DOI: 10.7860/JCDR/2017/26975.9870

Original Article

Orthopaedics Section

Muscle Activation Profiles and Co-Activation of Quadriceps and Hamstring Muscles around Knee Joint in Indian Primary Osteoarthritis Knee Patients

SANJEEV KUMAR SHARMA[1], SHIV LAL YADAV[2], U SINGH[3], SANJAY WADHWA[4]

ABSTRACT

Introduction: Osteoarthritis (OA) of knee is a common joint disease. It is associated with reduced knee joint stability due to impaired quadriceps strength, pain, and an altered joint structure. There is altered muscle activation in knee OA patients, which interferes with normal load distribution around the knee and facilitates disease progression.

Aim: Our primary aim was to determine activation patterns of the muscles i.e., quadriceps and hamstrings in knee OA patients during walking. We also studied co-activation of muscles around knee joint in primary OA knee patients including directed medial and lateral co-contractions.

Materials and Methods: This observational study was done at Department of Physical Medicine and Rehabilitation, All India Institute of Medical Sciences, New Delhi, India. Forty-four patients with medial compartment primary knee OA were included in study after satisfying inclusion and exclusion criteria. All the patients were assessed for mean, peak and integrated Root Mean Square (RMS), EMG values, muscle activation patterns and co-activation of muscles around knee joint by surface Electromyography (EMG) analysis of Vastus Medialis Obliques (VMO), Vastus Lateralis (VL), Semitendinosus (SMT) and Biceps Femoris (BF) muscles during gait cycle. The EMG waveform for each muscle was amplitude normalized and time normalized to 100% of gait cycle and plotted on graph. Quantitative variables were assessed for normal distribution and accordingly mean±SD or median (range), as appropriate, was computed.

Results: For primary OA knee, mean age 61±5 years, mean weight 63.7±10.1 kg, mean height 153.9±7.2 cm, and mean Body Mass Index (BMI) 26.8±3.0 kg/m^2 was found. The muscle activity of hamstrings (SMT muscle and BF) was increased during midstance, late stance and early swing phase of gait cycle as compared to quadriceps (VMO and VL) muscle activity respectively, suggesting co-contraction of opposing muscles around knee joint.

Conclusion: Patients with knee OA walk with increased hamstring muscle activity (during late stance and early swing phase) and reduced quadriceps recruitment. Altered neuro-muscular control around knee interferes with normal load distribution and facilitates disease progression in knee joint.

Keywords: Muscle activation patterns, Muscle co-activation, Muscle co-contraction, Surface electromyography

INTRODUCTION

Knee OA is one of the common joint diseases of humans especially in middle-to-elderly age group [1,2]. The exact aetiology of OA is still unknown but multiple risk factors (e.g. increasing age, previous damage to the articular surfaces i.e., meniscectomy, anterior cruciate ligament injuries; muscular weakness; deformities or mal-alignment of bones; proprioceptive deficits; obesity) predispose to this disease. The medial compartment of knee is most frequently involved because during midstance phase of gait cycle, about 60%-80% of load is distributed through medial compartment of normal knee [3]. Varus angulation of knee also contributes to the progression of OA knee by causing increased load to medial compartment of knee [4]. The presence and progression of OA knee is usually evaluated by reading knee skiagrams and scoring them by using standard Kellgren-Lawrence (KL) scale [5,6]. Rogers MW et al., found that patients with higher Body Mass Index (BMI) had increased likelihood of knee pain as compared to subjects with normal BMI in knee OA [7]. Metcalfe AJ et al., showed that bilateral knee OA is very common, as clinical and radiographic OA disease also develops early in non involved knee [8].

Human gait is resultant of force produced by coordinated activation of agonist and antagonistic muscle groups around joint. During a normal gait cycle, agonist and antagonistic group of muscles contract alternatively, with very low activity of agonist muscles during contraction of opposite group of muscle (antagonist) and vice-versa [9,10]. In normal knee joint, quadriceps group of muscles contracts to produce anterior shear force on tibia relative to femur, but there is simultaneous low-level activation of hamstring group of muscles which oppose excessive anterior translation of tibia relative to femur, producing stability of knee joint during knee extension [11]. During normal gait, both quadriceps and hamstrings are activated sequentially to prepare for heel-strike and control of the leg as foot-flat occurs. During early midstance (0%-20% gait cycle), the dominant muscle groups are knee extensors (represented by vastus lateralis), as they eccentrically stabilize the knee from heel-strike through foot-flat. During late swing (80%-100% gait cycle) the dominant muscle group are knee flexors (represented by biceps femoris), as they eccentrically decelerate hip flexion and knee extension to prepare for heel-strike [12].

Electromyographic (EMG) techniques are used to study motor unit activity during muscle activation, which indicates the size and number of active motor units and the rate of their discharge. Surface EMG techniques are used to study muscle activation patterns especially the timing and magnitude of muscle contraction. It has been found in previous studies that there is good correlation between indwelling and surface EMG recordings [10] for the superficial muscles examined.

