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# Vastus Medialis Obliquus Atrophy

## Does It Exist in Patellofemoral Pain Syndrome?

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**Background:** Quadriceps atrophy and in particular atrophy of the vastus medialis obliquus (VMO) muscle have been frequently related with patellofemoral pain syndrome (PFPS), despite very little objective evidence.

**Hypothesis:** Patients with PFPS exhibit atrophy of the VMO in comparison with healthy controls.

**Study Design:** Case-control study; Level of evidence, 3.

**Methods:** Forty-six patients with PFPS and 30 healthy control persons with similar age, gender, body mass index, and activity index distributions underwent magnetic resonance imaging (MRI) of the quadriceps. The muscle size was determined by calculating the cross-sectional area of the total quadriceps and its components.

**Results:** The cross-sectional area (CSA) of the VMO was significantly smaller in the PFPS group than in the control group ( $16.67 \pm 4.97 \text{ cm}^2$  vs  $18.36 \pm 5.25 \text{ cm}^2$ ) ( $P = .040$ ). A tendency was noted for a smaller total quadriceps CSA for the PFPS patients at midhigh level ( $66.99 \pm 15.06 \text{ cm}^2$  vs  $70.83 \pm 15.30 \text{ cm}^2$ ) ( $P = .074$ ).

**Conclusion:** This is the first study to examine VMO size in PFPS patients by MRI. Patients with patellofemoral problems exhibited atrophy of the VMO. Although it is not clear whether this atrophy is a result or a cause of PFPS, the results of this study do show that atrophy of the VMO is a contributing factor in PFPS. Longitudinal, prospective studies are needed to establish the cause-effect relation of VMO atrophy and PFPS.

**Keywords:** patellofemoral pain syndrome; vastus medialis obliquus; cross-sectional area; magnetic resonance imaging

Patellofemoral pain syndrome (PFPS) is a very common knee disorder in the general public and in athletes.<sup>8,9,25,28</sup> The exact cause is still unknown but has been proposed to be multifactorial. One of the main suggested contributing factors of patellofemoral pathological lesions is patellar malalignment or abnormal patellar tracking.<sup>¶</sup> Several factors are propounded to play a role in patellar maltracking such as abnormalities in the retinacular restraints<sup>5,14</sup> or

abnormalities of patella shape and position.<sup>19</sup> One other proposed main mechanism for abnormal patellar tracking in PFPS is an imbalance in the activity of the vastus medialis obliquus (VMO) muscle relative to the vastus lateralis (VL) muscle.<sup>7,19,29,31,32</sup> Under normal conditions, the VMO is able to counterbalance the lateral pull of the larger VL to ensure patellar stability. Therefore, the VMO is seen as an important medial stabilizer of the patellofemoral joint.<sup>18,24</sup> A disruption of this mechanical balance between the VMO and the VL has frequently been attributed to an insufficiency of the VMO secondary to atrophy, hypoplasia, inhibition, or impaired motor control.<sup>3,12</sup> Accordingly, it has often been suggested that PFPS is associated with decreased VMO muscle mass. However, it is striking to observe the scarcity of objective data and studies concerning VMO atrophy along with patellofemoral disorders. Only one study actually investigated VMO atrophy in PFPS patients in comparison with people without knee problems. Jan et al<sup>20</sup> found in their study using sonography a significant smaller VMO volume in people with PFPS compared with a healthy control group ( $1.8 \pm 1.5 \text{ cm}^3$  vs  $3.0 \pm 2.2 \text{ cm}^3$ ).

