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Knee osteoarthritis Disease: A review

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Abstract

Osteoarthritis of the knee is a complex peripheral joint disorder with multiple risk factors. The molecular basis of osteoarthritis has been generally accepted; however, the exact pathogenesis is still not known. Management of patients with osteoarthritis involves a comprehensive history, thorough physical examination and appropriate radiological investigation. The relative slow progress in the disease allows a stepwise algorithmic approach in treatment. Non-surgical treatment involves patient education, lifestyle modification and the use of orthotic devises. These can be achieved in the community. Surgical options include joint sparing procedures such as arthroscopyando osteotomy or joint-replacing procedures. Joint-replacing procedures can be isolated to a single compartment such as patellofemoral arthroplasty or unicompartmental knee replacement or total knee arthroplasty. The key to a successful long-term outcome is optimal patient selection, preoperative counselling and good surgical technique.

Keywords: Osteoarthritis; Knee joint; Total knee replacement; Cartilage

1. Introduction

Osteoarthritis (OA) is a shared form of degenerative joint disease distressing the western population. The knee is the principal peripheral joint affected subsequent in progressive loss of function, pain and stiffness [1]. It is estimated that approximately a tenth of the population aged over 50 years will be affected [2].

2. Etiology

OA is a complex disorder through multiple risk factors. These include both generalized constitutional factors [age, female sex, obesity, family history] and local adverse mechanical factors [trauma, occupational and recreational wear, malalignment, generalized laxity] [3,4]. There is a significant genetic component to the prevalence of knee OA, but the exact gene responsible is unknown. Classic twin studies have revealed that the influence of genetic factors is between 39% and 65%

3. Molecular basis of OA

Although the causes of OA are not entirely understood, biochemical changes in the subchondral bone, articular cartilage and synovial membrane are important in its process, a traumatic event or an originally defective matrix; the second view attributes OA to net loss of cartilage matrix due to failure of chondrocyte balancing synthesis and degradation; and the third hypothesis implicating extracartilagnious factors such as vascular changes, bony remodeling, microfractures and synovial changes as primary initiators with cartilage damage as a secondary affect. In OA, there is increased degradation of type II collagen fibrils. Collagenases only cleave within the type II collagen triple helix and not within

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types IX or XI collagen, which is most likely, degraded by proteolysis [7]. Aggrecans diminish in parallel to the severity of OA, as there is a net loss of matrix due to the inability of chondrocytes to compensate for loss of proteoglycan. This increased loss is due to the action of matrix metalloprotease [MMP] and aggrecanases. Although new aggrecan is synthesized to help with cartilage repair in the early stages of OA, it is lost to synovial fluid at later stages [8]. The composition of newly synthesized aggrecan may be different to the original, particularly in the content of keratan sulfate and chondroitin sulfate [9]. All these changes hamper the ability of cartilage repair. As a result of this altered extracellular matrix network, the water content increases. These in turn decrease the modulus of elasticity. The formation and accumulation of advanced glycation end products [AGEs] is one of the molecular events that has been suggested to be responsible for predisposing to the development of cartilage damage in OA [10]. There is increasing interest in defining the role of inflammation in OA, which is often associated with low-grade synovitis [11]. Symptoms and progression of cartilage degeneration have been associated with synovitis [12,13]. Studies have demonstrated that chondrocytes, osteoblasts and inflamed synovium all produce and contribute to various proinflammatory cytokines [14–17] .The proinflammatory cytokine IL-1b has been implicated as the most important factor responsible for the catabolic process in OA. Other proinflammatory cytokines such as TNF-a, IL-6, IL-8 have all been shown to contribute to the cartilage degradation process in OA. [14,18,19]

4. Diagnosis

The diagnosis of knee OA can be made by history and clinical examination and confirmed by radiography. A precise diagnosis also helps to rule out other causes of knee pain, such as pain referred from the hip or back amongst others.