OA knee is associated with reduced knee joint stability due to impaired quadriceps strength, pain, and an altered joint structure [13–15]. So to maintain joint function and reduce instability, subjects with knee OA generate compensatory muscle activity via hamstrings muscles [16]. However, Hubley-Kozey CL et al., found no difference in muscle strength of quadriceps and hamstrings between asymptomatic and symptomatic moderate OA knee patients [17]. Also, Hortobágyi T et al., revealed that the hamstring activity increased, 200 milliseconds before heel strike and throughout stance phase in OA knee, that may cause altered muscle activation and change in distribution of load around knee, resulting in disease progression [18].

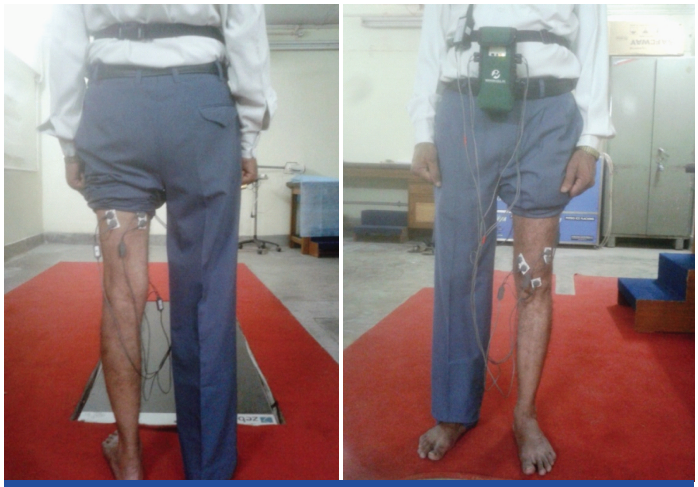
Our hypothesis is that muscle activation pattern at the knee is altered in knee OA and these patients utilize altered muscle recruitment patterns to stabilize knee during walking. So we planned a study to determine activation patterns of the muscles i.e., quadriceps and hamstrings in knee OA patients during walking. Our study also aimed to find relative contribution of each muscle (VMO, VL, SMT and BF) to knee stability and to study co-activation of muscles around knee joint in primary OA knee patients including directed medial and lateral co-contractions.

MATERIALS AND METHODS

This observational study was conducted at Department of Physical Medicine and Rehabilitation, All India Institute of Medical Sciences (AIIMS), New Delhi, India, of duration 21 months from July 2011 to April 2013. In this study, patients suffering from primary OA knee and satisfying inclusion and exclusion criteria were selected.

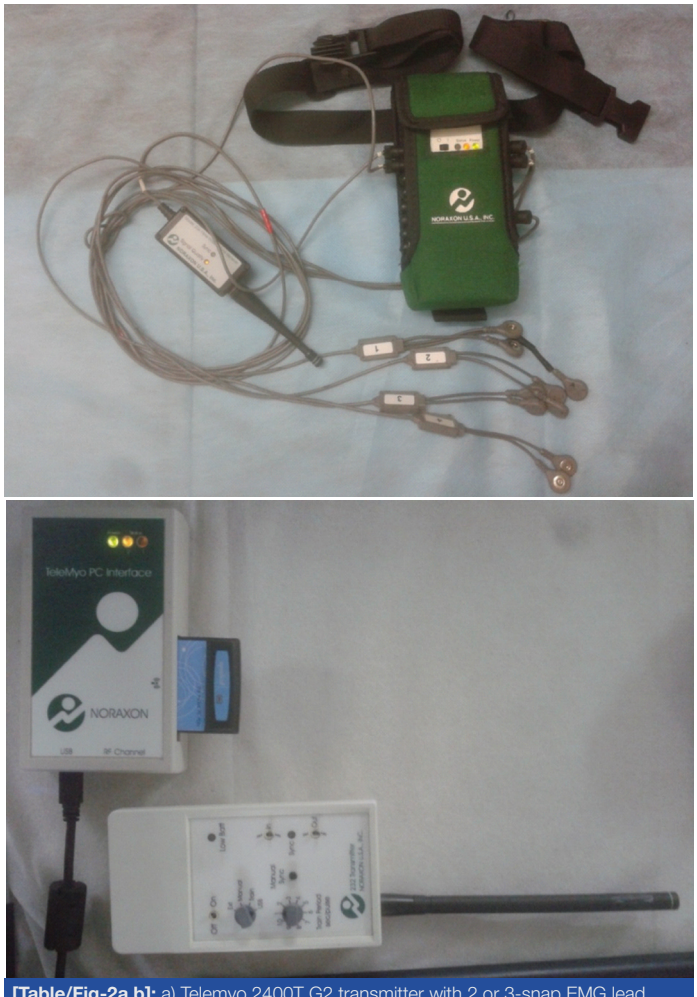
Patients aged between 40-70 years age of both gender; all diagnosed cases of primary OA knee as per American College of Rheumatology [19], 1986 clinical and radio-graphical criteria for knee OA and had grade II or III KL grade radiographic changes in the knee [5]; who were able to walk independently without support were included in study. Patients who were excluded from study were having any congenital abnormality, traumatic, infective condition or deformity subsequent to injury of knee joint which might have contributed to the development of secondary OA knee; any previous history of surgical intervention on knee joint; inflammatory arthritis (e.g., rheumatoid arthritis); Grade IV OA on KL scale; knee flexion deformity greater than 15°; inability to walk or patients who walked with the use of assistive devices. Informed written consent was taken from each participant prior to being enlisted in the study. Ethics approval was taken from Institutional Ethics Committee, prior to patient's enrolment in study. Complete history of patient's disease and detailed clinical examination was done. Few investigations like plain radiograph bilateral knee joints in standing position-antero-posterior and lateral views; complete haemogram; Erythrocyte Sedimentation Rate (ESR); serum uric acid levels were done for all the patients.

