COMPARISON OF ELECTRICAL ACTIVITY OF PATELLA STABILIZER MUSCLES BETWEEN ATHLETES WITH AND WITHOUT PATELLOFEMORAL PAIN SYNDROME

Nahid Khoshraftar Yazdi
Majid Mehdikhani
Assistant Professors, Faculty of Physical education and sport science- Ferdowsi University of Mashhad, Iran
Stephan Starischka, Prof. Dr.
Institute of Sport, Dortmund University, Germany
Elke Zimmermann, Prof. Dr. med
Department of sports medicine, Bielefeld University, Germany

Abstract:
The aim of this study was to compare the electrical activity of the Tensor fascia lata (TFL) and Vastus lateralis (VL) muscles as lateral stabilizers and the Vastus medialis oblique (VMO) muscle as medial stabilizer of the patella in athletes with and without patellofemoral pain syndrome (PFPS). Eleven healthy athletes (4 women, 7 men) and 9 athletes with PFPS (3 women, 6 men) ranging in age from 20 to 30 years performed the submaximal isometric knee extension contraction initiated from 45°of flexion and an extended position to 30° and 15°angles. The activation amplitude of the TFL, VL and VMO muscles were quantified from the recorded electromyographic (EMG) signals. The results showed that Electrical activity of the VMO muscle at 30° and 45° angles for the patients was significantly greater than those of the control subjects. No significant differences were demonstrated in VMO: VL and VMO: TFL across all knee flexion angles between groups. However, the mean value of the ratio of VMO: (TFL + VL) at an angle of 30° for the subjects with PFPS was significantly greater than that of the control group. It may be assumed that the higher electrical activity of the VMO at 30° angle in PFPS patients is an effort to prevent more lateral patella tracking. Since the TFL and VL muscles produce lateral force on the patella, both of them should be studied for consideration of lateral stabilizers of the patella in the patients with PFPS.

Key Words: Patellofemoral pain syndrome, Vastus lateralis, Vastus medialis oblique, Tensor fascia lata, Patella, EMG

Introduction

Patellofemoral pain syndrome (PFPS) is the most common knee complaint found in adolescents and young adults. Researchers showed an incidence as high as one in four, and even higher, among athletes (Cowan, Bennell & Hodges, 2002; Witvrouw, Lysens, Bellemans, Cambier & Vanderstraeten, 2001). Despite this high incidence, the exact cause of these disorders remains enigmatic (Crossley, Bennel, Green, Cowan & McConnell, 2002) and PFPS remains one of the most vexatious clinical challenges in rehabilitation medicine (Wilk, Davise, Mangine & Malone, 1998).

The major complaint of patients with PFPS is retropatellar pain during activities such as running, squatting, going up and down stairs, prolonged sitting, cycling, and jumping (Witvrouw et l., 2001). A combination of factors, such as abnormal lower limb biomechanics and abnormal lateral tracking of the patella may result in increased cartilage and subchondral bone stress, subsequent PFPS and subtle patellar malalignment or more overt patellar maltracking (Fredericson & Yoon, 2006; Miller, Sedory & croce, 1997; Souza & Gross; 1991).

Several researchers showed that PFPS is associated with specific quadriceps muscle atrophy, especially in the vastus medialis oblique muscle (VMO) (Boucher, King, Lefebvre & Pepin, 1992; Lefebvre et al., 2006). Insall (1982) suggested that mechanism of abnormal lateral tracking of the patella is an imbalance in the activity of the VMO muscle relative to the vastus lateralis muscle (VL). Owings and Grabiner (2002) stated that the rationale for an important biomechanical role of the VMO

in patellar tracking is substantiated by its physiologic and biomechanical properties. They believed that due to smaller maximum contraction force and slower maximum contraction velocity of the VMO, it would be expected that VL dominates the movement of the patella unless the activation patterns of these muscles account for this difference (Owings & Grabiner, 2002).

