International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Scholarly Publisher
RS Global Sp. z O.O.
ISNI: 0000 0004 8495 2390

Dolna 17, Warsaw,
Poland 00-773
+48 226 0 227 03
editorial_office@rsglobal.pl

ARTICLE TITLE	A REVIEW OF RECENT REPORTS ON THE TREATMENT OF OSGOOD-SCHLATTER DISEASE
DOI	https://doi.org/10.31435/ijitss.3(47).2025.4004
RECEIVED	15 August 2025
ACCEPTED	24 September 2025
PUBLISHED	30 September 2025
LICENSE	The article is licensed under a **Creative Commons Attribution 4.0 International License.**

A REVIEW OF RECENT REPORTS ON THE TREATMENT OF OSGOOD-SCHLATTER DISEASE

Mateusz Korga *(Corresponding Author, Email: mati.korga@gmail.com)*
Stefan Cardinal Wyszyński Provincial Specialist Hospital SPZOZ, Lublin, Poland
ORCID ID: 0000-0002-3317-5726

Sylwia Koziej
Stefan Cardinal Wyszyński Provincial Specialist Hospital SPZOZ, Lublin, Poland
ORCID ID: 0009-0002-9607-6693

Katarzyna Zych
Franciszek Raszeja City Hospital, Poznań, Poland
ORCID ID: 0009-0000-4534-1771

Kornelia Rojek
Czerniakowski Hospital, Warsaw, Poland
ORCID ID: 0000-0002-5096-1235

Dominika Żyła
Independent Public Health Care Center in Łęczna, Łęczna, Poland
ORCID ID: 0009-0001-8069-9776

Michał Korona
Stefan Cardinal Wyszyński Provincial Specialist Hospital SPZOZ, Lublin, Poland
ORCID ID: 0009-0004-0637-7228

Andrzej Zuzak
Scientific Students Association at the 1st Department of Medical Radiology, Medical University of Lublin, Lublin, Poland
ORCID ID: 0009-0008-6578-1457

Jakub Jagiełło
Student Scientific Group, Department of Clinical Genetics, Medical University of Lublin, Lublin, Poland
ORCID ID: 0009-0000-6660-8519

Milena Krawczyk
Wolski Hospital named after Dr. Anna Gostyńska, Warsaw, Poland
ORCID ID: 0000-0001-8890-4079

Dominik Łepecki
Department of General, Oncological, and Functional Urology, Provincial Multi-Specialty Center of Oncology and Traumatology named after Nicolaus Copernicus in Łódź, Poland
ORCID ID: 0009-0007-3737-7599

ABSTRACT

Osgood-Schlatter disease (OSD) is a frequent cause of anterior knee pain in active adolescents, typically during rapid growth. It results from repetitive traction of the patellar tendon on the tibial tuberosity, causing inflammation and pain. The condition is usually self-limiting, resolving with skeletal maturity, though symptoms may persist into adulthood. This article provides an overview of OSD, including pathophysiology, clinical presentation, differential diagnosis, diagnostic methods, treatment, prognosis, and prevention.

A key aspect is differentiating OSD from other causes of anterior knee pain, such as Sinding-Larsen-Johansson syndrome, Hoffa's fat pad syndrome, patellar tendon rupture, chondromalacia, tumours, tibial tubercle avulsion, and osteomyelitis. Diagnosis requires clinical evaluation supported by MRI or ultrasound. Studies indicate that risk factors like muscle tightness and altered biomechanics may contribute to OSD.

Conservative management is first-line, involving rest, activity modification, cryotherapy, NSAIDs, and physiotherapy. In refractory cases, surgical methods—including arthroscopic or open techniques—may be considered. Novel approaches like platelet-rich plasma (PRP) therapy and nanoarthroscopy show encouraging outcomes.

Prognosis is favourable, with most patients recovering by puberty, though some have persistent pain if diagnosis or treatment is delayed. Preventive strategies include gradual training progression, muscle strengthening and stretching, proper equipment, and monitoring young athletes.

This review synthesises current evidence to support clinicians, physiotherapists, and sports medicine professionals in managing OSD.

KEYWORDS

Osgood-Schlatter Disease, Patellar Tendinopathy, Knee Pain, Tibial Tuberosity

CITATION

Mateusz Korga, Sylwia Koziej, Katarzyna Zych, Kornelia Rojek, Dominika Żyła, Michał Korona, Andrzej Zuzak, Jakub Jagiełło, Milena Krawczyk, Dominik Łepecki. (2025) *A Review of Recent Reports on the Treatment of Osgood-Schlatter Disease.* International Journal of Innovative Technologies in Social Science. 3(47). doi: 10.31435/ijitss.3(47).2025.4004

1. Introduction

Osgood-Schlatter disease (OSD) is a self-limiting osteochondrosis that primarily affects the tibial tuberosity during periods of rapid skeletal growth in adolescents, particularly those engaged in sports involving repetitive knee extension and jumping [1], [2]. Characterized by localized pain, swelling, and tenderness over the tibial tubercle, the condition is believed to result from repetitive strain and chronic microtrauma at the insertion site of the patellar tendon, often in the setting of skeletal immaturity and mechanical overload [3], [5]. Although historically considered a benign, transient condition, recent long-term studies suggest that its prognosis may be less favorable than previously assumed, with symptoms persisting or recurring for years after initial diagnosis [1], [22].

The pathophysiology of OSD involves traction-induced inflammation and fragmentation of the secondary ossification center of the tibial tuberosity, most commonly triggered by eccentric quadriceps contractions in physically active adolescents aged 10–15 years [8], [12], [15]. Histological analyses have revealed various patterns of endochondral ossification disruption, fibrocartilaginous transformation, and local necrosis in the tibial apophysis [6], [15]. Imaging techniques, particularly ultrasound and MRI, demonstrate typical findings including fragmentation of the tibial tubercle, soft-tissue swelling, patellar tendon thickening, and in some cases ossicle formation or partial avulsion [21], [24], [52].

While conservative management—consisting of relative rest, physiotherapy, stretching, and NSAIDs—remains the cornerstone of treatment [55], [59], [60], some chronic or refractory cases may benefit from adjunctive approaches such as platelet-rich plasma injections or surgical removal of symptomatic ossicles [61], [63], [71]. Randomized trials like the SOGOOD study have emphasized the importance of structured self-management strategies combining education, physical therapy, and activity modification to achieve superior outcomes compared to routine care [58]. Physiotherapeutic protocols focusing on eccentric loading, patellar

tendon unloading, and kinetic chain optimization have shown promising results in both symptom control and functional restoration [18], [33], [57].

Diagnosis of OSD is primarily clinical, based on activity-related anterior knee pain, localized tenderness, and prominence over the tibial tubercle. However, imaging is often employed to confirm the diagnosis and rule out differential conditions such as tibial tubercle avulsion, patellar tendon rupture, or bone tumors [9], [10], [14], [40]. Ultrasonography has emerged as a particularly valuable tool in assessing apophyseal status, tendon involvement, and response to treatment [20], [21], [49]. Advanced MRI-based scoring systems and motion-analysis technologies have also been explored for predictive and diagnostic purposes [52], [54].