Subjects were asked to rate their knee pain in each leg immediately before testing on Visual Analogue Scale (VAS) after 20 meters plain surface walking. The subjects were asked to walk at their natural speed with comfortable shoes on ground. Then bipolar Ag/AgCl electrodes (single-use diagnostic paediatric ECG electrodes) with 1 cm diameter of effective caption area (0.79 mm^2 contact area) were placed in a bipolar configuration (20 mm centre-to-centre) over maximum muscle bulk area parallel to line of muscle fibres of VMO, VL, SMT and BF muscles of the involved leg i.e., most painful or functionally limited limb in OA patients [Table/Fig-1a,b]. Prior to application of the electrodes, the hair underlying the electrodes were removed using a standard disposable safety razor, and the skin was cleansed with alcohol according to SENIAM guidelines [20] (Surface EMG For Non-Invasive Assessment of Muscles). The electrode cables were attached to lower part of waist to avoid interference with movement of the leg. Muscle activity was recorded at 1000 Hz bandwidth with a 4-channel surface electromyography system (Telemyo® telemetric hardware system, Noraxon USA) [Table/Fig-2a,b] [19,20]. The differential amplifiers have a gain of 2000, an input



[Table/Fig-1a,b]: a) Electrode positioning over biceps femoris and semitendinosus; b) Electrode positioning over vastus medialis obliques and vastus lateralis.

impedance of 10 MΩ, and a common mode rejection ratio of 130 dB. A reference electrode was placed over patella. Crosstalk was assessed [21,22] and placement of the electrode was facilitated by palpating the muscle as the subject contracted the muscle against resistance [23]. Additionally accurate placement of the electrode was verified during the normalization process. If a good signal was not obtained when the individual performed a maximal isometric contraction of the muscle, then the electrode was repositioned. Then subjects were asked to walk bare-foot at their comfortable pace on testing walkway with electrodes attached and surface muscle activity for each muscle was assessed using Myo-research XP clinical edition (version 1.07.09) software. The video module contains a high-quality video camera with a stand, a wide-angle lens, synchronization cables and software extension. The recording



[Table/Fig-2a,b]: a) Telemyo 2400T G2 transmitter with 2 or 3-snap EMG lead connected and Inline wireless Sync Receiver connected; b) Telemyo 2440T G2 PC interface connected to USB cable.

and display of the images take place at a measuring rate of 50/60 images per second.

STATISTICAL ANALYSIS

Myo-research XP clinical edition (version 1.07.09) software corrected the raw EMG signals for subject bias, converted amplitudes into 1 Volt, full-wave rectified and low pass filtered the signals at 6 Hz using a zero-lag Butterworth filter [12]. The Root-Mean-Square (RMS) of the direct EMG signal was obtained by using a 100-ms smoothing window. The EMG waveforms for each muscle were amplitude normalized to the sub-maximal 0.1-s amplitude (peak RMS muscle activity) that was recorded by software itself and time-normalized to 100% of the gait cycle (duration of stance phase). The y-axis is normalized EMG amplitude in percentage muscle activity and the x-axis is percentage gait cycle. Then markers were placed at heel strike and then at toe off (for stance phase) and from toe-off to heel strike (for swing phase). Onset and offset times of the muscle activity were determined without normalization to time by visually identifying the time when the sEMG had a phasic increase in activation above baseline. Gait cycle was broken down into four sub-phases i.e., percentage of gait cycle as we have already done time-normalization of EMG data to 100% of gait cycle. Analysis of muscle activation patterns was done in following sub-phases:

1. Early stance 0%-20% of gait cycle;
2. Mid-late stance 20%-60% of cycle;
3. Early swing 60%-80% of cycle;
4. Late swing 80%-100% of cycle.

Comparison of mean, peak RMS and integrated EMG activity level of individual knee muscles during stance phase was done.