It is assumed that the medial tracking role of the VMO counteracts the laterally directed force of the VL on the patella (Grabiner, Koh & Miller, 1991). Lateral tracking of the patella may be due to inadequate medial control from the VMO in persons with PFPS (Gilleard, McConnel & Parsons, 1998).

The other soft tissue which influences the patella is illiotibial band (ITB). The ITB, the tendon of Tensor fascia lata (TFL), is an important dynamic lateral stabilizer of the patella (Chimera, Swanik, Buz Swanik & Straub, 2004; Mosher & Jackson, 2008; Recondo et al., 2000). With regard to the role of the VL and TFL on the lateral side of the patellofemoral joint and the VMO on the medial side, it can be assumed that the TFL as well as the VL contributes in making lateral force on the patella. Therefore, in order to prevent patella tracking, the VMO should counteract this force which is produced by the TFL and VL. If the VMO is not as strong as the TFL and VL, it has to recruit more motor units to dominate the force of the TFL and VL.

Since the EMG intensity provides a reliable estimate of the volume of recruited muscle, but not necessarily of the developed force (Roberts & Gabaldon, 2008), we expect that the VMO muscle has more activity in patients than that of a control group.

Although the electromyographic (EMG) activity of the VMO and the VL was evaluated in patients with PFPS by several investigators (Boucher et al., 1992; Gilleard et al., 1998; Owings & Grabiner, 2002), none of them considered the activity of the TFL against the VMO.

The purpose of this study was to examine the electrical activity of lateral and medial stabilizer muscles of patella including the VMO, the VL and the TFL and to compare their activities in subjects with and without PFPS.

Methods

Subjects volunteered to participate in this study and were placed in an experimental group and a control group based on the presence of PFPS symptoms with no evidence of any other specific pathologic condition. All group members were athletes who were active in sports such as running, football, basketball, and handball, for more than 10 years. The control group was composed of 4 women and 7 men with a mean age of 25.1 ± 3.2 years. They were healthy and athletic, and reported no history of knee injury.

The experimental group consisted of 3 women and 6 men with a mean age of 26.3 ± 2.6 years, who had history of PFPS with duration of symptoms was more than 6 months and intensity sufficient to limit function or cause the individual to seek intervention. These symptoms consisted of retropatellar pain during physical activities such as jumping, running, squatting, and going up or down stairs. Clinical criteria include pain on direct compression of the patella against the femoral condyles with the knee in full extension, tenderness of the posterior surface of the patella on palpation, pain on resisted knee extension and pain with isometric quadriceps muscle contraction against suprapatellar resistance with the knee in 15° of flexion. These clinical signs are summarized in Table 1. Participants were excluded if they had signs or symptoms of meniscal, bursa, ligament laxity or tenderness, tenderness over the patellar tendon, illiotibial band, or pes anserinus tendons, patellar apprehension sign, patellar dislocation and previous knee surgery. The subjects did not have pain at rest, and did not have pain during a submaximal isometric contraction of knee flexion. The study received ethics committee approval from the Ruhr-Universität Bochum Ethics commission of Germany.

Table 1. Clinical signs of the patients with PFPS

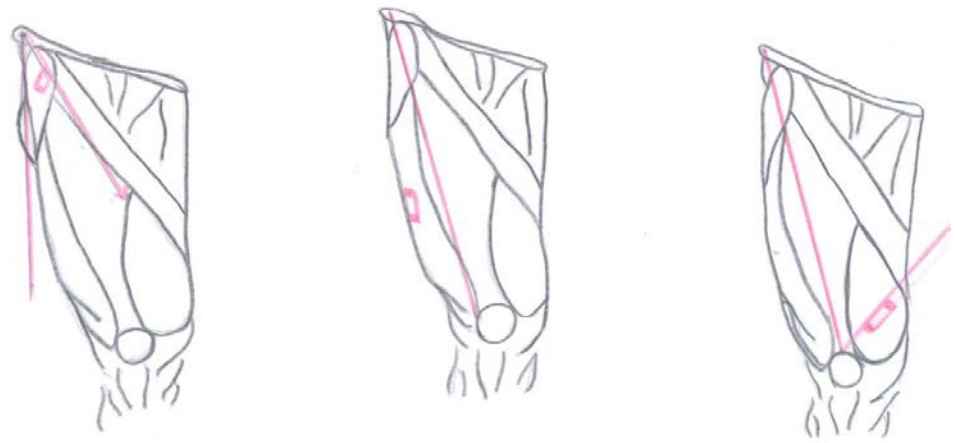
Pain on direct compression of the patella
Tenderness of the posterior surface of the patella on palpation
Pain on resisted knee extension
Pain with isometric quadriceps muscle contraction

