

Journal homepage: http://www.journalijar.com

INTERNATIONAL JOURNAL OF ADVANCED RESEARCH

RESEARCH ARTICLE

EFFECTIVENESS OF RETROWALKING IN OSTEOARTHRITIS OF KNEE – A REVIEW ARTICLE

K.Hrishikesh Yadav, Sindhu Shashidharan.

Manuscript Info	Abstract
Manuscript History:	Osteoarthritis (OA) also known as degenerative arthritis or degenerative joint disease or osteoarthrosis, is a group of mechanical abnormalities involving degradation of joints, including articular cartilage and subchondral bone. Walking is one of the most common movements all performences, and there is the potential that a person's walking style may damage their cartilage and thus cause the initiation of OA. Patients with knee Osteoarthritis seems to develop their own gait pattern and try to unload the affected structures during gait. More over patients with less severe knee osteoarthritis develop a gait pattern that differs from patients with severe osteoarthritis and control group of patients. This prolonged usage of secondary gait compensation creates greater imbalances of muscle, progressively reduces muscle strength, endurance, flexibility and later ending to deformity. Retro walking has more advantageous effects compare to forward walking. It increases stride rate, decreases stride length and increases support time and reduces overall range of motion of knee thereby increase active functional range. Considering the advantageous effect of Retro Walking with respect to forward walking in decreasing the compressive load on knee and improving the muscular strength in functional range. Retro walking training to overcome pain and physical dysfunction in patients suffering from chronic OA knee.
Received: 19 December 2015 Final Accepted: 16 January 2016 Published Online: February 2016	
Key words:	
*Corresponding Author	
K.Hrishikesh Yadav.	
	Copy Right, IJAR, 2016,. All rights reserved.

Introduction:-

Osteoarthritis (OA) also known as degenerative arthritis or degenerative joint disease or osteoarthrosis, is a group of mechanical abnormalities involving degradation of joints, including articular cartilage and subchondral bone¹.

The word 'osteoarthritis' originated from the Greek word "osteo", meaning "of the bone", "arthro", meaning "joint", and "itis", meaning inflammation, although the "itis" of osteoarthritis is somewhat of a misnomer – inflammation is not a conspicuous feature which is present in rheumatoid or autoimmune types of arthritis. Osteoarthritis traditionally was considered as a disease of articular cartilage. Now it is thought to involve the entire joint tissues, synovium, capsule, bone and ligaments leading to subchondral bone attrition and remodelling, meniscal degeneration, ligamentous laxity, fat pad extrusion, and impairments of neuromuscular control ^{2.3}.

OA is the most common form of arthritis⁴. In addition, OA knee patients may also experience poor neuromuscular control, slower walking speed, decresed functional activity and walking. The prevalence of knee osteoarthritis is 22% - 32% in India; commonly females are more affected than males as the age progress⁵.

More than half of those with arthritis are under 65 years of age. OA knee increases with age (older than 50 years), especially in women. According to a survey, anywhere from 6% to over 13% of men, but between 7% and 19% of women, resulting in a 45% less risk of incidence in men^{6} .

Additional factors that increase the risk of developing OA of the knee include genetics and obesity^{7-10.} Genetic factors appear to influence risk of developing primary OA though they may influence disease differently in men and women. Twin studies suggest that generalised OA in women has a heritability rate of 39 to 65%, with a concordance

rate in monozygotic twins of 0.64. Other risk factors includes joint hypermobility or instability, specific occupations, or sports stress (e.g., with high impact loading with farming or soccer), peripheral neuropathy, injury to the joint, history of immobilisation, repetitive knee bending or heavy weight lifting, and strong family history. Other causes are lower extremity misalignment, torn meniscal pathology, bone marrow lesion shown by MRI, and quadriceps weakness.^{11,12}

Classification:-There are two forms of OA:

1. Primary OA:-

Primary osteoarthritis is a chronic degenerative disorder related to but not caused by aging, as there are people well into their nineties who have no clinical or functional signs of the disease. The pathophysiology of osteoarthritis involves a combination of mechanical, cellular, and biochemical processes. The interaction of these processes leads to changes in the composition and mechanical properties of the articular cartilage. Cartilage is composed of water, collagen, and proteoglycans. As a person ages, the water content of the cartilage decreases as a result of a reduced proteoglycan content, thus causing the cartilage to be less resilient. Without the protective effects of the proteoglycans, the collagen fibres of the cartilage can become susceptible to degradation and thus exacerbate the degeneration. Inflammation of the surrounding joint capsule can also occur, though often mild compared to what occurs in rheumatoid arthritis.¹³

This can happen as breakdown products from the cartilage are released into the synovial space, and the cells lining the joint attempt to remove them. New bone formation, called "spurs" or osteophytes, can form on the margins of the joints, possibly in an attempt to improve the congruence of the articular cartilage surfaces. These bone changes, together with the inflammation, can be both painful and disabling¹⁴.

2. Secondary OA :-

This type of OA is caused by other factors but the resulting pathology is the same as for primary OA, i.e.

- Congenital or developmental disorders of joints
- Mechanical: limb length discrepancy, malalignment, hyperlaxity, Ehlers-Danlos syndrome, Marfan's syndrome
- Inflammatory: rheumatologic diseases, i.e., rheumatoid arthritis, SLE, all chronic forms of arthritis
- **4** Traumatic: injury to joints or ligaments, postsurgical
- ↓ Infective: septic arthritis, Lyme disease
- Hetabolic: haemochromatosis and Wilson's disease, gout, calcium crystal deposition, alkaptonuria
- Endocrine: diabetes, acromegaly, hypothyroidism, obesity
- **Weuropathic arthopathy**
- 4 Miscellaneous like haemophilia, osteonecrosis.¹⁵



Etiology:-

Exact etiology is unknown and multiple factors interact to cause this disorder.

- Age: Although advance osteoarthritis may occur in many young people in early 20's, the frequency of condition escalates markedly in advancing years. Furthermore, older people are found to have rapid radiological progression of osteoarthritis¹⁶.
- Sex: The Framingham Knee Osteoarthritis study suggests that knee osteoarthritis increases in prevalence throughout the elderly years, more so in women than in men. Females are found to have more severe OA, more number of joints are involved, and have more symptoms and increased hand and knee OA¹⁷. These observations and others reporting a painful form of hand osteoarthritis after the menopause suggest that loss of estrogen at the time of menopause increases a woman's risk of getting osteoarthritis, however few contrary reports are pouring in ¹⁸.
- Obesity: Obesity preceeds rather than follow knee osteoarthritis and indeed weight loss prevents development of knee osteoarthritis¹⁹
- Genetic: Hip osteoarthritis has a significant genetic component.²⁰ Nodal generalised osteoarthritis is a polyarticular form of osteoarthritis characterized by Heberden's nodes occurring mainly in women of perimenopausal age. Heberden's nodes appear to be inherited independently as an autosomal dominant trait with greater penetrance in women.²¹
- Bone density: Negative association has been reported between osteoporosis and osteoarthritis at certain sites particularly the hip ²².
- Cigarette smoking: Protective influence of smoking on knee osteoarthritis has been reported from various studies including Framingham study²³.
- Local factors: Major direct injury particularly if resulting in a fracture of articular surface is considered a cause of osteoarthritis²⁴. Trauma in college years (mean age 22) increases subsequent prevalence of osteoarthritis in subjects in their 60's.²⁵
- Joint location: OA is more common in hip and knee joint but occur rarely in ankle. Alteration in chondrocyte responsiveness to different cytokines may be the reason eg. knee chondrocytes exhibit more IL-1 receptors than ankle chondrocytes and knee chondrocytes express mRNA for matrix MMP-8.²⁶
- Other: Chondrocalcinosis, crystals in joint fluid cartilage, prolonged immobilization, joint hypermobility or instability, peripheral neuropathy, prolonged occupational or sports stress are the important risk factors for the causation of OA.²⁷

Abnormal Gait in Osteoarthritis Knee:-

Walking is one of the most common movements all performences, and there is the potential that a person's walking style may damage their cartilage and thus cause the initiation of OA¹³. As the disease progresses it effect on the patients gait. Patients with knee Osteoarthritis seems to develop their own gait pattern and try to unload the affected structures during gait. More over patients with less severe knee osteoarthritis develop a gait pattern that differs from patients with severe osteoarthritis and control group of patients ²⁹.

