A STUDY OF THE ROLE OF PLATELET RICH PLASMA IN THE TREATMENT OF KNEE OSTEOARTHRITIS

Dissertation submitted

in partial fulfillment of the requirements for the degree of

M S. DEGREE - BRANCH II

ORTHOPAEDIC SURGERY

STANLEY MEDICAL COLLEGE

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

April 2016

CERTIFICATE

This is to certify that the work entitled **" A STUDY OF THE ROLE OF PLATELET RICH PLASMA IN THE TREATMENT OF KNEE OSTEOARTHRITIS"** which is being submitted for M.S.Orthopaedics, is a bonafide work of **Dr. M.SERAN,** Post Graduate Student in the Department of Orthopaedics, Stanley Medical College, Chennai.

DEAN

Stanley Medical College,

Chennai

CERTIFICATE

This is to certify that the work entitled **" A STUDY OF THE ROLE OF PLATELET RICH PLASMA IN THE TREATMENT OF KNEE OSTEOARTHRITIS"** which is being submitted for M.S. Orthopaedics, is a bonafide work of **Dr. M.SERAN,** Post Graduate Student in the Department of Orthopaedics, Stanley Medical College, Chennai

He has completed the necessary period of stay in the Department and has fulfilled the conditions required for submission of this thesis according to the University regulations. The study was undertaken by the candidate himself and the observations recorded have been periodically checked by us.

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ECLARATION

I solemnly declare that the dissertation titled **"A STUDY OF THE ROLE OF PLATELET RICH PLASMA IN THE TREATMENT OF KNEE OSTEOARTHRITIS"** was done by me from August 2014 onwards under the guidance and supervision of

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This dissertation is submitted to The **Tamilnadu Dr. MGR Medical University, Chennai** towards the partial fulfillment of the requirement for the award of **M.S. Degree in Orthopaedics (Branch II)** to be held in April 2016.

Place : Chennai

Date : **DR.M.SERAN**

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INTRODUCTION

Osteoarthritis (OA) represents a failure of the diarthrodial, synovial lined joint. Among the elderly, knee Osteoarthritis is the leading cause of chronic disability[1]. Because of the increased lifespan and obesity the prevalence of osteoarthritis is on the rise in Indian population.

Osteoarthritis is age related, affecting more than 80% of people over the age of 55. OA in weight-bearing joints is strongly linked to body mass index. As life expectancy increases, and the rate of obesity reaches epidemic proportions, OA has become increasingly common. The pathogenesis involves an imbalance between normal cartilage derivative and repair mechanisms, which results in net cartilage loss, hypertrophy of bone, and osseous outgrowths called osteophytes. OA has a predilection for finger joints, knees, hips, shoulders, and the spine. Occurrence in an atypical joint, such as an elbow, can usually be traced to prior trauma, a congenital joint abnormality, underlying systemic disease, or a chronic crystalline arthropathy. The heterogeneity of OA arises from the many factors that can contribute to cartilage damage.

Symptomatic OA of the knee which is described as having pain during most days of a month along with radiologic evidence of arthritis has a prevalence of 22% to 39% in India[3, 4].

Osteoarthritis is a chronic disorder of synovial lined joints where there is progressive softening and disintegration of articular cartilage accompanied by new growth of cartilage and bone at the joint margins, cyst formation and sclerosis at subchondral regions of bone, mild synovitis and capsular fibrosis.

Osteoarthritis differs from simple wear and tear in that it is asymmetrically distributed, often associated with abnormal loading rather than frictional wear. It is not an inflammatory disorder although at times there are local signs of inflammation. In its most common form osteoarthritis is unaccompanied by any systemic illness.

RISK FACTORS

1. AGE:

Age is the most powerful risk factor for osteoarthritis. The cartilage shows diminished cellularity, reduced proteoglycan concentration, loss of elasticity and a decrease in breaking strength with advancing years.

2. INHERITANCE:

First degree relations had shown an increased incidence of osteoarthritis compared with controls. Mutations in COL2A1 genes have been associated with clinical phenotypes ranging from mild spondyloepiphyseal dysplasia to severe generalized osteoarthritis.

3. TRAUMA:

Articular cartilage may be damaged by trauma or other inflammatory disorders.

4. JOINT LOADING:

Increased mechanical stress in some part of the articular surface may lead on to development of osteoarthritis.

5. OBESITY

Obesity is one of the risk factor for both knee and hand osteoarthritis. People with higher ranges in body mass index are at increased risk of developing osteoarthritis. A weight loss of only 5 kg is associated with a 50% reduction in the odds of developing symptomatic knee osteoarthritis.

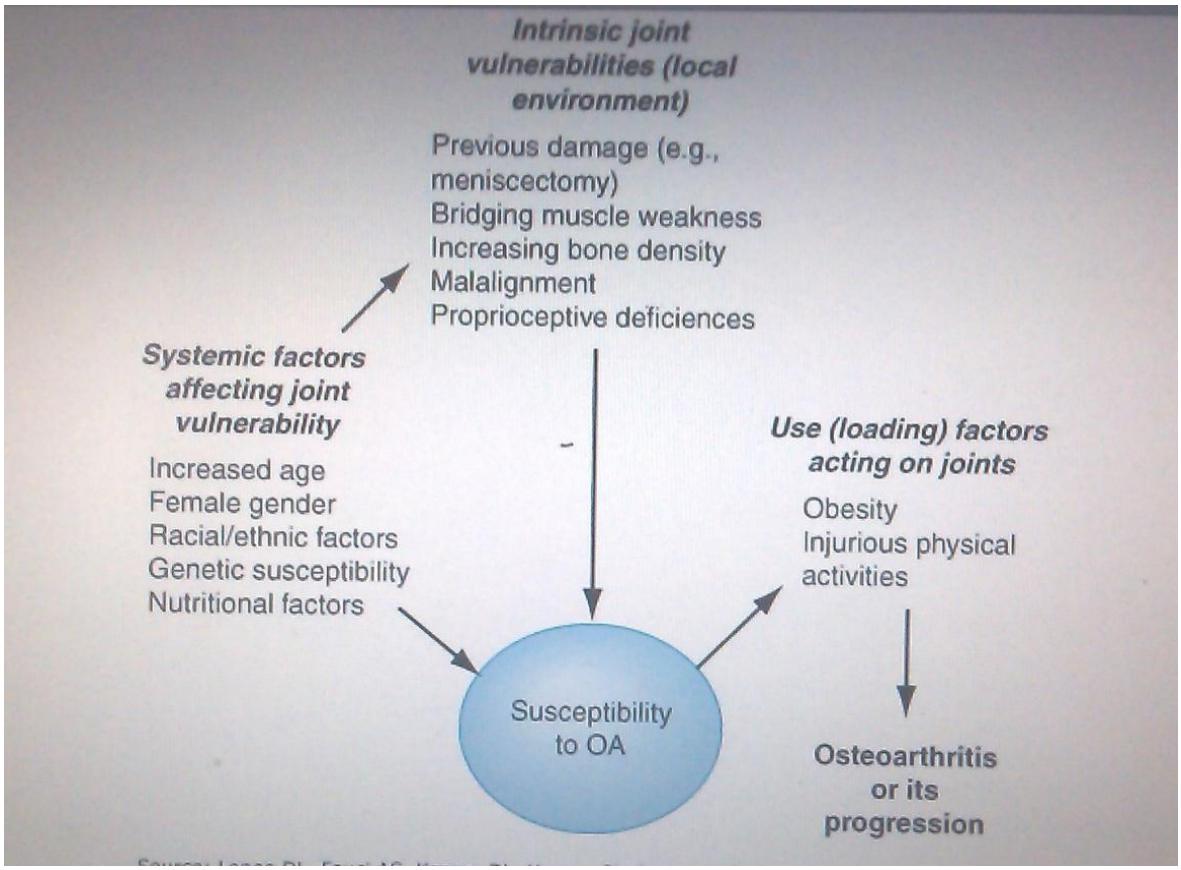
Three to six times body weight is transmitted across the knee during single-leg stance. Any increase in weight may be multiplied by this factor to reveal the excess force across the knee in overweight persons during walking.

Obesity is a well-recognized and potent risk factor for the development of knee OA and, less so, for hip OA. Obesity precedes the development of disease and is not just a consequence of the inactivity present in those with disease. It is a stronger risk factor for disease in women than in men, and in women, the relationship of weight to the risk of disease is linear, so that with each increase in weight, there is a commensurate increase in risk. Weight loss in women lowers the risk of developing symptomatic disease. Not only is obesity a risk factor for OA in weight-bearing joints, but obese persons having more severe symptoms from the disease.

Obesity's effect on the development and progression of disease is mediated mostly through the increased loading in weight-bearing joints that occurs in overweight persons. However, a modest association of obesity with an

increased risk of hand OA suggests that there may be a systemic metabolic factor circulating in obese persons that affects disease risk also.

Diagramatic representation of various risk factors culminating in susceptibility to osteoarthritis



6. FAILURE OF JOINT PROTECTIVE MECHANISMS

Joint protectors are structures in and around the joint that include joint capsule, ligaments, afferent nerve fibers, muscle, and bone. Joint capsule and ligaments limit excursion and fix range of motion, Synovial fluid reduces friction induced cartilage wear by providing a well lubricated gliding surface. This lubrication function depends on the molecule lubricin, a mucinous glycoprotein secreted by synovial fibroblasts. The concentration of synovial fibroblasts is found to diminish in the face an injury to the joint or synovial inflammation.

Mechanoreceptor sensory afferent nerves are present over the ligaments, tendons and skin. These mechanoreceptors, throughout a joint's range of motion fire with different level of frequencies and provide feedback by way of the spinal cord to muscles and tendons. Thus these muscles and tendons act as optimal joint protectors, anticipating joint loading as they assume the right tension at appropriate points.

Muscles and tendons that bridge the joint are key joint protectors. Their contractions at the appropriate time in joint movement provide the appropriate power and acceleration for the limb to accomplish its tasks. Focal stress across the joint is minimized by muscle contraction that decelerates the joint before impact and assures that when joint impact arrives, it is distributed broadly across the joint surface.

7. REPEATED USE OF JOINT

There are two categories of repetitive joint use, occupational use and leisure time physical activities. Workers performing repetitive tasks as part of their occupations for many years are at high risk of developing OA in the joints they use repeatedly. For example, farmers are at high risk for hip OA, and miners have high rates of OA in knees and spine, Even within a textile mill, women whose jobs required fine pincer grip [increasing the stress across the interphalangeal (IP) joints] had much more distal IP (DIP) joint OA than women whose jobs required repeated power grip, a motion that does not stress the DIP joints. Workers whose jobs require regular knee bending or lifting or carrying heavy loads have a high rate of knee OA. One reason why workers may get disease is that during long days at work, their muscles may gradually become exhausted, no longer serving as effective joint protectors.

While exercise is a major element of the treatment of OA, certain types of exercise may paradoxically increase the risk of disease. While recreational runners are not at increased risk of knee OA, studies suggest that they have a modest increased risk of disease in the hip.

However, persons who have already sustained major knee injuries are at increased risk of progressive knee OA as a consequence of running. Compared to non runners, elite runners (professional runners and those on Olympic teams) have high risks of both knee and hip OA.

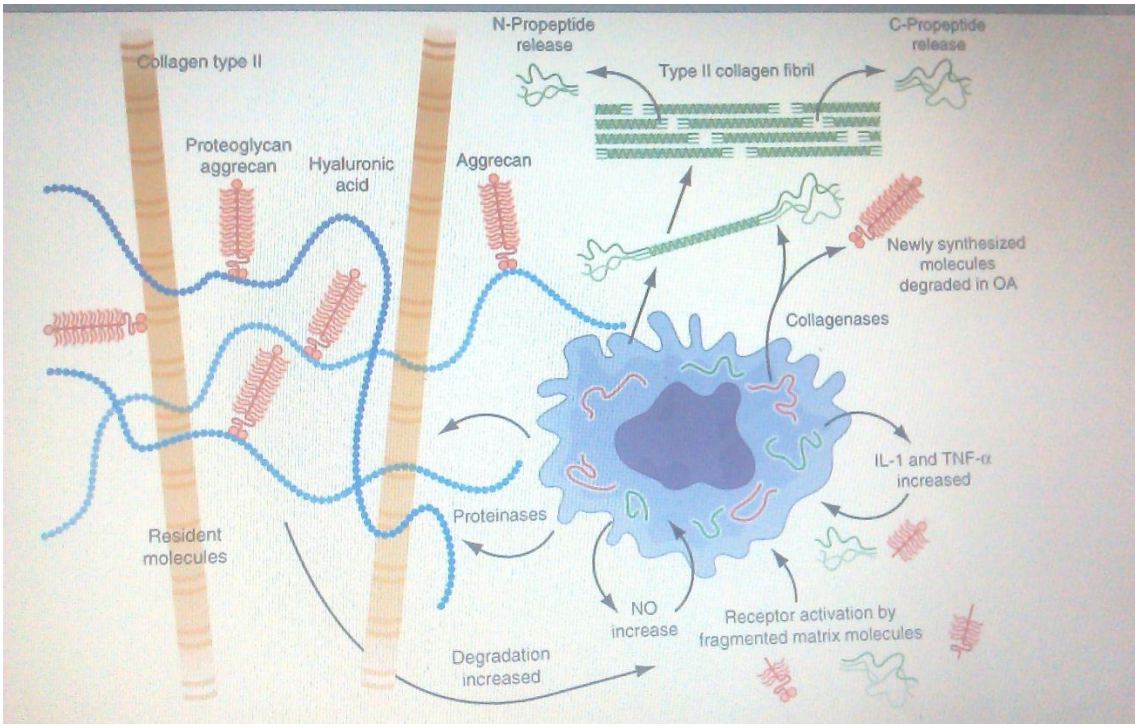
Given the widespread recommendation to adopt a healthier, more exercise-filled lifestyle; longitudinal epidemiologic studies of exercise contain cautionary notes. For example, women with increased levels of physical activity, either as teenagers or at age 50, had a higher risk of developing symptomatic hip disease later in life than women who were sedentary. Other athletic activities that pose high risks of joint injury, such as football, may thereby predispose to OA.

