Biomechanical analysis of Jumping in Badminton players and its contribution to the clinical manifestations of Osgood Schlatter Disease (OSD) – An Observational Study.

By

AKANSHA A. JOSHI

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Under the guidance of

Dr. PRIYADARSHINI MISHRA (PT)

Associate Professor

ABHINAV BINDRA SPORTS MEDICINE & RESEARCH INSTITUTE

Bhubaneswar, Odisha

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ABSMARI

Odisha University of Health Sciences

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Date:

Place: **Signature**

Akansha Joshi

University Registration no.- 23MP435003

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Name :

Signature of the Guide

Dr. Priyadarshini Mishra (PT)

Department of Electrotherapeutics and Electrodiagnosis

Date :

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Date : **Seal & Signature of the HOD**

Place : **Dr. Asifuzzaman Shahriyar Ahmed (PT)**

Department of Orthopaedic Physiotherapy

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Date : **Signature & Seal of the Principal**

Dr. Chinmaya Kumar Patra (PT)

Principal, ABSMARI

Place:

ODISHA UNIVERSITY OF HEALTH SCIENCES

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Date : **Signature of the Candidate**

AKANSHA JOSHI

Place:

LIST OF ABBREVIATIONS USED

Here are 10 abbreviations from the document:

1. ATT – Anterior aspect of the tibial tuberosity
2. GRF – Ground Reaction Force
3. JH – Jump height
4. NORM – Normality (test: Shapiro-Wilk)
5. OSD – Osgood-Schlatter Disease
6. p – Probability value (statistical significance)
7. PP – Peak Power
8. PV – Peak Velocity
9. ROM – Range of motion
10. SPSS - Statistical Package for the Social Sciences

ABSTRACT

Background: Osgood-Schlatter Disease represents a prevalent traction apophysitis affecting the tibial tubercle in adolescent athletes, particularly those engaged in sports requiring repetitive jumping movements. Badminton demands explosive lower-extremity activities, rapid deceleration patterns, and frequent directional modifications, creating substantial mechanical loading on the knee's extensor apparatus. Limited research has examined the specific biomechanical characteristics of jumping tasks in badminton athletes diagnosed with this condition.

Purpose: This investigation examined biomechanical parameters during vertical jumping in adolescent badminton players presenting with clinically diagnosed Osgood-Schlatter Disease, focusing on kinetic variables including peak velocity, peak power , jump height and GRF and kinematic variable including knee joint angle.

Methodology: Twenty-six competitive badminton players (mean age: 12.19 ± 2.42 years) with confirmed OSD diagnosis participated in this cross-sectional observational study. Three-dimensional motion analysis and force platform technology quantified jumping biomechanics in a controlled laboratory environment. Kinetic measurements encompassed peak velocity, maximum power generation, ground reaction forces, and jump height. Kinematic evaluation concentrated on sagittal plane knee joint angles during propulsion phases. Statistical examination included descriptive analysis, normality assessment via Shapiro-Wilk testing, and one-sample t-test comparisons with significance established at $p < 0.05$.

Results: Participants demonstrated elevated peak velocity (2.5 ± 0.1 m/s), enhanced power output (2549.3 ± 344.2 W), and increased ground reaction forces (1166.3 ± 107.7 N), yet exhibited reduced vertical displacement (30 ± 3 cm) and limited knee flexion angles (80.4 ± 3.2°).

Conclusion: Young badminton athletes with OSD display superior force-generation capabilities but demonstrate inefficient biomechanical patterns that limit functional performance while potentially increasing tibial tubercle stress. These findings support incorporating plyometric training, neuromuscular coordination enhancement, and kinematic correction strategies within comprehensive rehabilitation protocols for adolescent racquet-sport participants.

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INTRODUCTION

Osgood-Schlatter Disease (OSD) represents one of the most prevalent causes of anterior knee pain in children and adolescents, particularly affecting those undergoing rapid skeletal growth phases while simultaneously participating in high-impact sporting activities. This condition, characterized by inflammation and micro-trauma at the tibial tuberosity, emerges from the complex interplay between repetitive stress from quadriceps muscle activity and the vulnerable anatomical structures present during skeletal maturation, resulting in a constellation of symptoms including pain, swelling, and potential ossicle formation that can significantly impact an adolescent's ability to participate in physical activities and negatively influence their overall musculoskeletal development trajectory.[1]

The anatomical basis for OSD's predisposition during growth periods stems from the unique structural characteristics present before and during skeletal maturation, where initially the patellar ligament connects to the tibia through thick cartilage, but as the growth spurt commences, an ossification center appears within this cartilage and gradually fuses with the epiphysis, creating a period of structural vulnerability where the cartilage is progressively replaced with bone until, at the completion of the growth period, the patellar ligament attaches directly to the tibia through complete epiphyseal closure. This transitional period, during which the structures of the tibial tuberosity become particularly vulnerable before complete epiphyseal closure, represents the critical window during which OSD typically manifests, with the condition being thought to be most prevalent when these anatomical structures exist in their most susceptible state.[6][1]

The epidemiological profile of OSD reveals its significant burden on young athletic populations, with the condition predominantly affecting children aged 8-12 years among females and 12-15 years among males, coinciding precisely with the development of the secondary ossification center of the anterior tibial tuberosity during the apophyseal phase, which typically occurs around age 9 in girls and 11 in boys, though symptom onset frequently precedes these chronological markers. The prevalence of OSD varies considerably across different sporting populations, with particularly high incidence rates observed in sports characterized by explosive lower-limb actions, repetitive jumping movements, and high-impact loading patterns, including badminton, soccer, gymnastics, basketball, football, volleyball, and athletics, where the mechanical demands placed upon the extensor mechanism of the knee create optimal conditions for the development of traction apophysitis at the tibial tubercle.[5] Recent epidemiological studies have demonstrated that the condition affects up to 13% of adolescent athletes, with males showing a slightly higher predisposition than females, though this gender disparity may reflect differential participation rates in high-risk sporting activities rather than inherent biological susceptibility differences, while the condition's manifestation also demonstrates strong associations with periods of rapid skeletal growth, muscular development patterns, and training load characteristics that combine to create the biomechanical environment conducive to tibial tuberosity irritation.[8][5]

The pathophysiological mechanisms underlying OSD development involve a complex cascade of events initiated by repetitive tensile stress applied to the patellar tendon insertion at the tibial tuberosity, which during periods of rapid skeletal growth creates an imbalance between the mechanical demands placed upon the structure and its capacity to adapt to these stresses without sustaining micro-trauma. This pathological process is fundamentally characterized by minor avulsion fractures occurring at the anterior tibial ossification center as a result of repetitive active knee extension movements, where the immature bone and cartilage structures prove insufficient to withstand the cumulative mechanical loading imposed by high-intensity athletic activities, leading to chronic irritation, inflammatory responses, and eventual structural changes that manifest as the clinical signs and symptoms associated with the condition. The underlying pathophysiology involves increased vascularization at the tibial tuberosity as the body attempts to repair the ongoing micro-trauma, leading to enhanced blood flow to the affected area that can contribute to neovascularization, which may further perpetuate the inflammatory response and prolong symptom duration, creating a self-reinforcing cycle of tissue irritation and repair that characterizes the chronic nature of this condition.[6]

The clinical presentation of OSD follows a characteristic pattern that begins with gradual onset of activity-related pain localized at the tibial tuberosity, which initially may be mild and intermittent but progressively intensifies during the acute phase to become severe and continuous, significantly impacting the affected individual's ability to participate in normal daily activities as well as sporting pursuits. The distinguishing clinical features include thickening of the patellar tendon insertion that can be detected through palpation and is frequently associated with localized tenderness, while pain is particularly elicited during resisted knee extension or counter-resisted knee flexion, as these movements increase tensile forces on the patellar tendon and directly stress the inflamed tibial tuberosity structures.[4] Physical examination typically reveals pain, tenderness, and local swelling over the patellar tendon and tibial tuberosity, with the main feature being a painful and enlarged tibial tubercle accompanied by surrounding soft tissue swelling and painful, restricted mobility that interferes with normal functional activities including running, jumping, climbing stairs, and sports-specific movements that require explosive knee extension patterns.[7]

The diagnostic classification of OSD has evolved to incorporate both clinical severity and temporal progression patterns, with Eric J. Wall's three-stage classification system providing a framework for understanding disease progression and treatment planning, where Stage 1 is characterized by pain that resolves within 24 hours after physical activity cessation, Stage 2 involves pain that occurs during and after physical activity but does not restrict participation and fails to disappear within 24 hours, and Stage 3 represents permanent pain that limits not only physical activities but also everyday functional tasks, indicating significant disease severity that requires comprehensive management approaches. The radiological progression of OSD follows a predictable pattern beginning with acute stage findings where patellar tendon margins become blurred due to soft tissue swelling, progressing after three to four months to demonstrate bone fragmentation at the tibial tuberosity, then advancing to a sub-acute stage where soft tissue swelling resolves but bony ossicles remain visible, and finally reaching a chronic stage where bone fragments may fuse with the tibial tuberosity, potentially appearing radiologically normal despite persistent clinical symptoms.[10]

Badminton, as a racquet sport characterized by explosive lower-limb actions, rapid deceleration patterns, frequent directional changes, and repetitive jumping movements, imposes substantial mechanical demands on the extensor mechanism of the knee that create particularly high-risk conditions for OSD development in adolescent athletes. The sport's biomechanical requirements include powerful vertical jumps for overhead smashes, rapid lateral movements for court coverage, explosive forward lunges for net play, and quick directional changes that collectively place enormous stress on the quadriceps-patellar tendon-tibial tuberosity complex, making badminton players especially susceptible to traction apophysitis during periods of skeletal immaturity. Research examining badminton injury patterns has revealed that despite being classified as a non-contact sport, badminton carries a significant injury risk profile with prevalence rates approaching those observed in other high-intensity racquet sports such as tennis and squash, with lower extremity injuries, particularly those affecting the knee complex, representing a substantial proportion of badminton-related musculoskeletal problems.[21] The sport's unique movement patterns, including the characteristic badminton jump smash technique that requires maximal vertical displacement combined with overhead reaching and trunk rotation, create complex multi-planar loading conditions that challenge not only the primary extensor mechanism but also the secondary stabilizing structures around the knee joint, potentially contributing to altered biomechanical patterns that may predispose athletes to overuse injuries including OSD.[11]

The biomechanical analysis of jumping movements in athletic populations has emerged as a critical component in understanding injury mechanisms and performance optimization, with particular relevance to conditions like OSD where repetitive jumping activities represent the primary causative mechanism for symptom development. Jumping biomechanics encompasses both kinetic parameters, including variables such as peak velocity, peak power, ground reaction forces, and jump height that quantify the force-generating capacity and performance outcomes of the neuromuscular system, and kinematic parameters, particularly joint angle measurements that describe the spatial and temporal characteristics of movement patterns during jumping activities.[14] The production of maximum muscular power output has been recognized as a fundamental aspect of various athletic performances, with movement tasks such as jumping, sprinting, throwing, and kicking requiring maximization of velocity in either specific body segments or the entire body, which demonstrates

close relationships with the skeletal muscles' ability to produce high power outputs, making maximum power output of lower-body muscles a frequently explored parameter through maximum vertical jumping assessments.[21]

The complex relationship between jumping biomechanics and injury risk, particularly in relation to conditions like OSD, involves multiple interdependent factors including landing mechanics, muscle control patterns, muscle fatigue responses, flexibility characteristics, and musculoskeletal stiffness parameters that collectively represent an individual's landing technique, which has been identified as one of the most important factors related to injury potential in jumping sports.[4] Ground reaction forces generated during jumping and landing activities are directly related to the biomechanical characteristics of these movements, with one of the most critical factors being joint angle profiles, particularly knee flexion patterns, where natural knee flexion typically ranges from 15° to 55° at the time of landing, and greater knee flexion angles are associated with reduced ground reaction forces and consequently lower injury risk. The study of human jumping mechanics also focuses on performance optimization in terms of maximum height achievement, though increases in jumping height must be carefully considered as they coincide with requisite increases in landing height, creating greater mechanical demands and potentially elevated risks for landing-related injuries, highlighting the delicate balance between performance enhancement and injury prevention that must be maintained in athletic training programs.