4.1. Clinical examination

Physical examination should incorporate body habitus and gait pattern. Patient should be examined standing and supine looking for effusion, tenderness ROM and ligament instability. Gait should be assessed both in anteroposterior as well as mediolateral plane. The presence of an antalgic gait with knee flexion may suggest presence of a flexion deformity. Also evidence of either medial or lateral thrust should be noted. Limb alignment or deformity can be visualised on standing. Varus deformity is suggestive of medial compartment involvement, whilst valgus malalignment is suggestive of lateral compartment disease. These are indicative of longstanding disease process]. ROM both active and passive should be noted. Special tests include assessment of the integrity of collateral using varus/valgus stress test and also the assessment of cruciates. Examination of the hip and spine should be carried out to rule out referred pain

5. Treatment

The relative slow progression of the disease allows for stepwise algorithmic approach in management. Following systematic review of research evidence and expert consensus, the EULAR task force for OA developed evidence-based recommendations for the treatment of knee OA. Options include both non-surgical and surgical strategies.

5.1. Non-surgical

The aim of non-surgical option is patient education, pain control, delay the progression disease and to improve function. Life style modification. Exercises involving high-impact activity such as running on hard surfaces and jumping should be avoided, instead low-impact activities such as swimming, cycling should be encouraged. In patients with evidence of patellofemoral OA, activities such as stair climbing and squatting should be limited. The obese patient should be advised and encouraged to lose weight; obesity has been shown to be risk factor. for developing symptomatic OA in a large cohort study [26].

5.2. Orthoses and footwear

The aim of an orthosis is to reduce pain and improve function. The ideal candidate for an orthosis is a patient with passively correctable unicompartmental arthritis. A brace may function by improving the biomechanical axis of the deformity thereby unloading the compartment or by improving the perception of instability A recent Cochrane review has demonstrated that there is improvement in pain and function following the use of orthoses [25].

5.3. Physiotherapy

There is a good quality evidence that muscle strengthening and an aerobic exercise program is beneficial in the management of OA [28,29] Range-of motion exercises help to prevent the development of contractures. Periarticular muscle strengthening exercises tend to stabilise the knee and improve symptoms. Pharmacotherapy. Non-steroidal anti-inflammatory drugs [NSAIDS] are prescribed when the patient presents with exacerbation of pain and a swollen

knee. These agents act by blocking the proinflammatory agents such as prostaglandins and leuktines by reversibly blocking the cylooxygenase and lipooxygenese pathway. Selective COX2 inhibitors have an anti-inflammatory effect but are nephrotoxic. Due to its cardiovascular toxicity the COX2 inhibitor, Refecoxib, was withdrawn from the market in October 2004.

Intra-articular corticosteroids are indicated when there is an exacerbation of symptoms despite using NSAIDS. A systematic review has shown that intraarticular corticosteroids are efficacious in controlling pain in OA, but the effect lasts approximately one week [30]. Injectable hyluronate therapy has theoretical advantage in OA as a result of its viscoelastic, analgesic, anti-inflammatory and chondroprotective properties [31]. A Cochrane review revealed up to 5–13 weeks improvement in pain and function post injection following the use of Hyluron group of products [32]. Surgical management This can be broadly classified as joint preserving and joint-replacing procedure

5.4. Arthroscopy

Arthroscopic debridement of the knee, for OA is controversial. Despite this, it is one of the common procedures performed [33]. Randomized controlled trails have revealed that the symptomatic relief obtained is attributable to placebo effects [34,35]. However, in a selected group of patients, arthroscopy did provide beneficial effects. The factors which predicted better outcomes are younger age, mechanical symptoms such as locking, medial joint line tenderness, mild to moderate radiographic evidence of OA and presence of unstable degenerative tear of the meniscus [36–38].

5.5. Osteotomy

The goal of an osteotomy is to transfer the mechanical axis from the pathologic area to the normal compartment. In a normal knee, the mechanical axis travels from the center of the hip through the center of knee to the center of the ankle joint. In medial compartment OA with resultant varus deformity, the mechanical axis tends to move medially, this in turn leads to more stress on the medial compartment [38]. Conversely, in lateral compartment OA with a valgus deformity, the mechanical axis runs more to the lateral compartment. Proximal tibial osteotomy was popularised in Coventry [UK] in 1962 [39]. Several types of osteotomies have been described; these include opening wedge, closing wedge, oblique plane osteotomy and ball and socket osteotomy. However, the opening and closed wedge have been popularly used due to its technical ease. Regardless of the type of osteotomy, the aims are similar to normalize the mechanical axis and off-load the degenerate side.

5.6. Total knee replacement

Total knee replacement is reserved as the final option for patients with arthritis. Advances in the implant design, with better polyethylene wear properties and appropriate patient selection, reproducible results of 96% have been achieved at 10 years [66,67]. The timing of knee replacement is still debatable. Gidwani et al., [68] in a series of patients, have reported that good results can be obtained when intervention is carried out at earlier stages of radiological OA in a symptomatic patient. There is no correlation between radiological stages and symptoms [69]. The overall complication rate following primary TKR is 5% in patients. This includes infection about 1.5% and symptomatic deep vein thrombosis or pulmonary embolism is between 1 and 3 %.70 This increases up to 26% following revision knee replacement. One of the main risk factors for early revision as per the Swedish arthroplasty register is young age [71], hence, orthopaedic surgeons are often reluctant to offer TKR for young patients. Despite this, recent studies have shown promising results in younger patients. Gill et al., [72] in their cohort of patients who were <55 years when they underwent TKR, reported good or excellent score in all knees using the knee society score. Similarly, Duffy et al [67] at minimum follow-up of 10 years reported survivorship of 96% at 10 years and 85% at 15 years. The average at the time of surgery was 53 (range 29–55 years).

6. Conclusion

The knee is the commonest peripheral joint affected by OA. Initial treatment should include non-operative modalities in the form of patient education, exercises, life-style modification and analgesics. Once these are exhausted, a surgical option, which should be patient specific, should be considered. Arthroplasty should be used as the last option following appropriate patient counseling.

Compliance with ethical standards

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Disclosure of conflict of interest

The author declare that he have no conflict of interest.

References

[1] Lawrence RC, Helmick CG, Arnett FC, et al. Estimates of the prevalence of arthritis and selected musculoskeletal disorders in the United States. Arthritis Rheum. 1998; 41: 778–799.

[2] Felson DT. Epidemiology of hip and knee osteoarthritis. Epidemiol Rev. 1988; 10: 1–28.

[3] Radin EL, Yang KH, Riegger C, et al. Relationship between lower limb dynamics and knee joint pain. J Orthop Res 1991; 9: 398–405.

[4] Radin EL, Paul IL and Pollock D. Animal joint behavior under excessive loading. Nature 1970; 226: 554–555.

[5] Byers PH. Folding defects in fibrillar collagens. PhilosTrans R Soc Lond B Biol Sci. 2001; 356: 151–157.

[6] Lohmander LS, Ionescu M, Jugessur H, et al. Changes in joint cartilage aggrecan after knee injury and in osteoarthritis. Arthritis Rheum. 1999; 42: 534–544.

[7] Cs-Szabo G, Roughley PJ, Plaas AH, et al. Large and small proteoglycans of osteoarthritic and rheumatoid articular cartilage. Arthritis Rheum. 1995; 38: 660–668.

[8] Verzijl N, DeGroot J, Ben ZC, et al. Crosslinking by advanced glycation end products increases the stiffness of the collagen network in human articular cartilage: a possible mechanism through which age is a risk factor for osteoarthritis. Arthritis Rheum. 2002; 46: 114–123.

[9] Pessler F, Chen L, Dai L, et al. A histomorphometric analysis of synovial biopsies from individuals with Gulf War Veteran's illness and joint pain compared to normal and osteoarthritis synovium. Clin Rheumatol. 2008; 27: 1127–1134.

[10] Hill CL, Hunter DJ, Niu J, et al. Synovitis detected on magnetic resonance imaging and its relation to pain and cartilage loss in knee osteoarthritis. Ann Rheum Dis. 2007; 66: 1599–1603.

[11] Ayral X, Pickering EH, Woodworth TG, et al. Synovitis: a potential predictive factor of structural progression of medial tibiofemoral knee osteoarthritis e results of a 1 year longitudinal arthroscopic study in 422 patients.Osteoarthritis Cartilage. 2005; 13: 361–367.

[12] Henrotin YE, Labasse AH, Simonis PE, et al. Effects of nimesulide and sodium diclofenac on interleukin-6, interleukin- 8, proteoglycans and prostaglandin E2 production by human articular chondrocytes *in vitro*. Clin Exp Rheumatol. 1999; 17: 151–160.

[13] Haringman JJ, Ludikhuize J, Tak PP. Chemokines in joint disease: the key to inflammation? Ann Rheum Dis. 2004; 63: 1186–1194.

[14] Goldring MB. The role of cytokines as inflammatory mediators in osteoarthritis: lessons from animal models. Connect Tissue Res. 1999; 40: 1–11.

[15] Pola E, Papaleo P, Pola R, et al. Interleukin-6 gene polymorphism and risk of osteoarthritis of the hip: a 8 Scottish Medical Journal 0 [7] on June 23, 2016case–control study. Osteoarthritis Cartilage. 2005; 13: 1025–1028.

[16] Watterson JR, Esdaile JM. Visco-supplementation: therapeutic mechanisms and clinical potential in osteoarthritis of the knee. J Am Acad Orthop Surg. 2000; 8: 277.

[17] Bellamy N, Campbell J, Welch V, et al. Viscosupplementation for the treatment of osteoarthritis of the knee. Cochrane Database Syst Rev. 2006; 2: CD005321.

[18] Feeley BT, Gallo RA, Sherman S, et al. Management of osteoarthritis of the knee in the active patient. J Am Acad Orthop Surg. 2010; 18: 406–416.

[19] Moseley JB, O'Malley K, Petersen NJ, et al. A controlled trial of arthroscopic surgery for osteoarthritis of the knee.N Engl J Med. 2002; 347: 81–88.

[20] Kirkley A, Birmingham TB, Litchfield RB, et al. A randomized trial of arthroscopic surgery for osteoarthritis of the knee. N Engl J Med. 2008; 359: 1097–1107.

[21] Dervin GF, Stiell IG, Rody K, et al. Effect of arthroscopic de´bridement for osteoarthritis of the knee on health-related quality of life. J Bone Joint Surg Am. 2003; 85-A: 10–19.

[22] Hanssen AD, Stuart MJ, Scott RD, et al. Surgical options for the middle-aged patient with osteoarthritis of the knee joint. Instr Course Lect. 2001; 50: 499–511.

[23] Coventry MB. Upper tibial osteotomy for gonoathrosis: the evolution of the operation in the last 18 yrs and long term results. Orthop Clin North Am. 1979; 10: 191–210.

[24] Sharma AK. Campbell's Operative Orthopaedics. *Med J Armed Forces India*. 2004;60(1):91. doi:10.1016/S0377-1237(04)80177-1

[25] Ackroyd CE, Newman JH, Evans R, Eldridge JD, Joslin CC. The Avon patellofemoral arthroplasty: five-year survivorship and functional results. J Bone Joint Surg Br. 2007 Mar;89(3):310-5. doi: 10.1302/0301-620X.89B3.18062. PMID: 17356140.

[26] Duffy GP, Trousdale RT, Stuart MJ. Total knee arthroplasty in patients 55 years old or younger: 10- to 17-year results. Clin Orthop Relat Res. 1998; 356: 22–27.

[27] Callahan CM, Drake BG, Heck DA, et al. Patient outcomes following tricompartmental total knee replacement. A meta-analysis. JAMA 1994; 271: 1349–1357.

[28] Robertsson O, Knutson K, Lewold S, et al. The Swedish knee arthroplasty register 1975–1997. An update with special emphasis on 41,223 kneesoperatedonin1988–1997. Acta Orthop Scand. 2001; 71: 503–513.

[29] Kane RL, Saleh KJ, Wilt TJ, Bershadsky B, Cross WW 3rd, MacDonald RM, Rutks I. Total knee replacement. Evid Rep Technol Assess (Summ). 2003 Dec;(86):1-8. PMID: 15160414; PMCID: PMC4781011.