Mean RMS EMG

It is the time average of the full-wave rectified EMG over a specified period of time. It is measured in mV. In our study, we took stance duration (affected knee heel-strike to toe-off) as time period for calculation of mean EMG for each tested muscle.

Peak EMG

It is the peak RMS EMG activity of the full-wave rectified EMG over a specified period of time. It is measured in mV.

Integrated EMG

It depicts the area under the curve for a full-wave rectified EMG signal over a specified period of time. It is measured in mV·s. In our study, we took stance duration as time period for calculation of Integrated EMG for each tested muscle.

The amount of co-contraction was quantified using a Co-Contraction Area (CCA) calculation adapted from Damiano et al., a ratio between integrated antagonist and agonist signals during each designated phase of gait [24].

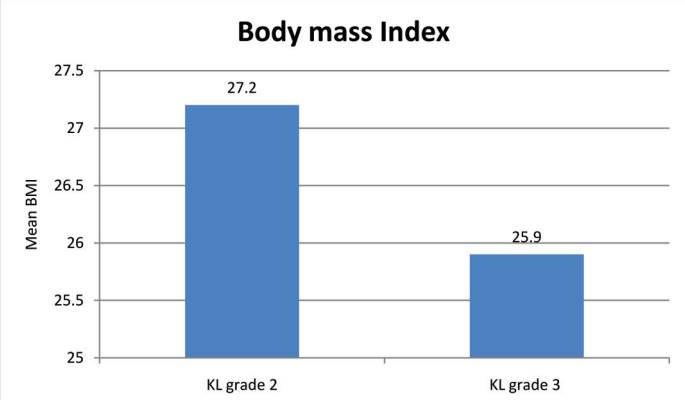
Directed Co-contraction Ratios (DCCR) of Agonists and Antagonists

1. **Medio-Lateral Directed Co-Contraction Ratio (ML-DCCR):** Ratio of net muscle activity over medial side of knee joint to lateral side of knee joint. It is calculated as (*Mean RMS EMG of SMT + Mean RMS EMG of VMO*) / (*Mean RMS EMG of BF + Mean RMS EMG of VL*).
2. **Medial (SMT)/ lateral (BF) hamstrings ratio (HAM-DCCR):** Ratio of mean RMS EMG activity of semitendinosus and biceps femoris. It is calculated as *Mean RMS EMG of SMT/Mean RMS EMG of BF*.
3. **Quadriceps- Hamstring coactivity Ratio (EF-DCCR):** Ratio of net muscle activity of knee extensors (Quadriceps) to knee flexors (Hamstrings) around knee joint. It is calculated as *(*Mean RMS EMG of VMO + Mean RMS EMG of VL*)/(*Mean RMS EMG of SMT + Mean RMS EMG of BF*)*.

For sample size calculation, a pilot study of primary OA knee patients (n=10) fulfilling our inclusion and exclusion criteria was done. The mean and standard deviation value of ratio (mean RMS activity of VL/mean RMS activity of biceps femoris) was found to be 111±33.38. Taking power of study as 80% and level of significance as 5%, the sample size is found to be 35 (including drop-outs of 20%). However, we were able to recruit 56 patients in our study, out of which 44 patients completed the study. Data was recorded on a predesigned performa and excel spreadsheet. Quantitative variables were assessed for normal distribution and accordingly mean±SD or median (range), as appropriate, was computed. Fisher's-exact test was used to evaluate correlation of BMI with KL grade of knee.

RESULTS

We included a total of 44 patients with primary OA knee with mean age 61±5 years, mean weight 63.7±10.1 kg, mean height 153.9±7.2 cm, mean BMI 26.8±3.0 kg/m^2. Majority of the affected females (73%) were housewives and affected males (27%) were either retired or employed in jobs involving prolonged standing. Thirty-three out of 44 patients patients had bilateral involvement of knees. In our study population, the mean duration of symptoms at the time of presentation was 7.6±4.6 years. Mean pain in knee on VAS scale was 8.08±0.8 with most pain aggravating factor found to be climbing upstairs (100% cases), followed by squatting and sitting cross-legged. However, all of the patients reported pain relief with lying supine and taking analgesics. Majority of our patients reported early morning stiffness lasting for less than 30 minutes but none reported features suggestive of joint instability or locking of knee. On clinical examination, all had medial joint line tenderness at knees and mild-to-moderate genu-varum deformity. No joint effusion or baker's cyst or significant ligament laxity were present at the time of evaluation. All patients had medial joint space narrowing in radiographs i.e., grade 2 and 3 according to K-L grading system for OA knee. There was no significant correlation between BMI and KL grade of patients however, patients with higher BMI reported



[Table/Fig-3]: Correlation between mean body mass index and KL grade of knee.

lower radiographic changes i.e., KL grade 2 changes in knee [Table/Fig-3]. The mean characteristics of muscles and gait cycle in knee OA patients are presented in [Table/Fig-4].