During adolescence, the rapid bone growth that characterizes this developmental period temporarily disrupts the balance between muscular strength and tendon elasticity, creating increased tension at the tibial tuberosity that, when combined with high levels of physical activity, leads to repetitive micro-trauma and inflammation at the site of patellar tendon attachment.[13] Muscular imbalances, particularly those involving quadriceps tightness and hamstring inflexibility, have been strongly associated with increased tibial traction forces, while biomechanical factors including over-pronation of the foot and knee valgus alignment may influence the degree of patellar tendon tension, thereby increasing susceptibility to tibial tuberosity irritation and subsequent OSD development. Studies have suggested that anatomical variations such as external tibial torsion, increased posterior tibial slope, and limited ankle dorsiflexion contribute to abnormal loading patterns that further exacerbate stress on the apophysis, creating a multifactorial risk profile that extends beyond simple overuse mechanisms to encompass complex biomechanical, anatomical, and developmental factors that interact to influence individual susceptibility to this condition.[14]

The morphological characteristics of the tibial tuberosity undergo predictable developmental changes that have been systematically classified according to bony maturity stages, with the Ehrenborg and Lagergren classification system dividing the maturation process into four distinct stages: the cartilaginous stage (stage C) characterized by a large amount of apophyseal cartilage without a secondary ossification center, the apophyseal stage (stage A) where the patellar tendon attaches to apophyseal cartilage but secondary ossification centers are visible in the apophysis, the epiphyseal stage (stage E) characterized by patellar tendon attachment to the bone surface with a thin layer of insertional cartilage still present, and the bony stage (stage B) where the patellar tendon attaches to the tubercle and apophyseal cartilage is completely absent. Research has demonstrated that tibial tuberosity maturation occurs earlier in female participants compared to males, and that the risk of OSD is greater in stage A than stage C and in stage E than stage A, while the risk of OSD increases with age in males but not in females, reflecting the complex interaction between chronological age, skeletal maturation, and gender-specific developmental patterns that influence disease susceptibility.[15]]

The assessment and monitoring of tibial tuberosity maturation through ultrasonographic evaluation has emerged as a valuable clinical tool for understanding OSD risk profiles and disease progression, with imaging assessments revealing progression of bone maturation stages as chronological age increases, with the majority of subjects above 14 years displaying complete bone development.[16] Ultrasonographic examination proves particularly useful for visualizing cartilage structures and detecting subtle bony abnormalities and ossification changes, though distinguishing between normal and abnormal states in skeletally immature youth can be challenging, especially during the transition from stage C to stage A where structural changes are subtle but clinically significant. Studies examining cartilage thickness patterns have demonstrated significant decreases with progression through maturity stages, with advanced age, development of secondary sex characteristics, and sports club affiliation all showing associations with cartilage thickness, though the impact of age and sports activity on cartilage thickness becomes less significant after adjusting for both tibial tuberosity maturity stages and secondary sex characteristic development, suggesting that skeletal maturity represents the primary determinant of structural vulnerability rather than chronological age or activity level alone.[5]

The multifactorial aetiology of OSD encompasses not only mechanical overload factors but also anatomical predisposition patterns and neuromuscular imbalance characteristics that collectively contribute to disease development and progression, with the most credible etiological theories focusing on variants of patellofemoral anatomy and alignment of the extensor mechanism associated with overuse injury patterns. [20] Risk factors for OSD development include regular sports participation during adolescent growth periods, short rectus femoris muscle belly configurations, more proximal and broader tibial attachment patterns of the patellar ligament, tight quadriceps and hamstring muscle groups, and biomechanical factors such as increased posterior tibial slope and limited ankle dorsiflexion that combine to create unfavourable loading conditions at the tibial tuberosity. The identification of these risk factors has important implications for prevention strategies, suggesting that interventions targeting muscular flexibility, particularly quadriceps and hamstring stretching programs, should be implemented as routine practice for children participating in regular sports activities, while biomechanical assessments and corrective interventions may help identify and address predisposing factors before symptom onset occurs.[17]

Despite the traditionally held belief that OSD represents a self-limiting condition that resolves with growth plate closure and therefore carries no long-term functional consequences, emerging research has challenged this assumption by demonstrating that knee joint impairments may persist in both patients who underwent treatment for OSD and those who received no therapy, with potentially negative effects on knee joint function being significantly more likely in athletes with previous OSD history compared to their peers without such history. These findings suggest that OSD may have more persistent effects than previously recognized, potentially leading to performance deficits and career disadvantages for affected athletes, emphasizing the importance of early recognition, appropriate treatment, and long-term monitoring of individuals with OSD history to minimize potential long-term complications and optimize functional outcomes.[13][18]

The selection and implementation of appropriate outcome measures represents a critical component in the comprehensive assessment of biomechanical factors contributing to OSD in badminton players, requiring sophisticated instrumentation and validated measurement protocols that can accurately quantify the complex kinetic and kinematic parameters associated with jumping performance and movement quality.[6] The kinetic parameters selected for this investigation encompass peak velocity, measured using advanced motion capture systems integrated with force plate technology to assess the maximum speed achieved during jumping movements, which provides crucial information about the neuromuscular system's capacity for rapid force generation and movement execution that directly relates to the mechanical demands placed upon the tibial tuberosity during explosive athletic activities. Peak power measurement, calculated through force plate analysis during jumping activities, represents another fundamental kinetic parameter that determines the maximum power output generated by the lower extremity musculature during explosive movements, providing insights into the athlete's ability to produce high rates of force development that are essential for badminton-specific movements such as vertical jumping for overhead smashes, rapid directional changes, and explosive court coverage patterns that characterize elite badminton performance.[14]

Jump height measurement, obtained through either jump mat technology or sophisticated motion capture systems, quantifies the maximum vertical displacement achieved during jumping activities and serves as a functional outcome measure that integrates multiple biomechanical factors including neuromuscular power, coordination efficiency, and technique optimization, while simultaneously providing information about the mechanical demands that must be absorbed during landing phases that directly influence the stress patterns experienced at the tibial tuberosity. [17] Ground reaction force analysis, recorded using high-frequency force plate systems, captures the complex force-time characteristics of jumping and landing activities, providing detailed information about the magnitude, timing, and distribution of mechanical loads transmitted through the lower extremity during athletic movements, with particular relevance to understanding the forces that contribute to repetitive stress at the patellar tendon insertion and subsequent development of OSD symptoms in susceptible individuals.[14][17]

The kinematic assessment components focus primarily on joint angle measurements during jumping activities, with particular emphasis on sagittal plane knee joint angles that provide critical information about movement patterns, range of motion utilization, and biomechanical efficiency during the propulsion and landing phases of jumping movements that are fundamental to badminton performance and injury risk assessment. Three-dimensional motion analysis systems equipped with multiple high-speed cameras and reflective marker sets enable precise quantification of joint angles throughout the complete jumping movement cycle, providing detailed information about knee flexion patterns, hip and ankle coordination, and inter-segmental timing that collectively influence the mechanical stress patterns experienced at the tibial tubercle and surrounding structures.[14]

The clinical interpretation of outcome measures requires careful consideration of the relationships between biomechanical parameters and OSD pathophysiology, with particular attention to identifying patterns that may contribute to increased mechanical stress at the tibial tuberosity or reflect compensatory movement strategies that develop in response to pain and dysfunction associated with the condition.[9] The integration of kinetic and kinematic findings provides comprehensive insights into the complex biomechanical factors that influence OSD development and progression, enabling the identification of specific targets for intervention strategies aimed at reducing symptom severity, improving functional capacity, and preventing recurrence or progression of the condition in affected athletes.[3]

The significance of this research lies in addressing the limited understanding of how specific biomechanical factors during jumping contribute to OSD development in adolescent badminton players, where the sport's unique demands for explosive lower-limb actions and high-impact loading create particular challenges for the developing musculoskeletal system. The identification of biomechanical factors such as kinetic variables including peak velocity, power, and jump height, combined with kinematic variables including joint angles, represents crucial information for developing targeted prevention and rehabilitation strategies aimed at reducing mechanical stress on the tibial tubercle while optimizing athletic performance in young athletes. Understanding these biomechanical relationships can inform evidence-based approaches to training program design, injury prevention protocols, and rehabilitation strategies that address the specific demands of badminton while respecting the developmental characteristics and vulnerability patterns of the adolescent athlete population, ultimately contributing to more effective management of this common but potentially impactful condition in youth sport populations.

AIM

To investigate the relationship between biomechanical jumping characteristics and clinical presentations of Osgood-Schlatter Disease in adolescent badminton players through comprehensive kinetic and kinematic analysis.

OBJECTIVES

1. To quantify kinetic parameters (peak velocity, peak power, vertical jump height, and ground reaction forces) during standardized vertical jumping tasks in badminton players with diagnosed OSD.
2. To assess kinematic variables, specifically sagittal plane knee joint angles during the propulsive phase of jumping movements in the study population.

HYPOTHESES

- **Null Hypothesis :** There is no significant relationship between the biomechanical parameters of jumping in badminton and the clinical manifestations of Osgood-Schlatter disease in children.
- **Alternate Hypothesis :** There is a significant relationship between the biomechanical parameters of jumping in badminton and the clinical manifestations of Osgood-Schlatter disease in children.

REVIEW OF LITERATURE

1. **Insights into Knee Pain: Risk Factors and Management Strategies for Osgood-Schlatter Disease(2025) :** Piotr Charzewski and Agnieszka Starzyk, aimed to comprehensively analyse the risk factors contributing to OSD and evaluate the effectiveness of various management strategies, including conservative treatment, rehabilitation programs, and surgical intervention for refractory cases. The findings highlighted that OSD is predominantly associated with repetitive tensile stress on the patellar tendon, exacerbated by quadriceps tightness, hamstring inflexibility, and rapid skeletal growth. Anatomical variations, such as increased tibial tuberosity prominence and altered patellar tendon mechanics, further predispose individuals to the condition. Limited ankle dorsiflexion and lower limb misalignment contribute to increased stress on the tibial tuberosity, intensifying symptoms.

2. **Optimizing Agility and Athletic Proficiency in Badminton Athletes Through Plyometric Training: A Review(2024)**: Saylee S Shedge, Swapnil U Ramteke , investigates the advantages of plyometric training in badminton, concentrating on the effects on agility, power, speed, and overall athletic performance. It also looks at how plyometric training affects jumping ability and explosiveness, with a concentration on vertical jumps and hard smashes. In addition, the research explores the function of plyometrics in injury prevention in the physically demanding sport of badminton.

3. **A systematic review of resistance training methodologies for the development of lower body concentric mean power, peak power, and mean propulsive power in team-sport athletes.(2024):**Patrick Cormier , Tomás T Freitas , aimed to systematically review training methods prescribed to develop lower-body power, determine their effectiveness for the development of lower-body mechanical power and their implementation in an annual training cycle amongst team-sport athletes. The absolute and relative outcome values of concentric mean power, peak power and mean propulsive power were extracted from 19 studies.