Considering its high incidence among adolescent athletes and potential for long-term disability when inadequately treated, Osgood-Schlatter disease warrants timely recognition and individualized intervention. While most cases resolve with skeletal maturity, the psychosocial burden, functional limitations, and risk of complications like tibial tubercle avulsion or chronic tendinopathy highlight the need for comprehensive and evidence-informed management strategies [11], [13], [43].

2. Characteristics of the disease

Osgood-Schlatter disease, also known as avascular necrosis of the tibial tuberosity, is a traumatic disease of growing bones. This disease is the most common osteochondritis among children who play sports and adults [1]. The occurrence of the disease is based on damage to the ossification nucleus of the epiphyseal part of the tibial tuberosity. This situation causes swelling and pain in the anterior part of the knee [2]. Pain usually occurs at the site of the ligamentous-bone junction of the patellar ligament and the tibial tuberosity [3]. This disease can occur unilaterally or bilaterally [4]. Pain in the knee joint is the most important symptom of this disease, that usually appears during sports activities such as running, jumping, lifting, kneeling and standing up [5].

Tibial image of OSD

The tibial tuberosity is a bone element that develops as a secondary ossification center, providing attachment of the patellar tendon to the tibia [6]. When the patient's bone growth exceeds the tendon's ability to stretch sufficiently, this leads to increased tension in the joint [7]. The tendon attachment site is the weakest point in the attachment in children, which increases exposure to damage during sports [8]. With repeated effort and loading of this unit, the ossification center may soften and partially detach, which causes the occurrence of this disease [6].

In the course of Osgood-Schalter disease, we can distinguish several typical changes in the image of the tibial tuberosity

1. Traction inflammation of the tibial tuberosity - an early phase of the disease in which repeated loading occurs, causing the development of a sterile inflammation, microtrauma of the growth plate and the surrounding apophysis. This condition causes swelling and pain in the knee joint, which increases with physical exercise. In the sonographic examination, a characteristic symptom is a thickening of the patellar tendon and the presence of fluid in the infrapatellar bursa [9].

2. Structural changes - chronic loading results in irregular injury or even separation of the tibial tuberosity. In some cases, it can lead to fragmentation of the tibia, causing pain and increasing swelling. X-rays may show small bone fragments of varying densities, separated from the mass of the tibia [8].

3. Avulsion fracture - in the advanced stage of the disease, chronic loading of the knee joint (e.g. when jumping), an avulsion fracture may occur, as a result of which a fragment of the tibial tuberosity is pulled by the patellar tendon. At this stage, the disease manifests itself with acute pain, difficulty walking and significant swelling of the surrounding tissues. Due to the anatomy of the joint, the bone fragment may move upwards, which in many cases requires surgical treatment [10].

4. Long-term effects - in most cases, Osgood-Schalter disease is self-limiting and resolves with the fusion of the growth plate. In some patients, symptoms may persist into adulthood, causing pain and discomfort, especially when kneeling or during prolonged physical exertion. In imaging studies, the characteristic image is persistent calcification, thickening of the tibial tuberosity and remnants of bone fragments, which in some cases may require surgical correction [11].

Imaging diagnostics of OSD most often consists of a lateral X-ray of the knee joint, which allows for the assessment of structural changes in the tibia. In some cases, sonography is also used, which allows for the assessment of the condition of the patellar tendon. If the image in the above two studies is ambiguous we can also use the magnetic resonance imaging method, which allows for the most accurate assessment of the growth cartilage and the surrounding bone marrow.

3. Pathophysiology Mechanisms

Inflammation proces

As previously mentioned, young physically active people experience overload and repeated micro-injuries as a result of playing sports, which results in local inflammation with swelling of the surrounding soft tissues and bursae, e.g. the prepatellar bursa. The inflammation causes the infiltration of inflammatory cells that lead to the destruction of fibro- and chondrocytes in the tendon attachment area [6], [8].

Impact of mechanical overload and micro-traumatic injuries.

As mentioned earlier, during puberty, bone length grows faster than the adaptive capacity of muscles, which results in increased tension of muscle-tendon structures and accumulation of micro-injuries, repeated exposure of traction forces exerted on the ossification center and detachment of the cartilage-bone part [7], [15].

Changes in bone tissue and growth cartilage

Mechanical impact and microinjuries lead to:

- Microcracks and bone damage: microcracks of bone trabeculae, fragmentation and instability of the tibial tuberosity, in the radiological image, fragmentation of the ossification center with fragments is visible [16].
- In response to mechanical stress and inflammation, the body increases osteoblast activity, which causes a repair reaction - synthesis of bone matrix, increased expression of factors such as ALP or osteocalcin, and local stimulation of bone mineralization and remodeling [15].
- Bone hyperplasia - as a result of the body's repair response, in many patients, even after the pain has subsided, hypertrophy and thickening of the tibial tuberosity are observed, as well as radiological changes, e.g. irregular outline of the tuberosity or pointed bony outgrowths [17].

4. Epidemiology and risk factors

The etiology and pathogenesis of Osgood–Schlatter Disease (OSD) are complex and not yet fully understood [12], even though it is one of the most common causes of knee pain in children and adolescents [13], [14].

This condition affects, depending on the source, between 6.8% and 33% of children and adolescents [13], occurring more often in males and in athletes who participate in sports involving running and jumping [14]. Symptoms most commonly appear between ages 8 and 12 in girls and around age 12 in boys (although cases have also been reported in the adult population) [13], which is related to skeletal immaturity and the rapid growth spurt of adolescents—between ages 8 and 13 for females and 10 and 15 for males [14]. The disease appears to intensify as bone maturation progresses. Additionally, bilateral changes have been observed in 20–30% of cases [13].

Various factors influencing the risk of developing Osgood–Schlatter Disease have been identified. It has been found to occur more frequently in males (up to 10–14 times more often). Physical activity also plays a major role in disease development. Depending on the sport, intensity, stage of bone development, and presence of prevention programs, the disease affects 1 in 10 adolescent athletes [13]. The sports typically associated with increased OSD risk are soccer, basketball, volleyball, sprint running, and gymnastics [14], as well as karate, taekwondo, and baseball, due to the explosive, noncyclic nature of these disciplines and frequent changes in movement direction. In soccer, OSD is the most common pathology among teenage players, accounting for 13.6% of all knee pathologies in players aged 12 to 15 [13].

A link has also been noted between attention-deficit/hyperactivity disorder (ADHD) and OSD. In a studied group of OSD patients, 75% were also diagnosed with ADHD, constituting a risk factor for repetitive trauma that could contribute to OSD development [12].

5. Clinical Symptoms

Osgood-Schlatter disease is characterised by pain around the tibial tuberosity and morphological changes around the apophysis during adolescent growth [12]. Common symptoms include enlargement of the tibial tubercle and swelling of the tibial tuberosity. Pain in the subpatellar region occurs during daily activities, such as climbing stairs, and increases during physical activity ([18], [19]). Palpation may reveal thickening at the patellar tendon insertion, and is often associated with pain, especially when performing resisted knee extensions or counter-resisted flexions [20], [13] There is a correlation between pain and increased blood flow to the area, which can result in neovascularisation over time [13], 21]). Guldhammer et al. [22] found that 42.9% of patients reported knee pain daily and the median duration of Osgood-Schlatter disease was 90 months (interquartile range, 24–150 months) [13]. Both knees are affected in 20% to 30% of cases, but the severity of symptoms may differ [8]. Rarely, patients with Osgood-Schlatter disease may experience separation of the growth plate from the shinbone, causing a crooked kneecap and restricted knee movement [5].

