Physical Medicine and Rehabilitation - International

Open Access

Austin Publishing Group

Review Article

Knee Osteoarthritis: A Review of Literature

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Received: October 24, 2014; **Accepted:** November 10, 2014; **Published:** November 13, 2014

Abstract

Osteoarthritis (OA) is accepted as a major public health problem. It is one of the major causes of impaired function that reduces quality of life (QOL) worldwide. OA is a very common disorder affecting the joint cartilage. As there is no cure for OA, treatments currently focus on management of symptoms. Pain relief, improved joint function, and joint stability are the main goals of therapy. The muscle weakness and muscle atrophy contribute to the disease process. So, rehabilitation and physiotherapy were often prescribed with the intention to alleviate pain and increase mobility. However, as exercise has to be performed on a regular basis in order to counteract muscle atrophy, continuous exercise programs is recommended in people with degenerative joint disease. Therapeutic exercise regimes either focus on muscle strengthening and stretching exercises or on aerobic activity which can be land or water based. This article presents on overview of the current knowledge on OA and focuses on biomechanics, etiology, diagnosis and treatment strategies, conservative treatment including the physical therapy management are discussed. This information should assist health care practioners who treat patients with this disorder.

Keywords: OA; Knee pain; Strengthening exercises; Stretching exercises; Pain severity; Hamstrings / quadriceps ratio

Introduction

Osteoarthritis (OA) is a common chronic condition resulting in pain, fatigue, functional limitations, increased healthcare utilization and high economic costs to society [1]. The burden of OA is projected to increase, due in part to obesity and population aging [2]. While the prevalence of OA increases with age [3], there is a growing recognition that OA affects people at younger ages. Recent US data demonstrated that half of people with symptomatic knee OA are diagnosed by age 55 [4].

Quadriceps strength deficits have been reported in 20%–70% of patients with knee OA. Any improvement in muscle strength or peak power of the lower extremities with decreased levels of particular pain may be important and is a strong predictor of functional ability [5]. As lower limb musculature is the natural brace for the knee joint, potentially important muscle dysfunction may arise from either quadriceps weakness or relative weakness of the hamstrings in comparison to the quadriceps, usually assessed as the hamstrings: quadriceps (H:Q) ratio. An H:Q ratio of greater than or equal to 0.6 is considered to be normal [6]. Thus, evaluation of muscle dysfunction in relation to the knee joint should examine both quadriceps strength as well as the balance of muscle strength [6].

The etiology of OA is related to repetitive mechanical loads and aging. Recent studies have separated the etiological factors into three main sub-groups: sex, anatomy, and body mass. The clinical manifestations are joint pain, stiffness; decreased range of joint movement, muscle weakness of the quadriceps and alterations in proprioception [7]. Decreased strength in the muscle groups involving the joints is significant because it causes progressive loss of function. These symptoms significantly restrict the individual's ability to get up from a chair, walk, or climb stairs [3]. Walking with a limp, poor alignment of the limb, and instabilities can also be observed in individuals with OA. During movements, crepitating can be heard because of arthritis of the irregular joint surfaces [2].

Clinical knee OA usually is managed in primary care [8] with analgesics and non pharmacological options, such as exercise [6]. Exercise has been shown to improve function, strength, walking speed, and self-efficacy and to reduce pain and the risk of other chronic conditions [9, 10]. Also, prevent or retard progression of the disease using physical and occupational therapy and exercise programs [3].

Plain radiographs are commonly used to classify OA subjects for the purposes of clinical studies and joint space narrowing is often used as a measure of disease progression. Although plain radiography is at present, the "gold standard" for evaluation of OA progression, it is brimful with problems related to the accurate reproduction of measurements of joint space width, especially in subjects who have knee OA [11].

A self-reported physical disability or assessment questionnaire most commonly used to assess the outcome of exercises for OA. The most popular specific questionnaire for knee OA is the Western Ontario and McMaster Universities OA index questionnaire (WOMAC) which reports pain, stiffness and daily function difficulties experienced by OA subjects [12].