4. **Understanding the Interactions Between Loading, Pain Dynamics, and Imaging Characteristics for Osgood Schlatter: A Cross-Sectional Study (2024) :** S. Holden, K. Lyng, J. L. Olesen**,** aimed to characterize clinical, pain and ultrasound imaging characteristics in participants with OSD compared to controls. It included adolescents diagnosed with OSD and matched controls.

5. **Tibial tuberosity maturation assessment by ultrasonography and screening for Osgood–Schlatter disease in male and female children and adolescent athletes: a cross-sectional study(2023)** : Stefan Cristian Dinescu, Doru Stoica aimed to describe the maturation pattern of the tibial tuberosity in association with chronological age in children and adolescent athletes. The study provides information about the gender offset of tibial tuberosity bone maturation and it also aimed to analyse the sequence of tibial tuberosity bone maturation through ultrasound and report distribution by chronological age and gender comparison. In addition, prevalence of OSD was observed clinically and by ultrasound examination. The imaging assessments of tibial tuberosity showed a progression of bone maturation stages as chronological age increased with the majority of subjects above the age of 14 years displaying complete bone development.

6. **Causes, short- and long-term consequences and treatment options of Osgood Schlatter’s disease (2022)** : Karel Madou stated that OSD is an inflammation of bone and cartilage on the shin head (tibia) immediately under the growth disc. This disease is sometimes caused by overuse of the leg. In the process, pieces of bone can detach from the shin and die (necrosis). The disease is therefore counted among the aseptic (i.e., non-infection-related) osteonecrosis. Typical symptoms are pain, swelling and tenderness of the knee. This article discusses aetiology, the natural course, as well as the long-term consequences and treatment options.

7. **Osgood-Schlatter Disease: Appearance, Diagnosis and Treatment: A Narrative Review(2022)** : Francisco Corbi , Sergi Matas suggested that excessive tractional loads should not be applied on the anterior tibial tuberosity through the patellar tendon in children aged 9 to 15. Since complications are very rare (except for pain caused by fragmentation), early detection and differentiation from anterior knee pain are crucial to prevent the complications. For this reason, muscular imbalances that favour higher tension (stiffness) in the patellar tendon should be prevented, particularly in sports with acyclic and explosive components. Moreover, flexibility and muscle rebalancing programs that contribute to reducing tension in the patellar tendon insertion might represent an interesting strategy.

8. **The Aetiology and Risk Factors of Osgood–Schlatter Disease: A Systematic Review (2022)** :Ludovico Lucenti, Marco Sapienza, aimed to systematically analyse the available literature about the aetiology and risk factors of OD. The literature was systematically reviewed using the PRISMA criteria to evaluate all studies published in the last 25 years (between 1996 and 2021) dealing with the aetiology of OD. The most credible theories seem to focus on the variants of patellofemoral anatomy and alignment of the extensor mechanism, associated with overuse injuries.

9. **The mid-term effect of Osgood-Schlatter disease on knee function in young players from elite soccer academies .(2022):** Bezuglova, Pirmakhanov,Ussatayevad , aimed to evaluate the effect of Osgood-Schlatter disease (OSD) on knee joint function in elite young soccer players. OSD among young soccer players, when symptoms resolve, continue about one month and they can return to regular training and participation in games. Wherein, the negative effects in knee joint function were significantly more likely in soccer players with previous OSD history when compared with their peers with no history of OSD. Potentially this may lead to performance deficits and disadvantages for their future careers and coaches and physicians should be informed.

10. **Narrative Review — Knee Pain in the Pediatric Athlete (2021) :** Anne Kuwabara1 &Emily Kraus sought to highlight common youth athlete knee conditions due to overuse or trauma and elucidate differences from the adult populations. Recent findings Overuse conditions presented include apophysitis, osteochondritis dissecans plica syndrome, and discoid meniscus. Traumatic conditions presented include patellar instability, patellar sleeve fracture, and patellofemoral osteochondral fractures. Summary Knee injuries affect a significant proportion of youth athletes. These injuries place athletes at higher risk of chronic pain and potentially osteoarthritis.

11. **Badminton Injuries in Elite Athletes: A Review of Epidemiology and Biomechanics.(2020) :** Dinshaw N. Pardiwala’s analytical review identifies the incidence, severity, and profile of badminton injuries in elite players, and discusses the biomechanical basis of these injuries. Although badminton is a non-contact sport, there is a significant risk of injuries. This prevalence of injuries is much higher than commonly assumed, and is almost similar to the incidence of injuries in other racquet sports such as tennis and squash.

12. **Activity Modification and Knee Strengthening for Osgood-Schlatter Disease: A Prospective Cohort Study.(2020):** Michael S. Rathleff, Lukasz Winiarski, investigated an intervention consisting of education on activity modification and knee-strengthening exercises designed for adolescents with OSD. An intervention consisting of activity modification, pain monitoring, progressive strengthening, and a return-to-sport paradigm was associated with improved self-reported outcomes, hip and knee muscle strength, and jumping performance.

13. **Osgood-Schlatter disease: a 2020 update of a common knee condition in children.(2020):** Hannah N Ladenhauf, Gerd Seitlinger, conducted a study to provide an up-to-date account on contemporary prophylaxis as well as diagnostic and therapeutic approaches. OSD is a mostly self-limiting apophysitis of the tibial tubercle and the adjacent patella tendon in young active patients with open physis. Prevention strategies include quadriceps and hamstring stretching and therefore should be implemented in everyday practice routines for children who partake in regular sports activities.

14. **Jump and Landing Biomechanical Variables and Methods – A Literature Review(2020)** : Juan Baus , John R Harry, stated that , noncontact lower extremity injuries are commonly related to jumping and landing activities. They presented an overview of relevant biomechanical variables that can be modified in training to improve jumping performance, landing mechanics, and consequently, reduce injury risks. They concluded that ,jumping and landing biomechanics are considerably different between male and female subjects for different experimental methods and in both cases, these kinematics factors can be improved over shorter- or longer-time training to develop a better landing strategy.

15. **Screening of the Maturity Status of the Tibial Tuberosity by Ultrasonography in Higher Elementary School Grade School children(2019) :** Maiko Ohtaka , Izumi Hiramoto, aimed to obtain screening data on the maturity status of the tibial tuberosity in schoolchildren of higher elementary school grades for risk management of Osgood–Schlatter disease (OSD). They concluded that, cartilage thickness decreased significantly with progression in the maturity stage, and, after adjusting the maturity stage, advanced age, development of secondary sex characteristics, and sports club affiliation were associated with cartilage thickness. However, the impact of age and sports club affiliation on cartilage thickness became insignificant after adjusting both the tibial tuberosity maturity stages and the development of secondary sex characteristics.

16. **Bony Maturity of the Tibial Tuberosity With Regard to Age and Sex and Its Relationship to Pathogenesis of Osgood-Schlatter Disease: An Ultrasonographic Study (2018):** Yoichi Kaneuchi, Kenichi Otoshi, surveyed the bone maturation process of the tibial tuberosity by age and sex and clarified its relationship to OSD. They concluded that tibial tuberosity matures earlier in female participants and that the risk of OSD is greater in stage A than stage C and in stage E than stage A. The risk of OSD increases with age in males but not in females.

17. **Risk assessment of the onset of Osgood–Schlatter disease using kinetic analysis of various motions in sports (2018):** Gento Itoh, Hideyuki Ishii, aimed to quantitatively identify the load on the tibial tubercle through a biomechanical approach using various motions that may cause Osgood-Schlatter disease, and to compare the load between different motions.

18. **Apophysitis of the tibial tuberosity (Osgood-Schlatter disease): a review (2016):** R Vaishya, AT Azizi, concluded that Osgood-Schlatter disease (OSD) is usually a self-limiting disease mainly seen in late childhood. The symptoms usually resolve without any specific treatment or with simple conservative treatment such as the restriction of activity or immobilization in a cast for three to six weeks. Surgery is rarely indicated for OSD.If the symptoms persist and become disabling to the patient, surgery may be indicated especially after the fusion of the apophysis. The excision of the ossicle is the common surgical method, and removal of any prominences on the tibial tuberosity is optional.

19. **The Science of Badminton: Game Characteristics, Anthropometry, Physiology, Visual Fitness and Biomechanics (2015**): Michael Phomsoupha, conducted a study focusing on the game characteristics, anthropometry, physiology, visual attributes and biomechanics of badminton. The review takes a global approach to badminton performance and to the interrelationship between various metabolic, physiological, biomechanical, technological and visual factors**.**

20. **Osgood Schlatter Disease: A Rare Condition in Young Athletes - A Case Study(2014)** : Hritvansingh Parmar, stated that Osgood Schlatter disease (OSD) classified as an apophysitis of the anterior aspect of the tibial tuberosity (ATT) is a common condition in an active youth athletes and is associated with growth spurts. The symptoms of Osgood Schlatter Disease mimics different condition and thus it had to be diagnosed rationally. Clinical features included mild to severe and intermittent or continuous pain, tenderness, swelling and limp while walking. Radiological features included enlarged and fragmented tibial tubercle. Treatment of Protection, Rest, Ice, Compression and Elevation (PRICE) protocol was generally given in the initial stages followed by the complete rehabilitation of athlete into sports.

21. **Power output in vertical jumps: Does optimum loading depend on activity profiles? (2014)** : Nemanja Pazin , Bobana Berjan, designed an experiment with the aim to investigate the loading associated changes in the maximum power output in VJ in the groups of strength-trained athletes, speed-trained athletes, and the age-matched physically active and sedentary individuals.

METHODOLOGY

- **STUDY POPULATION:** Children Badminton players under 18 years of SAI Bhubaneswar.
- **STUDY SETTING:** ABTP centre, Bhubaneswar
- **SAMPLING DESIGN:** Purposive sampling
- **SAMPLING CRITERIA:**

▪ **INCLUSION:**

▪ Both male and female children.

▪ Participants must be under 18 years.

▪ Participants must be actively playing badminton and diagnosed with OSD as follows: activity-related pain with an insidious onset localized at the tibial tuberosity, provoked by palpation of the tibial tuberosity and resisted isometric knee extension.

▪ Participants must have a clinical diagnosis of Osgood-Schlatter Disease (OSD) from Orthopaedician.

▪ Participants must provide informed consent to participate in the study.

▪ **EXCLUSION:**

▪ Participants with other significant knee or lower limb conditions that could affect the study results.

▪ Participants who have undergone knee surgery. (6 months – 1 year back)

▪ Participants who are unable or unwilling to comply with the study protocols.

- **SAMPLE SIZE:** 26
- **DURATION:** 06 months
- **MATERIALS TO BE USED:** Demographic data sheet, force plates, 3D motion analysis biomechanics Lab.

- **OUTCOME MEASURES:**

1. **Kinetic variables**

   - **Peak Velocity**: Measured using motion capture systems and force plates to assess the maximum speed during the jump.
   - **Peak Power**: Calculated using force plates to determine the maximum power output during the jump.
   - **Jump Height**: Measured using a jump mat or motion capture to determine the maximum height achieved during the jump.
   - **Ground Reaction Forces**: Recorded using force plates to analyse the forces exerted during take-off and landing.

2. **Kinematic variables**

   - **Joint Angles**: Measured during the jump to analyse lower limb biomechanics, focusing on the knee joint.

