distribution was based on data for asymptomatic subjects, with 420 N, 116 N, and 60 N applied through the VI/VML/RF, the VL and the VMO, respectively. The force distribution for a weak VMO was based on data for symptomatic subjects, with 432 N, 127 N and 27 N applied through the VI/VML/RF, the VL and the VMO, respectively. The delayed activation case used the same forces as the weak case, except with no force applied by the VMO to simulate the time period before the VMO becomes active, with the realization that the extension moment would decrease.

Patellofemoral forces and pressures were measured using thin film sensors (I-Scan 5051, Tekscan, Boston, MA). The sensors are 0.1 mm thick with 44 rows and columns of force-sensing elements (sensels) every 1.27 mm. The sensors were coated with surgical jelly for calibration and the in vitro measurements to minimize shear loads [25,26]. Calibration was performed on a material testing machine (858 mini-Bionix II, MTS, Eden Prairie, MN), with each sensor sandwiched between two sheets of 3.2 mm thick soft (30 Shore A) neoprene rubber (McMaster-Carr, Elmhurst, IL) to simulate the compliance provided by patellofemoral cartilage [26]. The sensor and rubber sheets were placed on the testing machine between two steel plates to produce an area of nearly uniform pressure. Following preconditioning with three loads to a minimum of 6000 N applied over 15 seconds, a series of 8 loads ranging from 500 N to 6000 N were applied to an area of 1600 mm² on the sensor. The applied force was plotted against the raw output from the sensor, and the data was fit with a second order polynomial to determine the calibration curve for the sensor.

All three loading conditions were applied with the knees at 40°, 60° and 80° of flexion. The order of the flexion angles was randomized, as was the order of loading conditions at each flexion angle. The sensor was positioned to cover the entire articular surface of the patella and left in place for all three loading conditions. The position of the patella ridge was palpated on the sensor while applying a 111 N force through the VI/VML/RF, and a line representing the ridge was identified. The three loading conditions were applied by redistributing weights among the weight holders, without unloading the sensor.

Tests were conducted at all three flexion angles with all three loading conditions for four separate cartilage conditions. After testing the native knee, the patella was everted without removing the muscle clamps for creation of a lateral lesion. The center of the lesion was a point approximately midway between the patella ridge and the lateral edge of the articular surface and approximately midway between the proximal and distal edges of the articular surface. The lesion was given a diameter of 12 mm, and was created by removing all cartilage from the lesion area with a scalpel. For the third cartilage condition, the lateral lesion was filled with silicone (Aquarium Sealant, DAP, Baltimore, MD) and allowed to cure for 90 minutes. The silicone was applied to reduce pressure concentrations within the lateral cartilage. For the fourth cartilage condition, a medial lesion was created using the same technique as used for the lateral lesion.

Force and pressure distributions were characterized in terms of the lateral force percentage, the maximum lateral pressure and the maximum medial pressure. The sensor output was averaged over 100 data points collected over 10 seconds. For one knee, the output from two sensels contacting medial cartilage became saturated during testing, with pressure levels dramatically larger than the surrounding sensels. The data from these sensels was treated as artifacts and replaced with an average of the surrounding sensels. The lateral force percentage was quantified as the percentage of the total joint compression applied to cartilage lateral to the patella ridge. For the maximum medial and lateral pressure, the maximum force applied to the medial and lateral cartilage, respectively, was divided by the sensel area. Because the line representing the ridge was within a band estimated to be approximately 5 mm wide that could be characterized as the ridge, the maximum medial and lateral pressure measurements excluded an area 5 mm wide along the patella ridge.

The statistical analysis focused on changes at each flexion angle due to varying the loading condition and creating lesions. The medial and lateral lesions were analyzed separately. The intact cartilage case was treated as the control for the lateral lesion, with the analysis focusing on the lateral force percentage and the maximum lateral pressure. The case with the lateral lesion filled with silicone and intact medial cartilage was treated as the control for the medial lesion, with the analysis focusing on the lateral force percentage and the maximum medial pressure. At each flexion angle, a two level ANOVA with repeated measures on both the loading condition and the cartilage condition was used to determine if creating a lateral or medial lesion significantly influenced the output over the three loading conditions. The ANOVA also determined if the output varied significantly between loading conditions for each flexion angle. When the loading condition influenced the output, a repeated measures ANOVA with a post-hoc Student-Newman-Keuls test was used for comparisons between individual loading cases for each cartilage condition at each flexion angle.

RESULTS

Increasing the force applied by the VMO shifted compression medially. For intact cartilage, the average lateral force percentage was 70% or greater at all three flexion angles with no VMO force applied (Fig. 2). With a normal VMO force applied, the average lateral force percentage was less than 65% at all three flexion angles. Similar trends in the lateral force percentage were noted for all cartilage conditions. The lateral force percentage varied significantly between all three loading conditions for all states of the cartilage at all flexion angles ($p < 0.01$). The presence of a lateral or medial lesion did not significantly influence the lateral force percentage ($p > 0.2$).