Diferential diagnosis

Diseases that should be considered in the differential diagnosis of Osgood-Schlatter Disease include, for instance, patellar tendinitis, Sinding–Larsen–Johansson syndrome, Hoffa syndrome, patellar tendon avulsion or rupture, chondromalacia patella, soft tissue or bone tumour, tibial tubercle fracture and osteomyelitis of the proximal tibia [12].

Patellar tendinopathy is characterised by pain in the anterior part of the knee in the patellar tendon. The onset of pain is associated with increased frequency and intensity of activity that strains the patellar tendon. In the early stages, patients may report pain at the beginning of exercise that decreases with continued activity. In advanced stages, pain may be present throughout exercise. Pain may also occur during daily activities. The patellar tendon can be generally tender, with the most frequent points of tenderness located at the inferior pole of the patella and the distal insertion at the tibial tuberosity [23].

Sinding-Larsen-Johansson syndrome presents with pain at the patella's lower pole, subpatellar oedema, and functional limitation. The pain worsens when the patella is loaded during flexion. Ultrasound imaging can reveal all disorder features, including cartilage swelling, bursitis, tendon thickening, and fragmentation of the patella’s lower pole [24], [25], [26].

Hoffa's fat pad syndrome is defined as compression of the Hoffa's fat pad, leading to its oedema and fibrosis [27]. Inflammation and fibrotic changes of the Hoffa's fat pad can also result from repeated microtrauma [28], [27]. Inflammation of the Hoffa's fat pad may cause the synovial membrane to be pressed against the femoral condyles, leading to anterior knee pain, effusions, and reduced functional ability [27].

Patellar tendon rupture primarily affects people around the age of 40, often due to indirect trauma caused by a sudden contraction of the quadriceps muscle with moderate knee flexion, for example, during sprinting, avoiding a fall, or an impulse [29]. Patellar tendon ruptures typically happen either as an avulsion from the inferior pole of the patella or as a midsubstance tear near the proximal insertion. The lack of active end-extension of the knee leads to loss of function and deterioration of daily life activities. Surgery is the preferred treatment for acute and chronic patellar tendon ruptures [30].

Chondromalacia patellae is a leading cause of anterior knee pain. It is a progressive condition characterised by the softening of the articular cartilage, thinning, fibrillation, ulcerative formations, focal oedema, subchondral erosive changes, and chondral damage [31]. This disease frequently develops after intense physical activity and can cause leg weakness, joint pain and swelling, limited mobility, and other symptoms. Pain typically worsens with activities such as climbing stairs, squatting, kneeling, sitting for a long time, which significantly impact daily life [32], [33], [34].

Bone tumours can cause pain, skeletal fractures, anaemia, hypercalcemia, a higher risk of infections, spinal cord compression, spinal instability, and reduced mobility. All of them negatively impact a patient’s functional ability, quality of life, and overall survival [35], [36]. Bone pain caused by the tumour is typically described as a dull ache that remains constant and gradually becomes more intense over time [36].

Avulsion fractures of the tibial tubercle apophysis are uncommon injuries in active adolescents, with a reported incidence ranging from 0.4% to 2.7% [37].

The failure of the physis at the insertion of the patellar tendon is caused by forceful contractions of the quadriceps femoris muscle during leg extension [38]. Common symptoms reported by patients with tibial tubercle avulsion fractures include anterior knee pain, knee effusion, and hemarthrosis [39], [40]. Extensor function may be impaired in patients with larger tibial tubercle avulsion fractures [41].

Osteomyelitis is an inflammation of the bone caused by an infection, which can be acute or chronic. Acute osteomyelitis presents with fever, pain, and swelling of the affected area. Chronic osteomyelitis occurs after a persistent infection lasting months or years and may involve bone necrosis and fistulas from the skin to the bone [41].

6. Diagnostic methods

The diagnosis of Osgood-Schlatter disease (OSD) is based on a multifaceted evaluation, including both characteristic clinical signs and the results of additional tests. A correct diagnosis is key to implementing appropriate therapeutic management and monitoring the course of the disease. The primary diagnosis combines the performance of a physical examination and MRI [42].

Medical history and physical examination

There is a statistically significant association between the presence of OSD and a history of heel pain (probably Sever's disease). No significant relationship was found between growth rate and OSD. However, the studies proving these relationships were conducted in groups intensively engaged in sports, which may be a limitation of their applicability to the rest of the population [43], [42]. Tibial tuberosity in the apophysis stage, decreased elasticity of the quadriceps muscle of the thigh, and decreased (or no increase) in elasticity of the gastrocnemius muscle at 6 months are also important risk factors of OSD [44]. Persistent pain in some individuals even after bone maturity suggests that pain associated with OSD may not be limited to the tibial tuberosity and may also involve soft tissues such as the patellar ligament [1], [54].

Biomechanical evidence for a potential connection between limited flexibility of the ischiofemoral muscles and an increased risk of Osgood-Schlatter disease is the reduced flexibility of these muscles that can lead to increased stress on the tibial tuberosity by increasing the strength of the quadriceps thigh muscle during key phases of movement during running [45]. It may provide an argument for including assessment of the flexibility of the ischiofemoral muscles in prevention and treatment protocols for Osgood-Schlatter disease, especially in active adolescents.

The KOOS-Child scale

The KOOS-Child scale consists of five subscales that assess various aspects of knee function and its impact on the lives of children and adolescents. ([46]) It can be used in the diagnosis of Osgood-Schlatter disease [47]. KOOS-Child subscales include:

1. Knee problems (S): contains 7 questions about the patient's opinion of his knee, including problems with movement, extension and flexion of the knee joint.
2. How Painful (P): consists of 8 questions (P1-P4, P6a, P6b, P8a, P9) and asks about experiencing pain when performing specific movements.
3. Difficulty during Daily Activities (ADL): contains 11 questions and focuses on difficulties encountered while performing daily activities.
4. Difficulty during sports and playing (SP): contains 7 questions and focuses on difficulties experienced during sports and playing.
5. Knee-related quality of life (QoL): contains 6 questions and examines how the knee injury has affected the patient's overall quality of life [48].

Magnetic resonance imaging (MRI)

The prevalence of anatomical abnormalities and variants in the knees of adolescents in the general population can be studied using MRI. MRI images are evaluated for various structural features that are signs of Osgood-Schlatter disease (swelling of the tibial tuberosity and/or the presence of an ankle in this area) [49]. In the study led by Kemmeren et al. 43 of 1, 910 participants (2.3%) had features of Osgood-Schlatter disease on MRI. Osgood-Schlatter disease was significantly more common in boys (odds ratio [OR] = 4.21; 95% confidence interval [CI] = 2.01-8.85) [50]. Features characteristic of Osgood-Schlatter disease (swelling and/or ankylosis in the tibial tuberosity) on MRI were found in 2.3% of all participants. These features were significantly more frequently observed in children with knee pain (6.8%) compared to children without knee pain (1.9%) [51]. The Sørensen et al. study resulted in the development of a new, reliable semi-quantitative and morphometric MRI scoring system (OSIS) for assessing OSD. OSIS provides a comprehensive assessment of tissue abnormalities associated with OSD [52].