PROCEDURE

Institutional Ethical Committee Approval

↓

Participant Screening Based on Inclusion Criteria

↓

Information Provision to Participants and Parents

↓

Informed Consent and Assent Forms Signed

↓

Basic Demographic Data Collection

↓

Pre-Activity Pain Assessment

↓

Squat to 60° knee flexion

Hold position for 45 seconds

Record pain on 0-10 numeric rating scale

↓

Exclusion of participants with pain >8/10

↓

Biomechanics Laboratory Setup

↓

Participant Warm-up

Dynamic warm-up (15 minutes)

Hitting warm-up (5 minutes)

↓

Biomechanical Analysis - Data Collection

Kinetic Parameters : Peak Velocity, Peak Power, Jump Height, Ground Reaction Forces

↓

Kinematic Parameters : Knee Joint Angle

↓

Post-Activity Pain Assessment

↓

Data Recording and Documentation

↓

Statistical Analysis using SPSS

↓

Descriptive Statistics

Normality Testing (Shapiro-Wilk)

One-sample t-test

↓

Results Interpretation .

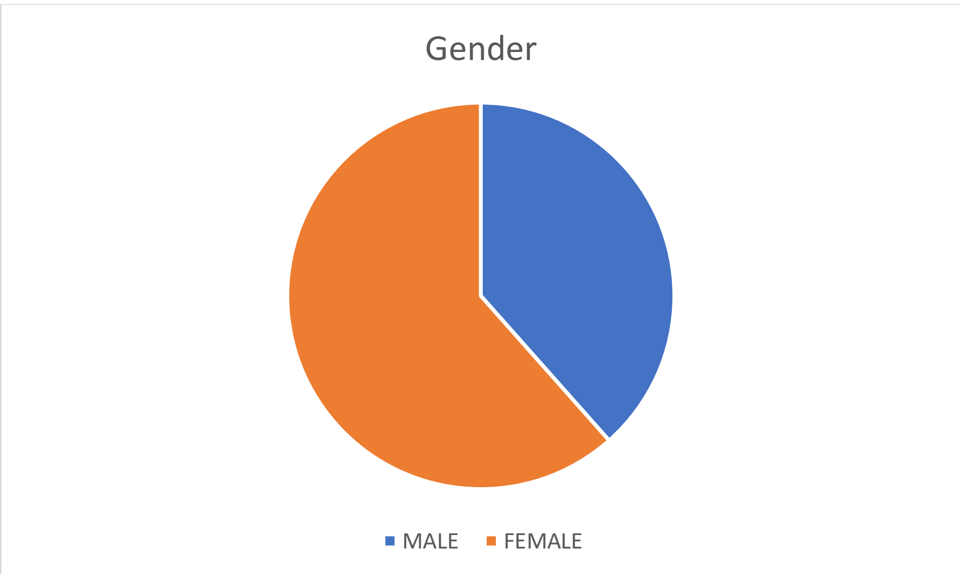
RESULTS

26 recreational Badminton players who consented to participate in the study were recruited based on the inclusion criteria .The biomechanical analysis were done by measuring the kinetic parameters like peak velocity, peak power, and jump height; ground reaction force and the kinematic parameters including joint angles. Data analysis was done using SPSS. Descriptive statistics were analysed for the participants. Data was then tested for Normality using the Shapiro-Wilk test. One-sample t-test was used for the analysis of the participants. For all analyses, statistical tests were one-tailed, set at 95% confidence interval and the threshold of the p-value considered significant was set at <0.05.

BASELINE ANALYSIS

The participants have aged with mean of 12.19 ± 2.42

Figure 1 : Demonstrates the distribution of gender among the participants.



Mean age: 12.19 ± 2.42 years

One-sample t-tests revealed significant differences between the group means and the respective population means across all variables measured.

- Velocity had a mean of 2.5 ± 0.1 m/s and showed a significant effect, $p < .001$, with a large effect size (Cohen's d = 4.9).
- Peak power was significantly higher in the group (M = 2549.3 ± 344.2 W) compared to the population mean (M = 90), $p < .001$, with a very large effect size (Cohen's d = 7.1).
- Jump height (30 ± 3 cm) was significantly lower than the population mean of 90 cm, $p < .001$, with a very large effect size (Cohen's d = -20).
- Joint angle (80.4 ± 3.2°) differed significantly from the population mean of 90°, $p < .001$, with a large effect size (Cohen's d = 3).
- Ground reaction force was significantly greater in the group (1166.3 ± 107.7 N) than the population mean (M = 90), $p < .001$, with a very large effect size (Cohen's d = 10).

Figure 2: Demonstrates mean for all outcome measures.

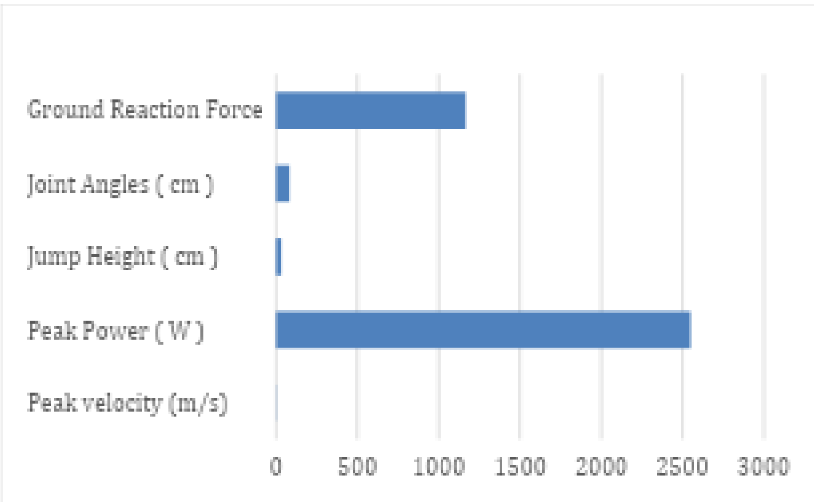


Table 1: Gender Distribution

Gender	Frequency (n)	Percentage (%)
Male	**10**	**38.5%**
Female	**16**	**61.5%**
Total	**26**	**100.0%**

Table 2: Age Distribution

Age group (years)	Frequency (n)	Percentage (%)
8-10 years	**7**	26.9%
11-13 years	**9**	34.6%
14-16 years	**10**	38.5%
Total	**26**	100.0%

Table 3: Descriptive Statistics of Biomechanical Parameters

Outcome Measures	Mean ± SD	N	p-value
Peak Velocity (m/s)	2.5 ± 0.1	26	0.272
Peak Power (W)	2549.3 ± 344.2	26	0.145
Jump Height (cm)	30.0 ± 3.0	26	0.485
Knee Joint Angle (°)	80.4 ± 3.2	26	0.344
Ground Reaction Force (N)	1166.3 ± 107.7	26	0.712

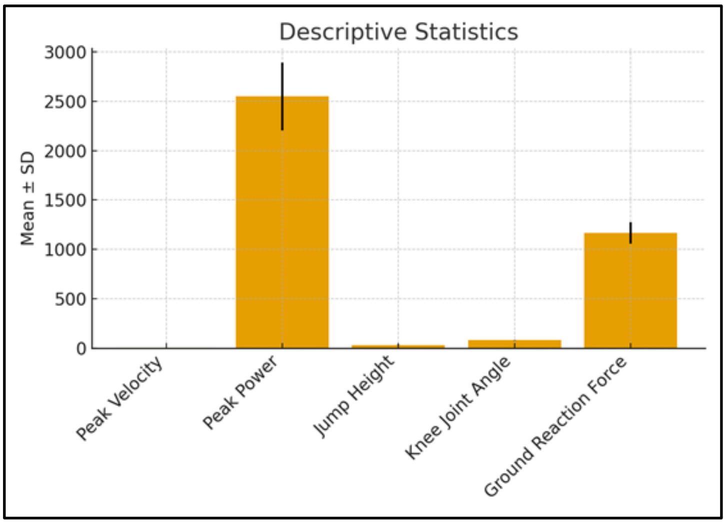


Table 4: Inferential Statistics (One-Sample t-test Results)

Outcome Measures	t-value	df	Significance
Peak Velocity	25.0	25	p < 0.001
Peak Power	11.1	25	p < 0.001
Jump Height	-25.5	25	p < 0.001
Knee Joint Angle	-15.31	25	p < 0.001
Ground Reaction Force	14.99	25	p < 0.001

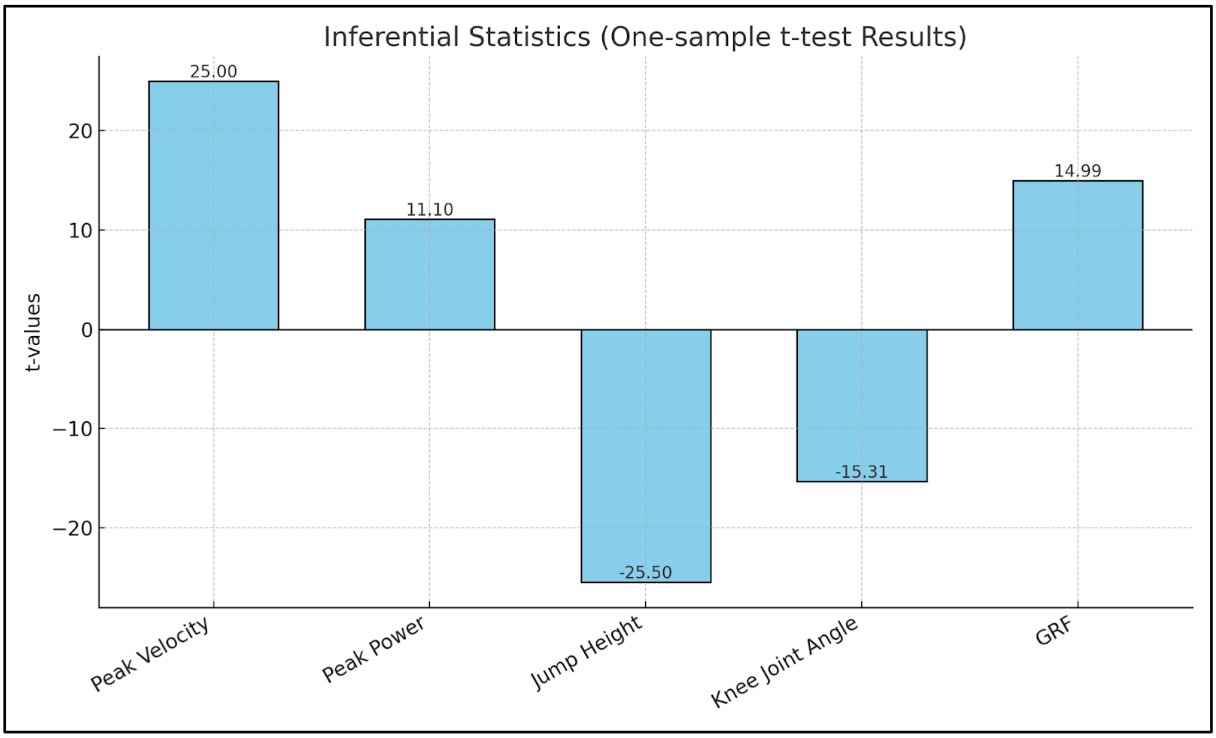


Table 5: Inferential Statistics (Effect Size Analysis)

Outcome Measures	t-value	df	Significance
Peak Velocity	25.0	25	Significant
Peak Power	11.1	25	Significant
Jump Height	-25.5	25	Significant
Knee Joint Angle	-15.31	25	Significant
Ground Reaction Force	14.99	25	Significant