Increasing the force applied by the VMO decreased the maximum lateral pressure. For intact cartilage, the average maximum lateral pressure was 12% to 18% larger for the VMO off than for a normal VMO at the three flexion angles. The maximum lateral pressure was significantly larger for the VMO off than for a normal VMO at each flexion angle (Fig. 3), with significant differences noted between all three loading conditions at 60° ($p < 0.05$). Creating a lateral lesion significantly increased the maximum lateral pressure at 60° and 80° ($p < 0.02$). With a lateral lesion, the average maximum lateral pressure was 17% to 27% larger for the VMO off than for a normal VMO at the three flexion angles, although the statistical variations between loading conditions were similar to those recorded for intact cartilage.

Increasing the force applied by the VMO increased the maximum medial pressure. With the medial cartilage intact and silicone within the lateral lesion, the average maximum medial pressure was 22% to 38% smaller for the VMO off than for a normal VMO at the three flexion angles (Fig. 4). The maximum medial pressure varied significantly ($p < 0.05$)

between all three loading conditions at all flexion angles, except for 80° for intact medial cartilage. Creating a medial lesion did not significantly increase the maximum medial pressure ($p > 0.4$).

Other noted trends were a shift in the area of contact from distal to proximal on the patella as the flexion angle increased, and an increase in the total compression applied to the cartilage as the VMO force was increased. At 40° of flexion, the contact area was typically distal to the medial and lateral lesions. At 80°, the contact area typically surrounded the proximal half of the lesions (Fig. 5). The average total compression increased by approximately 4% at the three flexion angles from the VMO off case to the weak VMO, and increased by another 2% from the weak VMO case to the strong VMO.

DISCUSSION

The current results indicate that improving VMO function reduces the load carried by the lateral cartilage of the patellofemoral joint. Increasing the force applied by the VMO consistently decreased the percentage of the joint compression applied to the lateral cartilage and consistently increased the maximum medial pressure. The slight increase in the compression as the VMO force increased contributed to the medial pressure increase being more consistent than the lateral pressure decrease, although the decrease in the maximum lateral pressure was still significant. The increase in compression was planned when representing elimination of delayed VMO activation. When the VMO force was increased from the weak to strong cases the other forces were decreased to maintain a consistent extension moment and minimize differences in compression. Nevertheless, compression increased by approximately 2%. Previous experimental studies have shown that decreasing the lateral force acting on the patella through medialization or anteromedialization of the patella tendon attachment on the tibia reduces patellofemoral pressures [25,27], although redistributing forces among the quadriceps muscles produces a less dramatic change in patellofemoral loading. A previous in vitro study showed that going from no force applied by the VM to a normal VM force decreased the maximum pressure [28]. The previous study did not show any trend for a pressure decrease when the VM force was increased from 50% of normal to normal, as was shown for the current study with the VMO. Unlike the current study, the previous study did not analyze medial and lateral pressures separately or decrease the other quadriceps forces when the VM force was increased. For the current study, using physiologically realistic muscle forces and minimizing experimental errors may have also contributed to more consistent trends than reported for the previous study.

The current study also shows that a lateral cartilage lesion increases the pressure applied to surrounding cartilage. Lateral lesions significantly increased the maximum lateral pressure at 60° and 80°, but not at 40° due to the distal area of contact. Lesions at the center of the medial facet did not significantly increase the maximum medial pressure, primarily because the majority of the compression was applied to the lateral cartilage. A previous in vitro study performed with knees with existing lesions also showed that lesions create pressure concentrations in the surrounding cartilage [7], although the pressure increase due to creating a lesion was not quantified. The current study also showed that improving VMO function tends to cause a greater decrease in the maximum lateral pressure when a lesion has elevated the lateral pressure, although the tendency for a greater decrease in the maximum lateral pressure did not lead to additional significant differences between loading cases with a lateral lesion present.

Muscle forces were applied to represent physiologically realistic loading conditions. The EMG data used to characterize the quadriceps forces provided an assessment of normal VMO function and of the VMO in a weakened state for patients with patellofemoral pain

and lateral malalignment [9]. For the normal quadriceps force distribution, the VMO force was 10% of the total quadriceps force, matching the percentage predicted based on the physiological cross-sectional areas (PCSA's) of the muscles [22]. The contribution of the VL to the normal quadriceps distribution was half of what would be used based on PCSA's. For the experimental set up used for this study, the difference in lateral force applied, as compared to a PCSA based force distribution, was approximately 5 N because grouping the VML with the RF and VI along the orientation of the femur reduced the medial force balancing the lateral force applied by the VL. Simulation of delayed activation of the VMO focused on representing the time period before the VMO becomes active, with the loading conditions for patients with pain applied with the VMO unloaded. The total quadriceps force was more than twice that used previously to characterize the influence of the VM on patellofemoral pressures [28]. The larger quadriceps force produces more realistic contact forces between the patella and trochlear groove.