Gait of a person has a major influence on their independence which will in turn be a great relief for patient's family in the day to day life. When a person is walking the body should bear weight, provide support for locomotion and maintain equilibrium to achieve that gait and the body alignment should set accordingly³⁰. As the mobility is important to the independence of the patient they strive to retain this ability even in the presence of severe impairment. Patient usually falls during walking. People may fall in the context of buckling ("giving way") of the knee. Subjects may also fall when they fail to regain balance after tripping over an obstacle³¹.

Biomechanical studies indicated individual with O.A. knee walk more slowly, with less knee excursion, increased adduction moment and with more joint stiffness. These secondary compensatory gait adaptation in O.A. knee patients helps in reducing pain by decreasing ground reaction loading on knee ³². This prolonged usage of secondary gait compensation creates greater imbalances of muscle, progressively reduces muscle strength, endurance, flexibility and later ending to deformity ^{33, 34}. Patients with OA knee tends to maintain an extended knee position at heel strike, thereby experiencing a rapid increase in ground reaction force and vertical loading rates. They make more rapid shift of the body weight from the contra lateral limb to the support limb which reduces medial and lateral distance between center of mass and knee joint center³⁵.

In chronic OA there is lack of hip abductor muscle strength, the contra lateral limb drops throughout the swing phase, leading to the movement of the trunk away from the support limb, similar to trendelenberg gait which results in increased medial lateral distance between the center of mass and knee joint center and greater varus alignment results in peak knee adduction moment³⁶. Also due to reduction of abductor moment in chronic OA, maintenance of pelvic level will alter. These changes contribute to increase in load on the medial compartment and thereby increase the risk of progression of disease.

Effect of Retro Walking in correcting Patho-mechanics of Osteoarthritis Knee:-

Retro walking walking has more advantageous effects compare to forward walking. During forward walking knee joint flexes, extends and then flexes in support phase, where as in retro walking walking knee initially extends, flexes and extends in support phase, prior to flexing and extending during swing. However Support swing ratio of backward walk is similar to forward walking with 60% support and 40% swing.

Retro walking walking increases stride rate, decreases stride length and increases support time. Back ward walking reduces overall range of motion of knee thereby increase active functional range. Muscular structure supporting ankle and knee reversed their role during retro walking. It provides the primary power producer and ankle plantar flexors shock absorber. Direction of knee joint shear force directed forward initially during retro walking where as backward in forward walking.

Some studies proved that retro walking produces significantly lower patellar compressive force than forward walking and Retro walking helps to reduce maximal vertical force and impulsive force on knee compare to forward forces because of Toe heel contact pattern³⁶.



From previous research works it stated numerous performance differences between retrowalking against forward walking. Increased stride rate and support time with decreased stride length is the hall mark of retrowalking. In this Toe heel contact pattern is seen and knee act as primary power producer and an ankle shock absorber ³⁷ and also Shear force is directed forward during retrowalking, where as backward in forward walking during the initial support phase there by decreases external compression load.

It also shown significant increase in hamstring stretch during each stride, E.M.G. activity shown greater in lower limb muscles in backward compared to forward walk. This study correlate the result where Extension Lag, VAS, could have improved because of retro walking effect in improving extensor muscle activation, gaining flexibility with reduce reaction and shear force directing on joint. Retro walking has shown to have many health benefits. Back ward walking is found to be beneficial in improving the body composition and cardio respiratory fitness than forward wlking on treadmill ³⁸.

Studies shown that retrowalking on treadmill of 15 degree slope increases higher firing rate of hamstring and quadriceps, where vastus medialis of quadriceps shown higher activation.