Muscle Activation Patterns

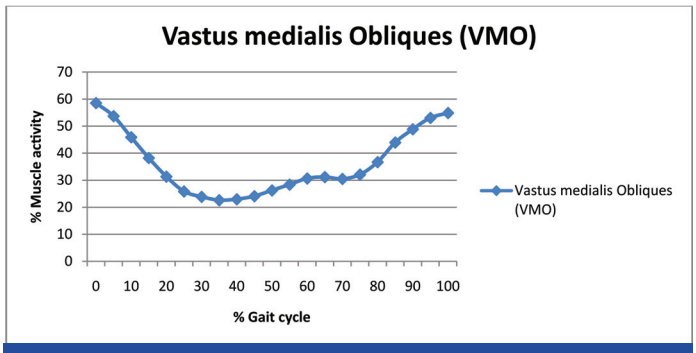
VMO: There is high muscle activity during early stance phase followed by decline in muscle activity during midstance phase. However, there is transient increased activity during late stance phase followed by decline during early swing phase. Again there is increased muscle activation in late swing phase which continues to early stance phase [Table/Fig-5].

VL: VL muscle also follows similar activation pattern to VMO, except there is no transient increased activity during late stance phase [Table/Fig-6].

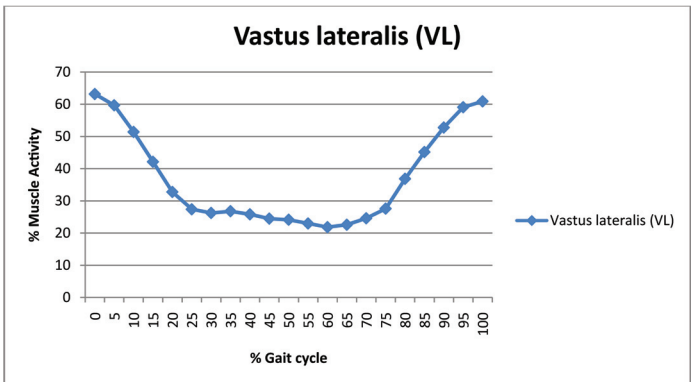
SMT: There is high muscle activity during early stance phase which rapidly declines during early part of midstance phase. However,

Variables (Unit)	Value (Mean± SD) n=44
VMO Mean RMS value (mV)	31.1±9.5
VMO Peak RMS value (mV)	64.4±19.4
VMO Integrated (mV*s)	3176.2±978.5
VLO Mean RMS value (mV)	30.9±11.3
VLO Peak RMS value (mV)	65.8±22.1
VLO Integrated (mV*s)	3134.3±1121.3
SMT Mean RMS value (mV)	35.0±9.5
SMT Peak RMS value (mV)*	65.2 (33.9-102.9)
SMT Integrated (mV*s)	3434.8±944.5
BF Mean RMS value (mV)	35.7±12.2
BF Peak RMS value (mV)	67.3±18.1
BF Integrated (mV*s)	3683.4±1226.5
Hamstring coactivity ratio= BF/VL*	110.3 (40.1-792.5)
BF/Bfmax ratio	53.5±12.2
VL/Vlmax ratio	48.0±10.5
VMO/SMT* ratio	87.2 (41.6-250)
SMT/BF (HAM-DCCR) ratio	106.2±37.3
VMO/VLO* ratio	103.7 (44.8-440.9)
EF-DCCR ratio	94.1±36.6
ML-DCCR ratio	1.1±0.3
Duration Stance phase (ms)	869.1±93.0
Duration swing phase (ms)	401.8±43.1
Duration gait cycle (ms)	1270.9±107.0
Stance/Swing phase duration	2.1±0.3
Gait Speed (m/s)	0.5±0.1
Knee Pain (VAS)	8.1±0.8

[Table/Fig-4]: Characteristics of knee osteoarthritis patients.
* Data represented in median(minimum–maximum) and Wilcoxon sign-rank test applied



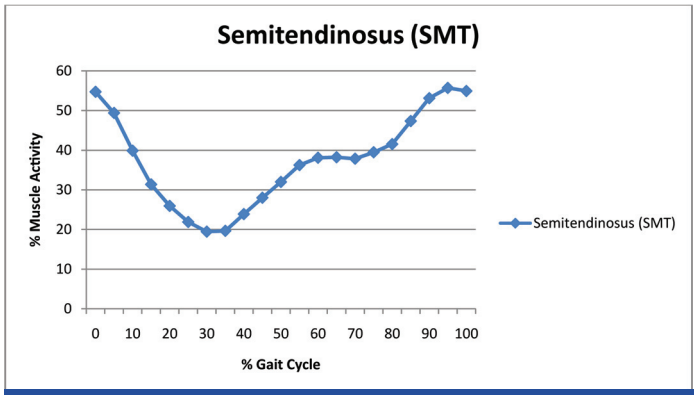
[Table/Fig-5]: Vastus medialis Obliques muscle activation pattern during gait cycle.