Before beginning the study, every subject signed an informed consent document.

The motor point of muscles was estimated by the following method (Rainoldi, Melchiorri & Caruso, 2004; Hermans HJ, Freiks B, 1997), (Figure 1):

Vastus Lateralis: Two anatomical landmarks (the anterior superior iliac spine (ASIS) and the superior lateral side of the patella (SLSP)) were determined. Electrodes were placed at 2/3 on the line from the ASIS to the SLSP in the direction of the muscle fibers.

Vastus medialis obliquus: Two anatomical landmarks (ASIS and the superior medial side of the patella (SMSP)) were determined. A quadriceps line was drawn from the ASIS to the SMSP. Electrodes were placed at 80% of the quadriceps line (starting from ASIS) with a medial inclination of 50°.



A B C

Figure 1. Schematic drawing of proper electrode positioning over the Tensor fascia lata (A), Vastus lateralis (B), and Vastus medialis oblique(C) muscles

Tensor fascia lata: Two anatomical landmarks (ASIS and greater trochanter (GT)) were determined. A line was drawn from the ASIS to the GT. Electrodes were placed at 50% of this line with an inclination of 30°.

Active bipolar Ag/AgCl surface electrodes (pre- amplification gain= 10, recording diameter, 1 mm; center-to-center distance, 20 mm) were placed on the motor point of the VL, the VMO and the TFL muscles of the tested leg. The subject's skin was prepared by shaving, then used abrasive paste for gentle local abrasion and finally cleaned with isopropyl alcohol. The resistance between the electrode pair was also measured by an EMG electrode impedance tester. The long axis of the electrodes was positioned over each muscle in the assumed direction of the underlying muscle fibers.

The protocol prescribed submaximal isometric contractions with 60% of maximal voluntary contraction (MVC) for the knee extension. A device was designed for displaying the exact degree of knee extension. We named it angle meter. Each subject was seated on a chair. The hip-trunk angle was approximately 100°.The angle meter was positioned so that knee flexion angle was 90° and the estimated center of knee joint rotation was on a level with the angle meter's axis of rotation. The subjects put their foot on the pedal of the angle meter. By moving the pedal upwards, the knee was extended and when the angle meter displayed 45°, the subjects had to maintain the position for 6 seconds (Figure 2). Afterward, they contracted isometrically again at 30° and 15° maintaining the contraction for 6 seconds at each stage. The test was repeated 3 times. A rest of 1 min was given between tests. The mean of the three measurements was used for analysis.