ARTICULAR CARTILAGE

Articular cartilage is composed of two major macromolecular species

(I)Proteoglycans:

Proteoglycans are responsible for the compressive stiffness and the ability to withstand loads, of the tissues.



(II) Collagen:

Collagen provides tensile strength and resistance to shear

Matrix metalloproteinases (MMP) such as collagenase, gelatinase and stromelysin are present in cartilage. These metalloproteinases can degrade components of the extra cellular matrix at neutral pH. Metalloproteinases are secreted in pro enzyme form and require proteolytic cleavage of n-terminal sequence for its activation.

MMP activity in the cartilage is basically a balance between proenzyme activation and inhibition of the active enzyme by tissue inhibitors. Aggrecan which is the major Proteoglycan in articular cartilage is degraded by aproteinase, named aggrecanase.

Interleukin -1 is a cytokine synthesized by synovial lining mononuclear cells and chondrocytes. Interleukin -1 stimulates the synthesis of proenzyme matrix metalloproteinases and tissue plasminogen activator.IL-1 suppresses PG synthesis by the chondrocyte, inhibiting matrix repair.

Human articular cartilage is primarily degraded by proteinase collagenase-3(MMP-13) and aggrecanase –1(ADAMTS4). Transcriptional activation by cytokines controls MMP-13 activity. Nuclear factor kappa –B and p38 mitogen activated protein kinase pathway, activates collagenase -3.

Interest currently exists in the inhibition of cartilage matrix degradation by therapeutic agents that block the p38 dependent transcriptional activity of MMP-13 or control extracellular activation of the secreted form of ADAMTS4.

The balance of the system lies with inhibitors of matrix degrading enzymes, e.g., tissue inhibitor of metalloproteinase and plasminogen activator inhibitor-1, which are synthesized by the chondrocyte and limit the activity of MMPs and Plasminogen activator, respectively.

Chondrocytes induced by cytokines synthesize prostaglandin E2, bone morph genetic protein 2 (BMP-2) and nitric oxide, which together have complex effects on matrix synthesis and degradation. Nitric oxide enhances proteinase activity and inhibits aggrecan synthesis.BMP-2 stimulates anabolic activity.

Healthy cartilages have a sluggish metabolism where synthesis and degradation of matrix is in balance. The cartilage in early OA or after an injury is highly metabolically active. The stimulated chondrocytes synthesize proenzymes and new matrix molecules, with those enzymes becoming activated in the matrix, causing release of degraded aggrecan and type 2 collagen into cartilage and into the synovial fluid. OA cartilage is characterized by gradual depletion of aggrecan, an unfurling of the tightly woven collagen matrix, and loss of type 2 collagen. With these changes comes increasing vulnerability of cartilage, which loses its compressive stiffness.

HERITABILITY AND GENETICS

OA is a highly heritable disease, but its heritability varies by joint. Fifty percent of the hand and hip OA in the community is attributable to inheritance, i.e., to disease present in other members of the family. However, the heritable proportion of knee OA is at most 30%, with some studies suggesting no heritability at all. Whereas many people with OA have disease in multiple joints, this "generalized OA" phenotype is rarely inherited and is more often a consequence of aging.

Emerging evidence has identified genetic mutations that confer a high risk of OA, one of which is a polymorphism within the growth differentiation factor 5 genes. This polymorphism diminishes the quantity of GDF5, which normally has anabolic effects on the synthesis of cartilage matrix.

RISK FACTORS IN THE JOINT ENVIRONMENT

Some risk factors increase vulnerability of the joint through local effects on the joint environment. With changes in joint anatomy, load across the joint is no longer distributed evenly across the joint surface, showing an increase in focal stress.

Major injuries to a joint also can produce anatomic abnormalities that leave the joint susceptible to OA. For example, a fracture through the joint surface often causes OA in joints in which the disease is otherwise rare such as the ankle and the wrist. Avascular necrosis can lead to collapse of dead bone at the articular surface, producing anatomic irregularities and subsequent OA.

Tears of ligamentous and fibrocartilaginous structures that protect the joints, such as the anterior cruciate ligament and the meniscus in the knee and the labrum in the hip, increase joint susceptibility and can lead to premature OA. Meniscal tears increase with age and when chronic are often asymptomatic but lead to adjacent cartilage damage and accelerated osteoarthritis. Even injuries that do not produce diagnosed joint injuries may increase risk of OA, perhaps because the structural injury was not detected at the time. For example, in the Framingham study subjects, men with a history of major knee injury had a 3.5-fold increased risk for subsequent knee OA knee.

Another source of anatomic abnormality is malalignment across the joint. This factor has been best studied in the knee, which is the fulcrum of the longest lever arm in the body. Varus (bowlegged) knees with OA are at exceedingly high risk of cartilage loss in the medial or inner compartment of the knee, whereas valgus (knock-kneed) malalignment predisposes to rapid cartilage loss in the lateral compartment. There is evidence that malalignment in the knee not only causes cartilage loss but leads to underlying bone damage, producing bone marrow lesions seen on MRI.

Malalignment in the knee often produces such a substantial increase in focal stress within the knee (as evidenced by its destructive effects on subchondral bone) that severely malaligned knees may be destined to progress regardless of the status of other risk factors.weakness in the quadriceps muscles bridging the knee increases the risk of the development of painful OA in the knee.

PATHOLOGY

The pathology of OA provides evidence of the involvement of many joint structures in disease. Cartilage initially shows surface fibrillation and irregularity. As disease progresses, focal erosions develop there, and these eventually extend down to the subjacent bone. With further progression, cartilage erosion down to bone expands to involve a larger proportion of the joint surface, even though OA remains a focal disease with non uniform loss of cartilage.

After an injury to cartilage, chondrocytes undergo mitosis and clustering. While the metabolic activity of these chondrocyte clusters is high, the net effect of this activity is to promote proteoglycan depletion in the matrix surrounding the chondrocytes. This is because the catabolic is greater than the synthetic activity. As disease develops, collagen matrix becomes damaged, the negative charges of proteoglycans get exposed, and cartilage swells from ionic attraction to water molecules. Because in damaged cartilage proteoglycans are no longer forced into close proximity, cartilage does not bounce back after loading as it did when healthy, and cartilage becomes vulnerable to further injury. Chondrocytes at the basal level of cartilage undergo apoptosis.

With loss of cartilage come alterations in subchondral bone. Stimulated by growth factors and cytokines, osteoclasts and osteoblasts in the subchondral bony plate, just underneath cartilage, become activated.

Bone formation produces a thickening and stiffness of the subchondral plate that occurs even before cartilage ulcerates. Trauma to bone during joint loading may be the primary factor driving this bone response, with healing from injury (including microcracks) producing stiffness. Small areas of osteonecrosis usually exist in joints with advanced disease. Bone death may also be caused by bone trauma with shearing of microvasculature, leading to a cut off of vascular supply to some bone areas.

At the margin of the joint, near areas of cartilage loss, osteophytes form. These starts as outgrowths of new cartilage and, with neurovascular invasion from the bone, this cartilage ossifies. Osteophytes are an important radiographic hallmark of OA. In malaligned joints, osteophytes grow larger on the side of the joint subject to most loading stress (e.g., in varus knees, osteophytes grow larger on the medial side).

The synovium produces lubricating fluids that minimize shear stress during motion. In healthy joints, the synovium consists of a single discontinuous layer filled with fat and containing two types of cells, macrophages and

fibroblasts, but in OA, it can sometimes become edematous and inflamed. There is a migration of macrophages from the periphery into the tissue, and cells lining the synovium proliferate. Enzymes secreted by the synovium digest cartilage matrix that has been sheared from the surface of the cartilage.

Additional pathologic changes occur in the capsule, which stretches, becomes edematous, and can become fibrotic.

SURGICAL ANATOMY

The knee joint is a synovial joint, the largest in the body. It is a modified Hinge joint in addition to flexion and extension a small amount of rotation of the leg is possible in the flexed position of the knee. It is a compound joint that includes two condoler joints between the femur and the tibia and a sellar (saddle) joint between the patella and the femur, the former being partly divided by menisci. The lateral and medial articular surfaces of the femur and tibia are asymmetrical.

The distal surface of the medial condyle of the femur is narrower and more curved than that of the lateral condyle. The lateral tibial articular surface is almost circular, the medial is oval with a longer anteroposterior axis, and these differences are reflected in the shapes of the menisci.

The articular surface of the patella is divided by a vertical ridge into a large lateral and a small medial surface; the latter is further subdivided by a faint vertical ridge into two smaller areas. The large lateral area articulates with the lateral condyle of the femur in extension and flexion. In extension the area next to it is in contact with the medial femoral condyle and the most medial area does not articulate with the femur. In flexion this surface is in contact with the medial condyle and the middle area is opposite the intercondylar notch of the femur.

LIGAMENTOUS SUPPORTS OF THE KNEE

Tibial collateral ligament is attached superiorly to medial femoral epicondyle and inferiorly attached to medial surface of tibia

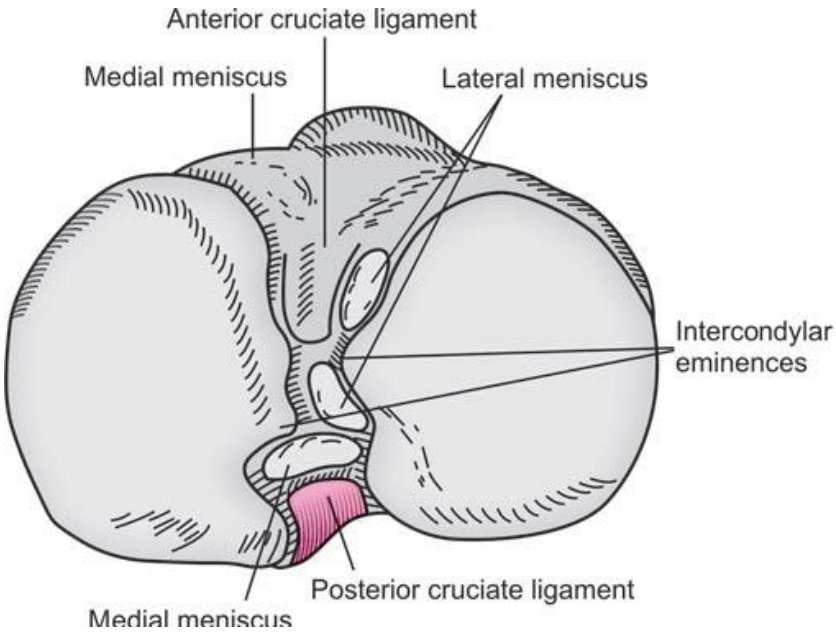
Fibular collateral ligament is attached, proximally to lateral epicondyle and below to head of fibula covered by the tendon of biceps femoris.

Oblique popliteal ligament, expansion from the tendon of semimembranous blends with the capsule at the back of the joint and ascends laterally to the intercondylar fossa and lateral femoral condyle. The popliteal artery lies on it, and genicular vessels and nerves penetrate it.

Arcuate popliteal ligament is a Y-shaped thickening of posterior capsular fibres. The stem of the Y is attached to the head of the fibula. The medial limb arches over the tendon of popliteus to the posterior edge of the tibial intercondylar area. The lateral limb ascends to the lateral femoral condyle with the popliteus tendon.

INTRAARTICULAR STRUCTURES

The Principal Intra articular structures are the medial and lateral menisci and the anterior and posterior cruciate ligaments.



The ACL originates from the anterior tibial eminence and courses through the intercondylar notch to insert on the posteromedial aspect of the lateral femoral condyle.

The fascicles of the ACL can be grouped into anteromedial and posterolateral bands, named for the anatomical tibial insertion sites. The anteromedial band tightens with flexion, while the posterolateral band is taut in extension.

The ACL serves as a primary restraint to anterior tibial translation. It is also an important secondary restraint to varus and valgus as well as tibial rotation.

Posterior cruciate ligament (PCL) takes origin from a depression between the posterior aspect of the two tibial plateaus. It inserts on to the lateral surface of the anterior portion of the medial femoral condyle.

Functionally, the PCL is composed of two bundles, anterolateral and posteromedial. The anterolateral band is tight in flexion and the posteromedial band tight in extension.

The PCL serves as the primary static restraint to posterior translation of the tibia.In addition, it is a secondary stabilizer to varus angulation and external tibial rotary displacement at 90° of knee flexion.

SURGICAL APPROACH

The surgical approach to the knee joint utilises a vertical anterior midline incision. On account of its variable course, the infrapatellar branch of the saphenous nerve may be at risk at the lower end of the incision. A medial skin flap is developed and the joint entered through an incision that divides the parapatellar retinaculum and extends above and below the patella as necessary.

The posterior approach to the knee joint utilises a S-shaped incision to avoid subsequent flexion contracture of the joint. The popliteal fascia is divided vertically and the interval between the tibial nerve and semimembranosus opened up by displacing the nerve and popliteal vessels laterally, and detaching the medial head of gastrocnemius from its femoral origin.

An anterolateral portal is most commonly used for diagnostic knee arthroscopy and is sited just above the joint line lateral to the patellar tendon. A corresponding anteromedial portal is used for the insertion of instruments. Aspiration is usually carried out from the side at the upper lateral margin of the patella, the needle entering the suprapatellar bursa.