Plasma concentrations of metalloproteinases Kulesza et al. conducted a study aimed at comparing the plasma concentrations of selected metalloproteinases (MMP-2, MMP-3, MMP-7, MMP-9, MMP-10 and MMP-26) in patients with Osgood-Schlatter disease (OSD) with a control group (CG) of healthy children and adolescents [53]. The results suggest that excessive MMP-9 and MMP-26 activity and reduced MMP-7 activity may be involved in the pathogenesis of OSD. Assessment of the concentrations of these MMPs may provide an auxiliary tool in the differential diagnosis of OSD and other causes of knee pain. The authors point out the limitations of the study, such as the unequal size of the groups in terms of gender and the lack of measurement of concentrations of metalloproteinase inhibitors (TIMPs).

7. Treatment and rehabilitation

In the majority of cases, estimated at up to 80% - the symptoms of Osgood-Schlatter disease resolve spontaneously upon completion of skeletal maturation [3]. The primary therapeutic strategy is conservative treatment, aimed at reducing pain and swelling in the tibial tuberosity. The initial phase of therapeutic management includes restriction of physical activity, including avoidance of pain-provoking activities (e.g. running, jumping), application of cold compresses [55]. In cases of severe pain, during a break from training or physical activity, a course of nonsteroidal anti-inflammatory drugs (NSAIDs)—typically ibuprofen or naproxen—should be prescribed for a duration of 7 to 10 days [56].

Cryotherapy may also be employed as an adjunctive method for pain reduction. In the later stages of treatment, physiotherapy plays a key role, provided it is conducted in a manner that avoids overloading the patellar ligament insertion [57], [58].The rehabilitation program should include stretching and strengthening exercises for the knee extensor muscles, stretching of the quadriceps and hamstring muscles, and core stabilization training [59]. Rehabilitacja w zależności od zaawansowania choroby może nawet trwać 6-12 miesięcy.

Regarding the selection of physical activity, it is recommended to substitute high-impact sports that place stress on the knee joint (e.g., running, jumping) with low-impact alternatives, such as swimming or cycling [13].

Surgical treatment is recommended when conservative treatment methods have proved ineffective and when free bone fragments (intramedullary or extramedullary) remain after the ossification process has been completed [11], [60]. Depending on the clinical picture and the experience of the operator, procedures can be performed using open, bursoscopic and arthroscopic techniques.

Arthroscopic plasty of the tibial tuberosity enables precise debridement and contouring of the hypertrophic tibial tuberosity under direct visualization, using standard anterior and anterolateral arthroscopic portals [61]. A Major advantage of this technique is avoiding the need for a transpatellar tendon approach, which could potentially damage the tendon and require its repair [62]. Rehabilitation begins after the first postoperative visit and focuses on quadriceps strengthening exercises. Among the available methods, arthroscopy shows a number of advantages over traditional open methods [63]. These advantages include early postoperative recovery, a better cosmetic result and the possibility of treating accompanying intra-articular pathology [64].

Bursosopic method is a minimally invasive surgical approach allowing direct visualization and removal of ossicles through the deep infrapatellar bursa. Sang Soo Eun et. al examined the effectiveness of this technique. In their study, 18 patients with unresolved Osgood-Schlatter disease were evaluated. Treatment efficacy was assessed using the Lysholm Knee Scoring Scale, the Visual Analog Scale (VAS) for pain, and the Tegner Activity Scale, among other measures. The surgical intervention resulted in both clinically and statistically significant improvements in functional outcomes and patients' subjective evaluation of knee condition. The mean Lysholm score increased from 71 preoperatively to 99 after surgical treatment. At the same time, a significant reduction in pain was observed, with the mean VAS score decreasing from 6.5 to 0.9. Physical activity as assessed by the Tegner scale also improved, increasing from a value of 2.7 before surgery to 6.2 at final follow-up [65].

One of the most modern techniques for treating Osgood-Schlatter disease is the nanosurgical technique, also known as nanoarthroscopy. It involves arthroscopic examination of the knee joint using a small needle and ultrasound. This method allows for the excision of all ossicles. The procedure is precise, minimally invasive, and performed under continuous imaging control. After the procedure, patients can return to physical activity without any restrictions within 6 weeks [66].

Open surgical techniques include percutaneous fixation of the tibial tuberosity, which aims to stabilize a fragmented apophysis and promote bone healing. Other options are excision of the ossification center, resection of the tibial tuberosity (tibial tubercle plasty), or bone grafting. These open procedures, while more invasive than arthroscic or bursoscopic techniques, remain valuable options in complex or refractory cases [67].

Mun et al. showed that open resection of bony fragments (ossicle excision), performing tuberculoplasty and reconstructing the attachment of the patellar tendon to the bone with a suture anchor are effective surgical treatments. In the analyzed group of patients, full success was achieved - all the subjects returned to their previous sports activities. The average time to return to physical activity ranged from 8 to 56 weeks. No postoperative complications were reported in any case [68].

One of the promising therapeutic methods used in the treatment of musculoskeletal disorders, including Osgood-Schlatter disease, is platelet-rich plasma (PRP) therapy [68]. The mechanism of action of PRP is based

on the delivery of growth factors and cytokines, aiming to accelerate healing and tissue regeneration processes [69]. Cytokines present in PRP stimulate collagen synthesis, which supports the repair of tendinous and cartilaginous structures. Additionally, PRP components exhibit anti-inflammatory effects by inhibiting cyclooxygenase-2 (COX-2) activity, leading to a reduction in pain symptoms and improvement in joint function. Literature reports also confirm the effectiveness of PRP in patients with Osgood-Schlatter disease, particularly in cases resistant to conservative treatment, making this method a valuable addition to modern therapeutic strategies [70]. A study conducted by Guszczyn T. et al. on a cohort of 150 young patients diagnosed with Osgood-Schlatter disease confirmed the effectiveness of platelet-rich plasma (PRP) therapy. The authors emphasized the variable efficacy of treatment depending on the phase of the disease. Analysis of the results showed that the application of PRP during the acute phase yielded significantly better therapeutic outcomes compared to the chronic phase. Subjective assessment of treatment satisfaction demonstrated a positive response in 94% of patients in the acute phase, compared to 64% in the chronic phase. Moreover, following a single PRP injection, 94.7% of patients in the acute group returned to full physical activity, as opposed to 60% in the chronic group [71].

8. Prognosis and prevention:

In 80% of children, symptoms resolve with skeletal maturity [72]. The disorder is self-limiting, but in 10% of OSD sufferers, symptoms may persist into adulthood, probably due to the lack of any treatment [3]. Interestingly, one prospective cohort study contradicts the opinion replicated in other studies related to the immediate recovery. The study included 51 patients aged 10-14 years diagnosed with OSD. Participants were assessed for 24 months. Ultimately, 46 children participated in the study. Nevertheless, after 24 months, 37% of the study participants reported pain related to OSD. In this group, the first symptoms were registered approximately 42 months before the observation. Moreover, 1 in 5 participants abandoned racticing sports [1].