Several muscle groups support the knee. The two main muscle groups that control knee movement and stability are the quadriceps and the hamstrings. The quadriceps and hamstrings muscles have the potential to provide dynamic frontal-plane knee stability because of their abduction and/or adduction moment arms [13]. Using a neuromuscular biomechanical model, the quadriceps and hamstrings not only have the potential to support frontal-plane moments but also actually do provide support to abduction-adduction moments [14].

Citation: Hafez AR, Alenazi AM, Kachanathu SJ, Alroumi AM and Mohamed ES. Knee Osteoarthritis: A Review of Literature. Phys Med Rehabil Int. 2014;1(5): 8.

In the frontal plane, balanced co contraction of the quadriceps and hamstrings leads to increased joint compression, which should assist in knee joint stabilization [15]. The diminished co activation between the quadriceps and hamstrings in women may contribute to greater knee joint instability in women than in men.

The strength relationship between the quadriceps femories and hamstring muscle have been measured and reported by various researchers [16, 17].That reported the isokinetic Q/H ratio for apparently healthy subject to be 1.70:1 and 1.37:1 at 60/sec and 180/sec angular velocities of limb movement respectively. The mean isometric Q/H ratio was hence found to be 1.43, a value below ratio reported for young healthy adult whose isometric Q/H ratio was 2:1 so the effect of Q/H ratio are different in OA due to relatively greater weakness in the quadriceps femories muscle [18].

Epidemiology

Osteoarthritis (OA) of the knee is the most common form of joint disease and prevalence of both radio graphically evident and symptomatic. The females having higher prevalence than males (11.4% vs 6.8%) [19]. The gender difference in prevalence has recently been emphasized in a meta-analyses, which provides evidence for a greater risk in females for prevalent and incident knee OA [20]. The meta-analysis also reported that females tend to have more severe knee OA radio graphically assessed than males and that the gender differences increase with age > 55 years. The prevalence of OA will increase as the population of the kingdom ages, especially if the incidence of obesity remains at over 50% in the 45+ age group [21].

Etiology & Pathogenesis

Modern imaging approaches recognize that OA is a whole joint disease which may involve multiple tissues which confer different phenotypes; subchondral bone in particular is integral to the pathogenesis and progression of OA. In particular, the area of subchondral bone at the femorotibial articulation is larger in OA knees than healthy controls and correlates with knee joint space narrowing, osteophytes [22].

Pathogenetically, knee OA is characterized by structural changes in and around the knee joint. The predominant structural changes are the loss of cartilage and the formation of osteophytes. These changes are easily demonstrated radio graphically, and objective measures of disease severity are based on the amount of joint space loss (a reflection of cartilage loss) and the presence of osteophytes [23]. Furthermore, the subchondral bone scleroses in the early phases of OA and this process, possibly involving micro fractures has been suggested to be pathogenetic factors in the process of cartilage degeneration [24]. In addition to these structural "hard tissue" changes, a number of changes in articular and periarticular soft-tissue occur with knee OA. These include synovial hyperplasia [25] and joint effusions [26]. Although knee OA is not classified as an inflammatory disease, a common sign of knee OA is synovial inflammation, detected using Ultrasonographic [25, 27]. In addition magnetic resonance imaging as well as orthoscopical inspection of the knee joint has also provided insights to the presence of inflammation in knee OA [28].

Pathophysiology

OA is viewed as a metabolically active, dynamic process, including both cartilage destruction and repair. These processes may be initiated by several biochemical and mechanical insults [29, 30]. The first OA change occurring in articular cartilage include a decrease in the superficial proteoglycan content, deterioration of superficial collagen fibrils, and an increase in the water content.