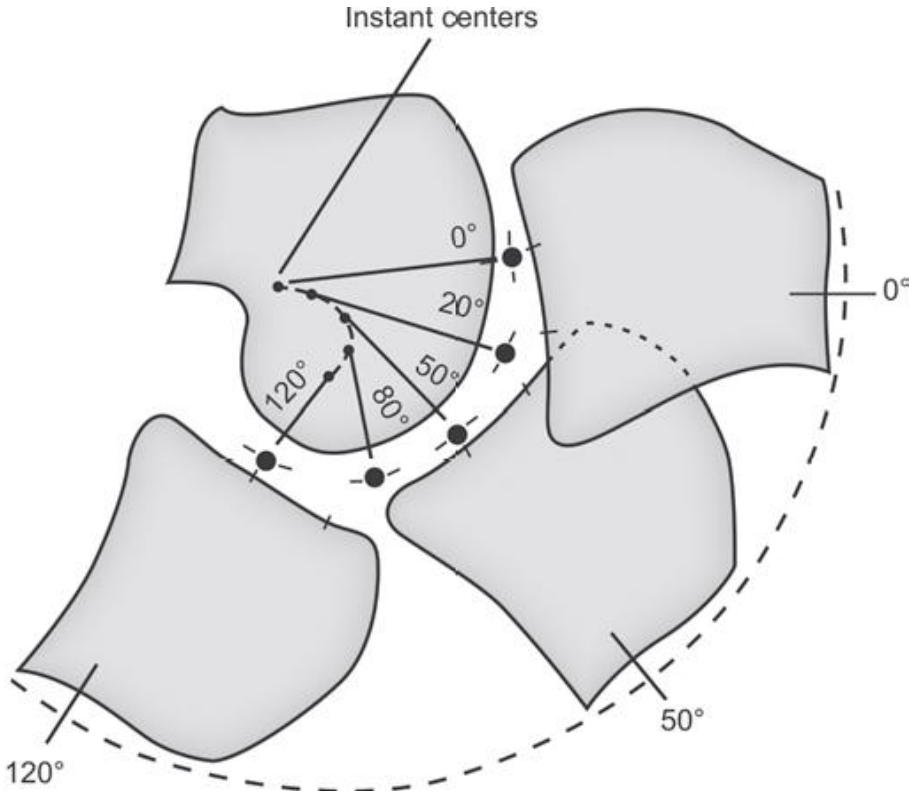
For injection the joint is entered at the lower border of the patella on either side of the patellar ligament. The needle tip must not damage the menisci or joint surfaces.

BIOMECHANICS OF THE KNEE

The knee joint transmits loads, participates in kinematic function, aids in momentum conservation, and provides a force couple for purposeful activities involving the foot.

In addition to flexion and extension occurring in the sagittal plane, constant abduction and adduction are occurring in the coronal plane, and internal and external rotation are occurring in the transverse plane.

Flexion and extension do not occur about a fixed transverse axis of rotation, but rather about a constantly changing center of rotation, i.e. polycentric rotation[8]. When plotted, the path of this changing center of rotation describes a J-shaped curve about the femoral condyles.



Flexion and extension of the knee are accomplished by both a rocking motion and a gliding motion between the femoral and tibial condyles. In a normal gait cycle, approximately 70° of flexion and extension occur during the swing phase, and 20° during the stance phase. Approximately 10° of abduction and adduction, and 10 to 15° of internal and external rotations occur during each gait cycle.

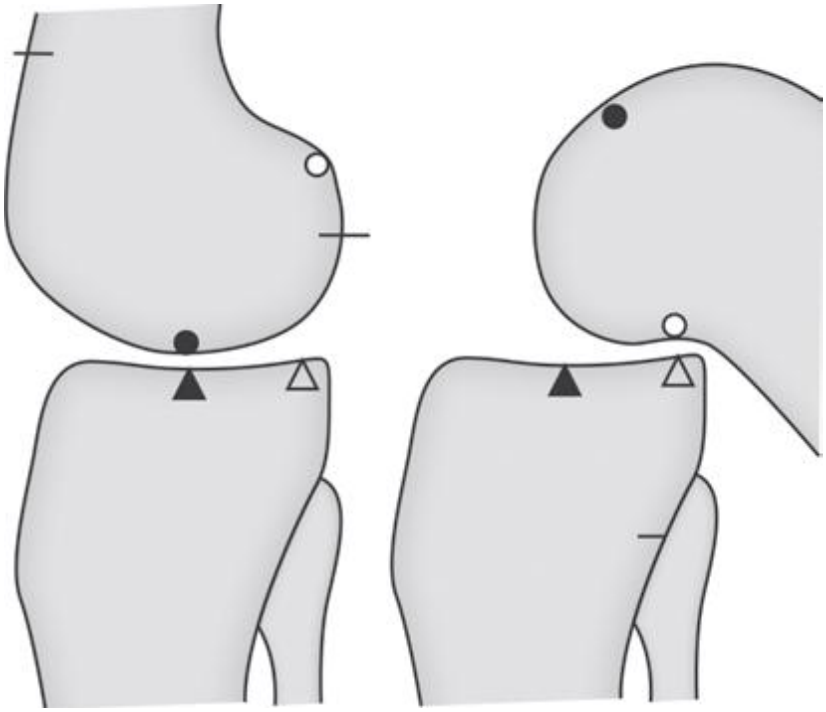


Diagram showing Movement of femur—relative to the tibia during flexion showing contact points, generated by combination of rocking and gliding.

Joint surfaces are subject to a loading force equal to three times the bodyweight in level walking. In climbing stairs, the force increases to four times the body weight. In a normally aligned knee, the weight bearing is shared across the medial and lateral tibial plateaus, 70% over medial and 30% over lateral. When malalignment exists, such as in varus or valgus deformity, a significant shift in the joint load to one side of the articulation occurs.

In a varus deformity almost 100% weight is borne by medial plateau. Corrective osteotomies about the knee attempt to reestablish a more normal weight bearing distribution across the joint surface of the knee.

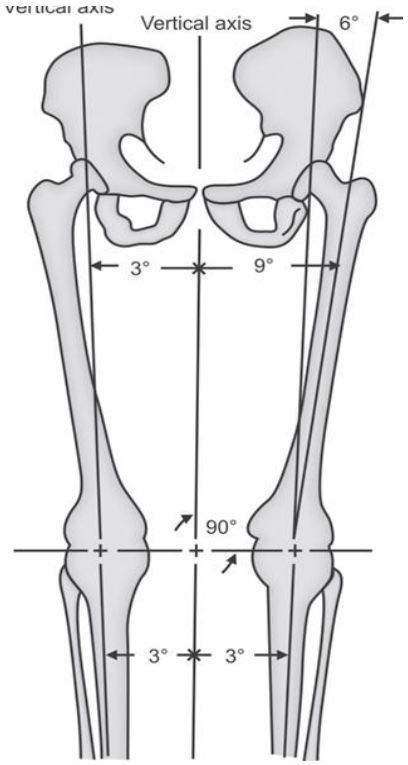
The mechanical axis of the lower limb extends from the center of the femoral head to the center of the ankle joint, and should pass through or near the center of the knee in a normally aligned lower limb. Because the hips are more widely separated than the knee and ankle, this mechanical axis is in 3° of valgue from the true vertical axis of the body which extends from the center of gravity to the ground.

The anatomic axis of the femur (femoral shaft axis) is in approximately 6 degrees of valgus from the vertical axis with variations according to body habitus.

Furthermore, the anatomic axis of the tibia is approximately 2 to 3° of varus from the mechanical axis by their measurement. The clinical implications of these facts are that when performing a total knee replacement, the femoral component should be placed in 9 ± 2° of valgus from the vertical axis, and the tibial component in 2 to 3° of varus.

The biomechanical functions of the patella allow for a wider distribution of forces on the distal femur. The patella prevents tendon-joint contact during flexion of the knee. It also effectively lengthens the lever arm of the quadriceps muscle through knee range of motion.

The menisci are important in reducing stress on the cartilage. Differential contributions of the medial and lateral menisci to load Transmission have been shown. The load across the medial compartment is borne and the meniscus carries 70% of the load transmitted.



Alterations in the vertical and transverse axes may occur with disruptions and derangements of the vertical axes of rotation shifts medially and vice versa. Because of the eccentricity of the femoral condyles the transverse axis of rotation constantly changes position (instant center of rotation) as the knee progresses from extension into flexion.

Changes in the "instant center of rotation" are often detectable kinematically and are responsible for many of the degenerative conditions about the knee joint.

CLINICAL FEATURES

Joint pain of OA is often described as deep ache localized to the involved joint. The pain is aggravated by joint use and relieved by rest but as disease progresses, it may become persistent. Articular cartilage is aneural, joint pain in OA must arise from other structures

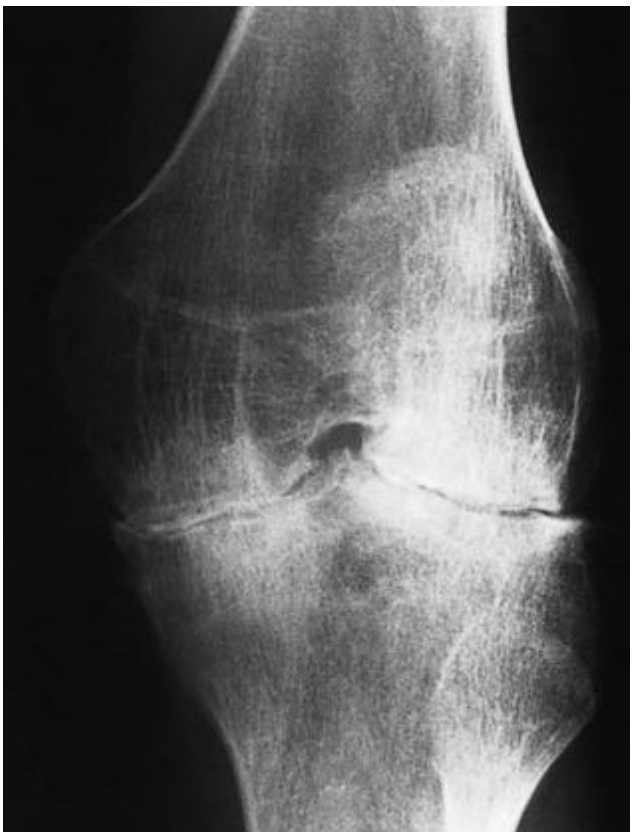
Source	Mechanism
Synovium	Inflammation
Subchondral Bone	Medullary Hypertension, Microfractures
Osteophytes	Stretching Of Periosteal Nerve Endings
Ligaments	Stretch
Capsule	Inflammation,Distension
Muscle	Spasm

Physical examination:

- Localized tenderness ,bony or soft tissue swelling
- Bony crepitus
- Joint effusion
- Restriction of mobility

THE CARDINAL SIGNS OF OSTEOARTHRITIS

- Narrowing of the ‘joint space’
- Marginal osteophytes
- Subchondral cysts
- Bone remodeling



MANAGEMENT

The initial conservative management includes:

1. Obesity is a well-known risk factor for the development of osteoarthritis and weight loss definitely slows down the progression of disease.
2. Avoidance of ground level activities reduces the mechanical stresses on the various compartments of knee and hence slows the progression of osteoarthritis. Avoidance of squatting, Indian toilet seat etc are strongly advised.
3. Strengthening of quadriceps and hamstrings and proper muscle balancing around the knee have been found to reduce the pain and disability of the knee in OA.[8,9,]There has been evidence from large randomized controlled trials that joint specific exercises reduce pain and improve function in knees as do the aerobic exercise regimens.

NSAIDs:

They are the most commonly used drugs for OA knee for reducing the pain and inflammation associated with the knee. Unfortunately their continuous use is associated with serious side effects. Paracetamol should be considered the drug of first choice as it is comparatively much safer. Gastritis, peptic ulcers and peptic ulcer rupture are well known complications of these NSAIDs. Besides, chronic renal failure is one of the most serious consequences of prolonged use.

Symptomatic Slow Acting Drugs for OA (SYSADOA) (glucosamine sulphate, chondroitin sulphate, diacerein, and hyaluronic acid).

There is evidence to suggest that these drugs may possess structural modification properties, but more methodological studies are required to prove the same.

Arthroscopic debridement:

Recent advances in instrumentation and a growing understanding of the pathophysiology of osteoarthritis have led to increased use of arthroscopy for the management of degenerative arthritis of the knee. Immediate symptomatic relief can be expected in almost all the patients. In properly selected patients arthroscopic debridement can provide long-lasting pain relief, improvement in quality of life, delay and in a few cases even obviate the need of reconstructive procedures.

Proximal/High Tibial Osteotomy (HTO):

Proximal tibial osteotomy is a well established procedure for the last 40 years known to decrease pain and improve functional results in unicompartmental knee arthritis.

HTO is indicated in patients with pain and disability resulting from osteoarthritis with weight bearing roentgenograms showing degenerative changes confined to one compartment with a corresponding varus or valgus deformity with the patient possessing sufficient muscle strength and motivation to carry out rehabilitation with crutches and having good vascular status.

Total Knee Arthroplasty (TKA):

Total Knee Arthroplasty is the only effective means of providing a painless and functional knee joint in painful arthritic joints. Knee pain caused by arthritis with or without deformity is the primary indication for total knee replacement.

Contraindications for Total knee arthroplasty include recent or current knee sepsis, a remote source of ongoing sepsis, extensor mechanism discontinuity secondary to muscular weakness and the presence of well functioning knee arthrodesis.

AIM OF THE STUDY

Osteoarthritis is a clinically heterogenous degenerative condition characterized by destruction of articular cartilage, due to uncoupling of balance between cartilage degeneration and regeneration.

The management of osteoarthritis[15] has varied from conventional therapy with physical education, non steroidal anti inflammatory drugs, intra articular glucorticoid injection, intra articular injection of hyaluronan etc. Advanced OA in whom aggressive medical management has failed to yield desired results are managed by joint replacement arthroplasty.

Pharmacological treatment of osteoarthritis with NSAIDSs is associated with an increased risk of GI disturbances along with an alarming rise in NSAID induced multisystem complications. Arthroplasty, though a definite treatment is usually reserved for advanced stages of OA. The concomitant post operative morbidity, cost issues and the need for technical expertise and revisions has precluded Arthroplasty from being a common form of treatment.

Autologous chondrocyte transplantation and attempts at cartilage repair using mesenchymal stem cells and autologous osteochondral plugs are curently in experimental stages.

The Aim of this study is to evaluate the effectiveness of Platelet Rich Plasma[17] in reducing pain and improving physical function, as Platelet Rich

Plasma provides a cocktail of growth factors directly into joint cavity. Platelet Rich Plasma is postulated to modify the disease process, unlike other methods of nonsurgical treatment which provide symptomatic relief.