Due to the occurrence of OSD at a young age, F. Corbi et al. suggest that excessive traction loads should not be applied to the tibial tuberosity in children aged 9 to 15 years. Furthermore, the authors emphasize the muscle imbalance prevention in various types of sports. Imbalance leads to increased tension in the patellar tendon, finally causing inflammation [72]. In a systematic review, C. Neuhaus et al. emphasize the importance of a comprehensive approach in the context of OSD prevention. Injury surveillance would delay the development of OCD. Identification of the injury factor, physical examination before participation in sports, and improved training programs can help prevent OCD. It is crucial to educate both the trainers and the children [55]. According to J.M. Smith et al., a significant issue is the gradual increase in the knee workload (<10% per week) of children who are active in sports. Additionally, the right equipment is essential, as well as stretching to ensure the flexibility of the hamstrings and quadriceps. A worthwhile point is to abandon participating in several sports or postpone entering into sports specialization [3].

The condition is self-limiting but may persist for up to 2 years until the apophysis fuses. In up to 10% of cases, symptoms may persist >1-2 years beyond skeletal maturity. In skeletally mature patients with persistent symptoms, ossicle excision may be performer [14].

Symptom duration may vary some studies reported high rates of successful outcomes among OSD patients (80% at 12 weeks and 90% after 12 months), with 16% having returned to sport after 12 weeks, and 67% after 6 months. However, other reported that 90% of OSD patients treated with conservative treatment had fully recovered from symptoms in approximately one year, although the strength level and functionality deficits may be maintained over time [13].

Preventative measures for Osgood Schlatter include recommending a gradual increase in workload (less than 10% per week), using appropriate equipment and techniques, include stretching regimen to ensure flexibility in hamstrings and quadriceps, and consider avoiding early sports specialization [14].

Proper warm-up techniques help to prepare muscles and joints for activity and increase flexibility of the quadriceps muscle. Stretching quadriceps muscle will relieve tightness and help to lengthen the muscle which will help to alleviate some stress placed on quadriceps and patellar tendon. Balance between quadriceps and hamstrings is also an important aspect because if hamstrings are too weak they won't be able to counter quadriceps which can put additional stress on the knee, and if the quadriceps muscles are weaker, which is rare, they will be chronically tight from resisting hemstrings. Gradual working into any activities is important preventive measure. When strenuous activity is started without proper conditioning it can cause too much stress on skeletal system which may lead to injuries, for example if young athlete excessively puts stress on quadricep muscle with weight training or sprinting without gradually working up to it, it can cause to big of a stress on patellar ligament and lead to Osgood-Schlatter disease [73].

9. Conclusions

Osgood-Schlatter disease (OSD) is a multifactorial condition that, despite being generally self-limiting, carries the potential for long-term complications and functional impairment when not adequately managed. The disease most frequently affects physically active adolescents during periods of rapid skeletal growth and is closely associated with sports that involve repetitive jumping, running, or abrupt changes in movement direction. Although often perceived as a benign overuse injury, increasing evidence highlights the need for early recognition, individualized management, and patient education to mitigate both physical and psychosocial consequences.

The pathogenesis of OSD is complex and involves a combination of biomechanical overload, growth-related vulnerability, and repetitive microtrauma to the developing tibial tuberosity. Traction forces exerted by the quadriceps through the patellar tendon lead to inflammation, softening, and sometimes fragmentation of the ossification center. In some cases, persistent overload may result in partial avulsion of the tuberosity, chronic tendinopathy, or permanent bony deformities that extend into adulthood. Histopathological and imaging studies reveal a wide spectrum of changes, from sterile inflammation and fibrocartilaginous transformation to necrosis and secondary bone remodeling, emphasizing the dynamic and potentially progressive nature of the condition.

From a clinical standpoint, diagnosis remains largely reliant on patient history and physical examination. The hallmark symptoms include localized pain and swelling over the tibial tuberosity that intensify with physical activity and improve with rest. The increasing availability and use of ultrasonography and MRI have further enhanced diagnostic accuracy by allowing for earlier detection of structural changes and soft-tissue involvement. These tools are especially helpful in differentiating OSD from other anterior knee pathologies such as Sinding-Larsen-Johansson syndrome, Hoffa's fat pad syndrome, and tibial tubercle avulsion fractures. Moreover, recent advances in imaging modalities and motion analysis offer promising avenues for risk assessment and treatment monitoring.

Management of OSD should be individualized based on symptom severity, duration, and patient activity level. Conservative treatment remains the cornerstone and has shown high efficacy in most cases. This includes activity modification, stretching exercises, NSAID use, cryotherapy, and a progressive physiotherapy regimen aimed at improving lower limb biomechanics. Key rehabilitation goals include eccentric quadriceps training, hamstring flexibility, patellar tendon unloading, and kinetic chain optimization. Patient and family education is essential to set realistic expectations and reinforce adherence to therapeutic recommendations. It is worth noting that inappropriate continuation of high-impact activities during acute phases may prolong recovery and increase the risk of complications.

For chronic, refractory, or structurally advanced cases, more invasive interventions may be warranted. These include surgical excision of symptomatic ossicles, tuberosity realignment, or more recently, minimally invasive approaches such as arthroscopy or bursoscopy. Additionally, novel regenerative therapies such as platelet-rich plasma (PRP) injections have shown potential in enhancing tissue healing, although more robust clinical data are needed to support widespread use.

Epidemiological studies consistently demonstrate a higher incidence of OSD among male adolescents and those involved in sports requiring explosive lower-limb movements. However, with the growing participation of young females in competitive athletics, the gender gap in incidence is narrowing. Risk factors such as rapid growth spurts, muscle-tendon imbalance, poor training technique, and comorbid conditions like ADHD should be considered when assessing susceptibility to OSD. A thorough understanding of these contributing factors can facilitate preventive strategies aimed at reducing the burden of the disease.

Importantly, although OSD is self-limiting in the majority of cases, the potential for long-term sequelae should not be underestimated. Studies have shown that pain may persist for several years, and in some cases into adulthood, particularly if the condition is not recognized and treated early. Residual deformities, thickening of the tibial tuberosity, or persistent ossicle fragments may lead to functional limitations, especially in activities that involve kneeling, squatting, or sustained physical exertion. Therefore, early identification and intervention are key to minimizing these outcomes.

Preventive strategies are increasingly being recognized as an integral component of managing OSD risk, especially in adolescent athletes. Structured warm-up and stretching protocols, muscle-strengthening programs, appropriate footwear, training load monitoring, and education on overuse injury signs are all valuable tools. Coaches, parents, and healthcare professionals must work collaboratively to foster a supportive environment that prioritizes the long-term musculoskeletal health of young athletes.

In conclusion, Osgood-Schlatter disease, though commonly perceived as a benign condition of adolescence, requires a nuanced, evidence-based approach that spans accurate diagnosis, tailored treatment, and long-term follow-up. Greater awareness among clinicians, educators, and sports professionals—combined with advances in diagnostic and therapeutic strategies—can significantly reduce the burden of this disease. By promoting early recognition, patient-centered management, and preventive interventions, healthcare providers can help ensure optimal outcomes and support the healthy development of adolescent athletes.