Experimental error was minimized for this study by loading the knees statically at individual flexion angles. For each combination of flexion angle and cartilage condition, the position of the patella ridge was determined a single time and three loading conditions were applied without unloading the knee or reorienting the sensor. Pressure seemed to be the most effective means of securing the sensor in the joint while minimizing artifacts in the output. Artifacts were noted for individual sensels contacting medial cartilage for one sensor, but eliminating the data from this knee would not have lessened the significant differences noted for the maximum medial pressure. Variations in the position of the patella relative to the sensor as the loading condition was varied could have influenced the lateral force percentage. As the loads were varied, changes in the location of the contact area on the sensor were minimal. Previous studies have shown that eliminating the force applied by the VMO tends to shift the patella laterally by 2 to 4 mm [29,30]. The total quadriceps force used for these studies was approximately equal to 25% of the quadriceps force used for the current study, thereby reducing the compression of the patella within the trochlear groove. For the normal loading condition, the percentage contribution of the VMO to the total quadriceps force was more than twice that used for the current study, thereby increasing the influence of the VMO on patella motion. Based on the maximum pressure measurements and the distribution of pressure on the sensors (Fig. 5), pressure shifting from the lateral to medial cartilage as the VMO force was increased is believed to be the primary cause of the trends noted for the lateral force percentage. The test set-up allowed assessment of cartilage lesions with minimal variations in tibiofemoral alignment or the orientations of quadriceps forces between cartilage conditions.

Limitations related to the ability to represent in vivo conditions should be noted. Anatomical conditions that can lead to patellofemoral malalignment, which are common for patients with patellofemoral disorders [3,31], were not modeled. In order to control the size and location of lesions, only knees with intact cartilage throughout the contact area were included. The lesions created included an abrupt change from intact cartilage to no cartilage, which is not typical in vivo. The current study did use a conservative lesion size, with an area 30%–40% smaller than the average size of lesions encountered in vivo [7,32]. A lateral release was performed to insert the sensor into the patello femoral joint. An in vitro study indicated that from 40° to 80° of flexion a lateral release has no significant influence on patellar medial translation, but increases medial tilt by approximately 1°, with the change having no significant influence on the maximum pressure [33]. The experimental set-up was designed to represent a knee extension exercise with resistance applied at the distal tibia. At each flexion angle, the position of the tibial rod was fixed distally, although minimal rotation of the tibia with respect to the rod was allowed to represent tibial rotation during the exercise. For this type of exercise, the redistribution of forces among the quadriceps muscles was assumed to have minimal influence on tibiofemoral kinematics. The conclusions drawn

are based on the assumption that delayed VMO activation and VMO weakness can be improved by training the VMO, which has been questioned [8,34], although some evidence suggests that improving VMO function is possible [12,15,16].

The current study indicates that improving VMO function in patients with patellofemoral disorders and impaired VMO function reduces the pressure applied to lateral cartilage within the patellofemoral joint. The pressure reduction can be magnified when a lateral cartilage lesion is present. Improving VMO function increases the pressure applied to medial cartilage, although the presence of a relatively small medial lesion does not increase the medial pressure. For patients with a lateral lesion, reducing pressure applied to areas of overloaded lateral cartilage could relieve symptoms.

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Figure 1.

A diagram of the experimental set-up. Letters are used to represent the knee (K), the sensor and handle (S), and the tissue clamps (C). The cables applying the forces representing the VMO, VL, and VI/VML/RF (VI) are also labeled.

Figure 2.

The average ($\pm$ standard deviation) lateral force percentage at all three flexion angles for all three loading conditions, for both intact cartilage and cartilage with a lateral lesion. Statistically significant differences between the loading conditions for each cartilage condition at each flexion angle are indicated ($a > b > c$ at $p < 0.01$). Similar values of the lateral force percentage and similar trends with the applied loading condition were recorded for the cartilage with silicone and intact medial cartilage and for the cartilage with a medial lesion.

Figure 3.

The average ($\pm$ standard deviation) maximum lateral pressure at all three flexion angles for all three loading conditions, for both intact cartilage and cartilage with a lateral lesion. Statistically significant differences between the loading conditions for each cartilage condition at each flexion angle are indicated ($a > b > c$ at $p < 0.05$, no letter means no significant differences for a data point, repeated b's mean no significant difference between two data points). Statistically significant differences between intact cartilage and cartilage with a lateral lesion over the three loading conditions at each flexion angle are also indicated ($A > B$ at $p < 0.02$).

Figure 4.

The average ($\pm$ standard deviation) maximum medial pressure at all three flexion angles for all three loading conditions, for both intact medial cartilage and cartilage with a medial lesion. Statistically significant differences between the loading conditions for each cartilage condition at each flexion angle are indicated (a > b > c at $p < 0.05$, repeated b's mean no significant difference between two data points).

Figure 5.

The pressure sensor output at 40° and 80° of flexion for two knees with a lateral lesion for the VMO off and a normal VMO force. The position of the patella ridge and the lesion with respect to each pressure plot is indicated. The highest pressure is recorded in the darkest areas. The pressure is concentrated distally on the patella at 40° and proximally at 80°. Increasing the force applied by the VMO decreased the lateral pressure and increased the medial pressure.