Platelet Rich Plasma is a cost effective tool that could obviate the need for Total Joint Arthroplasty, or atleast reduce the number of revision surgeries.

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PATHOPHYSIOLOGY OF CARTILAGE CHANGES IN OA

The Primary change is a defect in collagen network of matrix and evidence supports the concept that MMPs account for much of the loss of cartilage matrix in OA.

MMPs, plasmin and cathepsins all appear to be involved in the breakdown of articular cartilage in OA.TIMP and PAI- 1 work to stabilize the system while growth factors such as insulin like growth factor -1 and Transforming growth factor –β are implicated in repair process.

Nitric oxide (NO) plays a significant role in articular cartilage damage in OA. Nitric oxide stimulates synthesis of MMPs by chondrocytes. Chondrocytes are the major source of NO, the synthesis of which is stimulated by IL-1 and TNF and by shear stress.

Recent evidences on the beneficial effects of Platelet Rich Plasma In healing chronic conditions[12] such as lateral epicondylitis and plantar fasciitis has kindled interest on the usage of PRP in knee osteoarthritis.

Platelets play a central role in normal hemostasis. Platelets contain an abundance of growth factors and secrete numerous bio active proteins responsible for attracting mesenchymal stem cells, osteoblasts thus expediting tissue regeneration and healing.

Autologous Platelet rich plasma is a volume of plasma produced by centrifuging patient's whole blood, yielding a high concentration of platelets above baseline levels. Depending on the method of collection, the product may contain white blood cells above baseline levels. Platelets and white blood cells are sources of high concentration of cytokines.

Cytokines are well documented to modify processes such as cell migration, angiogenesis, and collagen synthesis and wound healing.

Platelet rich plasma delivers high concentrations of autologous Growth factors[6] contained in the alpha granules of platelets. Among these are

- Transforming Growth Factor-β is involved in phenotype expression, differentiation of chondrocytes, matrix deposition and decreasing the suppressing effect of IL1 on Proteoglycan synthesis in cartilage.
- Platelet Derived Growth Factor promotes maintenance of hyaline like phenotype, proliferation of chondrocytes and proteoglycan production.
- Insulin Growth Factor stimulates synthesis of proteoglycan Epidermal growth factor promotes differentiation of cells, epithelialisation and aids collagenases.
- Vascular endothelial growth factor promotes angiogenesis.
- Connective tissue growth factor stimulates vessel permeability and endothelial cell mitogenesis.

REVIEW OF LITERATURE

Patel et al in their study in 2013 compared the efficacy of platelet rich plasma in treating osteoarthritis with that of placebo and concluded that Group of patients treated with PRP fared better in all assessment parameters.

The study also concluded that a single dose of leucocyte filtered platelet when administered in concentrations more than ten times the normal amount to be as effective as two injections.

Khosbin et al in a systematic review with quantitative synthesis in 2013 concluded that intraarticular PRP injections may have beneficial effects in the treatment of adult patients with mild to moderate osteoarthritis. The study also reported increased incidence of non specific adverse events among patients treated with Platelet rich plasma.

Kalbkhani et al in 2014 studied the effect of PRP in experimentally induced OA in rabbits knee joint concluded that PRP group had near normal joint structure at 16 week post op interval and hence PR could potentially be used for the treatment of osteoarthritis.

Giuseppe Filardo in 2010 studied about platelet rich plasma intra articular knee injections for the treatment of degenerative cartilage lesions and osteoarthritis concluded that treatment with PRP injections can reduce pain and improve knee function and quality of life with short term efficacy.

Kon et al Trial - the effectiveness of PRP injections were compared to hyaluronic acid (HA) intra-articular injection therapy. The primary outcomes of pain reduction and symptoms and function improvements were measured through the International Knee Document Committee (IKDC) and Visual Analogue Scale (EQ VAS) scoring system.

Evaluation of the IKDC score in the PRP group showed a steady increase from 41.2 (baseline) to 62.7 and 64.0 at 2 and 6 months follow up, respectively.

OBJECTIVES

To evaluate the role of Autologous Platelet Rich Plasma in the treatment of patients presenting with primary osteoarthritis and to analyse whether it could be a cost effective disease modifying measure, bereft of major side effects and operative costs.

STUDY DESIGN

Randomized controlled trial. The patients were subjected to a standardized injection protocol and were assessed on variable such as pain, stiffness and physical function using WOMAC scale and for pain using visual analog scale at pre injection, 6 weeks post injection, 3 months and 6 months post injection.

MATERIALS AND METHODS

The patients attending the OPD of Orthopaedics Department at Stanley Medical College with complaints of bilateral knee pain were screened and those diagnosed as bilateral Knee Osteoarthritis were chosen for the study.

The Patients, classified either grade 0 to 4 on the Kellgren-Lawrence grading scale or grade 1 to 4 on the Ahlback scale, were included in the study after prior well informed written consent.

Hundred Patients were chosen and randomly divided into two groups of fifty each. Group I received intra articular injection Platelet Rich Plasma in to both knees served as study group. Group II received normal saline and served as control.

Randomization ensured that both the groups were comparable with respect to age, sex, height, weight, body mass index and pre injection WOMAC score.

EXCLUSION CRITERIA

- Immunosuppresed patients
- Patients with secondary osteoarthritis
- Patients with connective tissue disorders
- Patients with inflammatory disorder of joints
- Patients who have received steroid injections within past 6 months
- Patients with haemoglobin less than 10 mg%
- Patients with tumours, metabolic diseases of bone
- Patients with coexisting backache

KELLGREN LAWRENCE GRADING OF OSTEOARTHRITIS[10]

Grade 0: no radiographic features of OA are present

Grade 1: doubtful narrowing of joint space and possible osteophytic Lipping

Grade 2: definite osteophytes, definite narrowing of joint space

Grade 3: moderate multiple osteophytes, definite narrowing of joints space, some sclerosis and possible deformity of bone contour

Grade 4: large osteophytes, marked narrowing of joint space, severe sclerosis and definite deformity of bone contour

AHLBACK RADIOLOGICAL CRITERIA[1]

Grade 1: Joint Space Narrowing (Less Than 3 Mm)

Grade 2: Joint Space Obliteration

Grade 3: Minor Bone Attrition (0-5 Mm)

Grade 4: Moderate Bone Attrition (5-10 Mm)

Grade 5: Severe Bone Attrition (More Than 10 Mm)

The Department of Transfusion Medicine at Stanley medical college and Government Stanley Hospital had gracefully consented to prepare and provide Autologous Platelet Rich Plasma [7].

About 350 ml of venous blood was collected from the patient.

The Patient was blinded from knowing the amount of blood collected. The collected blood was centrifuged in a refrigerated centrifuge and Platelet Rich Plasma [11] was separated after removing red blood cells and buffy coat. The whole process of separating Platelet Rich Plasma was standardized and done under strict aseptic precautions.

The process yielded packed cells and fresh frozen plasma which was transfused back to the patient.

The Patients baseline platelet count and leucocyte count were determined and Platelet Rich Plasma was quantified as having eight to ten times the baseline valve of platelets. The Concentration of Platelets in final product were corroborated by the Department of Transfusion Medicine on a periodic basis.

We in this study did not use leucocyte filter and the final Platelet Rich Plasma contained minute traces of leucocytes. About 10 ml of blood was removed from the control group and was subjected to routine laboratory testing.

INJECTION PROTOCOL:

The Injection procedure was performed in Emergency operation theatre. The Patient was placed supine on the operation table. Parts painted and draped. Under sterile aseptic precautions 8 ml of Platelet Rich Plasma mixed with 2 ml of calcium gluconate was injected into the supra patellar pouch of knee or into the joint cavity from medial approach sterile bandaging given. The Patient is advised bed rest for 2 days.

The Patient is advised to avoid NSAIDS for 2 days before and after injection. Paracetamol in doses of 500mg is allowed in cases of febrile illness or discomfort due to pain. In the control group, 8ml of Normal Saline is injected in to the suprapatellar pouch of the patients.

The Patients are advised to carry on with their regular routine work from Day 2.

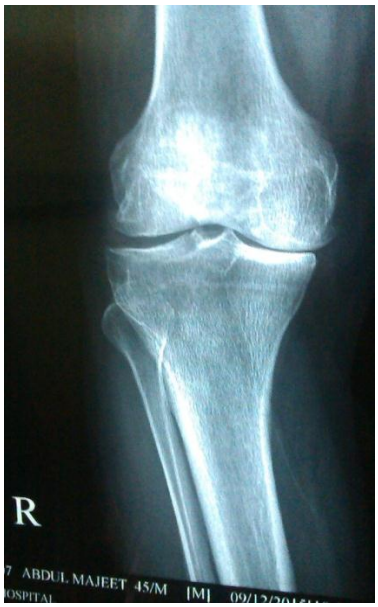
OUTCOME ANALYSIS:

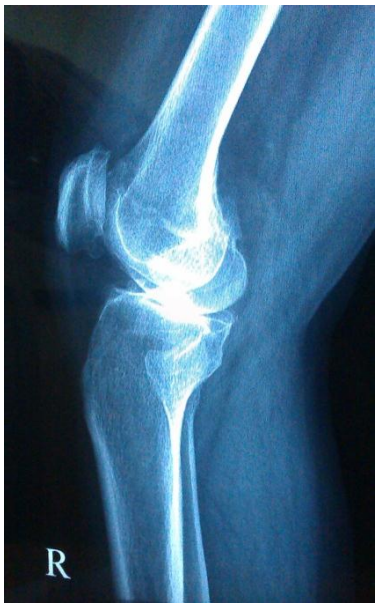
The study group and the control group are advised to follow up at 6 weeks, 3 months and 6 months .Outcome analysis for the efficacy was done for reduction in pain, reduction in stiffness and improvement in physical function using WOMAC scale.

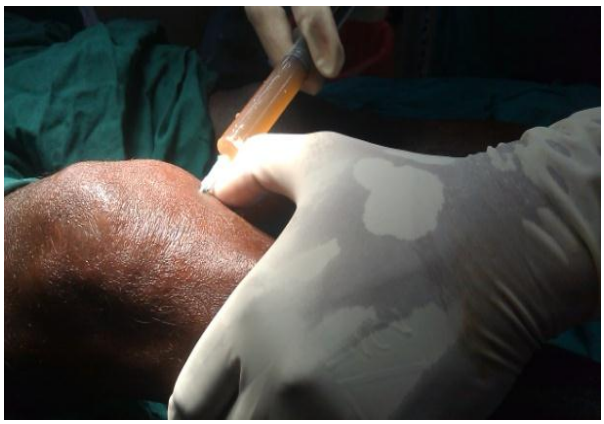
The Patients were also assessed for reduction in pain using Visual analog scale both at pre injection and at 6 months post injection.

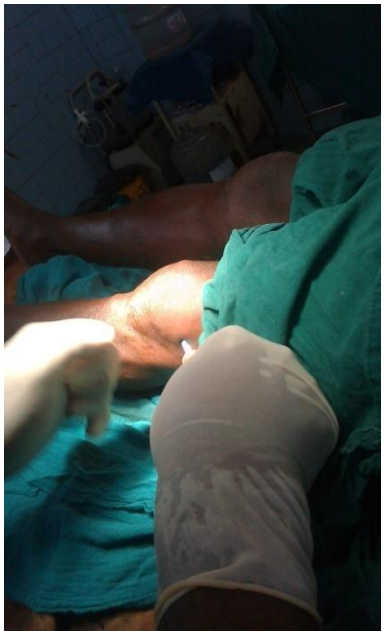
CASE ILLUSTRATION NO.1

Abdul majeet presented to the OPD with c/o chronic pain in both knees for the past 2 yrs. He had exhausted all conventional mode of therapies. The X ray showed clear joint space narrowing of medial compartment with marginal osteophytes. He was taken up for Platelet Rich Plasma.



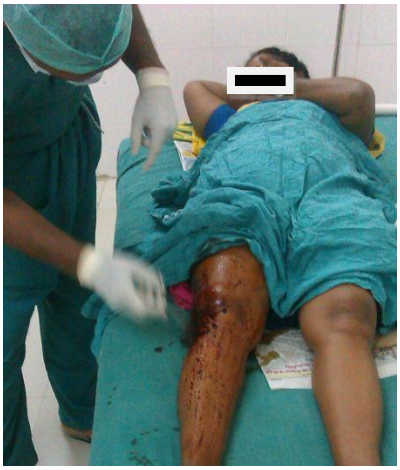


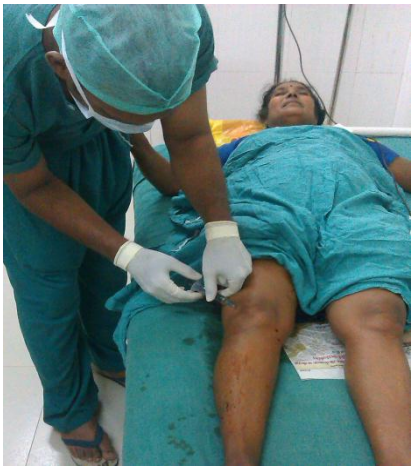


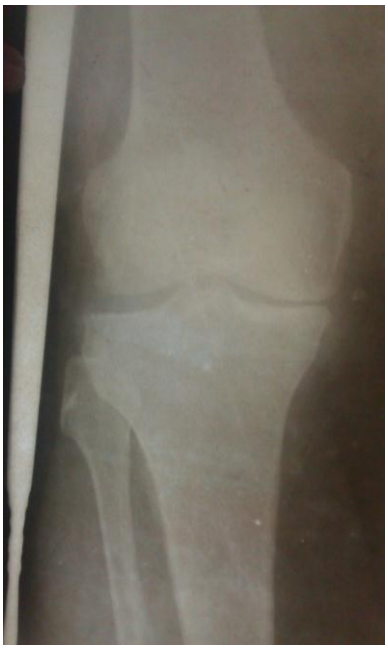


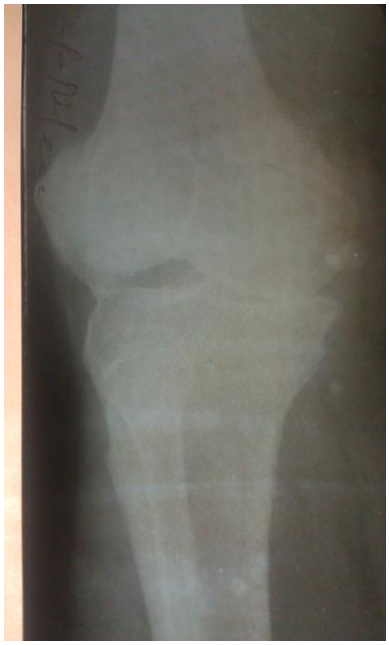
CASE ILLUSTRATION 2:

Valarmathi , 45/F, presented to OPD with c/o chronic knee pain .she was diagnosed as having Kellgren Lawrence grade 3 osteoarthritis in both her knees. She was treated by intraarticular Platelet Rich Plasma injection.