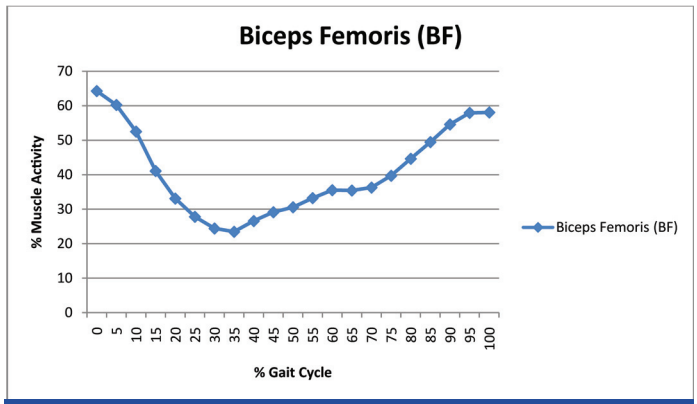
[Table/Fig-6]: Vastus lateralis muscle activation pattern during gait cycle.

muscle activity increases during late stance phase, plateaus during early swing phase, followed by increased muscle activation in late swing phase, which continues to early stance phase [Table/Fig-7].

BF: Biceps femoris follows similar activation pattern to SMT [Table/Fig-8].

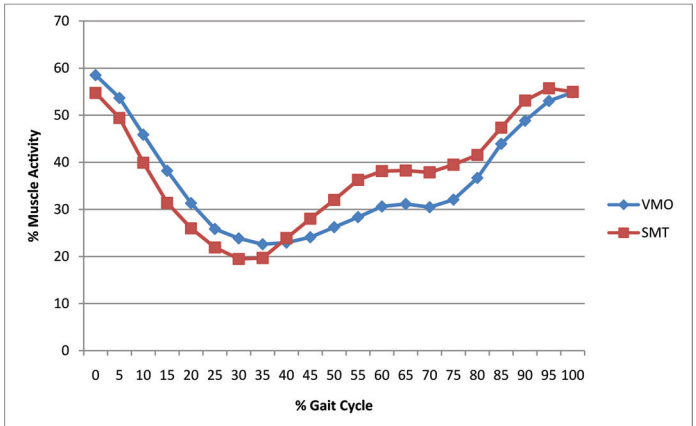


[Table/Fig-7]: Semitendinosus muscle activation pattern during gait cycle.



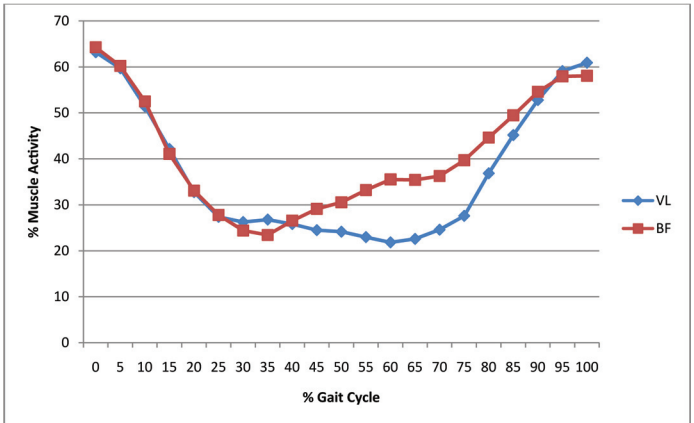
[Table/Fig-8]: Biceps femoris muscle activation pattern during gait cycle.

Comparison between muscle activation patterns: If muscles on medial side of knee i.e., VMO and SMT muscle activity patterns were compared to each other, it was found that there is increased muscle activity of VMO as compared to SMT in early stance phase followed by gradual increment in SMT muscle activity (suggesting co-activation of muscles) during mid-late stance phase. Also, there is higher muscle activity of SMT as compared to VMO during early as well as late swing phase [Table/Fig-9].



[Table/Fig-9]: Comparison of muscle activation patterns of vastus medialis obliques and semitendinosus during gait cycle.

If muscles on lateral side of knee i.e., VL and BF muscle activity patterns were compared to each other, it was found that similar muscle activation of both muscles during early stance phase followed by gradual increment in BF muscle activity as compared to VL suggesting co-activation of muscles during mid-late stance phase is seen. Also, higher muscle activity of BF as compared to VL is seen in early swing phase followed by gradual decrement of muscle activity of BF as compared to VL in late swing phase [Table/Fig-10].



[Table/Fig-10]: Comparison of muscle activation patterns of vastus lateralis and biceps femoris during gait cycle.