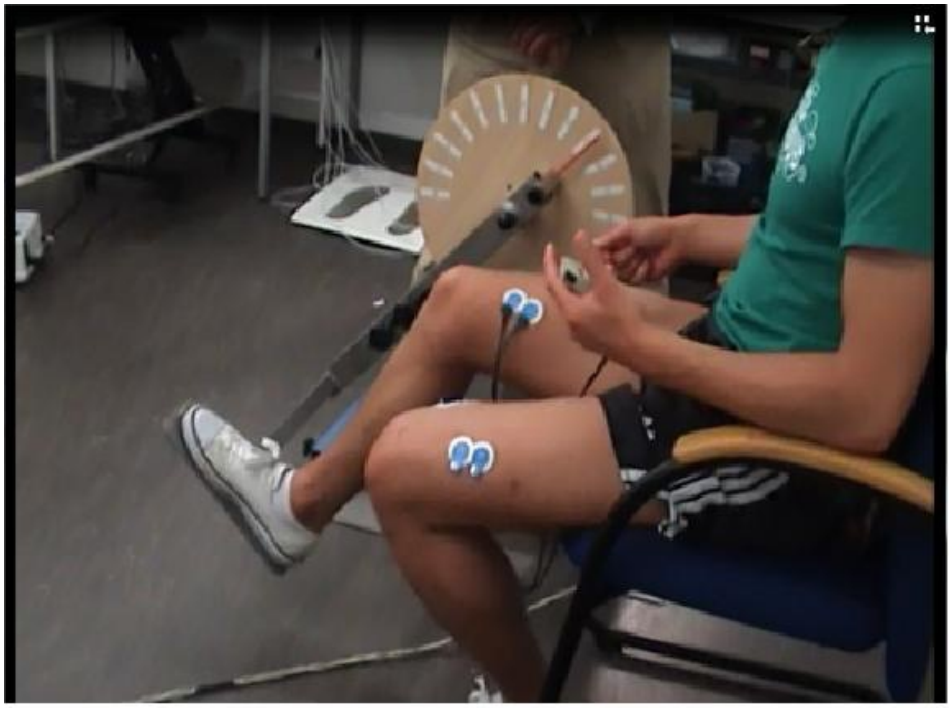


Figure 2. Measuring electrical activity of the muscles in different angles of knee extension

EMG raw signals were recorded by using a Noraxon Inc., Scottsdale, AZ, USA EMG system. Signals were amplified differentially (total gain= 1000; CMRR> 130db). A band-pass Butterworth filter with cut-off frequencies ranging from 15 to 500 HZ was applied. The transmitted signals were sampled at 1 kHz, input to an analog-to digital circuit (Data translation Inc., Marlboro, MA.USA, 16-bit resolution) and were stored. All signals processing was supported by the Noraxon MyoResearch XP software.

The activation signals of the VMO, VL and TFL were full-wave rectified through root mean square (RMS). Amplitude was analyzed by calculating IEMG in a window of 6 seconds and normalized by the mean value calculated within this time-window. Signals were also time normalized from 0-100% over these six seconds.

The t-test for independent variables was used to compare the activation amplitude of the VL, the VMO, the TFL muscles and their ratios between patients and control group. Statistical analysis was performed with SPSS Version 17.0, and a value of 0.05 was accepted as reflecting significance.

Results

Significant differences were identified in the activation amplitude of the VMO at 30° and 45°angles between the subjects with and without PFPS (P= 0.05, P= 0.03, respectively), (Table 2). According to the t-test analysis, VMO electrical activity in the subjects with PFPS was significantly greater than that of the subjects without PFPS. However, no significant differences were demonstrated in the activation amplitude of the VMO at an angle of 15° between the two groups (P= 0.76). In addition, there were no significant differences in the activation amplitude of the VL at 15° (P= 0.61), 30° (P= 0.55) and 45° (P= 0.66) angles between the subjects with and without PFPS. Furthermore, no significant differences were found in the activation amplitude of the TFL at 15° (P= 0.25), 30° (P= 0.67) and 45° (P= 0.89) angles between the two groups.

Table 2. The means and standard deviations of normalized VMO, VL and TFL IEMG value (%) for knee flexion angles in the subjects with and without PFPS

Group	Muscle	Knee Flexion Angle 45°	30°	15°
Control	VMO	22.3 ± 11.3	26.4 ± 11.5	72.3 ± 7.3
	VL	37.3± 18.1	40.7± 17.1	80.4± 8.9
	TFL	32.5± 16.2	45.2± 19.8	66.1± 11.3
Patellofemoral pain	VMO	34.6 ± 12.6	38.9 ± 15.3	73.6 ± 10.5
	VL	41.3± 21.4	46.2± 23.2	78.0± 11.0
	TFL	33.5± 16.2	41.7± 15.4	71.5± 8.0

There were no significant differences in the VMO: VL ratio across all knee flexion angles between the subjects with PFPS and the subjects without PFPS (P= 0.35, P= 0.09, P= 0.07, respectively from an angle of 15° to 45°). Similarly, no significant differences were identified in the VMO: TFL ratio at all knee flexion angles between the patients and control group (P= 0.39, P= 0.06, P= 0.12, respectively from an angle of 15° to 45°).