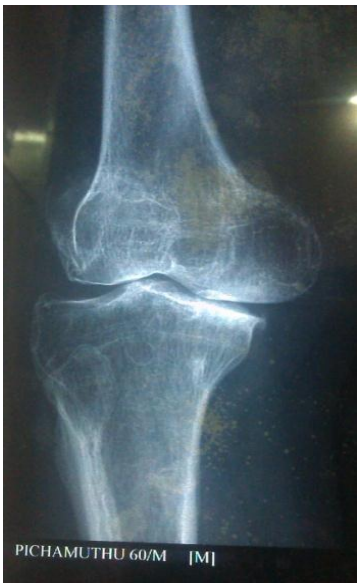


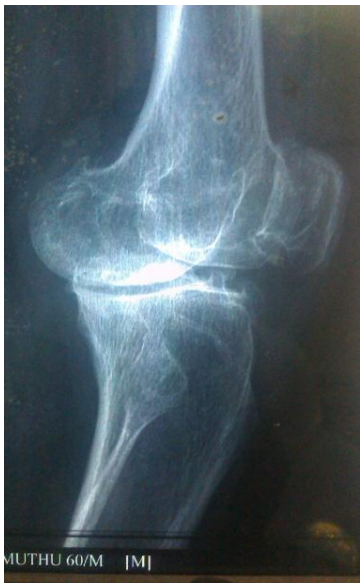




CASE ILLUSTRATION 3:

Pichamuthu 60/M, presented to the OPD with c/o chronic pain over both knees .He was facing difficulty in squatting, climbing stairs. Radiologic investigation revealed Kellgren-Lawrence grade 4 osteoarthritis. He was treated by intraarticular Platelet Rich Plasma injection.





THE WOMAC (WESTERN ONTARIO AND MCMASTER UNIVERSITIES) INDEX OF OSTEOARTHRITS[5, 17]

Overview:

The WOMAC (Western Ontario and McMaster Universities) index is used to assess patients with osteoarthritis of the hip or knee using 24 parameters. It can be used to monitor the course of the disease or to determine the effectiveness of Therapy

Scale of difficulty: 0 = None, 1 = Slight, 2 = Moderate, 3 = Very, 4 = Extremely

CIRCLE ONE NUMBER FOR EACH ACTIVITY

PAIN

1. Walking 0 1 2 3 4
2. Stair Climbing 0 1 2 3 4
3. Nocturnal 0 1 2 3 4
4. Rest 0 1 2 3 4
5. Weight bearing 0 1 2 3 4

STIFFNESS

1. Morning stiffness 0 1 2 3 4
2. Stiffness occurring later in the day 0 1 2 3 4

PHYSICAL FUNCTION

1. Descending stairs 0 1 2 3 4

2. Ascending stairs 0 1 2 3 4

3. Rising from sitting 0 1 2 3 4

4. Standing 0 1 2 3 4

5. Bending to floor 0 1 2 3 4

6. Walking on flat surface 0 1 2 3 4

7. Getting in / out of car 0 1 2 3 4

8. Going shopping 0 1 2 3 4

9. Putting on socks 0 1 2 3 4

10. Lying in bed 0 1 2 3 4

11. Taking off socks 0 1 2 3 4

12. Rising from bed 0 1 2 3 4

13. Getting in/out of bath 0 1 2 3 4

14. Sitting 0 1 2 3 4

15. Getting on/off toilet 0 1 2 3 4

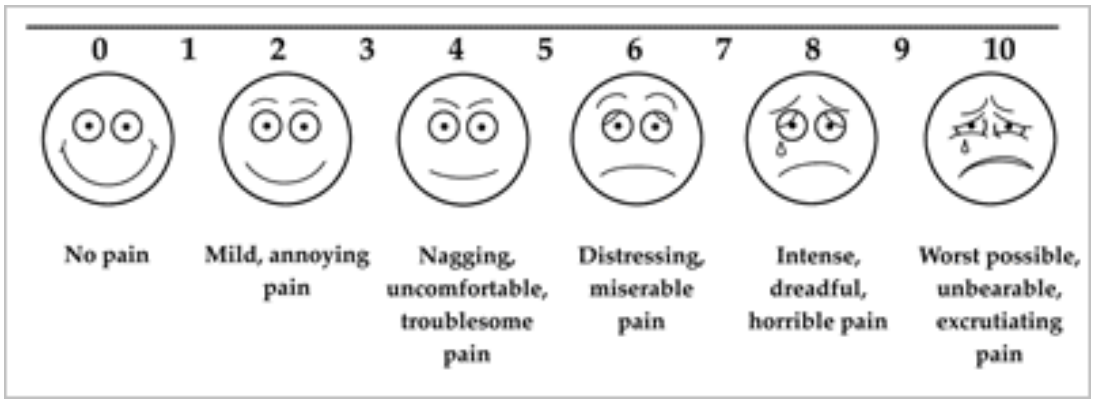
16. Heavy domestic duties 0 1 2 3 4

17. Light domestic duties 0 1 2 3 4.

Total Score: _____ / 96 = _____%

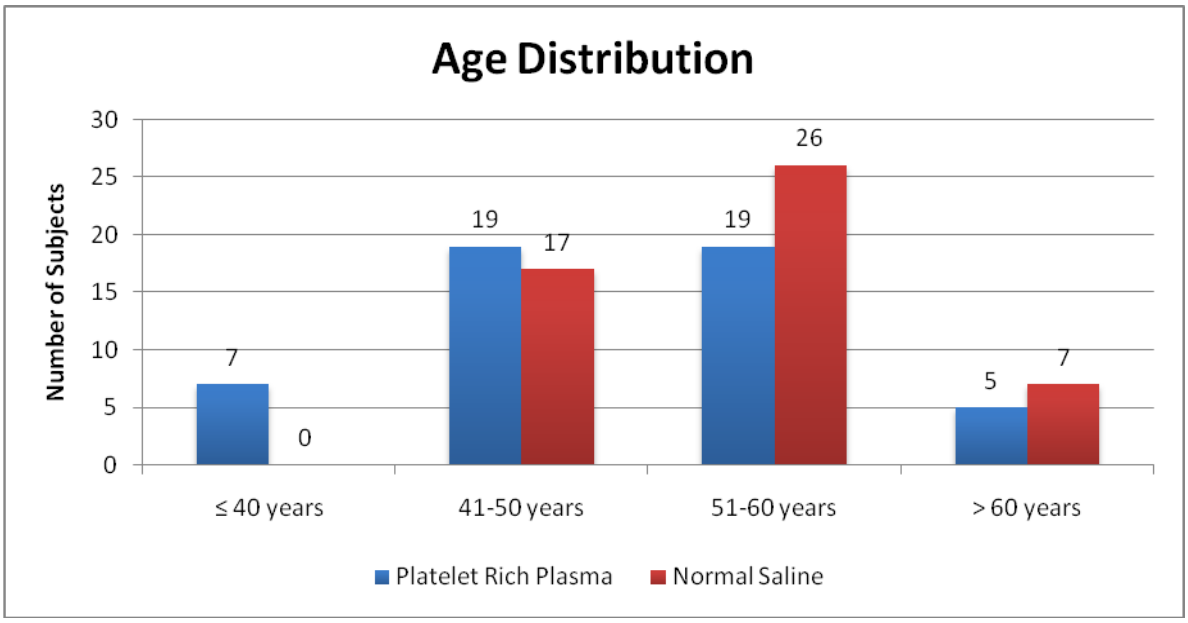
VISUAL ANALOG SCALE FOR PAIN

The Pain, the Patient perceive is graded on a visual scale and the score calculated.



STATISTICAL ANALYSIS

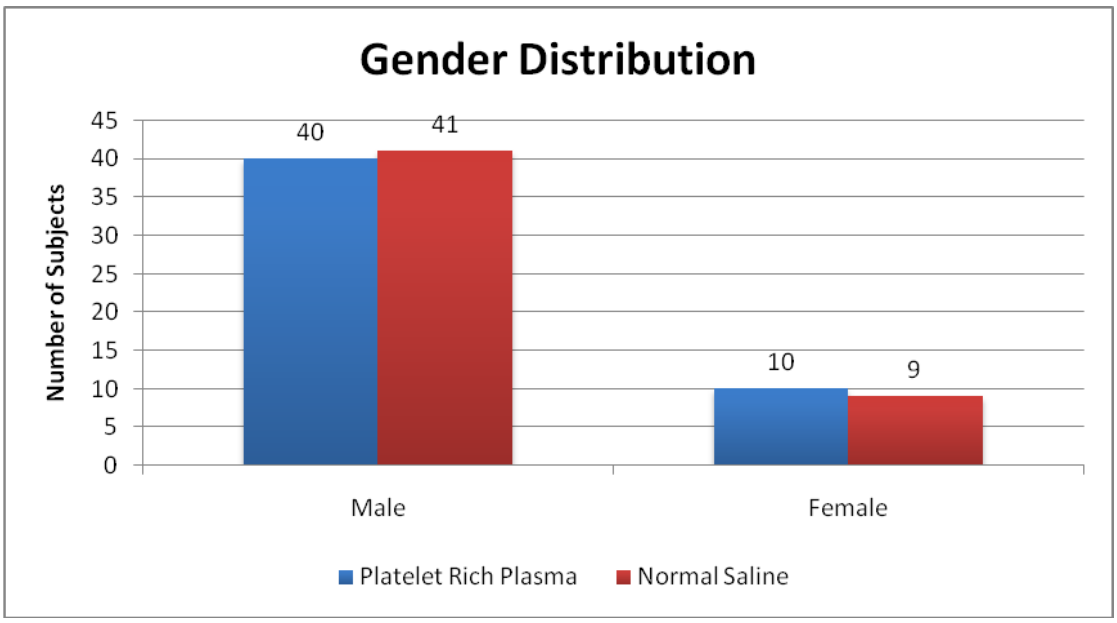
Age Distribution



Age Distribution	Platelet Rich Plasma	%	Normal Saline	%
≤ 40 years	7	14.00	0	0.00
41-50 years	19	38.00	17	34.00
51-60 years	19	38.00	26	52.00
> 60 years	5	10.00	7	14.00
Total	50	100	50	100

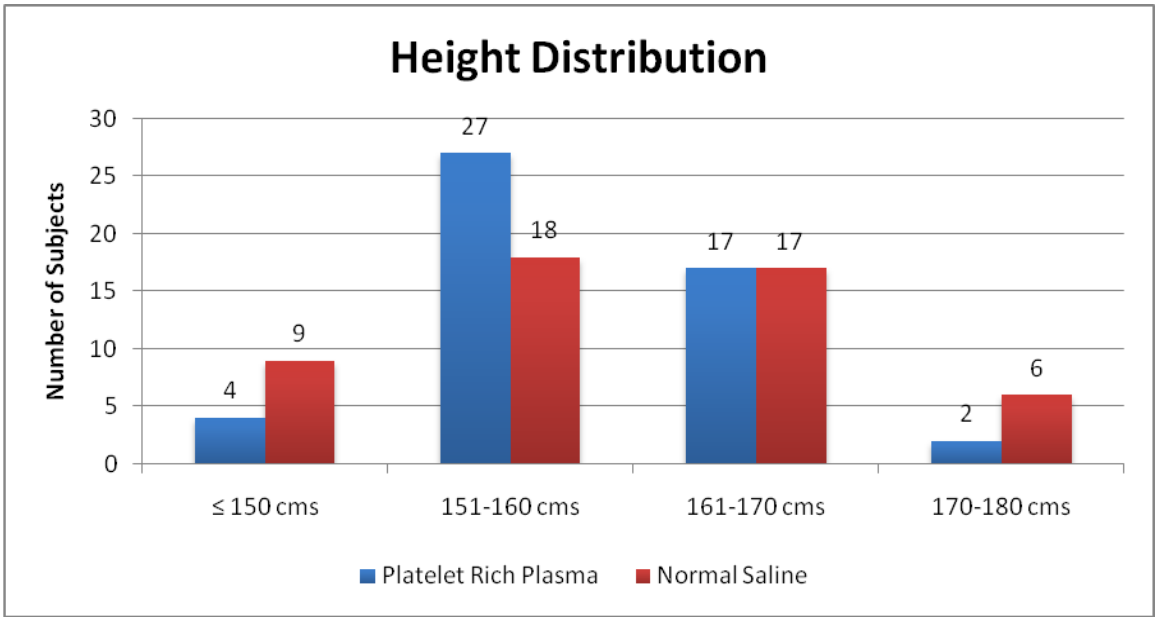
Age Distribution	Platelet Rich Plasma	Normal Saline
N	50	50
Mean	49.92	54.16
SD	7.72	5.36
P value Unpaired t Test	**0.1120**	

Gender Distribution



Gender Distribution	Platelet Rich Plasma	%	Normal Saline	%
Male	40	80.00	41	82.00
Female	10	20.00	9	18.00
Total	50	100	50	100
P values Chi Squared Test			**0.0833**	