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TABLE 1  
Demographic Data for Study Participants<sup>a</sup>

	PFPS (n = 46)	Control (n = 30)	P
Age, y	25.0 ± 7.4	21.6 ± 4.5	.052
Gender, male:female	21:25	13:17	.842
Weight, kg	68.9 ± 13.6	66.7 ± 13.0	.480
Height, cm	173.4 ± 9.9	173.1 ± 10.0	.924
BMI, kg/m <sup>2</sup>	22.8 ± 3.4	22.1 ± 2.9	.345
Activity index	8.4 ± 2.0	9.3 ± 1.5	.181
Dominant side, right:left	45:1	28:2	.326
Test side, dominant:nondominant	29:17	19:11	.980
Affected side, bilateral:unilateral	26:20	—	—
Duration of symptoms, mo	17.37 ± 30.2	—	—

<sup>a</sup>PFPS, patellofemoral pain syndrome; BMI, body mass index.

However, there are yet a few studies that did examine possible atrophy of the entire quadriceps in PFPS patients without any focus on the VMO in particular. Callaghan and Oldham<sup>6</sup> found a 3.38% difference in quadriceps cross-sectional area (CSA) between the affected and unaffected limbs in PFPS patients measured with ultrasound. However, the authors did not find any significant differences between the patients and the healthy control group. Doxey<sup>10</sup> noted a significant reduction in quadriceps thickness in individuals with chronic unilateral PFPS compared with the asymptomatic knees. This atrophy was detected by thigh girth measurements and B-mode ultrasound scans. Recently, a nuclear magnetic resonance imaging (MRI) study showed a decreased total volume and CSA of the quadriceps muscle in patients with unilateral PFPS.<sup>21</sup>

Clearly, there is a lack of well-documented comparative studies between PFPS patients and healthy controls concerning VMO atrophy. The goal of the present study was therefore to examine if PFPS patients actually exhibit a smaller size of the muscles (VMO and VL) that play a significant role in the dynamic balance of the patella.

## MATERIALS AND METHODS

### Participants

A total of 46 patients with PFPS were enrolled in this study. The diagnosis of PFPS and eligibility for the study were established by an experienced orthopaedic surgeon of the Ghent University Hospital or Jan Palfijn Hospital in Ghent. The criteria for PFPS were (1) peripatellar or retropatellar pain provoked by at least 2 of the following activities: prolonged sitting with flexed knees, stair climbing, squatting, running, kneeling, and jumping and (2) exhibition of 2 or more of the following clinical criteria on assessment: pain on direct compression of the patella against the femoral condyles with the knee in full extension, tenderness on palpation of the posterior edge at the medial and/or lateral border of the patella, pain on resisted knee extension, and pain on direct compression of the

patella against the femur during isometric quadriceps contraction with the knee in slight flexion. The inclusion criteria for this study included age between 12 and 40 years (to reduce the likelihood of osteoarthritic changes in the patellofemoral joint) and the presence of patellofemoral pain for a minimum of 3 months. The exclusion criteria consisted of (1) history or evidence of other knee disorders like patellar tendinopathy, ligament injury, bursitis, Osgood-Schlatter disease, meniscal injury, or osteoarthritis; (2) lower limb surgery or trauma <1 year previous; (3) patellar instability; and (4) pregnancy or other MRI contraindications like claustrophobia, implanted metals, and unremovable piercings. The patients did not receive any treatment for PFPS before entering the study. At the intake of the study, the patients completed the Kujala<sup>22</sup> scale, which objectified their pain and functionality. The Kujala score was on average 70.96 in this patient group, indicating reduced function.

The control group consisted of 30 healthy persons, recruited from the staff and the students from the Department of Rehabilitation Sciences and Physiotherapy of Ghent University, with no history of knee joint disorder. They also fulfilled the criteria of age and exhibited no MRI contraindications. The 30 controls were chosen to obtain similar age, body mass index (BMI), gender, and activity distributions (Table 1). The activity level was measured via the Baecke questionnaire.<sup>1</sup>

The mean duration of PFPS symptoms was 17 months. Twenty-six of a total of 46 PFPS patients reported bilateral knee pain. Only their more severely affected leg was used for the analysis. Nine patients with unilateral PFPS suffered from left patellofemoral pain, and 11 patients experienced pain at the right side. The corresponding dominant or nondominant leg was at random determined for the healthy control group. This resulted in 46 injured legs and 30 corresponding healthy legs. Twenty-nine dominant and 17 nondominant sides were tested in the patient group. Accordingly, the proportion in the control group was 19 dominant against 11 nondominant legs.