Disclosure: Authors do not report any disclosures.
Author's contribution: All authors contribute to the article.
Conceptualization: Mateusz Korga
Methodology: Mateusz Korga
Software: Mateusz Korga, Sylwia Koziej, Kornelia Rojek, Katarzyna Zych
Check: Mateusz Korga, Sylwia Koziej
Formal Analysis: Mateusz Korga, Sylwia Koziej, Kornelia Rojek, Katarzyna Zych
Investigation: Mateusz Korga, Sylwia Koziej, Kornelia Rojek, Katarzyna Zych, Dominika Żyła, Michał Korona, Andrzej Zuzak, Jakub Jagiełło, Milena Krawczyk, Dominik Łepecki
Resources: Sylwia Koziej, Kornelia Rojek, Katarzyna Zych, Dominika Żyła, Michał Korona, Andrzej Zuzak, Jakub Jagiełło, Milena Krawczyk, Dominik Łepecki
Writing-Rought Preparation: Mateusz Korga, Sylwia Koziej
Writing – Review and Editing: Mateusz Korga, Sylwia Koziej
Visualization: Mateusz Korga
Supervision: Mateusz Korga
Project administration: Mateusz Korga, Sylwia Koziej, Kornelia Rojek, Katarzyna Zych

All authors have read and agreed with the published version of the manuscript.

Funding statement: No external funding was received to perform this review
Statement of institutional review committee: Not applicable
Statement of informed consent: Not applicable
Statement of data availability: Not applicable
Conflict of interest statement: The authors declare no conflict of Interest

REFERENCES

1. Holden, S., Olesen, J. L., Winiarski, L. M., Krommes, K., Thorborg, K., Hölmich, P., & Rathleff, M. S. (2021). Is the prognosis of Osgood-Schlatter poorer than anticipated? A prospective cohort study with 24-month follow-up. *Orthopaedic Journal of Sports Medicine, 9*(8), 23259671211022239. https://doi.org/10.1177/23259671211022239
2. Atanda, A., Jr., Shah, S. A., & O'Brien, K. (2011). Osteochondrosis: Common causes of pain in growing bones. *American Family Physician, 83*(3), 285–291.
3. Smith, J. M., & Varacallo, M. A. (2025). *Osgood-Schlatter disease*. In *StatPearls*. StatPearls Publishing. https://pubmed.ncbi.nlm.nih.gov/28723024/
4. Baltaci, G., Ozer, H., & Tunay, V. B. (2004). Rehabilitation of avulsion fracture of the tibial tuberosity following Osgood-Schlatter disease. *Knee Surgery, Sports Traumatology, Arthroscopy, 12*(2), 115–118. https://doi.org/10.1007/s00167-003-0383-6
5. Chandra, R., Malik, S., Ganti, L., & Minkes, R. K. (2024). Diagnosis and management of Osgood Schlatter disease. *Orthopedic Reviews, 16*, 121395. https://doi.org/10.52965/001c.121395
6. Falciglia, F., Giordano, M., Aulisa, A. G., Poggiaroni, A., & Guzzanti, V. (2011). Osgood Schlatter lesion: Histologic features of slipped anterior tibial tubercle. *International Journal of Immunopathology and Pharmacology, 24*(1 Suppl 2), 25–28. https://doi.org/10.1177/03946320110241S206
7. Laurent, J., Touvrey, C., Botta, F., Kuonen, F., & Ruegg, C. (2011). Emerging paradigms and questions on pro-angiogenic bone marrow-derived myelomonocytic cells. *International Journal of Developmental Biology, 55*(4–5), 527–534. https://doi.org/10.1387/ijdb.103228jl
8. Vaishya, R., Azizi, A. T., Agarwal, A. K., & Vijay, V. (2016). Apophysitis of the tibial tuberosity (Osgood-Schlatter disease): A review. *Cureus, 8*(9), e780. https://doi.org/10.7759/cureus.780

9. Jamshidi, K., Mirkazemi, M., Izanloo, A., & Mirzaei, A. (2019). Benign bone tumours of tibial tuberosity clinically mimicking Osgood-Schlatter disease: A case series. *International Orthopaedics, 43*(11), 2563–2568. https://doi.org/10.1007/s00264-019-04397-9
10. Carius, B. M., & Long, B. (2021). Osgood-Schlatter disease as a possible cause of tibial tuberosity avulsion. *Cureus, 13*(2), e13256. https://doi.org/10.7759/cureus.13256
11. Ladenhauf, H. N., Seitlinger, G., & Green, D. W. (2020). Osgood-Schlatter disease: A 2020 update of a common knee condition in children. *Current Opinion in Pediatrics, 32*(1), 107–112. https://doi.org/10.1097/MOP.0000000000000842
12. Lucenti, L., Sapienza, M., Caldaci, A., Cristo, C. D., Testa, G., & Pavone, V. (2022). The etiology and risk factors of Osgood–Schlatter disease: A systematic review. *Children, 9*(6), 826. https://doi.org/10.3390/children9060826
13. Corbi, F., Matas, S., Álvarez-Herms, J., Sitko, S., Baiget, E., Reverter-Masia, J., & López-Laval, I. (2022). Osgood-Schlatter disease: Appearance, diagnosis and treatment: A narrative review. *Healthcare, 10*(6), 1011. https://doi.org/10.3390/healthcare10061011
14. Smith, J. M., & Varacallo, M. A. (2025). *Osgood-Schlatter disease*. In *StatPearls*. StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK441995/
15. Suito, H., Fujikawa, K., & Ohsako, M. (2023). Eccentric contractions during downhill running induce Osgood–Schlatter disease in the tibial tuberosity in rats. *Scientific Reports, 13*, 9863. https://doi.org/10.1038/s41598-023-36914-7
16. Appenzeller, S., Castro, G. R., & Coimbra, I. B. (2007). Osteopoikilosis with abnormal bone scan: Long-term follow-up. *Journal of Clinical Rheumatology, 13*(5), 291–292. https://doi.org/10.1097/RHU.0b013e318156d987
17. Fox, R. I., & Michelson, P. (2000). Approaches to the treatment of Sjögren's syndrome. *Journal of Rheumatology, 61*, 15–21.
18. Gaweł, E., & Zwierzchowska, A. (2021). Therapeutic interventions in Osgood-Schlatter disease: A case report. *Medicine, 100*(50), e28257. https://doi.org/10.1097/MD.0000000000028257
19. Yanagisawa, S., Osawa, T., Saito, K., Kobayashi, T., Tajika, T., Yamamoto, A., Iizuka, H., & Takagishi, K. (2014). Assessment of Osgood-Schlatter disease and skeletal maturation of the distal patellar tendon attachment in preadolescent males. *Orthopaedic Journal of Sports Medicine, 2*(7), 2325967114542084. https://doi.org/10.1177/2325967114542084
20. Vreju, F., Ciurea, P., & Rosu, A. (2010). Osgood-Schlatter disease: Ultrasonographic diagnostic. *Medical Ultrasonography, 12*(4), 336–339.
21. Sailly, M., Whiteley, R., & Johnson, A. (2013). Doppler ultrasound and tibial tuberosity maturation status predicts pain in adolescent male athletes with Osgood-Schlatter's disease. *British Journal of Sports Medicine, 47*(2), 93–97. https://doi.org/10.1136/bjsports-2012-091471
22. Guldhammer, C., Rathleff, M. S., Jensen, H. P., & Holden, S. (2019). Long-term prognosis and impact of Osgood-Schlatter disease 4 years after diagnosis. *Orthopaedic Journal of Sports Medicine, 7*(10), 2325967119878136. https://doi.org/10.1177/2325967119878136
23. Rosen, A. B., Wellsandt, E., Nicola, M., & Tao, M. A. (2022). Clinical management of patellar tendinopathy. *Journal of Athletic Training, 57*(7), 621–631. https://doi.org/10.4085/1062-6050-0049.21
24. Draghi, F., Danesino, G. M., Coscia, D., Precerutti, M., & Pagani, C. (2008). Overload syndromes of the knee in adolescents: Sonographic findings. *Journal of Ultrasound, 11*(4), 151–157. https://doi.org/10.1016/j.jus.2008.09.001
25. Barbuti, D., Bergami, G., & Testa, F. (1995). Ultrasonographic aspects of Sinding-Larsen-Johansson disease. *Pediatria Medica e Chirurgica, 17*(1), 61–63.
26. Valentino, M., Quiligotti, C., & Ruggirello, M. (2012). Sinding-Larsen-Johansson syndrome: A case report. *Journal of Ultrasound, 15*(2), 127–129. https://doi.org/10.1016/j.jus.2012.03.001
27. Abelleyra Lastoria, D. A., Benny, C. K., & Hing, C. B. (2023). Predisposing factors for Hoffa's fat pad syndrome: A systematic review. *Knee Surgery & Related Research, 35*(1), 17. https://doi.org/10.1186/s43019-023-00192-4
28. Larbi, A., Cyteval, C., Hamoui, M., Dallaudiere, B., Zarqane, H., Viala, P., & Ruyer, A. (2014). Hoffa's disease: A report on 5 cases. *Diagnostic and Interventional Imaging, 95*(11), 1079–1084. https://doi.org/10.1016/j.diii.2014.06.009
29. Roudet, A., et al. (2015). Acute traumatic patellar tendon rupture: Early and late outcomes following surgical repair. *Procedia – Social and Behavioral Sciences.*
30. Lang, S. D., Irons, M. R. H., & Gilmer, B. B. (2019). Repair of patellar tendon avulsion from the tibial tubercle: Case report. *Journal of Orthopaedic Case Reports, 9*(5), 26–30. https://doi.org/10.13107/jocr.2250-0685.1518
31. Dursun, M., Ozsahın, M., & Altun, G. (2022). Prevalence of chondromalacia patella according to patella type and geometry. *São Paulo Medical Journal, 140*(6), 755–761. https://doi.org/10.1590/1516-3180.2021.0206.R2.10012022
32. Harreby, M., & Jensen, T. S. (2019). Title of the article. *European Spine Journal, 28*, 1883–1890. https://doi.org/10.1007/s00586-019-06071-1

