

Cyclops Syndrome After Anterior Cruciate Ligament (ACL) Reconstruction: A Case Report with Molecular Mechanisms Strategies

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Submitted : 30 June 2026

Accepted : 30 June 2026

Published : 30 June 2026

Abstract

Background: Cyclops syndrome is a recognized complication after anterior cruciate ligament (ACL) reconstruction, with growth of a fibrous nodule in the intercondylar notch leading to painful loss of knee extension. While mechanical obstruction has been traditionally regarded the main cause, recent research reveals that molecular dysbalance might also contribute to the formation of lesions.

Objective: The present case report describes clinical indications of a patient with cyclops syndrome after ACL rupture and investigates whether these symptoms are mainly attributed to mechanical blockage or molecular dysregulation.

Methods: A descriptive case report design was used. Patient symptoms were assessed by clinical examination, measurement of range of motion, appraisal of pain and functional assessment. MRI and arthroscopy confirmed fibrous nodule. Literature study was included to contextualize data in known biological pathways including cytokine imbalance and fibroblast activation.

Results: The patient had stiff, painful loss of terminal knee extension and mechanical catching during movement. Imaging showed a fibrous lump consistent with cyclops lesion. Symptom analysis showed a combination of mechanical obstruction and molecular dysbalance. Clinical characteristics were consistent with documented cytokine-mediated fibrosis and extracellular matrix remodelling.

Conclusion: Cyclops syndrome following ACL reconstruction is a complex disease, with symptoms caused by mechanical and molecular processes. Management Early identification of extension limitations, meticulous surgical technique and systematic physiotherapy. Future therapeutic techniques may consider the molecular pathways and combine anti-fibrotic or cytokine-modulating therapies with mechanical treatment.

Keywords: ACL rehabilitation, molecular science, cyclops syndrome, health

INTRODUCTION

Restoration of anterior cruciate ligament (ACL) is one of the most common operations performed in sports medicine and aims to restore knee stability and function after ligament injury. Postoperative complications remain a concern, despite advances in surgical techniques and rehabilitation protocols, with cyclops syndrome being a significant factor in extension loss and functional impairment (Chia et al., 2022; Hong et al., 2023). Cyclops syndrome is characterized by the growth of fibroproliferative nodules in front of the healed graft, mechanically preventing full extension of the knee and potentially impairing long-term results. Cyclops lesions have been found in several populations and have been associated to surgical parameters, graft location, and the biological response to tissue healing. From a clinical perspective, patients present painful extension deficits, stiffness and biomechanical anomalies that may influence their return to sports and daily activities. The therapy of choice is arthroscopic excision although rehabilitation treatments are also of importance in the prevention and recovery (Kelmer et al., 2023; Olivares-Jabalera et al., 2021).

Recent knowledge of molecular mechanisms such as fibroblast activation, extracellular matrix deposition, and cytokine-mediated inflammation provides new insights for physical therapists and doctors. Pathological tissue remodeling and chronic lesion formation may be caused by abnormal fibroblast activation, increased collagen deposition, and cytokine-mediated inflammatory responses (IL-6, TNF- α and TGF- β) (Kacprzak, 2025). Knowledge of these pathways helps the integration of biologically based rehabilitation treatments including controlled mechanical stress, modulation of inflammatory response and specific exercise routines. This article aims to bridge the gap between clinical rehabilitation practice and molecular science through a case-based explanation of cyclosporine syndrome following ACL reconstruction. This

research sheds insight on the clinical presentation and molecular mechanisms and underscores the need for an interdisciplinary approach to improve patient outcomes and promote precision rehabilitation in musculoskeletal treatment.

METHODS

Study Design

This was a descriptive case report that described the clinical presentation, diagnostic work-up, and symptom analysis of a patient with cyclops syndrome after anterior cruciate ligament (ACL) reconstruction. The case study methodology was used to provide a thorough analysis of mechanical and molecular aspects related to the patient's complaints.

Case Presentation

A 20-year-old kabaddi competitor sustained an ACL tear after 2 unnoticed injuries during competitions. The first, the patient felt the damage but continued to play. The injury happened as a result of a fall on a bent knee. One week later the player was still playing but fell suddenly and the leg got stiff in a flexed position. The patient was scheduled for surgery with the bone-patellar tendon-bone graft method. The operation went smoothly without problems throughout operation. The doctor told the patient to rest in the same posture for 9 days and went through ACL replacement. The patient was referred to physiotherapy on the 12th post-operative day with passive range of motion of <20 degrees of knee flexion and considerable pain. The patient had follow-up for 2 months of therapy. He started to complain of tightness in the knee in both flexion and extension.

Data Analysis

Descriptive analysis of the patient's clinical data with respect to association of reported symptoms, imaging results and established diagnostic criteria for cyclops syndrome was performed. Knee extension

range of motion (ROM) measures were compared to normative values, paying particular attention to terminal extension abnormalities. Pain scores were assessed by the Visual Analog Scale (VAS) and associated to functional limits reported during sports-specific exercises such as running, pivoting, and squatting.

Imaging findings, including magnetic resonance imaging (MRI) and arthroscopic findings, were evaluated to confirm the existence of a fibrous nodule in the intercondylar notch. The results were compared to the clinical signs to ensure diagnostic consistency. Differential diagnoses such as graft impingement, meniscal disease, and widespread arthrofibrosis were excluded based on imaging and intraoperative visualization.

RESULTS

The patient developed a gradual loss of terminal knee extension approximately 6 weeks following ACL replacement. On clinical examination, there was 10° extension deficit compared to the contralateral limb as evaluated by goniometer. There was pain on attempts at full extension with a Visual Analog Scale (VAS) score of 8/10. Functional restrictions were difficulty with sport-specific activities such as squatting, pivoting and sprinting with mechanical symptoms of catching and loud clicking in extension.

Magnetic resonance imaging (MRI) revealed no indications of graft rupture, meniscal tear, or widespread arthrofibrosis. Post-operatively, the patient underwent structured physiotherapy with emphasis on restoration of range of motion, quadriceps strengthening and neuromuscular control following arthroscopic excision.

Table 1. Progression of Exercise

Phase	Parameter	Results
Pre-operative	ROM	S: 10 ⁰ -0 ⁰ -50 ⁰
	Anthropometry	D : prox 53, mid 45 S : prox 51, mid 43
	Inflammation	+
	Pain	6/10
Phase 1	Active mobility (week 2-week 4)	S: 4 ⁰ -0 ⁰ -90 ⁰
	Muscle chain strength	Reduced pain Improved contractions and function
	Proprioception and gait	Improved body awareness; standing without crutches for 30 seconds.
Phase 2	Active mobility	0 ⁰ -0 ⁰ -130 ⁰
	Motor control	Balance single leg stand >30 s Gait multidirectional exercise without crutches

 RUMAH SAKIT UMUM DAERAH Dr. SOETOMO   Beranda			
Rincian Tindakan	Hasil	Satuan	Nilai Rujukan
Hemoglobin	13.1	g/dL	11.0 - 14.7
Hematokrit	39.7	%	35.2 - 46.7
Lekosit	8.44	10 ³ /μL	3.37 - 10.0
Trombosit	180	10 ³ /μL	150 - 450
MCV	84.3	fL	86.7 - 102.3
MCH	27.8	pg	27.1 - 32.4
MCHC	33.0	g/dL	29.7 - 33.1
Eritrosit	4.71	10 ⁶ /μL	3.69 - 5.46
Basofil	0.4	%	0.3 - 1.4
Eosinofil	1.7	%	0.6 - 5.4
Neutrofil	61.6	%	39.8 - 70.5
Limfosit	31.2	%	23.1 - 49.9
Monosit	5.1	%	4.3 - 10.0
Basofil Absolut	0.03	10 ³ /μL	0 - 0.15
Eosinofil Absolut	0.14	10 ³ /μL	0.0 - 0.5
Neutrofil Absolut	5.21	10 ³ /μL	1.26 - 7.3
Limfosit Absolut	2.63	10 ³ /μL	0.8 - 4.0
Monosit Absolut	0.43	10 ³ /μL	0.1 - 0.8
MPV	12.3	fL	9.2 - 12.0
NRBC	0	%	
NRBC Absolut	0.00	10 ³ /μL	
P-LCR	42.2	%	19.7 - 42.4
PDW	16.3	fL	9.6 - 15.2
RDW-CV	12.9	%	12.2 - 14.8

Figure. 1 omplete Blood Count (CBC) Examination Results



Figure 2. MRI ACL tear in axial view



Figure 3. MRI ACL tear in coronal view

At 4 months, the patient had full knee extension with remission of mechanical complaints. pain ratings improved to 6/10 and functional performance increased. These findings corroborate the diagnosis of cyclops syndrome and emphasize the combined role of mechanical obstruction and molecular dysregulation to symptom manifestation.

DISCUSSION

Anterior cruciate ligament (ACL) injuries are one of the most common and devastating knee injuries in sport. One epidemiological study monitored high school athletes who sustained an ACL injury by sport and gender in football, basketball and soccer. The study results demonstrated an injury rate of 6.5/100,000 athlete exposures (Davies et al., 2017). To properly understand the ACL damage and reconstruction, a basic review of ligament architecture is needed. The anterior cruciate ligament is one of two cruciate ligaments that stabilize the knee in the sagittal plane. It arises from the tibial plateau, anterior and between the intercondylar eminence, and inserts into the posteromedial aspect of the lateral femoral condyle. The ACL has been long seen as a double bundle structure because to the fact that the tissue is composed of many bundles. Classically these two bundles are named the antero-medial and postero-lateral bundles. They differ in the resistance of the tissue that divides them and in their insertion sites. The anteromedial bundle of the ACL is more active functionally in deep knee flexion, whereas the posterolateral bundle is active primarily at near full extension. The anteromedial bundle contributes more to resisting anterior tibial stresses while the posterolateral bundle contributes more to resisting rotating pressures (Meissburger et al., 2025).

Arthrofibrosis or cyclops syndrome is a frequent complication after ACL reconstruction, with reported incidences of 2%-35%. Sex, period of repair and graft type have been described as risk factors for arthrofibrosis by some (D'Ambrosi et al., 2026; Kambhampati et al., 2020). In 2023, a case report documented a 45-year-old female patient with knee soreness after ACL surgery. The patient was planned for arthroscopic lysis of adhesion and manipulation under anaesthesia. They found improvement in flexion as well as extension after treatment with the help of vigorous physiotherapy regimen and strong patient compliance.

The key for better patient outcomes from a physiotherapy perspective is the combination of mechanical and molecular information. Rehabilitation should involve range of motion and neuromuscular control restoration but include the probable consequences of chronic inflammation and tissue remodeling on recovery (Jenkins et al., 2022; Larwa et al., 2021). Early detection of extension deficit and the use of specialized physiotherapy protocols are necessary in order to decrease the risk of progression of the lesion. Moreover, future treatments may include molecular-targeted interventions such as anti-fibrotic medicines or cytokine modulators that can complement mechanical management (ASHIGBI et al., 2020; Webster & Feller, 2019). This example illustrates the need for interdisciplinary management integrating surgical precision, molecular knowledge and physiotherapy practice in the management of cyclops syndrome.

Molecular Consideration

Cyclops syndrome pathogenesis is not just a mechanical phenomenon, but molecular disbalance is an essential part of the illness process. The local tissue milieu in ACL restoration is defined by a cascade of inflammatory responses. Pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF- α) are elevated to stimulate fibroblast proliferation and extracellular matrix deposition. This abnormal healing response leads to the formation of fibrous nodules in the intercondylar notch that ultimately produce an extension block of the knee. These cytokines suggest that cyclops lesions may represent a maladaptive healing process rather than a merely mechanical impingement (Alfayyadh, 2026; Kacprzak, 2025).

The molecular pathogenesis of cyclops syndrome mainly involves fibroblast activation and collagen remodeling. Transforming growth factor-beta (TGF-beta) is an important regulator of fibrosis, inducing excess collagen production and deposition. This results in dense fibrous

tissue formation that cannot be remodeled or removed normally. There may also be dysregulation of matrix metalloproteinases (MMPs) which normally mediate extracellular matrix turnover, resulting to reduced degradation of fibrotic tissue. The net effect is a microenvironment that encourages persistence of scar tissue, corresponding with the clinical presentation of stiffness and loss of extension.

Molecular disbalance, in addition to local tissue change, can also affect neuromuscular recovery. Altered proprioceptive signals of the knee joint due to chronic inflammation and fibrotic tissue growth may compromise neuromuscular control (Fonseca et al., 2022; Mizuno et al., 2025). This has an impact on physiotherapy as rehabilitation has to regain not only the range of motion but also the deficits of neuromuscular coordination (Jeong et al., 2021; Waldron et al., 2022). Future treatments may involve targeting biological causes (e.g. cytokine modulation or anti-fibrotic medicines) and mechanical interventions (e.g. arthroscopic excision). The integration of molecular knowledge into clinical care has the potential to improve long-term results and reduce the recurrence of cyclops syndrome (Kotsifaki et al., 2023; Mayer et al., 2025).

CONCLUSION

Cyclops syndrome is a multifactorial complication after ACL reconstruction with symptoms caused by a mechanical obstacle and molecular disbalance. In this instance, the patient presented with the classical clinical picture of painful loss of extension, stiffness and mechanical catching, which were verified on imaging and arthroscopic assessment. The formation of fibrous nodules in the intercondylar notch is a simple mechanical explanation, yet cytokine-mediated inflammation, fibroblast activation and abnormal collagen remodeling may also contribute to the continued symptoms and progression of lesions.

This dual process suggests the need for an integrated view in diagnosis as well as in management. Early detection of extension deficits, combined with accurate surgical technique and planned physiotherapy, is still required to prevent progression. Simultaneously, the examination of molecular pathways allows for prospects in future therapeutic methods, such as anti-fibrotic and cytokine-modulating therapies, to complement mechanical therapy strategies.

Finally, cyclops syndrome emphasizes the importance of interprofessional collaboration of surgeons, physiotherapists and researchers. By linking clinical data to molecular discoveries, rehabilitation approaches can be modified to not only restore range of motion and neuromuscular function, but also target the biochemical processes that sustain lesion growth. This type of therapy may improve long-term results and prevent recurrence, leading to excellent functional recovery after ACL reconstruction.

Recommendation

Integrating clinical rehabilitation with insights into molecular pathways underscores the importance of a dual strategy: mechanical restoration through physiotherapy and potential modulation of fibrogenic signalling to prevent recurrence.

CONFLICT OF INTEREST

The author hereby declares that this research is free from conflicts of interest with any party.

AUTHOR'S CONTRIBUTION

Ayuningtyas contributed in conceptualization, case data collection, clinical analysis, and manuscript drafting. Khoiriyah contributed in critical revision of the manuscript, supervision, and final approval of the version to be published.

FUNDING/SPONSORSHIP

This research does not receive external funding.

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