After receiving oral and written information, all patients signed informed consent forms. The study was approved by the local ethics committee of the Ghent University Hospital.

## Magnetic Resonance Imaging

Quadriceps anatomic CSA was obtained through MRI using a 3-T magnet (MAGNETOM Trio-Tim System, Siemens AG, Erlangen, Germany). Two body matrix coils on both legs were combined with a spine coil underneath as a receiver coil combination. The MRI scan was performed after 30 minutes of supine rest to control for the influence of posture-related fluid shifts on muscle size.<sup>4</sup> The patients were placed in a comfortable and relaxed supine position with straight legs. Two plastic tubes filled with water were fixed to the person's upper legs to indicate where the images should be taken. The body matrix coils restrained any leg movement during the scan procedure. All T2-weighted images were captured from both legs.

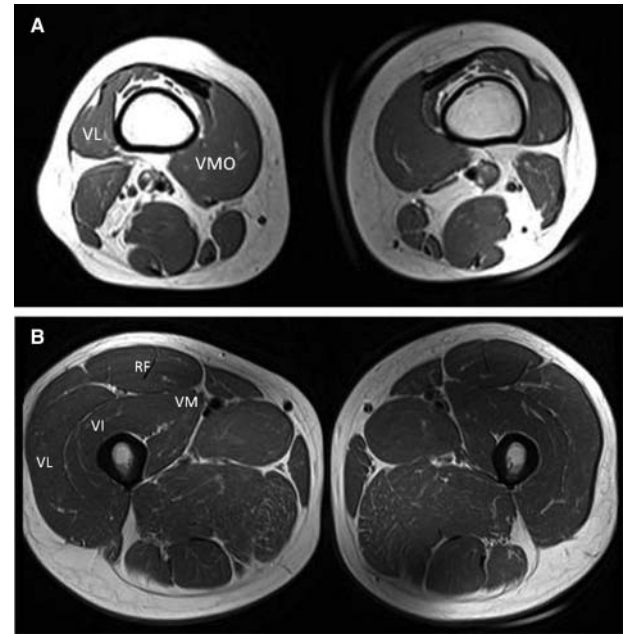
An initial localizing sequence was first performed to determine the position of the slices on the basis of the 2 tubes used as markers. One axial slice was taken at 2 cm above the superior border of the patella to bring the VMO clearly into view (Figure 1). The second slice was located at the middle of the distance from the spina iliaca anterior superior to the superior border of the patella (Figure 1). The following sequence parameters were used: field of view, 380 mm; repetition time, 3000 milliseconds; echo time, 38.0 milliseconds; voxel size,  $1.1 \times 0.8 \times 5.0$  mm; slice thickness, 5.0 mm.

## Data Management

The MRI images were transferred to an independent workstation (Leonardo, Siemens AG) and postprocessed with standard software (Syngo VB13, Siemens AG). The CSAs were measured by a trained observer by manually drawing contours around the muscle boundaries. This person was blinded to the participant's identity. The intramuscular fat was not quantified, and thus, the results represented the gross CSAs without any vascular and nerve bundle. At the patellar level, the CSAs of the VMO and the VL were measured. The CSAs of the following muscles were calculated at the middle of the thigh: VL, vastus medialis and vastus intermedius (VMVI) (could not be fully distinguished from each other on that level, so they have been considered as one entity), rectus femoris (RF), and the total quadriceps muscle (sum of all the quadriceps components). The ratio VMO:VL was calculated at the patellar level.