DISCUSSION

OA knee usually affects elderly age group [2,25] as also evident from our study. There was female preponderance (73%) observed which is in accordance to several previous reports where prevalence of OA knee was found more in females [26]. Most of the patients in our study were overweight or obese, as obesity is considered a risk factor for the development of this condition [7]. Majority (75%) of patients had bilateral involvement of knees as suggested by Metcalfe AJ et al., in a recent study [8]. Mild genu varum deformity was present in more than half of our patients which is a marker of disease severity and associated with risk for progression of knee OA. There is evidence to suggest that much of the effect of obesity on the severity of medial compartment knee OA can be explained by varus mal-alignment [25].

The muscle activation patterns found in this study were nearly similar to patterns reported by Mohamed O et al., in normal persons (without knee OA) during gait cycle [27]. However, few differences were found. In knee OA patients, muscle activity of hamstrings (SMT and BF) increases during late stance phase, which plateaus during early swing phase, followed by increased muscle activation in late swing phase; as compared to normal persons (without knee OA) having reduced or no muscle activity of hamstrings during late stance and early swing phases of gait cycle. It suggests that knee extension during late stance phase (by concentric contraction of quadriceps) and knee flexion during early swing phase (eccentric contraction of quadriceps) is being opposed by increased hamstrings activity (co-contraction) to provide stability to knee joint in knee OA patients.

In OA knee, there is increased hamstring muscles activity before heel-strike and throughout duration of stance phase as reported by Hortabagyi T et al., [18]. The muscle activity of SMT and BF was increased during midstance, late stance and early swing phase of gait cycle as compared to VMO and VL muscle activity respectively, suggesting co-contraction of antagonistic muscles around knee joint.

Medio-lateral directed co-contraction ratio (ML-DCCR) of 1.1±0.3 indicates relatively more co-contraction of medial (VMO, SMT) as compared to lateral group of muscles (VL, BF); suggesting more compressive forces (articular loading) over medial compartment of knee and more disease progression in medial compartment of knee. Medial (SMT)/lateral (BF) hamstrings ratio (HAM-DCCR) of 106.2±37.3 suggests higher muscle activation and recruitment of SMT during gait cycle in knee OA patients. Quadriceps-hamstring coactivity ratio (EF-DCCR) of 94.1±36.6 suggests reduced recruitment of quadriceps or heightened hamstring activity around knee during gait cycle. It suggests that interventions causing knee pain reduction or quadriceps strengthening exercises that could improve quadriceps activation in knee OA patients can substantially improve neuro-muscular control around knee and prevent disease progression. More studies are required in this regard.

LIMITATION

Patients who had kellegren-lawrence grade IV knee OA, medio-lateral knee instability and lateral knee compartment affection were not involved in this study. Future studies should be conducted on these patients, for better understanding of knee control during walking.

CONCLUSION

There is co-activation of muscles around knee joint in primary OA knee patients to stabilize the joint during walking. Patients with knee OA walk with increased hamstring muscle activity (during late stance and early swing phase) and reduced quadriceps recruitment. Altered neuro-muscular control around knee interferes with normal load distribution in the knee and facilitates disease progression.