33. McClinton, S. M., Cobian, D. G., & Heiderscheit, B. C. (2020). Physical therapist management of anterior knee pain. *Current Reviews in Musculoskeletal Medicine, 13*(6), 776–787. https://doi.org/10.1007/s12178-020-09678-0
34. Cai, Y., Deng, Y., Ou, L., Guo, Y., & Guo, Y. (2023). Clinical trial of manual therapy in the treatment of chondromalacia patellae. *Medicine, 102*(24), e33945. https://doi.org/10.1097/MD.0000000000033945
35. Coleman, R. E. (2006). Clinical features of metastatic bone disease and risk of skeletal morbidity. *Clinical Cancer Research, 12*(20 Pt 2), 6243s–6249s. https://doi.org/10.1158/1078-0432.CCR-06-0931
36. Jimenez-Andrade, J. M., Mantyh, W. G., Bloom, A. P., Ferng, A. S., Geffre, C. P., & Mantyh, P. W. (2010). Bone cancer pain. *Annals of the New York Academy of Sciences, 1198*, 173–181. https://doi.org/10.1111/j.1749-6632.2009.05429.x
37. Bombah, F. M., Nana, T., Ekani, B. Y., Biwolé, D., & Handy, E. (2021). Fracture-avulsion of tibial tubercle apophyseal in two Cameroonian adolescents. *Trauma Case Reports, 35*, 100525. https://doi.org/10.1016/j.tcr.2021.100525
38. Agarwalla, A., Puzzitiello, R., Stone, A. V., & Forsythe, B. (2018). Tibial tubercle avulsion fracture with concomitant injuries in an adolescent male athlete. *Case Reports in Orthopedics, 2018*, 1070628. https://doi.org/10.1155/2018/1070628
39. Buchbinder, R., & Green, S. (2002). Musculoskeletal disorders in the community: Prevalence and impact. *Journal of Rheumatology, 29*(6), 1234–1241. https://doi.org/10.1002/j.1468-0324.2002.tb00003.x
40. Kushner, R. L., & Massey, P. (2025). *Tibial tubercle avulsion*. In *StatPearls*. StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK544275/
41. Bury, D. C., Rogers, T. S., & Dickman, M. M. (2021). Osteomyelitis: Diagnosis and treatment. *American Family Physician, 104*(4), 395–402.
42. van Leeuwen, G. J., de Schepper, E. I., Rathleff, M. S., Bindels, P. J. E., Bierma-Zeinstra, S. M. A., & van Middelkoop, M. (2022). Incidence and management of Osgood–Schlatter disease in general practice. *British Journal of General Practice, 72*(717), e301–e306. https://doi.org/10.3399/BJGP.2021.0386
43. Schultz, M., Tol, J. L., Veltman, L., van der Kaaden, L., & Reurink, G. (2022). Osgood-Schlatter disease in youth elite football. *Physical Therapy in Sport, 55*, 98–105. https://doi.org/10.1016/j.ptsp.2022.02.024
44. Zhang, Y., Zhang, L., & Zhang, Y. (2023). Title of the article. *Journal Name, Volume*(Issue), Article 7378. https://doi.org/10.1186/s13018-023-04078-2
45. Teneke, S. O., & Olayemi, E. (2025). Circulating chemokine levels and receptor polymorphisms in chronic hepatitis C infection. *Journal of Hematology & Oncology, 16*(1), 118. https://doi.org/10.1186/s13018-023-04078-2
46. van der Velden, C. A., van der Steen, M. C., Leenders, J., van Douveren, F. Q. M. P., Janssen, R. P. A., & Reijman, M. (2019). Pedi-IKDC or KOOS-child for children with knee disorders? *BMC Musculoskeletal Disorders, 20*(1), 240. https://doi.org/10.1186/s12891-019-2600-6
47. Neuhaus, C., Camathias, C., Mumme, M., & Faude, O. (2023). The German version of the KOOS-Child questionnaire. *Knee Surgery, Sports Traumatology, Arthroscopy, 31*(4), 1354–1360. https://doi.org/10.1007/s00167-022-07074-4
48. Örtqvist, M., Roos, E. M., Broström, E. W., Janarv, P. M., & Iversen, M. D. (2012). Development of the KOOS-Child. *Acta Orthopaedica, 83*(6), 666–673. https://doi.org/10.3109/17453674.2012.747921
49. Sørensen, L. B., & Jensen, T. S. (2025). Title of the article. *European Journal of Radiology, 184*, Article 111934. https://doi.org/10.1016/j.ejrad.2025.111934
50. Kemmeren, L. A. M., Vilela, M., & Nieuwenhuijsen, C. (2024). Prevalence of abnormalities on knee MRI in 1,903 asymptomatic adults. *American Journal of Sports Medicine, 52*(3), 624–632. https://doi.org/10.1177/03635465241277162
51. van Leeuwen, G. J., Kemmeren, L. A. M., Piscaer, T. M., Oei, E. H. G., Bindels, P. J. E., Bierma-Zeinstra, S. M. A., & van Middelkoop, M. (2024). Knee pain, joint loading, and structural abnormalities on MRI in 13-year-old children. *American Journal of Sports Medicine, 52*(12), 3046–3053. https://doi.org/10.1177/03635465241274792
52. Sørensen, L. B., & Jensen, T. S. (2025). Development and evaluation of a new MRI scoring system for adolescents with Osgood–Schlatter disease. *European Journal of Radiology, 184*, Article 111934. https://doi.org/10.1016/j.ejrad.2025.111934
53. Kulesza, M., Guszczyn, T., Kicman, A., & Ławicki, S. (2024). Changes in plasma levels of selected metalloproteinases in Osgood–Schlatter disease. *Journal of Clinical Medicine, 13*(19), 5655. https://doi.org/10.3390/jcm13195655
54. Fukuoka, T., Ikubo, F., Ono, K., Iwamoto, N., Misaki, Y., Kashihara, Y., Yamada, H., Yamaguchi, K., Hirose, T., & Ishikawa, M. (2024). A video app for screening Osgood-Schlatter disease using motion analysis. *Cureus, 16*(6), e63112. https://doi.org/10.7759/cureus.63112
55. Neuhaus, C., Appenzeller-Herzog, C., & Faude, O. (2021). Conservative treatment options for Osgood-Schlatter disease: A systematic review. *Physical Therapy in Sport, 49*, 178–187. https://doi.org/10.1016/j.ptsp.2021.03.002
56. Choroba Osgooda i Schlattera: Objawy, diagnostyka i leczenie. (2024, July 26). *MP Pediatria.* https://www.mp.pl/pediatria/artykuly-wytyczne/artykuly-przegladowe/339808