## Statistical Analysis

All statistical analyses were performed using SPSS 18.0 for Windows (SPSS, Chicago, Illinois). For 21 patients, the CSA measurements were performed by 2 independent operators to determine the interobserver reliability. One month later, the measures on 15 patients were repeated by one of the observers. The intertester and intratester reliability was evaluated by the intraclass correlation coefficients. The normality of variables was evaluated by the Kolmogorov-Smirnov test, which demonstrated a normal distribution except for age. To evaluate the differences in CSA between



**Figure 1.** Magnetic resonance images of the quadriceps components. A, magnetic resonance image of a female patellofemoral pain syndrome (PFPS) patient at 2 cm above the superior border of the patella. VMO, vastus medialis obliquus; VL, vastus lateralis. B, magnetic resonance image of a male PFPS patient at the midthigh level. VL, vastus lateralis; VMVI, vastus medialis and intermedius; RF, rectus femoris.

the patients and the control group, a 2-way analysis of variance was used that was covariate adjusted for gender, BMI, and activity index. These parameters were used as covariates because of their close relationship with CSA. The statistical significance level was set at  $P < .05$ .

## RESULTS

### Reliability

The intraclass correlation coefficients for the intratester and intertester agreement for the CSA of the quadriceps components ranged respectively from .976 to .998 and from .672 to .989 depending on the muscles evaluated, indicating good to excellent reliability (Table 2).

### CSA Measurements

The CSA measurements of the quadriceps components at the patellar and midthigh level are presented in Table 3. A significant difference was noted between the CSA of the VMO of the injured leg of the PFPS patients and the corresponding legs of the healthy control patients ( $P = .040$ ). The CSA of the VMO was significantly smaller in the PFPS group than the control group ( $16.67 \pm 4.97$  cm<sup>2</sup> vs  $18.36 \pm 5.25$  cm<sup>2</sup>). At the middle of the thigh, the

TABLE 2  
Intraclass Correlation Coefficients<sup>a</sup>

			Intratester Reliability ICC (95% CI)	Intertester Reliability ICC (95% CI)
Patellar level	VMO	R	.998 <sup>b</sup> (.993-.999)	.989 <sup>b</sup> (.636-.998)
		L	.992 <sup>b</sup> (.976-.997)	.981 <sup>b</sup> (.922-.993)
	VL	R	.995 <sup>b</sup> (.985-.998)	.952 <sup>b</sup> (.878-.981)
		L	.989 <sup>b</sup> (.940-.997)	.887 <sup>b</sup> (.743-.953)
Midhigh level	VMVI	R	.976 <sup>b</sup> (.933-.992)	.782 <sup>b</sup> (.532-.907)
		L	.998 <sup>b</sup> (.992-.999)	.767 <sup>b</sup> (.496-.901)
	VL	R	.990 <sup>c</sup> (.976-.997)	.673 <sup>b</sup> (.322-.860)
		L	.991 <sup>b</sup> (.965-.997)	.672 <sup>b</sup> (.341-.855)
	RF	R	.997 <sup>b</sup> (.991-.999)	.859 <sup>b</sup> (-.015 to .967)
		L	.997 <sup>b</sup> (.991-.999)	.910 <sup>b</sup> (.094-.979)

<sup>a</sup>ICC, intraclass correlation coefficient; CI, confidence interval; VMO, vastus medialis obliquus; VL, vastus lateralis; VMVI, vastus medialis and intermedius; RF, rectus femoris; R, right side; L, left side.

<sup>b</sup> $P < .001$ .

<sup>c</sup> $P < .01$ .

CSA of the VL and the RF of the patient group differed significantly from those of the healthy individuals. A tendency was noted for a smaller total quadriceps CSA for the PFPS patients at the midhigh level ( $66.99 \pm 15.06 \text{ cm}^2$  vs  $70.83 \pm 15.30 \text{ cm}^2$ ) ( $P = .074$ ). There was no significant difference in the VMO:VL muscle size ratio at the patellar level.