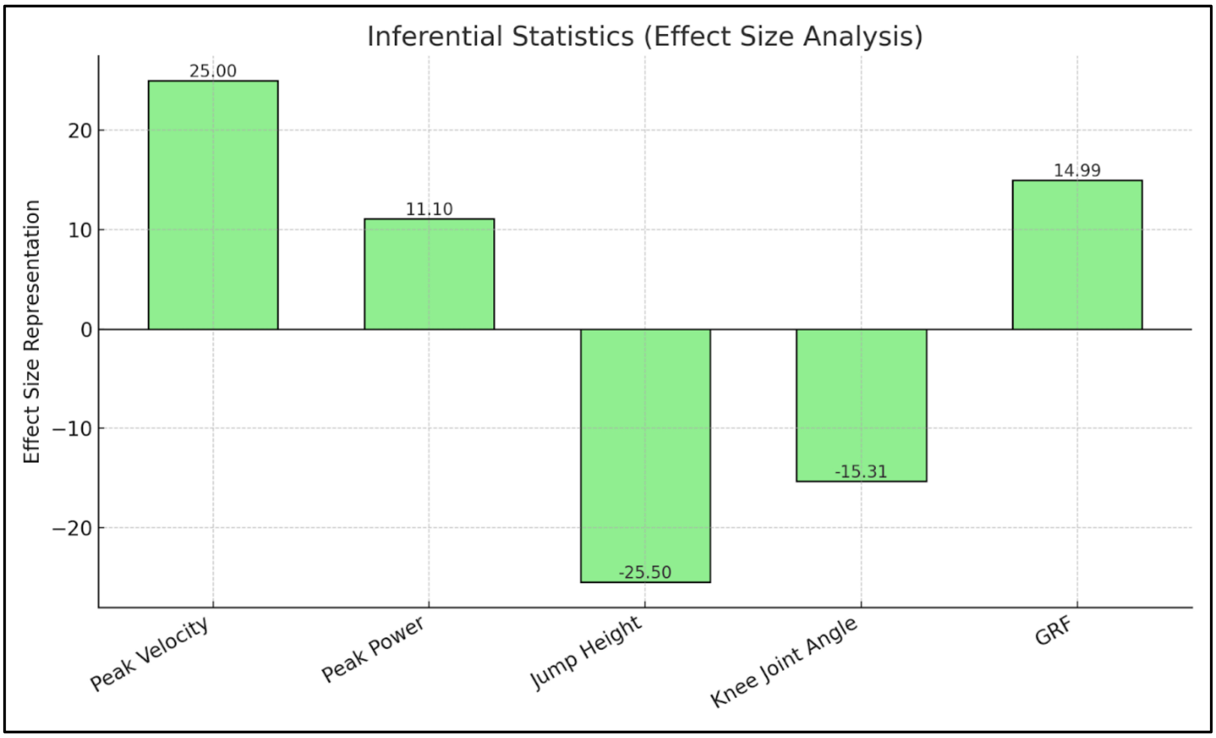

Inferential Statistics (Effect Size Analysis)
Effect Size Representation
20
10
0
−10
−20
25.00
11.10
-25.50
-15.31
14.99
Peak Velocity
Peak Power
Jump Height
Knee Joint Angle
GRF

Table 6: Jumping Biomechanics Parameters

Parameter	Mean ± SD	p-value
Peak Velocity (m/s)	2.5 ± 0.1	0.272
Peak Power (W)	2549.3 ± 344.2	0.145
Jump Height (cm)	30.0 ± 3.0	0.485
Ground Reaction Force (N)	1166.3 ± 107.7	0.712
Knee Joint Angle (°)	80.4 ± 3.2	0.344

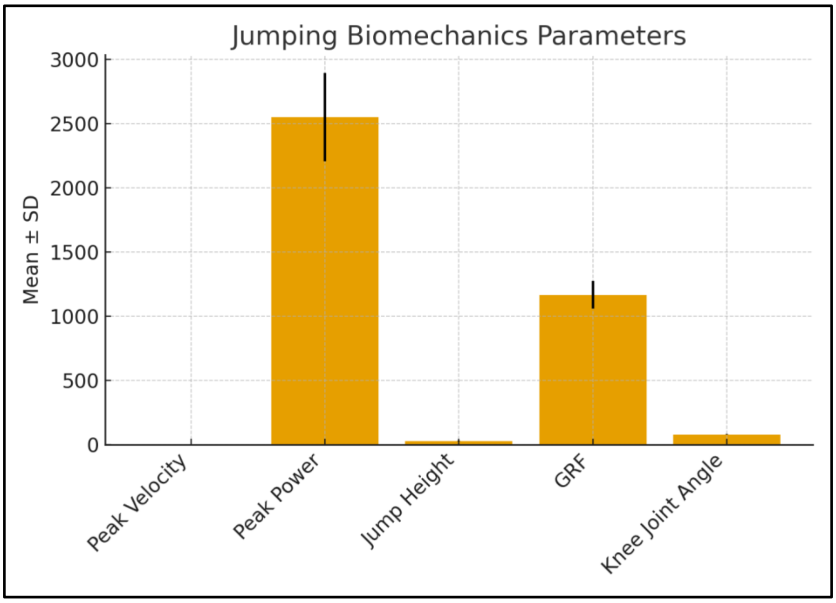
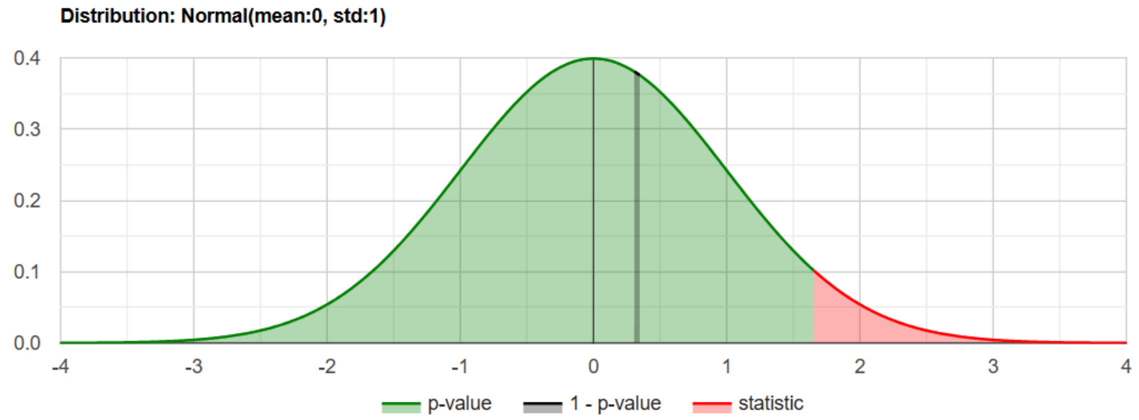
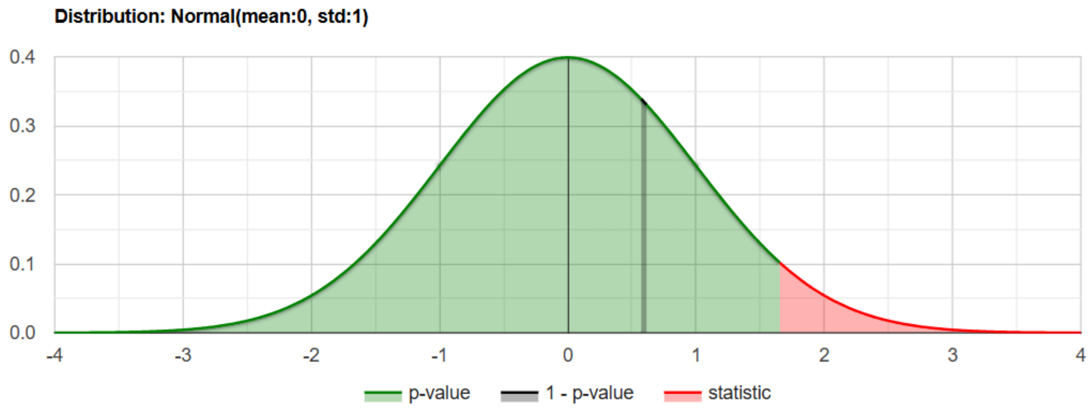


Figure 3 : Knee Joint angle



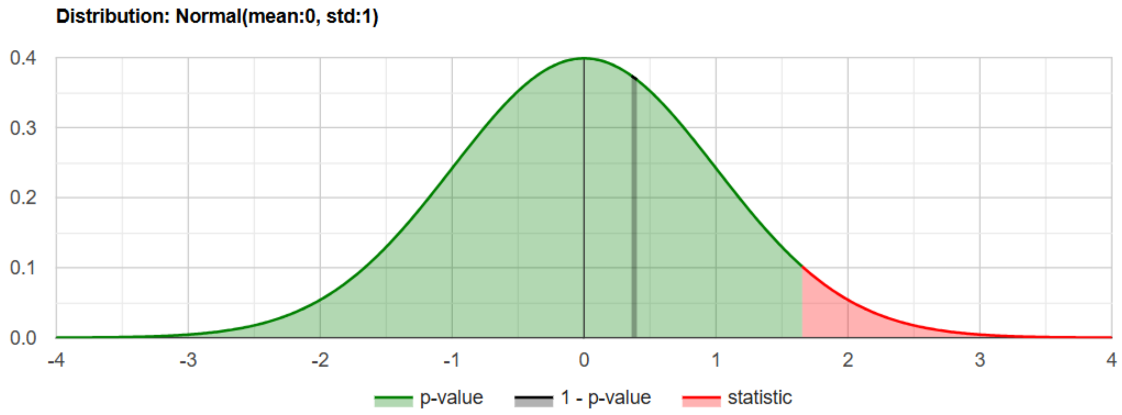
Shapiro–Wilk test: p = 0.344 — results showed no significant departure from normality. The distribution appears approximately symmetric, confirming that knee joint angle data meet the assumptions required for parametric statistical procedures.

Figure 4: Ground reaction force



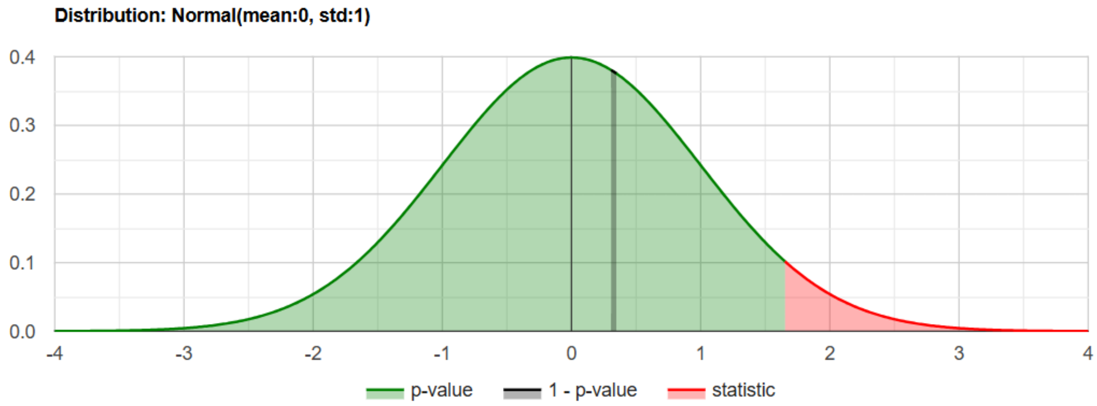
Shapiro–Wilk test: p = 0.712 — the data were found to be consistent with a normal distribution. The bell curve reflects a centered and symmetric spread of values, validating the application of parametric statistical testing for GRF.