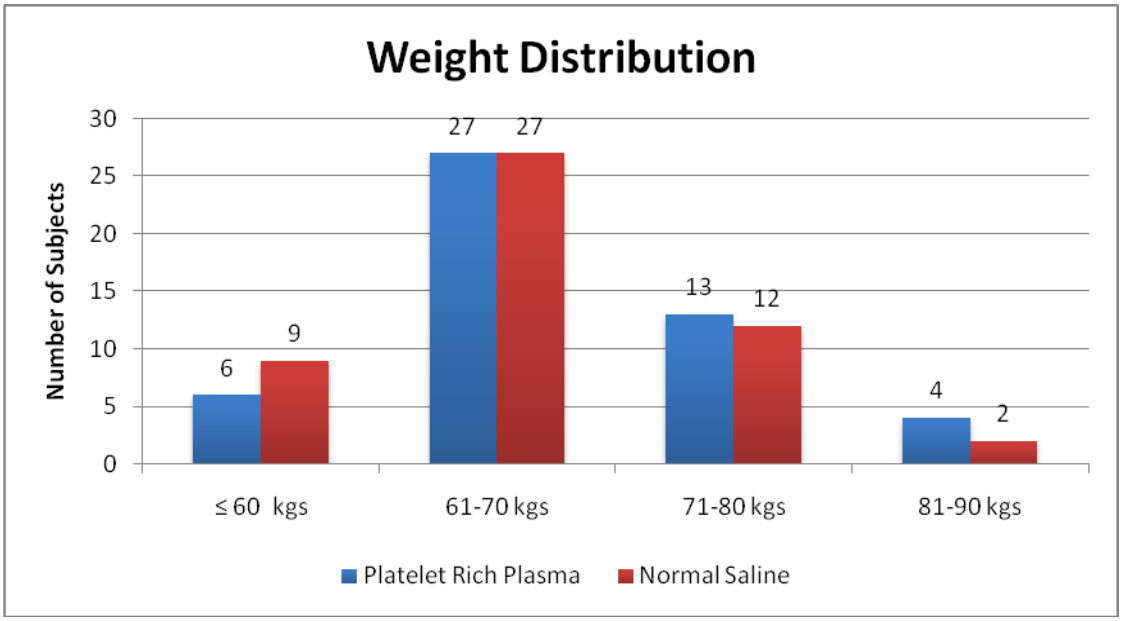
Height Distribution



Height Distribution	Platelet Rich Plasma	%	Normal Saline	%
≤ 150 cms	4	8.00	9	18.00
151-160 cms	27	54.00	18	36.00
161-170 cms	17	34.00	17	34.00
170-180 cms	2	4.00	6	12.00
Total	50	100	50	100

Height Distribution	Platelet Rich Plasma	Normal Saline
N	50	50
Mean	159.66	159.68
SD	6.63	8.39
P value Unpaired t Test	**0.9895**	

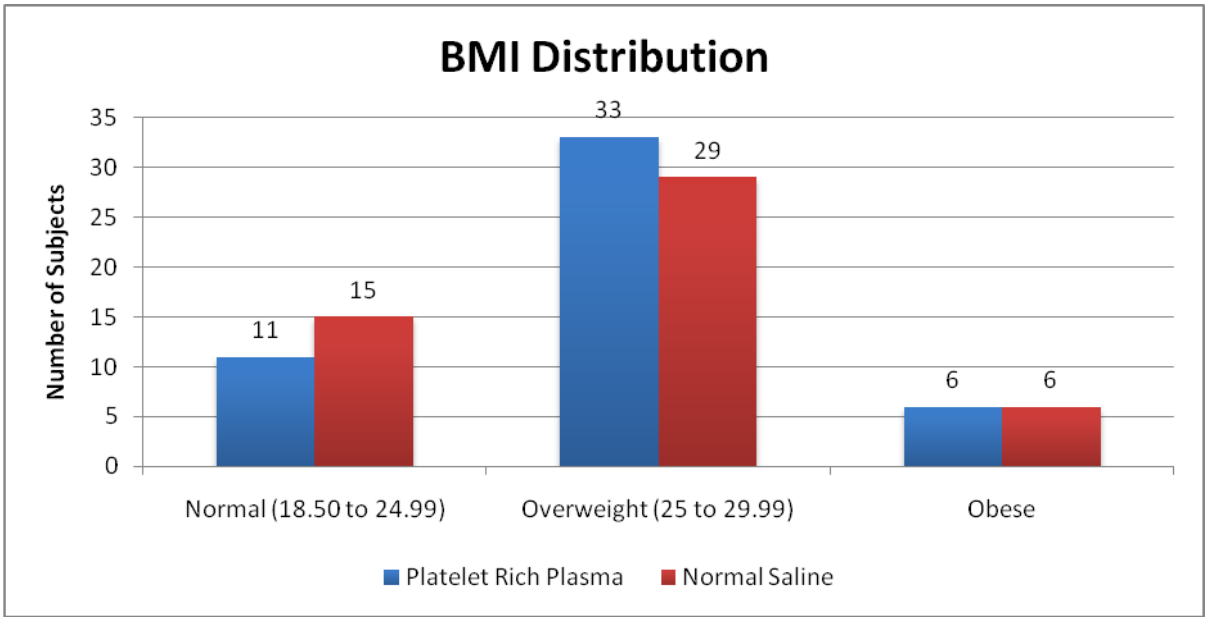
Weight Distribution



Weight Distribution	Platelet Rich Plasma	%	Normal Saline	%
≤ 60 kgs	6	12.00	9	18.00
61-70 kgs	27	54.00	27	54.00
71-80 kgs	13	26.00	12	24.00
81-90 kgs	4	8.00	2	4.00
Total	50	100	50	100

Weight Distribution	Platelet Rich Plasma	Normal Saline
N	50	50
Mean	68.62	67.66
SD	6.84	6.63
P value Unpaired t Test	**0.4777**	

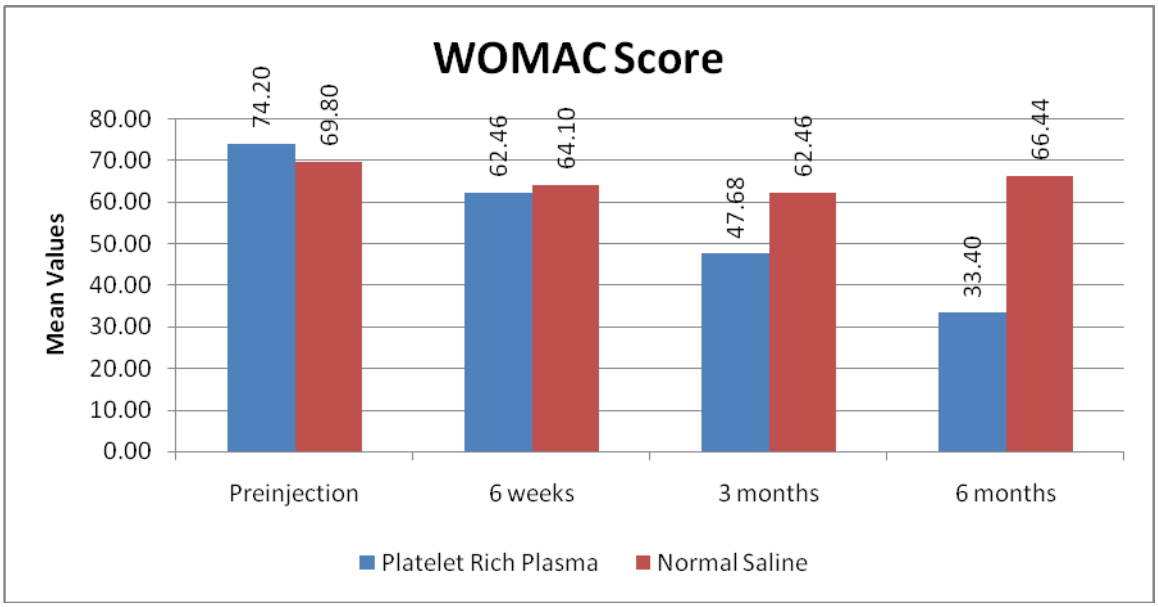
BMI Distribution



BMI Distribution	Platelet Rich Plasma	%	Normal Saline	%
Underweight (≤ 18.49)	0	0.00	0	0.00
Normal (18.50 to 24.99)	11	22.00	15	30.00
Overweight (25 to 29.99)	33	66.00	29	58.00
Obese	6	12.00	6	12.00
Total	50	100	50	100

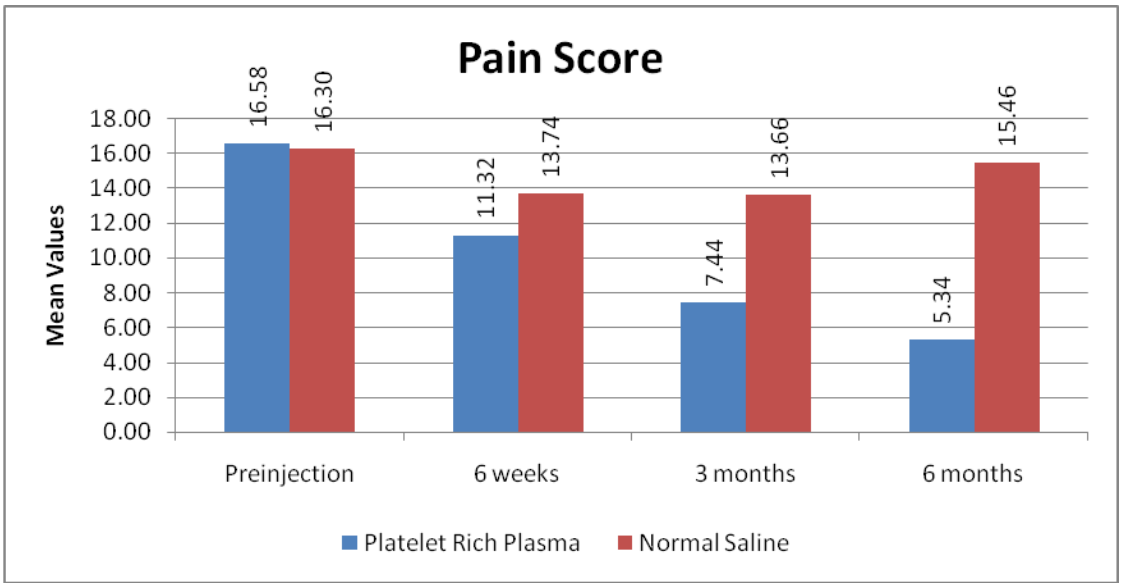
BMI Distribution	Platelet Rich Plasma	Normal Saline
N	50	50
Mean	26.97	26.64
SD	2.70	2.92
P value Unpaired t Test	**0.5507**	

WOMAC Score



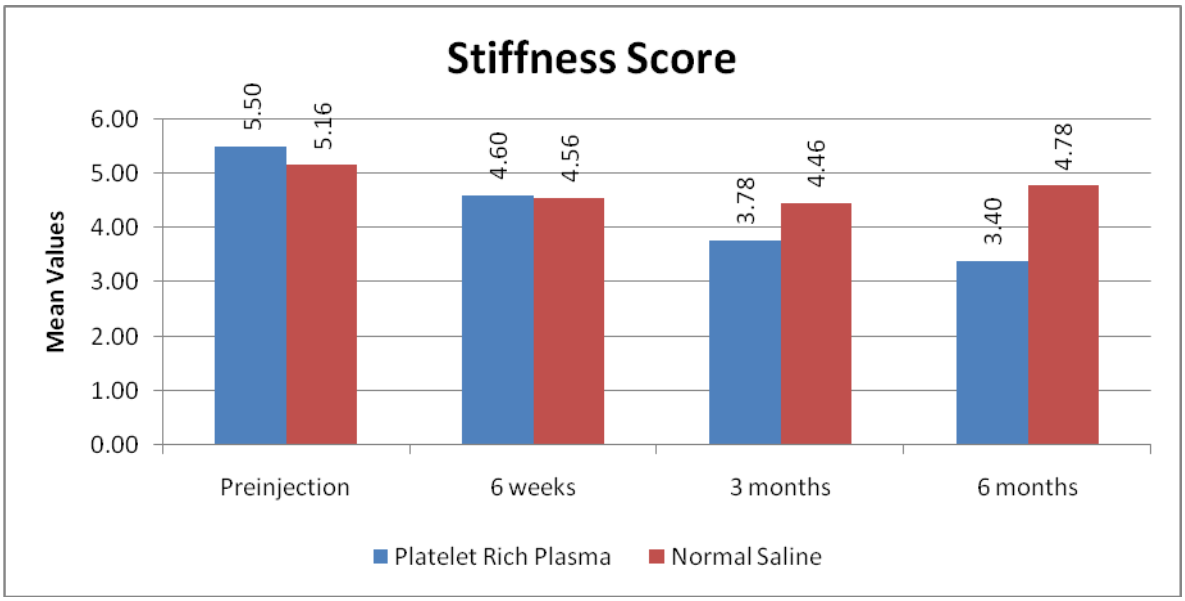
WOMAC Score		Preinjection	6 weeks	3 months	6 months
Platelet Rich Plasma	N	50	50	50	50
	Mean	74.20	62.46	47.68	33.40
	SD	4.85	6.60	8.15	7.59
Normal Saline	N	50	50	50	50
	Mean	69.80	64.10	62.46	66.44
	SD	4.68	5.50	5.44	5.01
P value Unpaired t Test		**0.1804**	**0.0000**	**0.0000**	**0.0000**