We found a significant difference in the VMO: (TFL+ VL) ratio at 30° of knee flexion between the two groups (P= 0.02). This ratio at 30° of knee flexion for subjects with PFPS (0.46) was significantly greater than for the subjects without PFPS (0.31), (Table 3).There was no difference in this ratio at 15° (P= 0.90) and 45° (P= 0.06) of knee flexion between the two groups.

Table 3. The VMO: VL, VMO: TFL and VMO: (TFL+Vl) ratios for knee flexion angles in the subjects with and without PFPS

Group	Ratios	knee flexion angle 45°	30°	15°
Control	VMO: VL	0.67± 0.29	0.68 ±0.22	0.90± 0.08
	VMO: TFL	0.79± 0.41	0.66 ± 0.33	1.14± 0.31
	VMO: (TFL+VL)	0.34 ± 0.13	0.31 ± 0.11	0.50 ± 0.07
Patellofemoral pain	VMO: VL	1.01± 0.51	0.98± 0.46	0.95± 0.12
	VMO: TFL	1.48± 1.31	0.99 ± 0.43	1.04± 0.17
	VMO: (TFL+VL)	0.48 ± 0.19	0.46 ± 0.14	0.49 ± 0.07

Discussion

In the current study, the electrical activity of muscles (VMO, VL and TFL) was investigated at 3 different angles of knee flexion (45°, 30°, 15°) during submaximal isometric contraction in the subjects with and without PFPS. The normalized EMG data of VMO and VL indicated that in subjects with PFPS, the activation amplitude of the VMO muscle during the contraction at 30° of knee flexion was significantly greater compared with that of the subjects without PFPS (P= 0.05). There was no significant difference in the activation amplitude of the VL muscle between the two groups at the same angle. However, Owings & Grabiner (2002) reported the activation amplitude of both muscles (VMO and VL) of the subjects with PFPS were significantly higher than those of the control subjects during eccentric contraction.

Following the data analysis, we found no significant differences in VMO: VL ratio across all knee flexion angles between the two groups. This finding has also been reported by previous studies (Boucher et al., 1992; Souza & Gross, 1991). Souza and Gross suggested that patients with PFPS may not differ from healthy individuals with regard to VMO: VL activation patterns. Since the TFL as well as VL, is a lateral stabilizer of the patella (Hughston, Andrews, cross & Moschi, 1976), it may be suggested to consider the TFL muscle as well as VL, when studying the ratio of lateral and medial dynamic stabilizer of the patella.

With regard to the role of the TFL/ ITB complex, which is an important lateral dynamic stabilizer of the patellofemoral joint, in particular from 0° to 30° of knee flexion (Hughston et al., 1976; Kanamiya et al., 2002), several studies suggested that TFL/ITB complex tightness may contribute to the development of PFPS (Puniello, 1993; Reider , Marshall & Warren, 1993).

On the other hand, some researchers indicated there is a correlation between hip internal rotation and PFPS (Berger, Crossett, Jacobs & Rubash, 1998; Ireland, Davis, Ballantyne & Willson, 2003). Berger et al (1998) stated that the direct correlation of combined (femoral and tibial) internal component rotation to the severity of the patellofemoral complication suggests that internal component rotation may be the predominant cause of patellofemoral complications in patients with normal axial alignment. Ireland et al. (2003) indicated that female runners, who have demonstrated significant knee valgus and hip internal rotation movements during running, are especially prone to PFPS. According to Press and Young (1998), internal rotation may be caused by a tight TFL and a weak gluteus medius . McConnel (2002) showed that decreased flexibility of TFL muscle is a contributing factor in the etiology of PFPS. Since TFL is an internal rotator of the hip and a lateral stabilizer of patella (Fredericson et al., 2000), we considered the electrical activity of this muscle. To the authors' knowledge there is no study that considers the electrical activity of the TFL muscle in the patients with PFPS.