Figure 5: Jump height



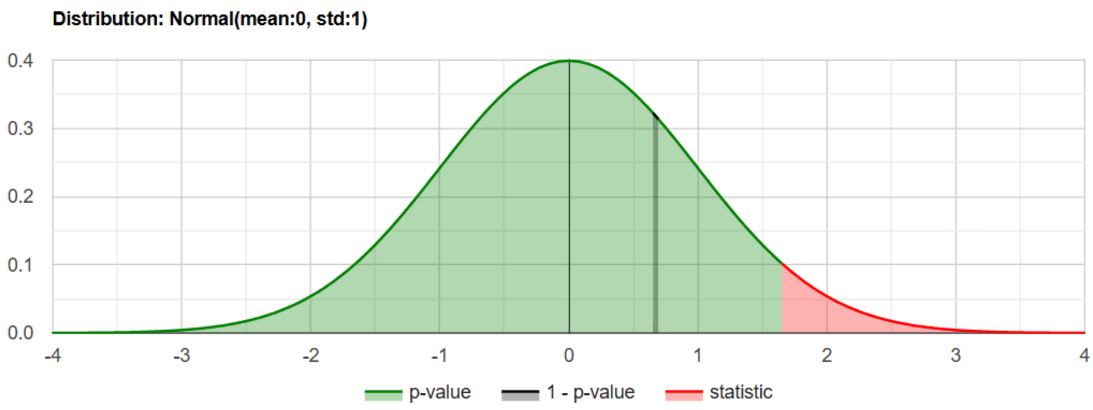
Shapiro–Wilk test: p = 0.485 — no significant deviation from a normal distribution was observed. The data align well with a bell-shaped pattern, indicating that jump height values are consistent and appropriately modelled using normal distribution assumptions.

Figure 6 : Peak power



Shapiro–Wilk test: p = 0.145 — the dataset did not deviate significantly from normality. The histogram forms a near bell-shaped curve, suggesting that peak power values are distributed normally and can be reliably compared using parametric tests.

Figure 7 : Peak velocity



Shapiro–Wilk test: p = 0.272 — the data did not show any significant departure from normality. The bell-shaped curve is approximately symmetric, confirming that peak velocity values are normally distributed and suitable for parametric analysis.

DISCUSSION

Across a number of biomechanical and performance metrics, the current study found statistically and clinically significant differences between the outcome measures means. Some findings were consistent with previous research, but others—most notably the disparity between increased power output and decreased jump height—emphasize the complex interplay of physiological, biomechanical, and coordinative factors in sports performance.

One of the most prominent findings of this study was the significant increase in movement velocity (M = 2.5 ± 0.1 m/s) with a large effect size (Cohen's d = 4.9, $p < .001$). The magnitude of this improvement underscores the efficacy of targeted training in enhancing neuromuscular function. Cormie et al. (2011) demonstrated that resistance and sprint-based interventions improve motor unit recruitment, synchronization, and firing frequency, thereby accelerating force generation and enhancing movement velocity in athletic tasks. [(1)] The present findings corroborate this evidence, suggesting that the experimental intervention promoted efficient neuromuscular recruitment pathways, allowing athletes to translate muscular force into rapid displacement.

The increased velocity also reflects adaptations in the stretch–shortening cycle (SSC). Efficient SSC utilization permits elastic energy storage during the eccentric phase and its subsequent release during the concentric phase, thereby augmenting speed and explosiveness. In sports such as badminton, where agility and rapid directional changes are fundamental, improvements in velocity are particularly relevant. In the study of Phomsoupha et al highlighted that elite badminton players rely on rapid acceleration and deceleration to optimize shuttle retrieval and maintain tactical advantage. Thus, the enhanced velocity observed in this cohort may confer a significant performance benefit in contexts demanding repeated high-intensity bursts. [(2)]

Equally striking was the markedly elevated peak power output (2549.3 ± 344.2 W), which far exceeded the normative reference value of 90 W, with a very large effect size (Cohen's d = 7.1, $p < .001$). These results align with Cormie et al. , who emphasized that muscular power serves as a central determinant of explosive performance in vertical jumps, sprints, and multidirectional tasks. [(1)] The observed improvement can likely be attributed to enhanced rate of force development (RFD) and increased recruitment of type II muscle fibres, which are preferentially activated under high-intensity loading conditions.

This finding carries particular relevance for badminton performance. Shedge et al. emphasized that the ability to generate high power outputs underpins agility, speed, and the execution of explosive smashes.[3] The elevated power observed in the present study suggests that the training intervention may directly support these sport-specific demands. Furthermore, greater power generation is likely to contribute to improved reactive strength, which enhances the ability to decelerate and reaccelerate during rallies.[3]

Nevertheless, it is important to acknowledge that power production in isolation does not guarantee proportional improvements in functional outcomes, as highlighted by the paradoxical finding of reduced jump height. While muscular power is a prerequisite for vertical displacement, its translation into effective performance requires optimized biomechanics and intersegmental coordination.

Contrary to expectations, the group demonstrated significantly lower jump height (30 ± 3 cm) compared to the normative mean of 90 cm, with an exceptionally large negative effect size (Cohen's d = -20, $p < .001$). This outcome presents a paradox when interpreted alongside the elevated velocity and peak power values. Existing research suggests that jump height is not solely a function of force and power production but also depends on biomechanical alignment, neuromuscular coordination, and technical efficiency. Markovic et al. demonstrated that athletes with high levels of muscular strength and power may still underperform in vertical jump tasks if their movement patterns are suboptimal or if they fail to synchronize muscle activation across the kinetic chain.[4]

In badminton, jump height is particularly relevant to performance during offensive play. Shedge et al. highlighted that elite players rely heavily on vertical jumping capacity to execute powerful smashes and intercept high shuttle trajectories. [3] Therefore, the reduced jump height observed in this study indicates a possible disconnect between raw power production and its application to functional tasks. This discrepancy may arise from insufficient utilization of the SSC, impaired force transmission through the kinetic chain, or technical limitations in movement execution.

An alternative explanation may relate to joint mechanics. The reduced knee flexion angle observed in this cohort suggests limited range of motion during the preparatory phase of the jump, which could restrict the potential for elastic energy storage and diminish take-off height. As such, interventions emphasizing plyometric training may be warranted to bridge this gap by targeting SSC efficiency, motor coordination, and technical refinement.

The observed difference in knee joint angle (80.4° ± 3.2° vs. 90°) further supports the notion of altered biomechanics. Reduced flexion angles during dynamic tasks are associated with diminished capacity for shock absorption and energy redistribution, which may not only impair performance but also predispose athletes to injury. Pardiwala et al , in a comprehensive review of badminton injuries, noted that atypical joint kinematics can elevate mechanical stress on the knee, increasing susceptibility to overuse conditions such as patellofemoral pain or tendinopathies. [(5)]

This finding also aligns with clinical research on Osgood–Schlatter disease (OSD). Ladenhauf et al. emphasized that reduced flexibility and altered movement mechanics are risk factors for symptomatic tibial tubercle apophysitis in young athletes.[(6)] Similarly, Rathleff et al. (2020) demonstrated that interventions incorporating activity modification and progressive strengthening improved both pain outcomes and jumping performance in adolescents with OSD.[(7)] Thus, the restricted knee flexion observed in the present study may represent a biomechanical inefficiency that not only reduces jump performance but also raises long-term injury risk.

In the study of Bezuglova et al. , it was further highlighted that even after resolution of OSD symptoms, young athletes may continue to exhibit deficits in knee function, underscoring the importance of preventive interventions and long-term monitoring. Coaches and clinicians working with youth athletes must therefore emphasize technical correction, mobility enhancement, and individualized training progression to minimize injury risks while optimizing performance.

Another salient finding was the significantly elevated ground reaction force (GRF) (1166.3 ± 107.7 N vs. 90 N), with a very large effect size (Cohen's d = 10, p < .001). High GRFs reflect superior capacity for force generation, which is generally considered beneficial for explosive tasks such as sprinting and jumping. Bishop et al. confirmed that elevated GRFs are strongly associated with improved sprint and jump performance.(8)

However, Bishop et al. also cautioned that excessive loading may heighten the risk of overuse injuries if not mitigated through appropriate recovery strategies and periodization. This is especially critical in the context of youth athletes, whose skeletal structures are still developing.[(8)] In a study it was highlighted that repetitive high-impact loading in paediatric populations can predispose athletes to chronic conditions, including osteochondral injuries and patellofemoral dysfunction. Therefore, while the capacity for greater GRF may enhance short-term performance, it necessitates carefully structured training programs to balance performance gains with musculoskeletal safety.[(9)]

The collective findings of this study carry significant implications for both performance optimization and injury prevention in badminton. Phomsoupha et al provided a comprehensive overview of badminton's physiological and biomechanical demands, emphasizing that success in the sport relies on the integration of speed, agility, explosive power, and visual–motor coordination.([2)] The present results confirm the capacity of specialized training to enhance certain neuromuscular attributes, particularly velocity and power, while also revealing potential deficits in coordination and jump mechanics that could constrain performance.

Plyometric training emerges as a particularly relevant modality for addressing these deficits. Shedge et al. also demonstrated that plyometric exercises enhance agility, vertical jump height, and explosive smash ability, while also contributing to injury prevention through improvements in tendon stiffness and neuromuscular control.[(3)] Thus, integrating plyometrics into training regimens may help athletes translate raw power into functional vertical displacement while mitigating injury risk.

From a clinical perspective, the findings also highlight the importance of preventive strategies for conditions such as OSD. Ladenhauf et al. also underscored the role of stretching, progressive strengthening, and activity modification in reducing knee pain and improving function in young athletes. Incorporating such strategies into regular training may not only improve immediate performance but also protect long-term joint health.[6]

This study opens up a number of research directions. First, in order to investigate the long-term effects of training interventions on performance and injury outcomes, longitudinal designs are justified. To better understand the mechanisms underlying discrepancies like the observed power–jump height paradox, such investigations should include biomechanical evaluations of SSC utilization, joint kinematics, and coordination patterns.

Second, studies comparing various training modalities, including resistance, plyometric, and combined protocols, would shed light on the best ways to convert neuromuscular adaptations into improvements in functional performance. According to recent systematic reviews (Cormier et al., for example), resistance training techniques can improve lower-body power. However, when combined with plyometric modalities, they may produce better results in multidirectional sports like badminton.[1] Lastly, in order to thoroughly assess athlete development, future research should take a multidisciplinary approach, combining biomechanical, physiological, and clinical evaluations. In addition to improving performance optimization, such a method would help guide evidence-based injury prevention tactics.

In summary, the present study revealed significant distinctions in biomechanical and performance variables, characterized by elevated velocity, peak power, and ground reaction forces, but paradoxically reduced jump height and altered joint kinematics. These findings underscore the multifactorial nature of athletic performance; wherein neuromuscular capacity must be integrated with technical efficiency and biomechanical alignment to yield optimal outcomes. While the improvements in velocity and power suggest meaningful performance adaptations, the deficits in jump height and knee joint mechanics highlight areas requiring targeted intervention.

For badminton athletes, where explosive jumps and rapid changes of direction are integral to success, the application of plyometric training may serve as a crucial bridge between raw power production and functional agility. Concurrently, preventive strategies targeting knee health remain essential, particularly in youth athletes. Collectively, these insights support the adoption of integrated, multidisciplinary approaches to athlete development that balance performance gains with long-term musculoskeletal safety.