Pain Score



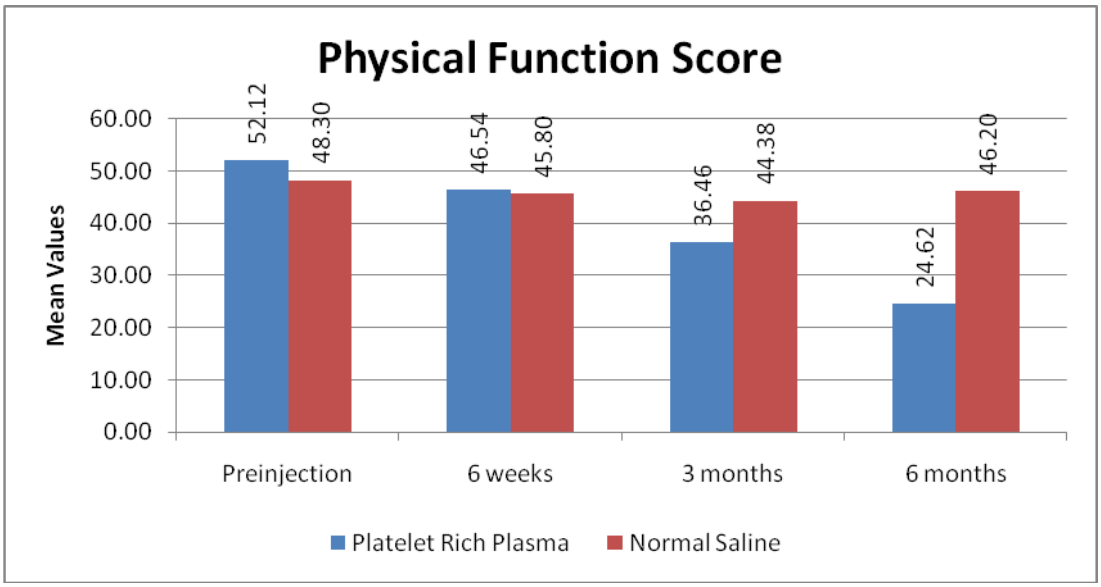
Pain Score		Preinjection	6 weeks	3 months	6 months
Platelet Rich Plasma	N	50	50	50	50
	Mean	16.58	11.32	7.44	5.34
	SD	3.08	2.76	1.93	1.42
Normal Saline	N	50	50	50	50
	Mean	16.30	13.74	13.66	15.46
	SD	2.39	2.28	2.35	1.94
P value Unpaired t Test		**0.6132**	**0.0000**	**0.0000**	**0.0000**

Stiffness Score



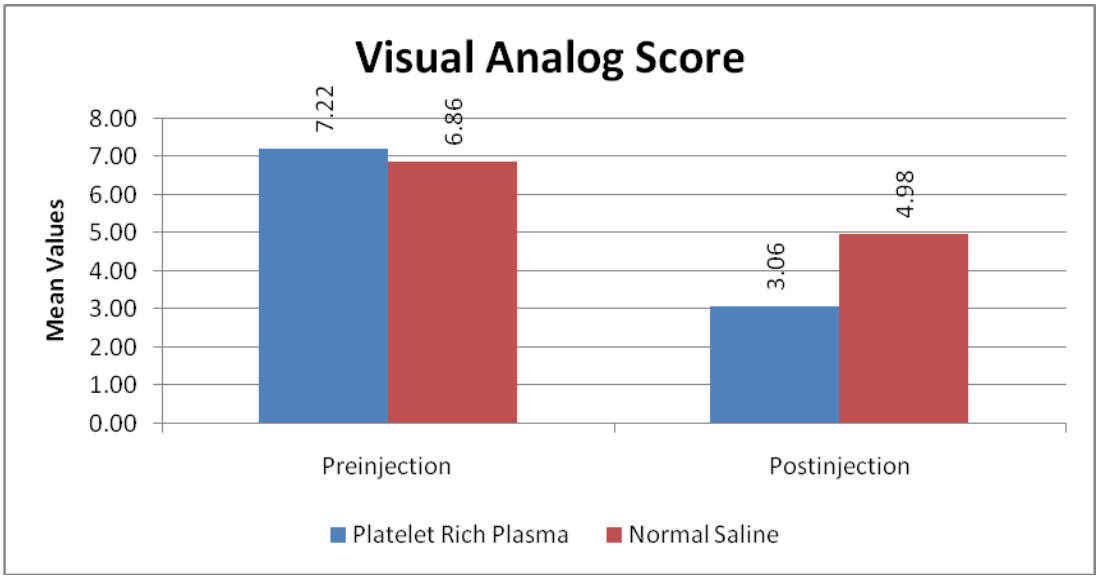
Stiffness Score		**Preinjection**	**6 weeks**	**3 months**	**6 months**
Platelet Rich Plasma	N	50	50	50	50
	Mean	5.50	4.60	3.78	3.40
	SD	1.22	1.20	1.09	1.14
Normal Saline	N	50	50	50	50
	Mean	5.16	4.56	4.46	4.78
	SD	0.93	0.81	0.73	0.84
P value Unpaired t Test		**0.1204**	**0.8453**	**0.0004**	**0.0000**

Physical Function Score



Physical Function Score		Preinjection	6 weeks	3 months	6 months
Platelet Rich Plasma	N	50	50	50	50
	Mean	52.12	46.54	36.46	24.62
	SD	3.77	4.53	6.49	6.33
Normal Saline	N	50	50	50	50
	Mean	48.30	45.80	44.38	46.20
	SD	3.17	3.91	3.88	3.67
P value Unpaired t Test		**0.3840**	**0.0000**	**0.0000**	**0.0000**

Visual Analog Score



Visual Analog Score		Preinjection	Postinjection
Platelet Rich Plasma	N	50	50
	Mean	7.22	3.06
	SD	0.97	1.24
Normal Saline	N	50	50
	Mean	6.86	4.98
	SD	0.81	0.89
P value Unpaired t Test		**0.0473**	**0.0000**

DISCUSSION

Osteoarthritis is a disorder of synovial joints characterized by focal loss of hyaline cartilage with proliferation of new bone and remodeling of joint contour, mainly due to uncoupling of balance between cartilage regeneration and degeneration. Osteoarthritis is a dynamic repair process of synovial joints that may be triggered a variety of insults.

We in our study had randomly chosen 100 patients with classic findings of Osteoarthritis and divided them in to two groups. Both the groups were comparable on baseline characteristics of age, height, weight, BMI, pre injection WOMAC score. Fifty of these patients were administered an intra articular injection of Platelet Rich Plasma and other fifty received Normal Saline.

The Efficacy of Platelet Rich Plasma in reducing pain ,stiffness and physical function were assessed and scored on WOMAC index for both study and control group. The Results were analysed using unpaired t-test and chi-square test.

Age distribution revealed mean age in group I to be 49.92 and the mean age in Group II was 54.16.The p-value derived using unpaired t-test is 0.1120, rendering age factor insignificant.

Gender distributions were comparable on both groups with 80 % being male 20% being female. The p-valve using chi square test is 0.0833 .The Gender factor was insignificant.

The mean height in group I was 159.66 and the mean height in Group II was 159.68.The p-valve using unpaired t test turned insignificant (0.9895).

The mean weight, in group I was 68.62 and group II was 67.66 with p-valve of 0.4777(insignificant).

The mean BMI was 26.97 in group I and 26.64 in Group II. The p-valve is 0.5507(insignificant).

Thus the study ensured that all patients were comparable on baseline characters.

The Global WOMAC showed a mean of 74.2 at pre injection period which decreased to 62.46 at 6 weeks follow up and 47.68 at 3 months and declining to 33.40 at 6 months.

The study showed a significant decrease in global WOMAC score, which was also consistent throughout the study period.

The Individual variables such as pain, stiffness and physical function were assessed. Mean score for pain showed a decrease from 16.58 to 11.32 at 6 weeks post injection .At the end of 6 months follow up, the mean was 5.34.

The mean score for pain in group II showed a marginal decreased from 16.30 to 13.74 at 6 weeks but returned to 15.46 at 6 months follow up.

The p-value using unpaired t-test showed significant improvement. Secondary variable stiffness showed significance difference at 3 months follow up and 6 months follow up.

The mean of Physical function decreased from a pre injection score of 52.12 to 24.62 at 6 months follow up in Group I.

Group II showed a marginal dip in mean scores from 48.30 to 45.80 and to 44.38 at 3 months .The scores leveled at 46.20 at the end of 6 months

Visual analog score showed a decrease in mean of 7.22 to 3.06 which denoted a change of patient's perception of pain from intense, dreadful, horrible pain to mild annoying pain in Group I.

Group II showed a marginal dip from 6.86 to 4. 98 on mean, showing insignificant change in pain.

CONCLUSION

The Epidemic of Modernization coupled with effective health care delivery has led to an expanded lifespan of human beings. The focus of health care providers is undergoing a drift towards non communicable and degenerative disorders.

Osteoarthritis represents a failure of diarthrodial joint, characterized by degenerative changes in articular cartilage of joint. The management of Osteoarthritis has undergone a sea change during the last century. Osteoarthritis has been managed by conservative methods like lifestyle changes, physiotherapy and surgical methods like joint replacement arthroplasty, depending upon the stage of the disorder.

A constant search for molecules that could aid in cartilage regeneration, thus interfering in disease process has thrown up surprises. One such ideology is, garnering the beneficial effect of growth factors in platelets to regenerate cartilage in a synovial joint.

Our study relied on injecting a highly concentrated mix of platelets into joint cavity and observing the patients for reduction in symptoms of pain, stiffness and improvement in physical function .our study has revealed a consistent reduction in pain and stiffness and a clear improvement in lifestyle of the patients.

Our study has thrown up an interesting choice of treatment modality using Platelet Rich Plasma in the treatment of Knee Osteoarthritis and it has proved efficacious in the observation period of six months.

COMPLICATIONS:

There were no major complications or incidences of infection in our study group and control group.

LIMITATIONS:

Long term follow up needed with M.R.I to assess the regeneration of cartilage.

MASTER CHART

GROUP-I PLATELET RICH PLASMA

S.NO	NAME	**AGE**	**SEX**	**HEIGHT**	**WEIGHT**	**BMI**	**PREINJECTION SCORE**	**WOMAC SCORE--6 WEEKS**	**WOMAC SCORE -3 MONTHS**	**WOMAC SCORE-6 MONTHS**	**VAS PRE INJECTION**	**VAS POST INJECTION**
1.	PERIYASAMY	52	M	160	65	25.4	82	72	66	52	8	4
2.	FATHIMA	56	F	152	69	29.9	86	80	72	66	8	5
3.	ARUMUGHAM	58	M	156	70	28.8	78	70	62	50	6	2
4.	MANIKANDAN	40	M	164	65	24.2	74	66	48	30	6	2
5.	ARUMUGHAM	38	M	160	64	25	72	60	30	24	6	2
6.	LOGANATHAN	56	M	148	62	28.3	70	60	44	30	6	2
7.	MUSTAFA	52	M	156	64	26.3	78	64	42	32	8	4
8.	YUSUF	46	M	152	63	27.3	74	52	32	26	6	2
9.	VALARMATHY	45	F	150	58	25.8	72	66	46	28	8	2
10	SHANMUGHAM	48	M	160	78	30.5	68	56	44	38	6	5
11	MOHAN	48	M	175	76	24.8	66	52	46	28	6	2
12	SURESH	45	M	168	64	22.7	66	52	44	32	6	4
13	PARTHASARATHY	60	M	162	70	26.7	74	52	40	36	6	2
14	ABDUL GAFFER	58	M	152	63	27.3	68	56	42	32	6	2
15	JEYAKUMAR	42	M	160	68	26.6	68	52	38	28	6	2
16	STELLAMARY	38	F	145	62	29.5	68	56	40	36	6	2
17	ESTHAR	48	F	142	66	32.7	66	46	40	32	6	3

18	PERIYASAMY	62	M	160	78	30.5	78	66	52	40	6	2
19	NAGAVATHY	56	F	156	56	23	78	66	54	30	8	2
20	VENKATESAN	56	M	164	66	24.5	72	66	52	40	6	4
21	MOHAN	48	M	160	68	26.6	70	66	52	32	8	2
22	GOVINDARAJ	45	M	158	68	27.2	72	62	54	38	6	2
23	RASUL	48	M	162	72	27.4	74	58	40	26	8	2
24	CHANDRASEKAR	56	M	170	75	26	68	56	38	24	8	3
25	SUBAA	48	F	156	66	27.1	78	62	42	30	8	5
26	ANWAR	44	M	160	68	26.6	76	66	52	30	6	3
27	SANTHANAM	52	M	170	74	25.6	74	66	54	40	8	6
28	ANNAMALAI	62	M	163	58	21.8	68	54	40	30	6	2
29	KARTHIK	48	M	160	69	27	72	66	44	32	8	5
30	MD.ASIF	56	M	162	78	29.7	70	62	50	40	8	5
31	JAYA	52	F	156	70	28.8	72	62	46	28	8	4
32	ANNAMMA	48	F	160	68	26.6	76	64	38	26	8	1
33	SIVA	56	M	158	68	27.2	70	66	52	38	8	4
34	ARUL	63	M	163	61	23	72	66	54	36	8	3
35	SHANMUGHAM	55	M	162	71	27.1	78	66	45	32	8	2
36	KUMAR	53	M	167	82	29.4	78	68	52	32	6	3
37	BOOPATHI	46	M	153	72	30.8	72	68	58	30	8	3
38	VIJAYA	45	F	158	60	24	78	62	42	28	8	3
39	VENKATESHWARULU	42	M	165	72	26.4	76	60	44	30	8	3
40	JOTHI	38	F	160	58	22.7	74	58	44	28	7	3
41	SOLOMON DOSS	53	M	157	81	32.9	78	66	52	40	8	5
42	RAVI	35	M	163	71	26.7	74	66	48	32	8	4

43	JAYARAMAN	41	M	156	67	27.5	82	68	56	40	8	6
44	SASIKUMAR	38	M	169	73	25.6	76	66	48	26	8	2
45	PERUMAL	42	M	173	82	27.4	84	70	58	32	8	3
46	VENKATESAN	38	M	168	69	24.4	74	52	40	26	8	2
47	UMAKANTH	58	M	157	86	34.9	78	65	49	28	8	3
48	PERUMAL	61	M	163	73	27.5	82	66	52	38	8	4
49	THIRUNAVUKARASU	59	M	154	59	24.9	80	72	58	36	8	3
50	SUBBAIAH	62	M	158	65	26	76	64	48	32	8	2

