

Understanding Post-ACL Injury Osteoarthritis: Pathophysiology and Management Insights

Rohit Rathore^{*1}, Drishti Pachauri², Vratika Arya³, Ritu Singh⁴, Reetuja⁵, Mukul Mudgal⁶, Priyanshi Aggarwal⁷, Aditya Maddhesiya⁸, Diksha Shubhangi⁹, Mohit Anand¹⁰

^{*1}Assistant Professor, Noida International University, SOAHS, Department of Physiotherapy, Uttar Pradesh, India.

²Assistant Professor, Noida International University, SOAHS, Department of Physiotherapy, Uttar Pradesh, India.

³Assistant Professor, Noida International University, SOAHS, Department of Radiology & Imaging Technology, Uttar Pradesh, India.

⁴Assistant Professor, Noida International University, SOAHS Department of Physiotherapy, Uttar Pradesh, India.

⁵PG Student, Department of Physiotherapy, SGT University, Haryana, India.

⁶Assistant Professor, Noida International University, SOAHS, Uttar Pradesh, India.

⁷Assistant Professor, Noida International University, SOAHS, Department of Physiotherapy, Uttar Pradesh, India.

⁸UG Student, Noida International University, SOAHS Department of Physiotherapy, Uttar Pradesh, India.

⁹UG Student, Noida International University, SOAHS Department of Physiotherapy, Uttar Pradesh, India.

¹⁰UG Student, Noida International University, SOAHS Department of Physiotherapy, Uttar Pradesh, India.

*Corresponding Author:

Rohit Rathore

Assistant Professor, Noida International University, SOAHS, Department of Physiotherapy.

Email ID: drrohitthephysio@gmail.com

Cite this paper as: Rohit Rathore, Drishti Pachauri, Vratika Arya, Ritu Singh, Reetuja, Mukul Mudgal, Priyanshi Aggarwal, Aditya Maddhesiya, Diksha Shubhangi, Mohit Anand, (2025) Understanding Post-ACL Injury Osteoarthritis: Pathophysiology and Management Insights. *Journal of Neonatal Surgery*, 14 (9s), 586-590.

ABSTRACT

PTOA or post-traumatic osteoarthritis is arising as a result of joint injuries, ACL damage being the most predominant. ACL tears or injuries are prevalent among working individuals of diverse backgrounds. The PTOA after ACL injury develops is associated with mere structure, biology, and mechanical factors, which can also be termed neuromuscular, and altogether disturb the homeostasis of the joint, leading to increased cartilage wastage and smoother equators or erosion. Techniques in surgeries and rehabilitation still the rate of PTOA is extremely high due to many factors more than 50-90 have been observed within a decade. This review helps me understand the mechanisms of PTOA progression, measures for preventing it using ACL reconstruction, ACL-R, and the changes in the method of diagnosis and treatment. The need for early-stage intervention is underscored by multi-PTOA rehabilitation. Strapping the gaps is crucial to decreasing the impact of PTOA, as surgeons improve the lives of people suffering from it.

Keywords: PTOA, anterior cruciate ligament (ACL) injury, cartilage degeneration, adhesions, inflammation, ACL reconstruction (ACL-R), biomarkers, imaging, neuromuscular training, and rehabilitation.

1. INTRODUCTION

OA has emerged as one of the most common causes of disability globally, leading to significant socio-economic challenges because of its effect on mobility and the overall quality of life. OA has always been perceived as a degenerative disease associated with aging; however, there is a specific subtype that arises due to distinct joint injuries- post-traumatic osteoarthritis (PTOA). PTOA is estimated to account for almost 12% of the symptomatic OA cases, and even more so in the younger population where primary injuries such as ligament tears or fractures are common in active/working populations (Lieberthal et al., 2015). This subtype has the advantage of having a definite 'starting point', making it more amenable for intervention compared to idiopathic OA (Wang et al., 2020).

Injuries to the Anterior Cruciate Ligament (ACL) are among the leading causes of PTOA (post-traumatic osteoarthritis). The ACL is essential in controlling knee joint stability and preventing excessive displacement and rotation of the tibial bone relative to the knee. ACL injuries are prevalent among athletes involved in high-impact sports with sudden stopping, rotating,

or directional change, such as basketball. The general population has an annual incidence of 68.6 ACL injuries per 100,000 individuals, increasing three- to five-fold for women participating in pivot sports (Friel & Chu, 2013; Nordenvall et al., 2014). Estimates suggest that between 50% and 90% of individuals with ACL injuries develop PTOA within 10 to 20 years, with asymptomatic cases often emerging sooner among high-risk groups (Sanders et al., 2016).

The progression of PTOA after ACL injury stems from a myriad of reasons which include the patient's age, body mass index (BMI), meniscal tears, time to treatment, and rehabilitation. Following ACL injury, the 'set joint-estate' is prepared for deterioration due to the cyclic inflammation, constant use, and deterioration of soft and hard tissues within the joint (Lieberthal et al., 2015; Titchenal et al., 2017). In addition, other injuries such as meniscus tears and cartilage damage significantly worsen the chances of PTOA, thus requiring careful attention for multi-faceted trauma.

A regrettably deviating attention concerning PTOA and ACL injury is the goal of this review. It depicts the preventative role of ACL reconstruction (ACL-R) surgery for PTOA, analyzes diagnostics, and assesses ACL-R rehabilitation protocols. This review aims to shift the focus to incorporating strategies in evidence frameworks alongside policy-making meant for PTOA by underscoring knowledge voids along with evaluative frameworks.

Incidence and Risk Factors: The ACL performs a key function in stabilizing the knee joint by preventing forward movement of the tibia and rotation. Its tearing could result in the development of joint laxity and PTOA as side effects of PTOA (Wang et al., 2020). The most crucial ones are:

- **Age:** Elderly people have an imbalance between cartilage replenishment and its breakdown, predisposing them to PTOA (Lieberthal et al., 2015).
- **Body Mass Index (BMI):** An increased BMI as well as obesity further aggravate joint loading and initiate inflammatory pathways that precipitate cartilage damage. (Friel & Chu, 2013).
 - **Activity Levels:** High-impact mechanical forces associated with specific movements accelerate cartilage degeneration. (Andriacchi & Favre, 2014).
 - **Meniscal Status:** Accompanying a meniscal injury greatly increases the risk of developing PTOA. (Wang Et Al., 2020).
 - **Delayed Treatment:** PTOA risk and severity increase with the delay in surgical treatment following the injury. (Nordenvall Et Al, 2014).

ACL Reconstructive Surgery and the development of PTOA After an ACL-R Surgery, the mechanical stability of the joint is restored, thus relieving the joint from excessive forces and secondary injuries. Secondary injuries are also prevented. Most commonly performed, the interplay between ACL-R and the development of PTOA is still poorly elucidated. Many determinants serve the role of ACL-R and its efficiency in postponing PTOA:

1. **Graft type:** The type of graft chosen will strongly influence the outcomes of the surgery. Bone-patellar tendon bone autografts provide greater strength, however, result in increased complications, whilst hamstring autografts have lower complication rates along with better functional outcomes. (Wang et al., 2020).
2. **Surgical Technique:** The improvement of the anatomic reconstruction methodologies strives to more accurately emulate the motions of the knee. Graft placement, as well as tensioning, are critical processes for restoring the mechanics of the joint and preventing abnormal mal-adaptive loading (Murray & Fleming, 2013).
3. **Biomechanical Restoration:** Although there is an enhancement in the stability of the joint following an ACL-R, its ability to restore normal joint kinematics is not fully possible. Observed changes to the lower extremity rotational mechanics include increased internal rotation of the tibia and unequal distribution of loads across the knee joint. These changes in mechanical function are a factor in the increased rate of cartilage deterioration and progression of PTOA (Friel & Chu, 2013).
4. **Inflammatory Response:** Joint inflammation may become worse because of surgical trauma. Post-surgical bleeding into the joint space, combined with upregulation in key cytokines such as IL-1 β , TNF- α , and IL-6, enhances the severity of inflammation and cartilage degradation. In the long-term, managing inflammation improves functional outcomes after surgery (Wang et al., 2020).
5. **Secondary Injuries:** ACL-R appears to have a protective effect against further damage to the meniscus or cartilage, both vital for the progression of PTOA. Nonetheless, these protective effects can be lost as a result of poor rehabilitation protocols or a slow return to activity (Sanders et al., 2016).
6. **The Timing of Surgery:** ACL injury surgeries performed sooner tend to have a lower secondary joint damage incidence compared to functional outcome injuries. PTOA risk is aggravated by postponing surgical intervention, as it raises the chance of meniscal tears and joint instability. (Luc et al., 2014).

Furthermore, ACL reconstruction does not automatically result in non-existent PTOA risk. Even after surgery, patients continue to suffer from joint degeneration due to already di, dysfunctional cartilage, altered biomechanics, and inflammatory

processes. Other surgical approaches adjunct treatment options should be developed to optimize the protective capability of ACL reconstruction.

A Dualistic Approach to the Progression Mechanisms of PTOA

1. Structural Components:

- About half of ACL injuries come with cartilage tears, often accompanied with the meniscus and subchondral bone, resulting in disrupted joint servomechanism (Friel & Chu, 2013).
- After an injury, subchondral bone remodeling redistributes load, which causes acceleration in the wear of cartilage (Wang et al., 2020).

2. Biological Components:

- An array of catabolic processes on cartilage is triggered by homeostasis, due to disease- modulated components such as IL-1 β , IL-6, and TNF- α (Friel & Chu, 2013).
- Increased concentration of ECM degrading matrix metalloproteases (MMPs) accelerates the loss of cartilage.

3. Mechanical Factors:

- After an injury or surgical arthroscopy, the abnormal joint kinematics change bearing distribution patterns which increases the damage on articulating surfaces (Wang et al 2020).

4. Neuromuscular Factors:

- Decreased joint stabilization results from deficits in proprioception, muscle strength, especially of the quadriceps and hamstrings, predisposing to increased joint degeneration (Friel & Chu, 2013).

Diagnostic Advances Accurate identification of PTOA is vital to the successful management of the condition. Tools available for diagnosis at the moment include:

- **Imaging Techniques:**

- **MRI:** More advanced procedures such as T1rho and T2 mapping evaluate the cartilage and more importantly its biochemical changes prior to incurring structural damage (Wang et al., 2020).
- **PET/CT:** Grant molecular aspects of vision into the joints for detection of early deterioration.
- **Biomarkers:** The forward stages of PTOA are signified by increased levels of MMPs, lubricin, and pro-inflammatory cytokines in synovial fluid (Friel & Chu, 2013).

Prevention Strategies: The most efficient method continues to be preventing the ACL injury. Neuromuscular training programs like FIFA 11+ have demonstrated the capability of reducing ACL injury rates by as high as 50% (Wang et al., 2020). Components include:

- Strength and balance training
- agility training
- correct application instruction

Therapeutic Approaches: Modern treatment approaches focus on controlling symptoms and slowing progression:

1. **Pharmacological Interventions:** Preclinical studies suggest the use of anti-inflammatory drugs (IL-1 inhibitors) and antioxidants as potential treatment options (Friel & Chu, 2013).
2. **Surgical Innovations:** Bio-enhanced ACL repair is among newer methods directed towards improving stability of a joint as well as minimizing degeneration.
3. **Rehabilitation Programs:** Emphasis is placed on the restoration of control through the neuromuscular system and enhanced biomechanics of the joint.

2. DISCUSSION

The development of PTOA in the context of ACL injuries illustrates the complex interplay of contributing factors. Current treatment methods, such as reconstructing the ACL, focus on restoring the stability of the knee, yet fail to counter the biological and mechanical processes associated with joint pathological changes. PTOA also has critical structural components, such as cartilage and meniscal damage, which are exacerbated by biological components like chronic inflammation and cytokines that upset the equilibrium of cartilage synthesis and resorption. There have been strides in imaging and biomarker development, but PTOA diagnosis remains challenging due to the absence of simple tools that are widely available and reasonably priced.

Focused and tailored approaches to prevention and early detection are equally important. The identification of effective neuromuscular training programs for ACL injury reduction highlights the role of active primary prevention. PTOA is often the end result of various conditions and active pathways prevention require novel, class-specific to inflammatory passages, could greatly slow down the onset of PTOA. There is a clear gap in human research; most proposals lie in preclinical phases, including surgical advancements that enhance functionality but further neglect joint degeneration mechanics. Surgeons need to develop strategies that intuitively imitate the movement of an uninjured joint for better long-term outcomes.

Rehabilitation programs also play a core role in managing PTOA. These programs should strive to balance the restoration of physical functions with the rehabilitation of neuromuscular, proprioceptive, and other higher-level integration systems. The input of orthopedists, physiotherapists, and other researchers may be important for maximizing the benefits and developing better methods.

Conclusion and Future Directions

Although it is well managed, PTOA still poses a challenge for individuals with ACL injuries as it is highly complex and multifactorial in nature. Existing approaches are helpful to an extent, but do not completely stop or slow down the progression of the condition. Research moving forward needs to address the following gaps:

Improving Imaging Techniques: PTOA diagnosis is highly reliant on sensitive imaging methods accompanied by well-defined imaging biomarkers, all of which are strived to be made more affordable.

Specific Inflammatory Pathways: Developing targeted systemic therapies, biologics, and novel pharmaceutical compounds aimed at inflammation and repair of damaged cartilage tissue.

- ACL reconstruction surgeries should focus on the use of tissue-engineered grafts known for their enhanced healing and durability to replicate the biomechanics of natural joints.
- Multidisciplinary rehabilitation programs need to be established to address and integrate biomechanics, neuromuscular, and psychological aspects for holistic recovery.
- Individualized pre-emptive measures for those identified as high risk should utilize markers that are genetic, biomechanical, and biochemical, thus enhancing selection precision and targeted preventative strategies.
- Longitudinal Studies: Performing extensive and enduring research exploring the effectiveness of newly developed therapies and interventions in different populations.
- Addressing these gaps will require collaboration amongst clinicians, researchers, and industry professionals. By developing more precise diagnostic tests and effective PTOA treatment options, as well as placing greater focus on preventative strategies, the PTOA burden and patients' quality of life globally stand to greatly benefit.

REFERENCES

- [1] Wang, L.-J., Zeng, N., Yan, Z.-P., Li, J.-T., & Ni, G.-X. (2020). Post-traumatic osteoarthritis following ACL injury: Mechanisms and interventions. *Arthritis Research & Therapy*, 22(57). <https://doi.org/10.1186/s13075-020-02156-5>
- [2] Friel, N. A., & Chu, C. R. (2013). The role of ACL injury in the development of posttraumatic knee osteoarthritis. *Clinical Sports Medicine*, 32(1), 1-12. <https://doi.org/10.1016/j.csm.2012.08.017>
- [3] Titchenal, M. R., Chu, C. R., & Andriacchi, T. P. (2017). The progression of OA following ACL reconstruction. *American Journal of Sports Medicine*, 45(12), 2827-2837. <https://doi.org/10.1177/0363546517710217>
- [4] Sanders, T. L., et al. (2016). Incidence and progression of PTOA after ACL injury. *American Journal of Sports Medicine*, 44(5), 1186-1191. <https://doi.org/10.1177/0363546515626538>
- [5] Shyamala, N., & Anand, S. (2018). Management of orthodontic emergencies-to act or not. *Inter Journal of Oral Health Dent*, 2018, 4.
- [6] Luc, B., Gribble, P. A., & Pietrosimone, B. G. (2014). Osteoarthritis prevalence following anterior cruciate ligament reconstruction: A systematic review and numbers-needed-to-treat analysis. *Journal of Athletic Training*, 49(6), 806-819.
- [7] Andriacchi, T. P., & Favre, J. (2014). The role of ambulatory mechanics in the initiation and progression of knee osteoarthritis. *Current Opinion in Rheumatology*, 26(5), 533-537.
- [8] Murray, M. M., & Fleming, B. C. (2013). Use of bioactive scaffolds to enhance healing of the anterior cruciate ligament. *Journal of Orthopaedic Research*, 31(4), 583-591.
- [9] Palmieri-Smith, R. M., & Thomas, A. C. (2009). A neuromuscular mechanism of posttraumatic osteoarthritis associated with ACL injury. *Exercise and Sport Sciences Reviews*, 37(3), 147-153.
- [10] Nordenvall, R., Bahmanyar, S., Adami, J., Mattila, V. M., & Felländer-Tsai, L. (2014). Cruciate ligament

reconstruction and risk of knee osteoarthritis: The association between cruciate ligament injury and posttraumatic osteoarthritis. *The American Journal of Sports Medicine*, 42(12), 2853-2859.

- [11] Harkey, M. S., Blackburn, J. T., & Padua, D. A. (2015). Quadriceps function and impaired joint mechanics following ACL reconstruction: A review of evidence. *Sports Health*, 7(3), 239-245.
 - [12] Henrotin, Y., & Mobasheri, A. (2018). Natural products for promoting joint health and managing osteoarthritis. *Current Rheumatology Reports*, 20(11), 72.
 - [13] Frobell, R. B., Roos, H. P., Roos, E. M., Rantam, J., & Lohmander, L. S. (2010). Treatment for acute anterior cruciate ligament tear: Five year outcome of randomised trial. *BMJ*, 340, c232.
 - [14] Menendez, M. I., et al. (2017). Molecular imaging of osteoarthritis: A review. *Frontiers in Physiology*, 8, 224.
 - [15] Chang, J., et al. (2017). Preventive strategies to slow cartilage degeneration following joint injury. *Current Rheumatology Reports*, 19(11), 50.
-