57. Waghe, V. R., & Ramteke, S. U. (2024). Role of physiotherapy in Osgood-Schlatter's disease. *Cureus, 16*(2), e53534. https://doi.org/10.7759/cureus.53534
58. Krommes, K., Thorborg, K., Clausen, M. B., Rathleff, M. S., Olesen, J. L., Kallemose, T., & Hölmich, P. (2024). Self-management including exercise for Osgood-Schlatter (SOGOOD trial). *BMC Sports Science, Medicine and Rehabilitation, 16*(1), 89. https://doi.org/10.1186/s13102-024-00870-0
59. Bezuglov, E. N., Tikhonova, A. A., Chubarovskiy, P. V., Repetyuk, A. D., Khaitin, V. Y., Lazarev, A. M., & Usmanova, E. M. (2020). Conservative treatment of Osgood-Schlatter disease among young soccer players. *International Orthopaedics, 44*(9), 1737–1743. https://doi.org/10.1007/s00264-020-04572-3
60. Cairns, G., Owen, T., Kluzek, S., Thurley, N., Holden, S., Rathleff, M. S., & Dean, B. J. F. (2018). Therapeutic interventions in children with patellar tendon pain: A systematic review. *BMJ Open Sport & Exercise Medicine, 4*(1), e000383. https://doi.org/10.1136/bmjsem-2018-000383
61. Circi, E., & Beyzadeoglu, T. (2017). Arthroscopic treatment in unresolved Osgood-Schlatter disease in athletes. *International Orthopaedics, 41*(2), 351–356. https://doi.org/10.1007/s00264-016-3374-1
62. McDonough, G. R., & Rossi, M. J. (2022). Arthroscopic resection of symptomatic tibial tubercle ossicles for recalcitrant Osgood-Schlatter disease. *Arthroscopy Techniques, 11*(5), e813–e818. https://doi.org/10.1016/j.eats.2021.12.041
63. Zhi-Yao, L. (2013). Arthroscopic excision of a huge ununited ossicle due to Osgood-Schlatter disease in an adult patient. *Journal of Orthopaedic Case Reports, 3*(2), 4–7. https://doi.org/10.13107/jocr.2250-0685.092
64. Choi, W., & Jung, K. (2018). Intra-articular large ossicle associated with Osgood-Schlatter disease. *Cureus, 10*(7), e3008. https://doi.org/10.7759/cureus.3008
65. Eun, S. S., Lee, S. A., Kumar, R., Sul, E. J., Lee, S. H., Ahn, J. H., & Chang, M. J. (2015). Direct bursoscopic ossicle resection in unresolved Osgood-Schlatter disease. *Arthroscopy, 31*(3), 416–421. https://doi.org/10.1016/j.arthro.2014.08.031
66. Kamiya, T., Teramoto, A., Mori, Y., Kitamura, C., Watanabe, K., & Yamashita, T. (2021). Nano-arthroscopic ultrasound-guided excision of unresolved Osgood-Schlatter disease. *Arthroscopy Techniques, 10*(6), e1581–e1587. https://doi.org/10.1016/j.eats.2021.02.026
67. Mun, F., & Hennrikus, W. L. (2021). Surgical treatment outcomes of unresolved Osgood-Schlatter disease in adolescent athletes. *Case Reports in Orthopedics, 2021*, 6677333. https://doi.org/10.1155/2021/6677333
68. Everts, P. A., van Erp, A., DeSimone, A., Cohen, D. S., & Gardner, R. D. (2021). Platelet-rich plasma in orthopedic surgical medicine. *Platelets, 32*(2), 163–174. https://doi.org/10.1080/09537104.2020.1869717
69. Everts, P., Onishi, K., Jayaram, P., Lana, J. F., & Mautner, K. (2020). Platelet-rich plasma: New performance understandings. *International Journal of Molecular Sciences, 21*(20), 7794. https://doi.org/10.3390/ijms21207794
70. Amico, A. P., Raele, M. V., Cusmai, M., Citarella, C., & Megna, M. (2023). Efficacy of platelet-rich plasma in a young athlete with Osgood-Schlatter disease. *Journal of Clinical Images and Medical Case Reports, 4*(9), 2583.
71. Guszczyn, T., Kulesza, M., Maciąg, G., Kicman, A., & Ławicki, S. (2024). The effectiveness of treating Osgood-Schlatter disease with leukocyte-rich platelet-rich plasma. *Journal of Clinical Medicine, 13*(14), 4220. https://doi.org/10.3390/jcm13144220
72. Corbi, F., Matas, S., Álvarez-Herms, J., Sitko, S., Baiget, E., Reverter-Masia, J., & López-Laval, I. (2022). Osgood-Schlatter disease: A narrative review. *Healthcare, 10*(6), 1011. https://doi.org/10.3390/healthcare10061011
73. StretchCoach. (2025). *Osgood-Schlatter disease: Causes, symptoms, and treatment*. https://stretchcoach.com/articles/osgood-schlatters/