The loss of proteoglycans and collagen results in diminished cartilage stiffness [31]. Subsequently, the chondrocytes increase the synthesis of cartilage matrix proteins, the destruction of components in the extracellular matrix accelerates, and the thickness of cartilage may even increase. At the same time, calcified cartilage and subchondral bone become thicker in a response to the increased formation and resorption of the subchondral bone [32].

Ultimately, the concentration of proteoglycans decreases and collagen fibrillation declines due to diminished repair capabilities of chondrocytes. This process leads to splits of the cartilage extending down to bone. The degenerated cartilage with the disrupted collagen network cannot regenerate, and this pushes the OA tissue to the point of no return [32]. On the other hand [33], postulated that the repetitive impulsive loading my first induce trabecular micro fractures in the subchondral bone. According to this theory, subsequent remodeling increases the stiffness and thickness of the subchondral bone in an attempt to dampen impact forces. As a consequence, the overlying cartilage may become overloaded and break down resulting in cartilage degeneration and loss.

Risk Factors

Knee OA is a multi factorial disease. The cause of OA remains unknown, thought there is clear evidence for major risk factors, such as age, obesity, joint trauma, and heavy work load [34]. The risk factors can be divided into systemic (for e.g. age, gender, genetics, and overweight) and local biomechanical factors, such as joint injury and malalignment, overweight, and muscle weakness [35]. Abnormal mechanical loading in various sport activities or during heavy work may activate the biochemical cascade that leads to joint degeneration and pain, but also even in normal mechanical loading if the cartilage is impaired [35].

Aging is the most significant risk factor for knee OA [36]. Knee OA is more common in obese subject than in subjects of normal weight [37]. For example, obese women with body mass index (BMI) of 30-35kg/m^2 had a four times higher risk for knee OA than non-obese women [38]. The corresponding was 4.8 for men. Obesity is also a major risk factor for the incidence of bilateral knee OA, whereas local mechanical factors are more often associated with unilateral OA [38]. The effect of obesity on OA has been thought to be mediated through the increased mechanical loading of the knee and hip. This would lead in cartilage damage in these weight-bearing joints. However, obesity is also associated with hand OA, which has given rise to the hypothesis that both mechanical and metabolic factors may mediate the effects of obesity on joints [39].

Joint injury increases the risk for knee OA [40]. After knee injury, women had a three-fold and men a 5 to 6 fold risk for developing of knee OA, compared to healthy controls. Injuries to the anterior cruciate ligament associate most clearly with the incidence of knee OA (15-20%). As many as 50-70% of patients with complete anterior cruciate ligament rupture, accompanied by concomitant injuries

to the meniscus or other ligaments, exhibit radiographic knee OA changes after 15-20 years [41]. Furthermore, at 10 to 20 years after anterior cruciate ligament or menisci injury, on average, half of those patients have symptomatic knee OA [42]. Total meniscectomy after an isolated meniscus tear has been a significant risk factor for knee OA, the relative risk being 14.0 after 21 years [43]. Partial meniscectomy can also contribute to the development of knee OA [44].

Heavy physical activity and occupational load are important risk factors for the incidence of knee OA [45]. Heavy physical activity may increase the risk of especially among obese individuals [46]. On the other hand, regular and moderate physical exercise has been shown to be associated with a decrease in the development of knee OA [47]. However, most of the clinical or epidemiological studies have concluded that jogging exercise at moderate intensity or recreational physical activity do not increase the risk for knee or hip OA, provided that the weight-bearing joints have not been injured [48]. The increased risk for knee OA is also associated with those occupations that entail prolonged or repeated knee bending. The risk may be even higher in those activities containing both knee bending and mechanical loading [49].

With respect to the other mechanical risk factors, knee malalignment has been reported to be associated with the development and progression of knee OA [50, 51]. Furthermore, the severity of malalignment can predict the decline in physical function Genetic factors also seem to account for the existence of knee OA to a degree ranging from 39-65% independently of the known environmental or demographic confounders [52]. This suggests that the articular cartilage of some individuals is congenitally vulnerable to mechanical wear and tear. However, the prevention of OA is still a challenging task although there is a considerable body of evidence about the definite causal risk factors, such as obesity, joint injury and occupational load.