GROUP II- NORMAL SALINE

S.NO	NAME	AGE	SEX	HEIGHT	WEIGHT	BMI	PREINJECTION SCORE	WOMAC SCORE--6 WEEKS	WOMAC SCORE -3 MONTHS	WOMAC SCORE-6 MONTHS	VAS PRE INJECTION	VAS POST INJECTION
51.	KUMAR	48	M	165	69	25.34	68	60	66	68	8	7
52.	KALAIARASI	55	F	163	58	21.83	72	68	66	68	7	5
53.	SAROJA	61	F	158	62	24.84	74	65	62	70	8	6
54.	MARKANDAN	62	M	160	69	26.95	72	70	68	74	6	4
55.	EASWARAN	48	M	172	74	25.01	72	70	68	68	8	5
56.	THANGADURAI	46	M	178	73	23.04	68	60	60	62	7	4
57.	SELLAPANDIAN	60	M	167	69	24.74	72	70	70	70	7	5
58.	RAJA	58	M	159	70	27.69	68	58	56	62	6	4
59.	GUNA	49	M	163	68	25.59	66	66	58	58	6	5
60.	SHANMUGHAM	53	M	164	68	25.28	66	52	58	60	7	5
61.	ANBU	59	M	172	68	22.99	74	62	62	68	8	6
62.	KANNAN	49	M	165	63	23.14	66	58	56	66	6	6
63.	DHIVYA	53	F	167	59	21.16	64	54	52	60	7	5
64.	DHANABAKIYAM	63	F	156	68	27.94	73	62	69	75	6	4
65.	DHANASEKARAN	57	M	147	56	25.92	66	68	60	60	6	4
66.	ABDUL SHERIFF	59	M	159	79	31.25	72	62	60	66	8	6
67.	KALAIMATHI	54	F	163	65	24.46	68	62	58	60	7	5
68.	HARI	49	M	158	68	27.24	70	69	63	66	6	4
69.	MD.ASIF	61	M	163	72	27.1	72	60	65	70	7	4
70.	FATHIMA	53	F	153	68	29.05	69	59	58	70	6	4

71.	BABI	49	M	143	64	31.3	76	70	64	70	7	5
72.	THYAGU	63	M	173	81	27.06	78	68	66	70	8	4
73.	GANAPATHY	49	M	165	67	24.61	68	60	58	60	7	4
74.	YOGI	58	M	159	73	28.88	73	73	68	68	8	5
75.	SATHISH	52	M	156	68	27.94	69	64	58	60	6	5
76.	RAHAMATHULLA	59	M	166	72	26.13	74	68	63	69	8	6
77.	KAMALUDEEN	49	M	171	83	28.38	76	69	69	70	8	6
78.	CHINNATHAMBI	53	M	158	59	23.63	62	57	54	58	7	4
79.	ELUMALAI	48	M	148	57	26.02	65	66	60	58	6	4
80.	MANOHARAN	54	M	158	63	25.24	67	64	60	62	6	5
81.	KUMAR	55	M	172	69	23.32	70	68	63	66	7	6
82.	DEVA	59	M	164	79	29.37	68	66	66	66	6	4
83.	KRISHNAN	63	M	156	58	23.83	69	67	60	62	7	6
84.	BOOMINATHAN	56	M	165	63	23.14	70	64	65	70	6	5
85.	MARIMUTHU	49	M	147	59	27.3	68	63	60	68	7	6
86.	ELAVARASAN	56	M	146	63	29.56	56	48	48	56	6	4
87.	ARUMUGHAM	57	M	159	76	30.06	68	58	56	70	6	5
88.	PONNIAH	53	M	165	68	24.98	66	60	58	68	6	5
89.	KAMARAJ	46	M	158	67	26.84	72	66	62	68	8	7
90.	NAGOOR KANI	57	M	164	63	23.42	80	72	72	78	8	6
91.	CHANDRAN	56	M	145	69	32.82	78	72	72	72	7	6
92.	HANUMANTH	48	M	152	79	34.19	76	70	70	70	6	5
93.	SOLAI	58	M	167	75	26.89	72	70	70	70	7	4
94.	KANTHA	49	F	147	59	27.3	68	66	68	70	6	4

95.	KUMAR	44	M	142	68	33.72	58	58	58	58	6	5
96.	SANKU	45	M	159	68	26.9	67	65	66	68	7	4
97.	ARUNA	49	F	149	58	26.12	68	60	58	68	6	4
98.	KRISHNAN	56	M	158	63	25.24	74	68	66	70	8	6
99.	AMEEN	62	M	157	71	28.8	68	60	60	68	7	5
100.	JEYAM	59	F	163	75	28.23	74	70	70	70	8	6

WOMAC SCORE DETAILED- GROUP I

s.no	Name	**Preinjection score**			**6 weeks**			**3 months**			**6 months**		
		pain	**stiffness**	**Physical function**	**pain**	**stiffness**	**Physical function**	**pain**	**stiffness**	**Physical function**	**pain**	**stiffness**	**Physical function**
1	PERIYASAMY	18	8	56	14	6	52	12	4	50	8	4	40
2	FATHIMA	18	8	60	15	8	57	15	6	51	12	6	48
3	ARUMUGHAM	15	6	57	11	6	53	7	6	49	6	6	38
4	MANIKANDAN	20	6	48	16	4	46	10	2	36	6	2	22
5	ARUMUGHAM	18	4	50	12	4	44	5	2	23	4	2	18
6	LOGANATHAN	18	6	46	13	6	41	8	5	31	6	4	20
7	MUSTAFA	16	8	54	10	6	48	6	6	30	4	6	22
8	YUSUF	17	6	51	7	4	41	6	4	22	4	4	16
9	VALARMATHY	18	8	46	15	6	45	7	4	35	6	4	18
10	SHANMUGHAM	14	6	48	10	4	42	6	2	36	6	2	30
11	MOHAN	12	4	50	8	4	40	8	4	34	4	4	20
12	SURESH	12	4	50	8	4	40	6	4	34	4	4	24
13	PARTHASARATHY	15	4	55	8	4	40	7	3	30	4	3	29
14	ABDUL GAFFER	13	3	52	9	3	44	5	2	35	5	2	25
15	JEYAKUMAR	13	3	52	8	2	42	7	2	29	6	2	20
16	STELLAMARY	10	4	54	7	3	46	5	2	33	5	2	29

17	ESTHAR	8	4	54	4	2	40	4	2	34	4	2	26
18	PERIYASAMY	17	5	56	12	3	51	7	3	42	5	3	32
19	NAGAVATHY	22	6	50	12	6	48	8	5	41	6	3	21
20	VENKATESAN	16	5	51	12	4	50	8	4	40	6	2	32
21	MOHAN	12	4	54	9	3	54	5	3	44	4	2	26
22	GOVINDARAJ	14	6	52	9	6	47	8	6	40	6	6	26
23	RASUL	15	5	54	10	4	44	6	4	30	5	3	18
24	CHANDRASEKAR	17	6	45	12	5	39	7	4	27	5	2	17
25	SUBAA	20	6	52	12	4	46	7	4	31	5	4	21
26	ANWAR	19	6	51	14	6	46	10	4	38	6	4	20
27	SANTHANAM	15	7	52	11	6	49	8	5	41	6	3	31
28	ANNAMALAI	16	5	47	10	4	40	8	4	28	6	3	21
29	KARTHIK	22	5	45	18	4	44	9	3	32	6	3	23
30	MD.ASIF	20	6	44	14	6	42	7	5	38	5	5	30
31	JAYA	19	7	46	14	6	42	8	4	34	6	4	18
32	ANNAMMA	17	6	53	12	4	48	6	4	28	4	2	20
33	SIVA	16	6	48	12	6	48	8	4	40	7	4	27
34	ARUL	17	5	50	14	4	48	10	4	40	8	4	24
35	SHANMUGHAM	18	6	54	12	5	49	7	3	35	5	3	24
36	KUMAR	15	5	58	10	5	53	7	3	42	5	3	24
37	BOOPATHI	14	6	52	12	5	51	8	4	46	4	3	23

38	VIJAYA	18	6	54	10	5	47	6	3	33	4	3	21
39	VENKATESHWARULU	20	5	51	12	4	44	7	4	33	6	4	20
40	JOTHI	19	5	50	13	4	41	8	3	33	6	3	19
41	SOLOMON DOSS	16	6	56	10	5	51	6	5	41	4	5	31
42	RAVI	13	5	56	9	5	52	6	3	39	4	2	26
43	JAYARAMAN	21	6	55	12	5	51	9	4	43	6	4	30
44	SASIKUMAR	17	4	55	10	4	52	8	4	36	4	4	18
45	PERUMAL	22	6	56	15	5	50	9	4	45	4	3	25
46	VENKATESAN	14	5	55	7	3	42	5	3	32	4	3	19
47	UMAKANTH	18	7	53	13	5	47	7	5	37	5	4	19
48	PERUMAL	19	5	58	13	4	49	9	4	39	5	4	29
49	THIRUNAVUKARASU	20	6	54	16	5	51	10	3	45	6	3	27
50	SUBBAIAH	16	4	56	10	4	50	6	4	38	5	3	24

WOMAC SCORE –GROUP II –NORMAL SALINE

s.no	Name	PREINJECTION SCORE			6 WEEKS			3 MONTHS			6 MONTHS		
		pain	stiffness	Physical function	pain	stiffness	Physical function	pain	stiffness	Physical function	pain	stiffness	Physical function
1.	KUMAR	16	6	46	13	4	43	16	4	46	14	4	50
2.	KALAIARASI	18	6	48	16	6	46	17	4	45	17	5	46
3.	SAROJA	20	6	48	15	4	46	15	5	42	20	5	45
4.	MARKANDAN	15	6	51	14	6	50	16	5	47	19	6	49
5.	EASWARAN	15	5	52	13	5	52	15	4	51	13	5	50
6	THANGADURAI	16	6	46	12	6	42	12	6	42	14	6	42
7.	SELLAPANDIAN	18	6	48	16	6	48	16	6	48	16	6	48
8.	RAJA	18	4	46	13	3	42	13	3	40	16	4	42
9.	GUNA	17	4	45	17	4	45	14	4	40	14	4	40
10.	SHANMUGHAM	15	4	47	10	4	38	14	4	40	14	4	42
11.	ANBU	15	6	53	13	4	45	13	4	45	15	6	47
12.	KANNAN	17	4	45	13	4	41	13	4	39	18	4	44
13.	DHIVYA	17	4	43	12	4	38	12	4	36	14	4	42
14.	DHANABAKIYAM	18	5	50	12	4	46	16	4	49	16	4	55
15.	DHANASEKARAN	14	6	46	14	6	48	12	4	44	12	4	44

16.	ABDUL SHERIFF	13	6	53	10	4	48	10	4	46	14	4	48
17.	KALAIMATHI	18	4	46	14	4	44	14	4	40	14	4	42
18.	HARI	16	6	48	15	6	48	12	6	45	15	6	45
19.	MD.ASIF	13	6	53	10	4	46	13	6	46	15	6	49
20.	FATHIMA	14	4	51	10	4	45	10	4	44	14	6	50
21.	BABI	20	6	50	14	6	50	12	4	48	14	6	50
22.	THYAGU	19	6	51	15	4	49	15	4	47	15	5	50
23.	GANAPATHY	16	6	46	12	5	43	12	5	41	14	5	41
24.	YOGI	18	5	50	18	5	50	15	5	48	15	5	48
25.	SATHISH	14	4	51	12	4	48	10	4	44	12	4	44
26.	RAHAMATHULLA	15	6	53	11	5	52	11	5	47	16	6	47
27.	KAMALUDEEN	17	6	53	15	5	49	15	5	49	16	5	49
28.	CHINNATHAMBI	14	4	44	13	3	41	10	3	41	14	3	41
29.	ELUMALAI	15	5	45	15	5	46	15	5	40	14	4	40
30.	MANOHARAN	17	5	45	15	5	44	15	5	40	16	5	41
31.	KUMAR	20	5	45	18	5	45	13	5	45	16	5	45
32.	DEVA	19	5	44	17	5	44	17	5	44	17	5	44
33.	KRISHNAN	14	4	51	14	4	49	12	4	44	14	4	44
34.	BOOMINATHAN	15	4	51	13	4	47	14	4	47	15	4	51
35.	MARIMUTHU	16	6	46	13	4	46	13	4	43	16	4	48
36.	ELAVARASAN	12	4	40	12	4	32	12	4	32	14	4	38

37.	ARUMUGHAM	13	5	50	11	4	43	9	4	43	18	4	48
38.	PONNAIAH	16	4	46	10	4	46	10	4	44	16	4	48
39.	KAMARAJ	19	5	48	15	5	46	13	4	45	15	5	48
40.	NAGOORKANI	22	7	51	20	5	47	20	5	47	22	6	50
41.	CHANDRAN	20	7	51	16	5	51	16	5	51	16	6	50
42.	HANUMANTH	18	6	52	16	6	48	16	6	48	16	6	48
43.	SOLAI	19	5	48	15	5	50	17	5	48	17	5	48
44.	KANTHA	13	5	50	11	5	50	13	5	50	15	5	50
45.	KUMAR	12	4	42	12	4	42	12	4	42	12	4	42
46.	SANKU	14	4	49	15	4	46	18	5	43	18	5	45
47.	ARUNA	16	4	48	14	4	42	12	4	42	17	5	46
48.	KRISHNAN	18	6	50	14	4	50	14	4	48	16	4	50
49.	AMEEN	13	5	50	13	4	43	13	4	43	17	5	46
50.	JEYAM	18	6	50	16	4	50	16	4	50	16	4	50

PROFORMA

S.No :

Patient name :

Age/Sex : IP No:

Occupation :

Address :

Phone no : Date of Admission:

Presenting complaints:

History of Presenting illness

Past History

Occupational History:

Family History:

General Examination:

Regional Examination:

Provisional Diagnosis:

Radiologic Examination:

Final Diagnosis:

Interventional Procedure: Platelet Rich Plasma injection:

Outcome Analysis WOMAC score:

Visual analog score: