Updates in Knee Joint Osteoarthritis

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Abstract

Background: Osteoarthritis (OA) of knee is one of the most common debilitating conditions associated with pain and limitation in daily living activities which negatively affect quality-of-life. It is a disease of the entire joint, involving not only the joint lining but also cartilage, ligaments, and bone. It is characterized by breakdown of the cartilage, bony changes of the joints, deterioration of tendons and ligaments, and various degrees of inflammation of the synovium.

Aim of the Work: To discuss the incidence and updates in Pathophysiology of Osteoarthritis (OA) of knee and demonstrate updates in management to solve this problem.

Study Design: Narrative review article.

Conclusion: The current review revealed that Osteoarthritis (OA) of knee joint has multi mechanism and increases the risk of complications bed redden as such, accounts for burden on economics budget.

Keywords: Osteoarthritis - knee joint – chronic pain management-VAS score.

1. INTRODUCTION

Osteoarthritis (OA) of knee is one of the most common debilitating conditions associated with pain and limitation in daily living activities which negatively affect quality-of-life. It is the most common form of joint disease, and among the top 10 causes of disability worldwide (1). Osteoarthritis is a degenerative disease characterized by joint pain, tenderness, stiffness, joint swelling, restricted movement and joint deformities (2).

Osteoarthritis (OA) refers to a clinical syndrome of joint pain with multifactorial pathology that is characterized by the gradual loss of articular cartilage, osteophyte formation, sub-chondral bone remodeling, and inflammation of the joint (3). It is a disease of the entire joint, involving not only the joint lining but also cartilage, ligaments, and bone. It is characterized by breakdown of the cartilage, bony changes of the joints, deterioration of tendons and ligaments, and various degrees of inflammation of the synovium (4).

Scott and Kowalczyk reported that a cohort study found that radiologic features of knee osteoarthritis were very common in adults: 13% of women 45–65 years of age (an incidence of 3% per year). Osteoarthritis commonly affects middle age to elderly population. Investigators have theorized the increase in osteoarthritis in women during menopause may partially be attributed to the hormonal factors (5).

In recent years, an increasing number of patients are being diagnosed with osteoarthritis, which has a notable impact on human health (6, 7). With aging of the population and increasing obesity, OA arises as a major public health problem and an important financial burden for the global economy.
2. PATHOPHYSIOLOGY AND CAUSES OF OA

The causes of osteoarthritis are complex, and the pathogenesis related to this disease is not well understood (8). Osteoarthritis is divided into primary and secondary osteoarthritis according to the presence of local and systemic risk factors (9). Osteoarthritis is frequently diagnosed as rheumatoid arthritis or ankylosing spondylitis in clinical differential diagnosis. It is the most common disease of arthritis and can occur together with other types of arthritis (10).

Diagnostic criteria for osteoarthritis have been developed by the American College of Rheumatology. These criteria are outlines in Box 1

Another classification of knee osteoarthritis is Kellgren and Lawrence grading scale. It is based on radiological imaging and consists of different grades:

Grade 1: doubtful narrowing of joint space and possible osteophyte lipping;

Grade 2: definite osteophytes and possible narrowing of joint space;

Grade 3: moderate multiple osteophytes, definite narrowing of joint space and some sclerosis and possible deformity of bone ends; and

Grade 4: large osteophytes, marked narrowing of joint space, severe sclerosis and definite deformity of bone ends (14).

Criteria for diagnosis of knee osteoarthritis: (15, 16, 17).

Clinical criteria: Age older than 50 years—Bony enlargement—Bony tenderness—Crepitus—No palpable warmth—Stiffness for <30 minutes.

Laboratory criteria: Erythrocyte sedimentation rate <40 mm/hour—Rheumatoid factor <1:40—Synovial fluid analysis: clear, viscous, white blood cell count <2,000/μL (2.00 x 10⁹ per L)

Radiographic criteria: Presence of osteophytes.

The differential diagnoses of knee chronic pain and osteoarthritis include: conditions involving soft tissue of knee such as bursitis, iliotibial band syndrome, ligamentous instability (medial and lateral collateral ligaments), meniscal pathology; other forms of arthritis like gout and pseudogout, rheumatoid arthritis and septic arthritis; referred pain from neuropathy or radiculopathy; and other diagnoses such as avascular necrosis, patellofemoral pain syndrome and tumor. (11, 12)
Previous studies have indicated that agents targeting rheumatoid arthritis are relatively ineffective at present (18). Therefore, there is an urgent requirement for more efficient treatments for osteoarthritis with minimal side effects (19). To date, there is no effective cure for OA. Main goals are to reduce pain, rigidity and swelling, and to improve function on the short term. On the long-term, the objectives are to reduce joint damage and to decrease destruction of the joint. (20)

3. TREATMENT OF OA

In recent years, non-surgical treatments for knee osteoarthritis have become more widely used, such as PRP, corticosteroid injection and hyaluronic acid (21).

As regards conservative treatment for OA knee, various treatment modalities are recommended by clinical guidelines. Although different kinds of nonsurgical options are currently available for the treatment of knee joint osteoarthritis, they are mainly aimed at the symptomatic relief, but not joint cartilage regeneration (22-23).

The non pharmacological modalities include patient education, exercises and self management, lifestyle modifications such as dieting and weight reduction, walking supports (canes/crutches), bracing, shoe and insole modification, local hot/cold applications, acupuncture, and electromagnetic therapy.

Physical therapy is generally used, with exercises to maintain range of motion and strength.

Pharmacologic therapies can be in the form of analgesics (paracetamol), NSAIDs, opioids, and slow-acting so-called nutraceuticals (glucosamine and chondroitin sulfate). If orally administered drugs are ineffective (24).

Intra-articular injections with corticosteroids seem to be safe. (25) More than one clinical guidelines reported high recommendations that these injections reduce pain for short term, especially if accompanied by effusion. (26, 27)

Glucosamine is one of the building blocks of cartilage, which can be taken as a tablet as a supplement to the diet, or sometimes as an injection. (28) The clinical guidelines of the American Academy of Orthopedic Surgeons reported strong recommendation of not prescribing glucosamine for symptomatic patients while other three guidelines (29-37) stated fairly low level strength of recommendation for conditional use or even uncertainty about it.

4. MECHANISM OF ACTION OF PLATELET RICH PLASMA

Autologous platelet concentrate suspended in plasma, also known as PRP, can be prepared from samples of centrifuged autologous blood. Exposure to a solution of thrombin and calcium chloride degranulates platelets, results in the formation of platelet gel and this stimulate the...
release of growth factors and bioactive molecules. Therefore, platelets actively participate in healing processes by delivering a broad spectrum of growth factors (insulin-like growth factor, transforming growth factor β-1, platelet-derived growth factor, and many others) and other active molecules (e.g., arachidonic acid metabolites, cytokines, chemokines, ascorbic acid, extracellular matrix proteins, and nucleotides) to the injured site. These factors altogether contribute to comprehensive roles of PRP, including anti-inflammation, angiogenesis, chondrogenesis, chondrocyte proliferation, bone remodeling, coagulation, and cell differentiation and this, in turn, reduces inflammation, pain & can then be used as an adjunct to surgery with the intent of promoting hemostasis and accelerating healing.

Use of platelet-rich plasma is considered experimental / investigational for all orthopedic indications. This includes, but is not limited to, use in the following situations:

A. Primary use (injection) for the following conditions (41-44):
   1) Achilles tendinopathy
   2) Lateral epicondylitis
   3) Osteochondral lesions
   4) Osteoarthritis
   5) Plantar fasciitis

B. Adjunctive use in the following surgical procedures:
   1) Anterior cruciate ligament reconstruction
   2) Hip fracture
   3) Long-bone nonunion
   4) Patellar tendon repair
   5) Rotator cuff repair
   6) Spinal fusion
   7) Subacromial decompression surgery
   8) Total knee arthroplasty.

5. CONCLUSION

By reviewing current literature and our own experience, we feel that IA injections of HA, PRP, and corticosteroids given together for knee OA are an effective non-operative modality of treatment. In combination, they seem to have better efficacy than any of them given alone. These injections are clinically safe and have promising and very positive effects for patient satisfaction. When the heterogeneity of OA is considered, it is difficult to categorize which patients and what level of disease would be ideal indications for IA injections.

REFERENCES

associated with increased risk of incident radiographic osteoarthritis and worsening cartilage damage in the following year. Eur Radiol 27: 404–413, 2017.


[34] Werner S, Grose R. Regulation of wound healing by growth factors and cytokines. Physiol Rev 2003;83:835–70.