As the TFL and the VL play the role of the dynamic lateral stabilizers of the patella, the VMO represent a dynamic medial stabilizer of the patella. This function of VMO counteracts the force of lateral stabilizers on the patella. Due to balances in lateral and medial stabilizing forces, patellar tracking is prevented. Therefore, an insufficiency of these muscles can have an effect on the patella and develop patellar malalignment and consequently PFPS.

In the present study, we found no significant differences in VMO: TFL ratio between two groups across all knee flexion angles, however, significant differences were demonstrated in VMO: (TFL+VL) ratio at 30° of knee flexion for the two groups. This ratio was greater for the subjects with PFPS (0.46) than for the subjects without PFPS (0.31). This result showed that there was no significant differences in (TFL+ VL) at an angle of 30° of knee flexion between two groups, but the electrical activity of the VMO was significantly higher in patients than that of the healthy subjects. Powers (2000) believed that increase motor unit activity of the vastus medialis appeared to be in response to meeting the increased demand of providing patellar stability. With regard to the significant differences in VMO: (TFL+ VL) ratio between patients and control group, it may be assumed that VMO as a medial stabilizer of the patella recruits more volume of active motor units thus preventing more patella tracking. Roberts and Gabaldon (2008) stated that it is generally assumed the EMG intensity provides a reliable estimate of the volume of recruited muscle, but not necessarily of the developed force. According to Roberts and Gabaldon (2008), high activation level of the VMO of the subjects with PFPS is not necessarily associated with developed force in this muscle. Powers (2000) suggested that increased motor unity activity of the Vastus medialis muscle appears to be associated with abnormal patellar kinematics in women, but it is not necessarily a cause of abnormal

patellar kinematics. It may be presumed that VMO with more activation and more recruited motor unit tries to counteract the lateral forces of VL and TFL on the patella and prevent more patella tracking.

This finding may provide evidence that PFPS is associated with a disruption in the control of the VMO during the isometric contraction at 30° of knee flexion. Furthermore, according to the results of the present study, it may be suggested to study the lateral stabilizers of the patella in the patients with PFPS, TFL as well as VL to be considered.

In the light of the results at 30° of knee flexion in the current study and another study (Orchard, Fricker, Abud & Mason, 1996) about the Iliotibial friction syndrome in runners, which shows that friction occurs at 30° of knee flexion, it may be assumed that this angle plays an important role in injuries occurring in runners.

We recognize that two limitations were present in our experimental design. The subjects' symptoms of patellofemoral pain varied in duration of pain. Nevertheless, as a group, these subjects were associated with a pattern of activation amplitude that was different from that of control subjects at angles of 30° and 45°. It may be assumed that PFPS, regardless of pain duration, effects on activation amplitude of VMO at above mentioned angles.

The other limitation that we found in our study was daily activities of the subjects which it was not possible to control them. Although all subjects were active in sports such as running, football, basketball and handball, their daily activities may be different. However, this limitation was similar for both groups.

Conclusion:

In the present study, the electrical activity of the VMO at 30° angle of knee flexion was significantly higher in the patients than that of the control group. It may be assumed that high electrical activity of the VMO in the patients is an effort for counteracting lateral force, which is produced by lateral stabilizers, and subsequently preventing patella tracking. With regard to no significant differences in VMO: VL and VMO: TFL ratios across all knee flexion angles between the two groups, and significant differences in VMO: (TFL+ VL) ratio at 30° angle of knee flexion, it is suggested that in consideration of lateral stabilizers of the patella, the TFL and VL should be assessed together.

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