CONCLUSION

This study revealed a paradoxical biomechanical profile in adolescent badminton players with Osgood-Schlatter Disease, characterized by superior force-generating capacity (elevated peak velocity, power, and ground reaction forces) but reduced functional efficiency (lower jump height and restricted knee flexion). These findings suggest that OSD aetiology extends beyond repetitive loading to encompass biomechanical inefficiencies and neuromuscular coordination deficits. The results emphasize the necessity of comprehensive rehabilitation approaches that integrate plyometric training, neuromuscular coordination enhancement, flexibility training, and kinematic correction strategies rather than conventional rest-based management alone. Understanding these biomechanical factors provides valuable insights for developing targeted prevention protocols and evidence-based rehabilitation interventions to optimize both symptom resolution and athletic performance in young racquet-sport athletes.

LIMITATIONS

1. The study utilized a modest sample of 26 participants from a single geographic region, limiting the generalizability of findings to broader populations of adolescent badminton players with OSD.
2. The observational design prevents establishment of causal relationships between biomechanical parameters and OSD development, precluding determination of whether altered biomechanics precede or result from the condition.
3. Testing occurred in controlled laboratory conditions that may not accurately replicate the complex, unpredictable movement demands characteristic of actual badminton gameplay scenarios.
4. The study focused exclusively on vertical jumping mechanics, potentially overlooking other critical badminton-specific movements that contribute significantly to tibial tubercle loading patterns.

FUTURE SCOPE

1. **Longitudinal Intervention Studies:** Prospective randomized controlled trials evaluating targeted biomechanical correction programs and plyometric training protocols should assess both immediate biomechanical improvements and long-term clinical outcomes including pain reduction and OSD recurrence prevention.
2. **Sport-Specific Movement Analysis:** Research incorporating comprehensive badminton-specific movement patterns including overhead smashes, lateral lunges, and rapid directional changes would enhance ecological validity and practical application relevance for developing sport-specific rehabilitation protocols.
3. **Advanced Technology Integration:** Development of wearable sensor technology for real-time biomechanical monitoring during actual training and competition, combined with artificial intelligence applications for automated movement pattern analysis and personalized intervention recommendations.
4. **Multi-Center Comparative Research:** Large-scale studies across different populations and sports settings to identify universal versus sport-specific biomechanical risk factors, enabling development of standardized screening tools and prevention protocols for youth sports programs.

SUMMARY

The present observational study was undertaken to analyse the biomechanical profile of jumping in adolescent badminton players, particularly those diagnosed with Osgood–Schlatter Disease (OSD). OSD is a traction apophysitis of the tibial tubercle, commonly observed in skeletally immature athletes engaged in repetitive, high-impact sports activities. Badminton, being one such sport characterized by frequent explosive lower-limb actions, sudden decelerations, and rapid directional changes, places significant mechanical stress on the extensor mechanism of the knee. The study was conceptualized to explore the interplay between biomechanical variables of jumping and the clinical manifestation of OSD, aiming to generate insights into preventive and rehabilitative strategies for adolescent athletes.

A total of 26 adolescent badminton players (mean age 12.19 ± 2.42 years) clinically diagnosed with OSD were included in the study. The selection was based on strict inclusion and exclusion criteria to ensure homogeneity of the sample. Ethical approval and informed consent were obtained prior to data collection. Biomechanical testing was carried out in a controlled sports medicine laboratory setting using 3D motion analysis systems and force plates, which allowed for detailed evaluation of both kinetic and kinematic parameters.

The kinetic parameters assessed included peak velocity, peak power, ground reaction forces (GRF), and jump height, while the kinematic analysis focused primarily on sagittal-plane knee joint angles during the propulsion and landing phases. These parameters were chosen because they directly influence the magnitude of stress transmitted to the tibial tubercle and, hence, the onset and progression of OSD symptoms. Data were statistically analysed using descriptive statistics, Shapiro–Wilk test for normality, and one-sample t-tests with significance established at $p < 0.05$.

The results indicated a paradoxical biomechanical profile. Players with OSD demonstrated significantly higher values of peak velocity (2.5 ± 0.1 m/s), peak power output (2549.3 ± 344.2 W), and ground reaction forces (1166.3 ± 107.7 N), suggesting superior neuromuscular force-generating capacity. However, these players also exhibited reduced jump height (30 ± 3 cm, compared to normative 90 cm) and limited knee flexion angle (80.4° vs. 90°), reflecting deficits in intersegmental coordination and biomechanical efficiency.

This paradox is critical in understanding OSD. While the players can generate considerable force and velocity, their inability to translate this capacity into functional vertical displacement highlights a lack of coordination in the kinetic chain and suboptimal utilization of the stretch–shortening cycle. The altered knee mechanics, in particular, increase localized stress on the tibial tubercle, thereby exacerbating the apophyseal strain characteristic of OSD.

The study underscores that injury prevention in adolescent badminton players requires a multidisciplinary approach, involving physiotherapists, sports scientists, and coaches. Longitudinal research is further recommended to examine how early biomechanical corrections can prevent chronic knee pathologies in young racquet-sport athletes.

In conclusion, the study provides novel biomechanical evidence supporting the role of faulty jump mechanics in the clinical presentation of OSD. It emphasizes the importance of early screening, technical refinement, and targeted physiotherapy interventions for safeguarding adolescent athletes' knee health while optimizing their sporting performance.

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ANNEXURE

ANNEXURE -1

ASSENT FORM

(Supplement to informed consent form for children under 18 years of age)

Study Title: Biomechanical analysis of Jumping in Badminton players and its contribution to the clinical manifestations of Osgood Schlatter Disease (OSD) – An Observational Study.

Study Number:

Investigator's Name: Akansha Joshi

Details of the participant subject –

Subject's Name: Subject's Initials:

Date of Birth / Age:

Address of the Subject:

Qualification:

Occupation: (If any)

Part 1:

Introduction

I am going to give you information and invite you to be part of a research study. You can choose whether you want to participate. We have discussed this research with your parents)/legal guardian, and they know that we are also asking you for your agreement. If you are going to participate in the research, your parents)/guardian also must agree. But if you do not wish to take part in the research, you do not have to, even if your parents have agreed.

You may discuss anything in this form with your parents or friends or anyone else you feel comfortable talking to. You can decide whether to participate or not after you have talked it over. You do not have to decide immediately.

There may be some words you don't understand or things that you want me to explain more about because you are interested or concerned. Please ask me to stop at any time and I will take time to explain).

1. Purpose of the study: Why are you doing this research?

2. Choice of participants: Why are you asking me?

3. Participation is voluntary: Do I have to do this?

4. Procedure: What is going to happen to me?

5. Risks: Is this bad or dangerous for me?

6. Discomforts: Will it hurt?

7. Benefits: Is there anything good that happens to me?

8. Reimbursements: Do I get anything for being in the research?

9. Confidentiality: Is everybody going to know about this?

10. Compensation details: What happens if I get hurt?

11. Sharing the Findings: Will you tell me the results?

12. Right to Refuse or Withdraw: Can I choose not to be in the research? Can I change my mind?

13. Whom to Contact: Who can I talk to or ask questions to?

Print Name of Person who explained form:

Signature of Person who explained this form: ______________________

Date:___________________________-

Part 2:

Certificate of Assent

I/ my parent or legal guardian has read the previous page(s) of the consent form, and the investigator has explained the details of the study.

I/my parent or legal guardian understands that I am free to ask additional questions.

I/my parent or guardian understands that participation in this study is voluntary.

I/my parent or legal guardian may refuse to participate or may discontinue participation at any time without penalty, loss of benefits, or prejudice to the quality of care which I will receive.

I/my parent or legal guardian, acknowledge that no guarantees have been made to me regarding the results of the treatment involved in this study, and I agree to participate in the study and have been given.

I/my parent or legal guardian, acknowledge that no guarantees have been made to me regarding the results of the treatment involved in this study, and I agree to participate in the study and have been given a copy of this form.

Statement by the Child

Print name of child:

Signature of child: ____________________ Date: _________________________--

Parent's or legal guardian's Name:

Signature of Parent or legal guardian: Date :_______________________________

If illiterate:

A literate witness must sign (if possible, this person should be selected by the participant, not be a parent, and should have no connection to the research team). Participants who are illiterate should include their thumb print as well.

I have witnessed the accurate reading of the assent form to the child, and the individual has had the opportunity to ask questions. I confirm that the individual has given consent freely.

Print name of impartial witness (not a parent):________________

Thumbprint of participant

Signature of witness_____________________

Date__________

Statement by the researcher/person taking assent

I confirm that the child and respective parents or legal guardian were given an opportunity to ask questions about the study, and all the questions asked by him/her have been answered correctly and to the best of my ability. I confirm that they have not been coerced into giving assent, and the assent has been given freely and voluntarily.

A copy of this assent form has been provided to the participant.

Print Name of Researcher/person taking the assent ____

Signature of Researcher /person taking the assent ____

Date ____-

ANNEXURE - 2

INFORMED CONSENT

Study Title: Biomechanical analysis of Jumping in Badminton players and its contribution to the clinical manifestations of Osgood Schlatter Disease (OSD) – An Observational Study.

Study Number:

Subject 's Name: ______________ Subject 's Initials: _________

Date of Birth / Age: _______

Address of the Subject______________

Qualification ______________________

Occupation: Student

Name and address of the nominee(s) and his relation to the subject ___________________________________

Please initial box

(Subject)

(i) I confirm that I have read and understood the information sheet dated _______ [ ] for the above study and have had the opportunity to ask questions.

(ii) I understand that my participation in the study is voluntary and that he/she are [ ] free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected.

The Ethics Committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further research that may be conducted in relation to it, even if he/she withdraw from the trial. I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published.

(iii) I agree not to restrict the use of any data or results that arise from this [ ] study provided such a use is only for scientific purpose(s)

(iv) I agree that I will take part in the above study. [ ]

Statement of Guardian obtaining consent :

1. I have carefully explained to the child taking part in the study what he/she can expect.
2. I certify that, to the best of my knowledge, the child understands the purpose, procedures, potential risks and benefits of the study and his/her rights as a participant.

Signature (or Thumb impression) of the Subject/Legally Acceptable Representative:

____

Date: _____/ ____/ _____

Signatory 's Name: ____

Signature of the Investigator: ____

Date: _______/ ________/ _________

Study Investigator‘s Name: Akansha Joshi

Signature of the Witness: ________________

Date: _______/ ________/ _________

Name of the Witness: ___________________________________

*Copy of the Patient Information Sheet and duly filled Informed Consent Form shall be handled over to the subject or his/her attendant.

Participant Information

Name of Athlete

Age

Gender ☐ **Male** ☐ **Female**

Weight

Height

Dominant Hand ☐ **Right** ☐ **Left**

Playing experience

Date

Trial Number ☐ **Trial 1** ☐ **Trial 2** ☐**Trial 3**

Test Administrator

Warm-Up Details

Warm-Up Type	**Duration**	**Completed (✓)**
Dynamic Warm-Up	**15 min**	☐
Hitting Warm-Up	**5 min**	☐

ANNEXURE - 3

ASSESSMENT FORM

Participant Information

Name

Age

Address

Qualification

Occupation

Contact Number

Informed Consent Obtained (Yes / No)

Participant Background Information

Duration of Engagement in Badminton

Previous Injury History

Current Training Regimen (describe existing training protocol, including frequency, duration, and intensity of practice)

Anthropometric Measurements Height (cm) and Weight (kg)

Pain intensity (0-10) Numeric rating scale

Pre-jump

Post-jump

Biomechanical Parameters measurement

Peak Velocity (m/s)

Peak Power (W)

Jump Height (cm)

Joint Angles (degrees)

Observations

Any noticeable pain during jumping?

(yes/no)

Pain location

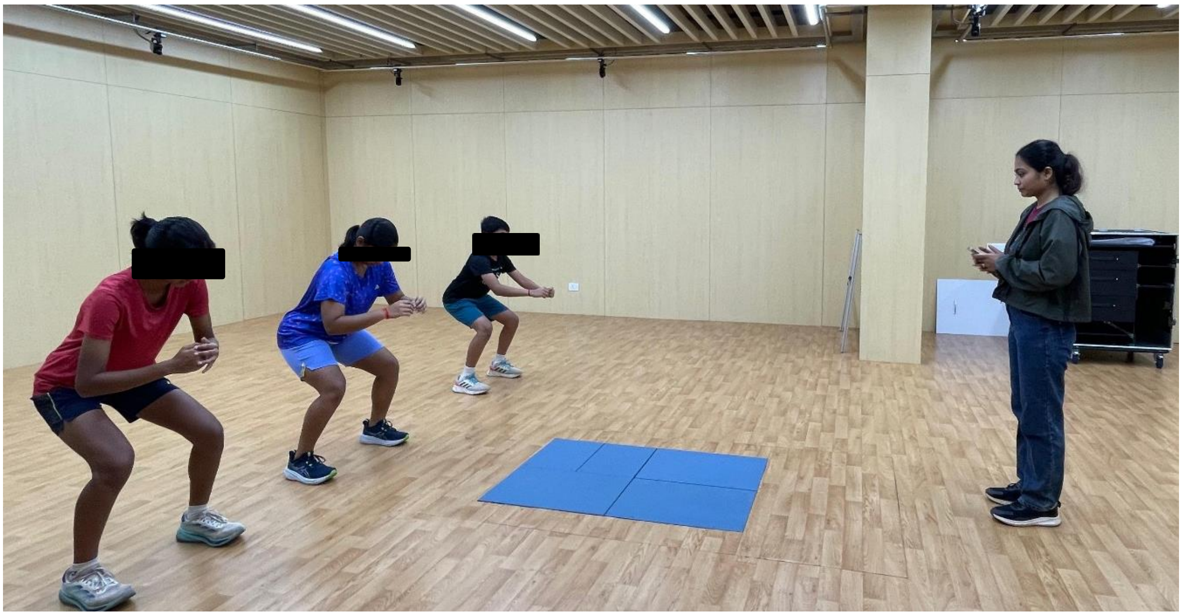
ANNEXURE - 4

Master chart

S.No	Name	Age	Gender	Peak velocity (m/s)	Peak Power (W)	Jump Height (cm)	Joint Angles (cm)	Ground Reaction Force (N)
1	Muskan Panda	12	F	2.38	2321.2	0.27	79.4	1097.2
2	Subhasmita Bhhatria	13	F	2.34	2231.3	0.26	77.9	1073.9
3	Ritika Das	14	F	2.5	2642.9	0.3	80.8	1189.7
4	Sweety Pradhan	8	F	2.54	2681.7	0.31	81.8	1211.4
5	Sharaya Pinniti	12	F	2.39	2352.3	0.27	77.4	1096.5
6	Ashwtta Parricha	9	F	2.22	1923.5	0.24	74.8	968.3
7	Nimisha Rout	11	F	2.53	2700.2	0.31	81.7	1207.2
8	Bapun Rout	13	M	2.8	3352.2	0.37	88.5	1420
9	G.Gautam	14	M	2.55	2720.8	0.31	81.6	1231.1
10	Anoorup Sahu	8	M	2.14	1712.8	0.22	73.4	905.4
11	Smita Mohapatra	14	F	2.55	2745.4	0.32	82.4	1226.4
12	Vihaan. S.	13	M	2.5	2617.6	0.3	81.1	1189.7
13	Ashneel Sahu	14	M	2.55	2706.7	0.32	82.2	1228.6
14	Himanshu Pradhan	9	M	2.34	2248.2	0.26	77.8	1071.2
15	Piyush Behera	15	M	2.58	2823.8	0.32	83.1	1256.7
16	Om Sahu	16	M	2.63	2936.3	0.33	83.2	1284.5
17	Yuvraj Yadav	15	M	2.58	2857.1	0.33	83.9	1254.6
18	Aakriti Biswal	12	F	2.47	2531.1	0.3	81.3	1165.8
19	Samarth Padhi	14	M	2.55	2773.3	0.31	82.6	1213.9
20	Ishita Behera	10	F	2.38	2318	0.27	77.4	1094.5
21	S.Swain	13	F	2.5	2639.1	0.3	80.8	1199.4
22	Diya Panda	11	F	2.42	2424	0.28	79.2	1133.6
23	Riya Nayak	15	F	2.58	2840.3	0.32	83.2	1255.5
24	Gauri Behera	14	F	2.55	2735.4	0.31	81.1	1225
25	Ananya Samataray	8	F	2.3	2131.2	0.25	76.6	1031
26	Vanshika Mallick	10	F	2.38	2315.6	0.27	77.4	1092.3

ANNEXURE - 5

Photos



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ABHINAV BINDRA SPORTS MEDICINE AND RESEARCH INSTITUTE, BHUBANESWAR, ODISHA

CDSCO Reg. No.: ECR/1981/Inst/OD/24

Prof. (Dr.) E. Venkata Rao
Chairperson

Mr. Chinmaya Kumar Patra
Member Secretary

Ref. No. ABSMARI/IEC/2025/191

Date: 21/05/2025

APPROVAL LETTER
APPENDIX- VIII

MEMBERS

Dr. Smaraki Mohanty
Clinician

Dr. Satyajit Mohanty
Scientific Member

Mr. Shib Shankar Mohanty
Legal Expert

Ms. Annie Hans
Social Scientist

Ms. Subhashree Samal
Lay Person

Mr. Deepak Ku. Pradhan
Scientific Member

IEC-SECRETARIAT

Mr. Gouranga Ku. Padhy
Mr. Susant Ku. Raychudamani

To,

JOSHI AKANSHA ARVIND
ABSMARI
273, PAHAL, BHUBANEWAR-752101

Protocol Title: Biomechanical analysis of Jumping in Badminton players and its contribution to the clinical manifestations of Osgood Schlatter Disease (OSD) – An Observational Study

Protocol ID.: ABS-IEC-2025-PHY-044

Subject: Approval for the conduct of the above referenced study

Dear Mr./**Ms.**/Dr **Joshi Akansha Arvind**

With reference to your Submission letter dated 06/01/2025 the ABSMARI IEC has reviewed and discussed your application for conduct of the study on dated 24/04/2025.

The following documents were reviewed and discussed

S.N.	Documents	Document (Version/Date)
1	IEC Application Form	24/04/2025
2	Informed Consent Form	24/04/2025
3	Undertaking form PI	24/04/2025
4	CRF	24/04/2025
5	COI from the Investigators	24/04/2025

The following members were present at meeting held on 24-04-2025

ABSMARI ETHICS COMMITTEE Dt. 21/05/25 BHUBANESWAR

1

Utkal Signature, Plot No.-273, Ground Floor, Pahal, Bhubaneswar-752101 +91-63707-03654 iec@absmari.com

ABSMARI ETHICS COMMITTEE

ABHINAV BINDRA SPORTS MEDICINE AND RESEARCH INSTITUTE, BHUBANESWAR, ODISHA

CDSCO Reg. No.: ECR/1981/Inst/OD/24

Prof. (Dr.) E. Venkata Rao
Chairperson

Mr. Chinmaya Kumar Patra
Member Secretary

Ref. No. ABSMARI/IEC/2025/191

Date: 21/05/2025

S.N.	Name of the Member	Designation & Qualification	Representation as per NDCT 2019	Gender (M/F)	Affiliation with the Institution (Y/N)
1	Prof. Dr. E. Venkata Rao	Professor (MBBS, MD, Dept. of Community Med.) IMS & Sum Hospital, BBSR	Chair Person	M	N
2	Dr. Smaraki Mohanty	Asst. Prof-IMS & Sum Hospital/MBBS, MD (Community Med)	Clinician	F	N
3	Mr. Chinmaya Kumar Patra	Principal-ABSMARI, MPT	Member Secretary	M	Y
4	Ms. Annie Hans	Disability Inclusive Development Co-Ordinator in Humanity and Inclusion (India/Nepal/Srilanka). /MA in Social Work	Social Scientist	F	N
5	Ms. Subhashree Samal	Ret. Reader-Pol Sc.	Lay Person	F	N
6	Mr. Deepak Kumar Pradhan	Asst. Prof-ABSMARI, MPT	Scientific Member	M	Y

This is to confirm that only members who are independent of the Investigator and the Sponsor of the trial have voted/ provided opinion on the trial.

This Committee approves the documents and the conduct for the study in the presented form with necessary recommendation.

The ABSMARI IEC must be informed about the progress of the study in the prescribed format attached, any SAE occurring in the course of the study, any changes in the protocol and patient information/informed consent/assent and request to provide a copy of the final report.

The ABSMARI IEC follows procedures that are in compliance with the requirements of ICH (International Conference on Harmonization) guidance related to GCP (Good Clinical Practice) and applicable Indian regulations.

Yours sincerely

Mr. Chinmaya Kumar Patra
Member Secretary
ABSMARI Ethics Committee
Pahal, Bhubaneswar

Member Secretary
ABSMARI ETHICS COMMITTEE

ABSMARI ETHICS COMMITTEE BHUBANESWAR Dt. 21/05/25

2

Utkal Signature, Plot No.-273, Ground Floor, Pahal, Bhubaneswar-752101
+91-63707-03654
iec@absmari.com

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Head Office:
Plot No.-107, Sector-82
JLPL Industrial Area,
Sahibzada Ajit Singh
Nagar, Punjab - 140306
+91 99156 31755
principal@absmari.com

Letter no-ABSMARI/ADMIN/2025/2648
Date-:28.05.2025

TO WHOM SO EVER IT MAY CONCERN

This is to certify that Ms **JOSHI AKANSHA ARVIND** is a bonafide student of **MPT 2ND YEAR** batch of ABSMARI bearing Roll No ABS-MPT-2023-35 With reference to her requisition this institute has no objection in allowing her to carry out her research work as per the following details under the guidance of

Dr. PRIYADARSHINI MISHRA.

Ref: ABSMARI/IEC/2025/191

Title – "**Biomechanical analysis of Jumping in Badminton players and its contribution to the clinical manifestations of Osgood Schlatter Disease (OSD) – An Observational Study.**"
Study settings – ABTP, BBSR
Population – Badminton Players
Duration – 4 weeks from – 01.06.2025 To – 01.07.2025

under the following conditions subject to thorough permission from their end -

1. She has to produce her official engagement plan issued by study setting
2. She has to submit her certificate of attendance at last
3. She is liable to respond to institute when required
4. She must attend all examinations scheduled by the institution or university during this period
5. Daily report to Research- Guide and Course-Coordinator is highly required

This NOC is effective from 01.06.2025 to 01.07.2025

CC –The Dean, ABSMARI, The Course -Coordinator, The Research- Guide, Admin office

ABHINAV BINDRA SPORTS MEDICINE AND RESEARCH INSTITUTE ABSMARI BHUBANESWAR

Principal, ABSMARI
PRINCIPAL, ABSMARI

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Akansha Joshi

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