Diagnosis

Pain is the predominant symptom of knee OA with the pain being generally related to joint use and with relief at rest. As OA progresses, pain may become more persistent and can appear also at rest and during the night. For a patient with symptoms, the inability to have restorative sleep may reduce the pain threshold via associated fatigue and reduced well-being [53]. The extent of the pain is usually linked to the severity of radiographic OA changes, but not necessarily [54]. Subjective pain correlates strongly with the psychological status, such as depression and anxiety [55]. In clinical practice, the subjective pain can be estimated with a visual analogue scale (VAS) from 0 t0 10 mm [56].

The mechanism of pain production in OA is not clear. The disease process may affect all intra capsular and periarticular tissues of the synovial joint loading to many possible sources of pain. The articular cartilage is aneural and a vascular tissue. However, it has a rich sensory innervations exists in other joint tissues [53]. It has been suggested that several processes in bone or/and subchondral bone such as elevated intraosseus pressure, bone marrow oedema, structural changes, and periosteal stretching may associate with the joint pain [57]. On other hand, the capsular mechanoreceptors may be stimulated by intra-articular hypertension, and the ischemia caused by mild synovitis may activate nociceptors. One factor is muscle weakness e.g. the weakness in the QFM has been shown to be a better determinant of pain and disability than any radiographic feature in knee OA [58]. Thus, in OA of large joints the periarticular structures interfere often with the pain [53].

Another typical feature in knee OA is short-lived morning stiffness, which is distinct from the more prolonged and often generalized joint stiffness characteristic of rheumatoid arthritis. The early morning stiffness, occasionally severe is believed to be related to inflammation. Patients with knee OA describe stiffness as a difficulty to rise from a chair, slowness of movements, or clumsiness later in the day [59].

Knee OA is the greatest contributor to impairment in functional ability of OA patients. The disability can be extensive containing mobility limitation, difficulty to cope with activities of daily living and social isolation. The principal contributors to disability are believed to include pain, reduce range of joint movement as well as muscle weakness [53, 60]. Recently, some studies suggested that in end- stage knee OA, the major attributes of self-reported disability was pain, obesity and antero-posterior (A-P) laxity of the knee joint. The effect of comorbidities on health-related quality of life was also considerable [61]. Subjective physical functioning could be qualitatively estimated using the Western Ontario and McMaster Osteoarthritis Index (WOMAC) and Lequesne questionnaires [62].

Clinical Findings

There are several signs in knee OA that can be identified during the clinical inspection. These include limping due to joint pain, decreased walking speed as well as reduced stride length and frequency [63]. Squatting may have become difficult for a patient suffering from knee OA. The deformity of the knee joint is usually a sign for advanced knee OA. Clinically detectable varus or valgus instability in the knee joint is regarded as a late sign of the disease. Coarse crepitus is considered to indicate the loss of congruency of the joint [53].

Tenderness can be identified with palpation of the knee joint. Tenderness along the joint-line points to an intra capsular origin for pain and point-tenderness away from the joint-line is indicative of a periarticular lesion. Reduced range of movement (ROM) easily measured with a goniometer is associated with physical impairment. The decreased ROM is mainly caused by osteophytes formation, remodeling, capsular thickening and can be accentuated by soft tissue swallowing. Muscle wasting and weakness are difficult to examine but can be present in knee OA. The classical signs of inflammation, such as heat, pain and effusion indicate synovitis in knee OA. Laboratory tests do not play any role in the diagnosis of knee OA but they can help in the differential diagnosis [53].

Laboratory Findings

A typical clinical presentation of OA does not require laboratory testing. The erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) are usually normal. The full blood count is normal. Rheumatoid factor and antinuclear antibodies are usually negative. Note that these antibodies may be low positive and of no significance in elderly patients and in some chronic condition. Synovial fluid analysis usually indicates a low white cell count <2000ul. Laboratory

investigations should only be performed if secondary causes of OA are clinically suspected [64].

Radiological Findings

The plain radiograph serves as the primary investigation in the diagnosis of knee OA, as well as in assessing the severity of the disease. The advantages of radiography are evident: it is cost-effective and relatively safe and its availability is excellent. However, the subjective pain and radiographic changes do not necessarily correlate with each other [54].

Typical radiographic features in knee OA include joint space narrowing, osteophytes, subchondral bone sclerosis, cyst formation, osteochondral bodies and bone deformity. Loss of cartilage is an early and cardinal feature of OA leading to joint space narrowing in plain radiographs [65]. The thickness of articular cartilage varies between individuals and joint surfaces [66]. Therefore, no reference values for thickness of joint space exist. The osteophytes are a hallmark of OA, these being formed at joint margins by endochondral ossification. They can be regarded as a repair attempt and indicate redistribution of abnormal joint loading. Cysts are also typical radiographic findings in OA and occur mainly within the areas of bony sclerosis at sites of increased pressure transmission. Disintegration of the joint surface in OA results in the formation of osteochondral fragments. As these fragments are released into the joint space, they appear characteristically with the other established features of OA [65].

Management

The current management of OA, which includes both non-pharmacologic and pharmacologic modalities, is primarily directed toward pain control and reduction in functional limitation.

Non-pharmacological therapy

Education: Patient education is an ongoing, integral part of management. The practitioner should address aspects of the disease process, benefits and risks of treatment options. Empowering the patient, by involving them in shared decision making and providing them with positive skills directed at lifestyle changes, goes a long way to ensure treatment adherence [64].

Reduction of adverse mechanical factors: Obesity is a risk factor for the development and progression of OA in the knee and hip. It remains one of the strongest modifiable risk factors for OA and weight reduction is an effective primary and secondary disease prevention strategy. Weight loss improves pain and function, particularly for knee OA and to a lesser extent, hip OA. It should be achieved by a combination of correct dietary habits (eat correctly, regularly and less) and exercise. Many patients with OA share other chronic cardiac and metabolic disease and the benefits of weight loss are substantial.

All patients with lower limb OA should be advised about appropriate footwear. A shoe with soft thick soles and no raised heel is recommended. Lateral or medial wedged insoles can be used to reduce pain and improve function in patients with medial or lateral tibiofemoral OA, respectively [64].

Assistive devices: The use of a cane, frame or wheeled walker in patients with hip or knee OA reduces mechanical loading and pain. Patient should be educated on the proper use of canes. The cane or crutch should be held in the hand contralateral to, and moved together with, the affected limb. The total length of the cane should be equal to the distance between the upper border of the greater trochanter of the femur and the bottom of the heel of the shoe. This should result in elbow flexion of about 20°.

Knee braces can be used in patients with OA and mild-to-moderate varus or valgus malalignment. Overuse and unnecessary use of braces my worsen joint instability by contributing to muscle atrophy. They should only be used when there is a flare of inflammation, to protect the joint during unusual activity and when all other treatment modalities have failed [64].

Ice and Heat

The periodic application of superficial heat or cold is a relatively safe and low cost treatment that can be recommended in isolation or in combination with other treatments for patient with knee OA [67].

Ice is essential during the acute phase of pain and is also useful after exercising. Ice therapy causes capillary vasoconstriction with decreased blood flow and decreased metabolic activity. The resulting decreased inflammatory edema which help in preventing further damage and will provide analgesia [68]. It is the safest anti-inflammatory "medication" but its successful use requires discipline. Applying ice for 10-20 minutes after activity is reasonable [69].

Heat application causes increased capillary blood pressure and increased cellular permeability, with resultant increased swelling and edema. Heat should thus be used after the initial swelling and edema phases have stabilized. The effect of heat is to increase blood flow and local metabolic activity with relaxation of muscle spasm [68].

Manual Therapy

Taping

Taping the knee, in particular the patella is a physiotherapy treatment strategy recommended in the management of knee OA by some clinical guidelines [70]. Knee taping involves the application of adhesive rigid strapping tape to the patella and/or associated soft tissue structures. The mechanism by which taping reduces pain is not clear, but my include changes in patellar alignment [71] and enhanced function and activation of muscles [72].

Electrotherapy

Transcutaneous electrical nerve stimulation (TENS) is recommended in most guideline as safe adjunctive modalities for pain relief. Although, acupuncture may provide relief to some patients, there is less universal support for its use [64].

Therapeutic Exercises

In recent years, there have been numerous studies that have demonstrated the effectiveness of exercise and physical activity for individuals with knee osteoarthritis (OA) [73]. Although exercise and physical activity programs have been found to be beneficial the overall effects of this intervention have been found to yield small to moderate effects at best for individuals with knee OA [74]. For example a systematic review of the effectiveness of exercise for reducing pain and improving disability.

Therapeutic exercise is a form of physical activity that is provided

under the supervision of appropriate health professional for specific treatment goals. Regular physical activity is associated with lower mortality rates for both older and younger adults. Moreover, it is associated with a decreasing risk for a wide range of disease and conditions, such as cardiovascular disease, osteoporosis, falling, cancer, diabetes, blood pressure and osteoarthritis.

The main reasons for prescribing exercise in general include (1) achieving therapeutic goals, (2) improving general health and reducing secondary disability, and (3) modifying possible risk factors in disease progression [75]. Minor summarized the potential benefits of physical activity and exercise on OA as follows:

1. Minimizes or slows the pathological process that takes place in the OA joint. Exercise increasing cartilage nutrition and remodeling, increases the synovial blood flow, decreases swelling, and improves muscle strength. Thus, the pathological effect of exercise may include slowing the cartilage degeneration process, decreasing bone stiffness, decreasing joint effusion and improving muscle strength.

2. Decreases impairments that occur from OA by reducing the main impairment factors. Exercise helps in decreasing pain, improving strength and endurance, and improving range of motion and connective tissue elasticity.

3. Decreases functional limitation by improving walking speed, gait and physical activity and decreasing depression and anxiety.

Because muscle weakness plays such an important role in development of OA, it is increasingly evident that exercise plays a critical role in the management of the condition. Although, activity avoidance by knee osteoarthritis patients is common, exercise is an effective non-pharmacological treatment for knee OA [76]. The American College of Rheumatology (ACR) has approved regular exercise as a therapeutic approach for the management of knee OA [77]. Systematic reviews of non-pharmacological interventions have documented the effectiveness of exercise in reducing pain and disability [74]. Evidence suggests that stretching, strengthening and aerobic exercise decrease pain and improve muscular strength, functional ability and psychological well-being [78]. Exercise increases muscle endurance, improves proprioceptive acuity and decrease arthrogenic muscle inhibition of the quadriceps [79].

Quadriceps weakness is one of the most common and disabling impairments seen in individuals with knee osteoarthritis (OA) [80]. Sufficient quadriceps and hamstrings strength, both isometric and dynamic, is essential for undertaking basic activities of daily living such as standing and walking [81]. Muscle strength testing has revealed that those with knee OA have a 25% to 45% loss of knee extension strength [82] and a 19% to 25% loss of knee flexion strength [83], compared with similarly aged controls. There are 3 factors thought to contribute to knee extension and flexion weakness in those with knee OA: muscle atrophy, failure of voluntary muscle activity, and apparent weakness from increased antagonist muscle co-contraction [84].

Strengthening

Strengthening exercise is commonly recommended. Patients with knee OA tend to have reduced muscle strength as a consequence of reductions in physical activity and pain inhibition [85]. The quadriceps are the largest group of muscles crossing the knee joint and have the greatest potential to generate and absorb forces at the knee. Many clinical studies have shown consistent improvements in knee extension strength after training, as well as reductions in pain and physical disability in people with knee OA [86].

Strengthening the hamstring muscle has been found to enhance the functional ability of the deficient knee [87]. This is probably due to the fact that, which an overall increase in both the hamstring and quadriceps strength, and increase in the hamstring to quadriceps ratio (H:Q), anterior-lateral subluxation of the tibia may be minimized [83].

Stretching

Stretching should be carried out in conjunction with strengthening exercises. If a specific muscle group is restricted, more emphasis may be placed on these areas but there must be stretching of all the major muscle groups of the lower limb, because they all have an effect on the biomechanics of the knee. Patients should be instructed to hold a stretch for 20-30 seconds for it to be effective [58].

Stretching includes the quadriceps, hamstring muscles, iliotibial band (ITB), and Achilles tendon [88]. Tightness of the ITB can affect normal patella excursion. The distal ITB fibers blend with the superficial and deep fibers of the lateral retinaculum, and tightness in the ITB can contribute to lateral patellar tilt and excessive pressure on the lateral patella. Because the ITB is a very dense and fibrous tissue, the effectiveness of stretching is questionable [89].

A recent Cochrane review identified 32 trials investigating a variety of land-based therapeutic exercise program [86]. Results of a meta-analysis showed mean treatment benefits for both knee pain and physical function. Although there is less robust research into the effects of aquatic exercise, a small to moderate effect on function and a small to moderate effect on quality of life have been reported in another relatively recent Cochrane review [90]. Typical physiological changes as a result of an effective exercise regime may include improvements in muscle strength, neuromuscular control and range of motion, joint stability and fitness [91].

Non-steroidal anti-inflammatory drug

The purposes of symptomatic treatment of OA of the knee are to control joint pain and to improve joint function. The well-known pharmacological approach for symptomatic treatment includes oral administration of paracetamol, NSAIDs, opioids, and intra-articular corticosteroid injections. Although paracetamol should be prescribed as the preferred oral analgesic, it has been reported that the majority of patients with OA would prefer NSAIDs to paracetamol [92].

Surgery

Referral for joint replacement surgery should be considered in patients who experience persistent pain and reduced function that are refractory to non-surgical therapies, and which impact markedly on their quality of life [93, 94]. Total or partial joint arthroplasty surgeries are highly invasive procedures, requiring surgical resection of all or parts of the joint and insertion of prosthesis. Many patients who undergo total knee arthroplasty (TKA) experience improved function and decreased symptoms, many others continue to have some degree of ongoing pain. A recent investigation of post-TKA

symptoms reported chronic pain in 88% of patients who have had the surgery [92]. Current evidence does not support arthroscopic lavage and/or debridement as part of unselected knee OA treatment [95, 96]. Several surgical options are available for severe thumb base OA when conservative therapies have failed [97].

Conclusion

Knee osteoarthritis (OA) is a major public health concern worldwide and one of the foremost causes of chronic disability in older adults. Preventive care is dependent upon identification of risk factors for development of incident knee OA. The symptoms are often associated with significant functional impairment, as well as signs and symptoms of inflammation, including pain, stiffness and loss of mobility. Conservative treatment has documented the effectiveness of exercise in reducing pain and disability. Evidence suggests that stretching, and strengthening exercise decrease pain and improve muscular strength, functional ability and psychological well-being. Exercise increases muscle endurance, improves proprioceptive acuity and decrease arthrogenic muscle inhibition of the quadriceps.

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Phys Med Rehabil Int - Volume 1 Issue 5 - 2014
ISSN : 2471-0377 | www.austinpublishinggroup.com

Citation: Hafez AR, Alenazi AM, Kachanathu SJ, Alroumi AM and Mohamed ES. Knee Osteoarthritis: A Review of Literature. Phys Med Rehabil Int. 2014;1(5): 8.