## DISCUSSION

The purpose of this study was to investigate if quadriceps and, in particular, VMO atrophy is present in patients with PFPS. The main results indicated that patients with PFPS show a less developed quadriceps and, in particular, a significantly smaller CSA of the VMO compared with healthy controls. There was no significant difference in the CSA of the VL at the patellar level between both groups. The ratio of VMO and VL at the patellar level was not significantly different between the patient and the control group. This was probably due to the fact that the VL of the patients was also smaller than the VL of the controls, although this difference was not significant. At the middle of the thigh, the CSA of the VL and the RF of the patient group was significantly smaller than those of the healthy individuals. There was only a borderline significance for general atrophy of the total quadriceps on the midhigh level. The differences of the VL and RF were even more explicit than the difference of the VMO. However, as at the midhigh level the VL and RF do not play an active role in the stabilization of the patella, we consider that the most important finding of this study is the significant atrophy of the VMO at the patellar level because it is an accepted fact in the literature that within the quadriceps the VMO is the most important dynamic stabilizer of the patellofemoral joint.<sup>16,24,27</sup>

Comparing the outcome of the present study with other research is hardly possible as almost no research exists on VMO atrophy in PFPS patients. Nevertheless, VMO

atrophy is a commonly cited accompaniment to PFPS, despite no objective supporting evidence. Perhaps clinicians assess VMO atrophy exclusively based on visual inspection. Actually, the distal portion of the VM appears as a prominence that turns into a bulge by contraction. In this respect, Lieb and Perry<sup>24</sup> stated that the clinical prominence of the VM may be attributed to the extreme obliquity of its distal fibers, the lowness of the insertion, and the thinness of the fascial covering. To the best of our knowledge, the study of Jan et al<sup>20</sup> was the only report focusing on VMO differences between PFPS patients and healthy controls. They revealed also a smaller muscle size of the VMO in the PFPS patients compared with the healthy individuals. However, it should be noted that no MRI measurements were performed as the VMO volume was measured by sonography. Nevertheless, together with the quoted study of Jan et al,<sup>20</sup> the present study shows objectively that patients with PFPS demonstrated a less developed VMO than healthy controls.

There may be different points of view to explain the phenomenon of a smaller VMO size in patients with PFPS in comparison with healthy controls. First, as a result of their pain, patients might have spared their injured leg(s), and this disuse possibly led to the atrophy of some of the quadriceps components. Moreover, the pain might have led to reflex inhibition, known to induce muscle atrophy. Hence, VMO atrophy might be a protective measure to reduce load on a painful patella. In contrast, Leroux et al<sup>23</sup> concluded that the VMO atrophy found in PFPS would not be associated with direct monosynaptic pathways, reflex inhibition that originates from nociceptor activation such as type IV joint receptors. According to these authors, the specific atrophy could be more likely elucidated by the functional differences observed by Lieb and Perry<sup>24</sup> between the quadriceps components. The VMO inserts directly on the patella and acts in tracking the patella medially without extending the knee, whereas the VL and the proximal fibers of the VM insert on the quadriceps femoris tendon and assist the RF in knee extension.<sup>24</sup>

TABLE 3  
CSA Measurements of the Quadriceps Components in PFPS Patients and Healthy Controls<sup>a</sup>

		PFPS Mean CSA (SD), cm <sup>2</sup>	Control Mean CSA (SD), cm <sup>2</sup>	Corrected Mean Difference (95% CI)	P
Patellar level	VMO	16.67 (4.97)	18.36 (5.25)	-2.02 (-3.94 to -0.10)	.040 <sup>b</sup>
	VL	5.90 (3.30)	6.59 (2.66)	-0.90 (-2.26 to 0.46)	.192
	VMO:VL	3.53 (1.99)	3.02 (0.86)	0.54 (-0.25 to 1.34)	.179
Midhigh level	VMVI	31.47 (8.28)	33.26 (8.92)	-1.61 (-4.44 to 1.21)	.259
	VL	24.83 (5.43)	27.19 (6.28)	-2.62 (-4.79 to -0.44)	.019 <sup>b</sup>
	RF	11.23 (2.59)	12.11 (3.18)	-1.21 (-2.02 to -0.37)	.006 <sup>b</sup>
	Total Q	66.99 (15.06)	70.83 (15.30)	-4.52 (-9.50 to 0.46)	.074

<sup>a</sup>Corrected mean difference is the mean difference after correction for gender, body mass index, and activity index. PFPS, patellofemoral pain syndrome; CSA, cross-sectional area; SD, standard deviation; CI, confidence interval; VMO, vastus medialis obliquus; VL, vastus lateralis; VMVI, vastus medialis and intermedius; RF, rectus femoris; VMO:VL, ratio of VMO in relation to VL.