REFERENCES

[1] Felson DT, Lawrence RC, Dieppe PA, Hirsch R, Helmick CG, Jordan JM, et al. Osteoarthritis: new insights. Part 1: the disease and its risk factors. Ann Intern Med. 2000;133(8):635–46.
[2] Felson DT. Osteoarthritis. In: Anthony S F et al, editor. Harrison's Principle of Internal Medicine. 17th ed. USA: McGraw Hill; 2008. pp. 2158–65.
[3] Johnson F, Leitl S, Waugh W. The distribution of load across the knee. A comparison of static and dynamic measurements. J Bone Joint Surg Br. 1980;62(3):346–49.
[4] Cerejo R, Dunlop DD, Cahue S, Channin D, Song J, Sharma L. The influence of alignment on risk of knee osteoarthritis progression according to baseline stage of disease. Arthritis Rheum. 2002;46(10):2632–36.
[5] Kellgren JH, Lawrence JS. Radiological assessment of osteo-arthrosis. Ann Rheum Dis. 1957;16(4):494–502.
[6] Spector TD, Cooper C. Radiographic assessment of osteoarthritis in population studies: whither Kellgren and Lawrence? Osteoarthritis Cartilage. 1993;1(4):203–06.
[7] Rogers MW, Wilder FV. The association of BMI and knee pain among persons with radiographic knee osteoarthritis: A cross-sectional study. BMC Musculoskelet Disord [Internet]. 2008[cited 2017 Jan 20];9(1). Available from: http://bmcmusculoskeletdisord.biomedcentral.com/articles/10.1186/1471-2474-9-163
[8] Metcalfe AJ, Andersson ML, Goodfellow R, Thorstensson CA. Is knee osteoarthritis a symmetrical disease? Analysis of a 12 year prospective cohort study. BMC Musculoskelet Disord. 2012;13:153.
[9] Grasso R, Zago M, Lacquaniti F. Interactions between posture and locomotion: motor patterns in humans walking with bent posture versus erect posture. J Neurophysiol. 2000;83(1):288–300.
[10] Ivanenko YP, Poppele RE, Lacquaniti F. Five basic muscle activation patterns account for muscle activity during human locomotion. J Physiol. 2004;556(Pt 1):267–82.
[11] Bernardi M, Solomonow M, Baratta RV. Motor unit recruitment strategy of antagonist muscle pair during linearly increasing contraction. Electromyogr Clin Neurophysiol. 1997;37(1):3–12.
[12] Winter DA, Yack HJ. EMG profiles during normal human walking: stride-to-stride and inter-subject variability. Electroencephalogr Clin Neurophysiol. 1987;67(5):402–11.
[13] Hurley MV. Muscle dysfunction and effective rehabilitation of knee osteoarthritis: What we know and what we need to find out. Arthritis Care Res. 2003;49(3):444–52.
[14] Pietrosimone BG, Hertel J, Ingersoll CD, Hart JM, Saliba SA. Voluntary quadriceps activation deficits in patients with tibiofemoral osteoarthritis: a meta-analysis. PM&R. 2011;3(2):153–62.
[15] Slemenda C, Brandt KD, Heilman DK, Mazzuca S, Braunstein EM, Katz BP, et al. Quadriceps weakness and osteoarthritis of the knee. Ann Intern Med. 1997;127(2):97–104.
[16] Bennell KL, Hinman RS, Metcalf BR. Association of sensorimotor function with knee joint kinematics during locomotion in knee osteoarthritis. Am J Phys Med Rehabil. 2004;83(6):455-463; quiz 464-466, 491.
[17] Hubley-Kozey CL, Deluzio KJ, Landry SC, McNutt JS, Stanish WD. Neuromuscular alterations during walking in persons with moderate knee osteoarthritis. J Electromyogr Kinesiol Off J Int Soc Electrophysiol Kinesiol. 2006;16(4):365–78.
[18] Hortobágyi T, Westerkamp L, Beam S, Moody J, Garry J, Holbert D, et al. Altered hamstring-quadriceps muscle balance in patients with knee osteoarthritis. Clin Biomech. 2005;20(1):97–104.
[19] Altman R, Asch E, Bloch D, Bole G, Borenstein D, Brandt K, et al. Development of criteria for the classification and reporting of osteoarthritis: Classification of osteoarthritis of the knee. Arthritis Rheum. 1986;29(8):1039–49.
[20] Hermens HJ, Freriks B, Merletti R, Stegeman D, Blok J, Rau G, et al. European recommendations for surface electromyography. Roessingh Res Dev. 1999;8(2):13–54.
[21] Shiavi R, Bugle HJ, Limbird T. Electromyographic gait assessment, Part 1: Adult EMG profiles and walking speed. J Rehabil Res Dev. 1987;24(2):13–23.
[22] Winter DA, Fuglevand AJ, Archer SE. Crosstalk in surface electromyography: Theoretical and practical estimates. J Electromyogr Kinesiol. 1994;4(1):15–26.

[23] Kendall FP, McCreary EK. Muscles: Testing and Function. In: 4th ed. Baltimore: Williams and Wilkins; 1993. pp. 265.
[24] Damiano DL, Martellotta TL, Sullivan DJ, Granata KP, Abel MF. Muscle force production and functional performance in spastic cerebral palsy: Relationship of cocontraction. Arch Phys Med Rehabil. 2000;81(7):895–900.
[25] Martin DF. Pathomechanics of knee osteoarthritis. Med Sci Sports Exerc. 1994;26(12):1429–34.
[26] Dwivedi SN, Kumar G. Prevalence and determinants of disability in the rural elderly population in northern India. [cited 2017 Jan 20]; Available from: http://iapmr.org/ijpmr/ijpmr0502/20050203.pdf.
[27] Mohamed O, Perry J, Hislop H. Relationship between wire EMG activity, muscle length, and torque of the hamstrings. Clin Biomech. 2002;17(8):569–79.

PARTICULARS OF CONTRIBUTORS:
1. Assistant Professor, Department of Physical Medicine and Rehabilitation, RUHS College of Medical Sciences, Jaipur, Rajasthan, India.
2. Professor, Department of Physical Medicine and Rehabilitation, All India Institute of Medical Sciences (AIIMS), New Delhi, India.
3. Professor, Department of Physical Medicine and Rehabilitation, All India Institute of Medical Sciences (AIIMS), New Delhi, India.
4. Professor, Department of Physical Medicine and Rehabilitation, All India Institute of Medical Sciences (AIIMS), New Delhi, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Sanjeev Kumar Sharma,
11,12 Roop Nagar, 2nd Mahesh Nagar, Tonk Phatak, Jaipur, Rajasthan, India.
E-mail: drsanjsharma@gmail.com

FINANCIAL OR OTHER COMPETING INTERESTS: None.

Date of Submission: **Jan 24, 2017**
Date of Peer Review: **Feb 16, 2017**
Date of Acceptance: **Mar 09, 2017**
Date of Publishing: **May 01, 2017**