Retro walking increases vo2max these above changes correlate with result of present study where there was improvement in WOMAC INDEX, Extension lag, Dynamic balance occurred due to its greater impact in increasing extension moment, improvising strength in functional range with decline compression force assist in improving physical function.

Conclusion:-

Considering the advantageous effect of Retro Walking with respect to Forward walking in decreasing the compressive load on knee and improving the muscular strength in functional range. Retro walking training to overcome pain and physical dysfunction in patients suffering from chronic OA knee.

It is the concluded that implementation of retro walking as an exercise in rehabilitation of OA Knee patients can reduce excessive abnormal adduction torque on knee joint and also improve functional activities.

Reference:-

- 1) Di Cesare P, Abramson S, Samuels J. Pathogenesis of osteoarthritis. In: Firestein GS, Kelley WN, eds. *Kelley's Textbook of Rheumatology.8th ed.* Philadelphia, Pa.: Saunders Elsevier; 2009: 1525-40.
- 2) Di Cesare P, Abramson S, Samuels J. Pathogenesis of osteoarthritis. In: Firestein GS, Kelley WN, eds. *Kelley's Textbook of Rheumatology.8th ed.* Philadelphia, Pa.: Saunders Elsevier; 2009: 1525-40).
- 3) Sellam J, Berenbaum F. The role of synovitis in osteoarthritis. Nat Rev *Rheumatol* 2010; 6: 625-35.
- 4) Felson DT, Zhang Y. An update on the epidemiology of knee and hip osteoarthritis with a view to prevention. *Arthritis Rheum* 1998; 41: 1343-55.
- 5) A Majan, S Varma, V Tandon etal .osteoarthritis. J,Ap 2005: 53.
- 6) Kraus VB. Pathogenesis and treatment of osteoarthritis. *Med Clin North Am* 1997; 81: 85-112.
- 7) Loughlin J. Genetic epidemiology of primary osteoarthritis. Curr Opin Rheumatol 2001.
- 8) Sinusas K. Osteoarthritis: diagnosis and treatment. *Am Fam Physician* 2012; 85(1): 49-56.
- 9) Lee R, Kean WF. Obesity and knee osteoarthritis. *Inflammopharmacology* 2012; 20(2): 53-8.
- 10) Sridhar MS, Jarrett CD, Xerogeanes JW, Labib SA. *Obesity and symptomatic osteoarthritis of the knee*. J Bone Joint Surg Br 2012; 94(4): 433-40.
- 11) Blagojevic M, Jinks C, Jeffery A, Jordan KP. Risk factors for onset of osteoarthritis of the knee in older adults: a systematic review and meta-analysis. *Osteoarthritis Cartilage 2010*; 18(1): 24-33.
- 12) Cooper C, Snow S, McAlindon TE et al. Risk factors for the incidence and progression of radiographic knee osteoarthritis. *Arthritis Rheum* 2000; 43(5): 995-1000.
- 13) Hinton R, Moody RL, Davis AW, Thomas SF. Osteoarthritis: diagnosis and therapeutic considerations. *Am Fam Physician* 2002; 65(5): 841-8.
- 14) Hinton R, Moody RL, Davis AW, Thomas SF. Osteoarthritis: diagnosis and therapeutic considerations. *Am Fam Physician* 2002; 65(5): 841-8.
- 15) Ruddy S, Harris ED, Sledge CB, Kelley WN. Kelly's Textbook of rheumatology. 6th ed. Philadelphia: Saunders, 2001; 1410.
- 16) Martin JA, Buckwalter JA. Aging, articular cartilage chondrocyte senescence and osteoarthritis. *Biogerontolog* 2002;3:257-64.)
- 17) Parazzini F; Progretto Menopausa Italia Study Group. Menopausal status, hormone replacement therapy use and risk of self-reported physician-diagnosed osteoarthritis in women attending menopause clinics in Italy. *Maturitas* 2003 20;46:207-12
- 18) Zhang Y, McAlindon TE, Hannan MT, et al. Estrogen replacement therapy and worsening of radiographic knee osteoarthritis: the Framingham Study. *Arthritis Rheum* 1998;41:1867-73

- 19) Felson DT, Ahange Y, Anthony JM, et al. Weight loss reduces the risk for symptomatic knee osteoarthritis in women. *Annals of Internal Medicine* 1992; 116:535-9.
- 20) Lanyon P, Muir K, Doherty S, Doherty M. Assessment of a genetic contribution to osteoarthritis of the hip: sibling study. *British Medical Journal* 2000; 321:1179-83.
- 21) Doherty M, Jones A, Cawston T. Osteoarthritis. In: *Oxford Textbook of Rheumatology, 3rd Ed.* (Eds: Isenberg, D.A. et al.), Oxford University Press, 2004; pp. 1091-1118.
- 22) Felson DT. Epidemiology of hip and knee osteoarthritis. Epidemiologic Reviews 1988; 10:1-28.
- 23) Wright V. Post-traumatic osteoarthritis a medico-legal minefield. British Journal of Rheumatology 1990; 29:474-8.
- 24) Gelber AC, Hochberg MC, Mead LA, et al. Joint injury in young adults and risk for subsequent knee and hip osteoarthritis. *Annals of Internal Medicine* 2000; 133:321-8.
- 25) Dicesare PE, Abramson SB. Pathogenesis of osteoarthritis. In :Harris ED, Budd RC, Genovese MC et al (editors). *Kelley's Textbook of Rheumatology, volume II, 7th edition*, Elsevier Saunders. 2005. pp.1493-1513.
- 26) Ruddy S, Harris ED, Sledge CB, Kent NN. *Kelly's Textbook of Rheumatology, 6th Ed.*, Philadelphia. WB Saunders, 2001; pp. 1410.
- 27) Mundermann A, Dyrby CO, Andriacchi TP. Secondary Gait Changes in Patients with Medial Compartment Knee Osteoarthritis, Arthritis & rheumatism 2005; 52(9): 2835–2844.
- 28) Whittle M.W. Gait analysis an introduction. (3rd edition).*Butterworth heinemann* 2006; 150.
- 29) Pijnapppels M, Van der Burg PJ, Reeves ND, Van Dieën JH. Identification of elderly fallers by muscle strength measures. *Eur J Appl Physiol*. 2008; 102:585-92.
- Mundermann, A., Dyrby, C.O. and Andriacchi, T.P., Secondary Gait Changes in Patients with Medial Compartment Knee Osteoarthritis; Increased Load on Ankle, Knee, Hip During Walking, Arthritis Rheum :2005. 52; 2835-2844.
- 31) Huang Effects of Severity of Degeneration of Gait Pattern in Patients with Knee Osteoarthritis of Knee In Press 2008.
- 32) Kaufman KR, Hughes.C, Morrey.B.F, et al Gait Characteristic of Patients with Knee Osteoarthritis journal of biomechanics, 2001 34 ;907-915.
- 33) Annegret Mundermann, Chris O Dyrby and Thomas. P. Andriacchi, September 2005-Secondary gait changes in patients with medial compartment knee osteo arthritis.
- 34) Flyns T.W., Connery. S.M., Smutok, M.A, Zeballos, R.J. & Weisman I.M. comparison of cardiopulmonary responses to forward and backward walking and running , 1994; Medicine *and science in sports and exercise*, 1994 26; 89-94.
- 35) Mundermann, A., Dyrby, C.O. and Andriacchi, T.P., Secondary Gait Changes in Patients with Medial Compartment Knee Osteoarthritis; Increased Load on Ankle, Knee, Hip During Walking Arthritis; *Rheum*, 2005, 52; 2835-284.
- 36) Arata A.W., *Kinematic And Kinetic Evaluation Of High Speed Backward Running* university of oregon microform publication 1999.
- Ashwini Dangi, Utkarsha Nirbhavane., International Journal of Scientific and Research Publications, Volume 4, Issue 4, April 2014 1 ISSN 2250-3153