<sup>b</sup>Significant between PFPS patients and control group ( $P < .05$ ).

A second possible explanation is that VMO atrophy might be indicative of VMO insufficiency, which may have led to a muscular imbalance between the dynamic knee stabilizers. If the VMO is no longer able to counteract the lateral pull of the larger VL, abnormal lateral tracking of the patella may occur, which has often been proposed as an important contributing factor of PFPS.

However, as this was a cross-sectional study, it is impossible to draw conclusions about VMO atrophy being the effect or the cause of PFPS. As a smaller VMO was demonstrated, it can only be stated that it plays a role in this disorder without any cause or effect assumptions.

To our knowledge, this is the first study to examine the muscle size of the VMO in PFPS patients compared with healthy controls by means of MRI. The choice of MRI was based on the accepted knowledge that MRI is the typical gold standard for assessing muscle size.<sup>2,11,26</sup> Magnetic resonance imaging has several advantages in comparison with computed tomography, ultrasound, and other imaging methods. The soft tissue contrast is superior, and the individual thigh muscles are more clearly delineated.<sup>2,34</sup> The observability of vessels and nerves on MRI makes it possible to exclude them from the measurements.<sup>34</sup> Also, MRI does not expose the participants to ionizing radiation and is considered to be safe.<sup>11</sup> Unfortunately, MRI is known to be expensive and time consuming.<sup>34</sup>

An important limitation of this study was its design. A cross-sectional study is not able to prove cause and effect relationships. It was demonstrated that the CSA of the VMO was significantly smaller in the patient group in comparison with the healthy control group. However, the results of this study did not resolve the question whether PFPS causes the decrease in muscle size or the decrease in muscle size results in PFPS. Longitudinal prospective research will be needed to enlighten the cause-result issue.

Another consideration is the use of the term "atrophy." Because of its cross-sectional design, this study only showed that the VMO size was smaller in the PFPS patients in comparison with the control group. However, this study cannot determine whether the patients also had a smaller VMO size before their patellofemoral

complaints started. Consequently, it may be possible that within the PFPS patients, the VMO did not atrophy but was just developmentally smaller compared to those of the healthy controls.

Another interesting value for this study would be the measurement of the quadriceps strength. Although it is not possible to measure the maximum strength of the individual quadriceps components separately, it would be interesting to examine the relationship between the differences in the entire quadriceps force and the differences in quadriceps CSA in PFPS patients and healthy controls. This may provide more insight to what extent muscle atrophy may account for muscle weakness as the current data on this subject are rather limited and vague. Callaghan and Oldham<sup>6</sup> found a poor correlation between CSA and peak torque changes of the lower limb extensors (not specifically quadriceps torque) and concluded that PFPS patients have weaker extensor muscles that cannot be explained by muscle atrophy. Thus, there may be more subtle mechanisms other than muscle size limiting quadriceps function. More research will be needed to shed light on this matter.

In conclusion, this study is the first to examine VMO size in PFPS patients by MRI. The CSA of the VMO was 2 cm<sup>2</sup> smaller in patients with PFPS in comparison with healthy control individuals. Because there was no significant difference in the CSA of the VL at the patellar level, it seems that the VMO muscle was disproportionately smaller in comparison with the VL. A tendency was noted for a smaller CSA of the total quadriceps for the PFPS patients at the midhigh level. Whether the VMO atrophy is a cause or effect of PFPS is still an unanswered question. Further research, among other things, on the relationship between quadriceps strength and atrophy will throw some more light on the origin of VMO atrophy in PFPS.

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