

# The Potencies of Autologous-conditioned Plasma for the Treatment of Osgood–Schlatter Disease: A Literature Review

Panji Sananta<sup>1\*</sup>, Yudha Anantha Khaerul Putra<sup>1</sup>, Eka Novi Fuzianingsih<sup>2</sup>

1) Orthopaedic and Traumatology Department, Faculty of Medicine, Universitas Brawijaya – Dr. Saiful Anwar General Hospital, Malang, Indonesia

\*[panjisanta@ub.ac.id](mailto:panjisanta@ub.ac.id)

[yudhaananthakp@gmail.com](mailto:yudhaananthakp@gmail.com)

2) Master of Immunology Study Program, Postgraduate School, Universitas Airlangga, Surabaya, Indonesia

[ekanoviya402@gmail.com](mailto:ekanoviya402@gmail.com)

## Abstract

Osgood-Schlatter disease (OSD) is characterized by inflammation of the patellar ligament at the tibial tuberosity resulting from growth-related overuse. Traction apophysitis of the anterior component of the tibial tuberosity (ATT) happens when the quadriceps muscle contracts at the point where the proximal tibial apophysis attaches to the tibia. The recommended interventions include the administration of analgesics, the provision of instruction on physical activity, and the implementation of physiotherapy. However, the duration of signs and symptoms can be extended. Autologous-conditioned plasma (ACP) injections are increasingly being employed in treating various musculoskeletal diseases due to their elevated concentration levels of growth factors compared to whole blood. The Autologous Concentrated Platelets (ACP) solution consists solely of platelet-rich plasma (PRP) devoid of leukocytes. This concentrated PRP will be administered via injection into the affected trauma region. The present literature evaluation used the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) methodology. The current literature review demonstrates that autologous conditioned plasma (ACP) is a suitable alternative therapy for osteochondritis dissecans (OSD). The efficacy of OSD treatment can be enhanced by administering autologous conditioned plasma (ACP) into the affected region. Using autologous conditioned plasma (ACP) to treat orthopaedic and traumatological conditions, such as OSD and other musculoskeletal injuries, has much potential as a therapeutic intervention in orthopaedics and traumatology.

**Keywords** : autologous-conditioned plasma; Osgood–Schlatter disease; patellar ligament; and platelet-rich plasma.

## Abstrak

*Osgood-schlatter disease (OSD) ditandai dengan peradangan pada ligamen patella pada tuberositas tibialis yang diakibatkan oleh penggunaan berlebihan yang berhubungan dengan pertumbuhan. Apofisititis traksi pada komponen anterior tuberositas tibialis (ATT) terjadi ketika otot paha depan berkontraksi pada titik di mana apofisis tibialis proksimal menempel pada tibia. Intervensi yang direkomendasikan meliputi pemberian analgesik, pemberian instruksi tentang aktivitas fisik, dan pelaksanaan fisioterapi. Namun, durasi tanda dan gejala dapat diperpanjang. Injeksi autologous-conditioned plasma (ACP) semakin banyak digunakan untuk*

*mengobati berbagai penyakit muskuloskeletal karena tingkat konsentrasi faktor pertumbuhannya yang lebih tinggi dibandingkan dengan darah lengkap. Larutan autologous concentrated platelets (ACP) hanya terdiri dari platelet-rich plasma (PRP) tanpa leukosit. PRP pekat ini akan diberikan melalui injeksi ke daerah trauma yang terkena. Evaluasi literatur saat ini menggunakan metodologi Preferred Reporting Items for systematic reviews and meta-analyses (PRISMA). Tinjauan literatur saat ini menunjukkan bahwa ACP merupakan terapi alternatif yang cocok untuk osteochondritis dissecans. Efektivitas pengobatan OSD dapat ditingkatkan dengan memberikan ACP ke bagian yang terkena. Menggunakan ACP untuk mengobati kondisi ortopedi dan traumatologi, seperti OSD dan cedera muskuloskeletal lainnya, memiliki banyak potensi sebagai intervensi terapeutik dalam ortopedi dan traumatologi.*

**Kata kunci** : *autologous-conditioned plasma; patellar ligament; penyakit Osgood–Schlatter; and platelet-rich plasma*

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## INTRODUCTION

Osgood-Schlatter disease (OSD) is an inflammation of the patellar ligament at the tibial tuberosity caused by growth-related overuse of the tibial tuberosity (1,2). Quadriceps muscle contractions cause the anterior component of the tibial tuberosity (ATT) traction apophysitis at the proximal tibial apophysis insertion (3,4). This causes minor avulsion fractures, including traumatic partial avulsion of the tibial tuberosity at the patellar tendon insertion. Acute OSD could also result from trauma to the ATT (5). Its appearance coincides with the maturation of the secondary ossification center of the anterior tibial tuberosity (apophyseal phase), which typically occurs between ages 11 and 9 in boys and girls, respectively. Nevertheless, indications and symptoms commonly arise between ages 8 and 12 for the former and between ages 12 and 15 for the latter (6). Analgesics, physical activity education, and physiotherapy are suggested for treatment. Nonetheless, the occurrence of signs and symptoms may be more prevalent. Autologous-conditioned plasma (ACP)

injections are increasingly used for musculoskeletal disorders (7). The growth factors in ACP will influence the essential mechanisms for tissue enhancement (such as the modulation of inflammatory processes, chemotaxis, cell proliferation, migration, and matrix synthesis and differentiation). These treatments' therapeutic applications are safe and minimally invasive. Moreover, the preparation of ACP from autologous blood is straightforward (8).

## METHODS

This literature review was created following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) Guidelines. The author uses a search engine from PubMed, Nature, and Google Scholar with the keywords: "Autologous-conditioned plasma, Osgood–Schlatter disease, and Patellar ligament". The range of the literature was published from 2013 to 2023 in English by choosing the full article, journal, and literature review. The journal will undergo a scanning

process to exclude scientific journals that are irrelevant to the keywords.

## **DISCUSSION**

### **Pathophysiology**

The pathophysiology which causes OSD is multifactorial, including mechanical, morphological, functional, environmental, and psychosocial factors.

### **Mechanical Factor**

The leading cause of Osgood-Schlatter disease (OSD) is the tight and forceful pull of the patellar tendon and the associated muscle on the apophyseal cartilage of the anterior tibial tuberosity. The physiological changes that take place during a period of rapid growth further exacerbate this. The transmission of stress loads from the quadriceps to the cartilage occurs via the patellar tendons and quadriceps. This process can result in a cartilaginous avulsion, as the ossification center tends to solidify and progress toward the formation of bone tissue. As a result, it was found that the support leg experienced a more significant occurrence of occupational stress disorder (OSD) as a result of the tractional pressures exerted during eccentric contraction of the quadriceps (9). Bone fragmentation has been observed towards the conclusion of evolutionary processes (10). Enomoto and colleagues established a reduced capacity for deformation in the patellar tendon that corresponds to the magnitude of the applied tractional force. This evidence supports the notion that mechanical and anatomical variables, such as patella infera, patella alta, shortening of the rectus femoris, or tightness or inflammation in the quadriceps muscle tendon, are the primary contributors

to the development of Osgood-Schlatter disease by segment avulsion (11).

### **Functional Factor**

It is important to stress the strength loss between the quadriceps and the hamstrings, considering how the agonist and antagonist muscles in each tissue work together now (12). For academic writing, the user's text needs to have information in it. Several other factors, especially in boys, have been found to significantly affect how much strength and muscle mass they gain during puberty (13). Some of these factors are the movement patterns, like sprints, jumps, and kicks, which can increase stress. Also, the thigh muscle has become less flexible. Also, it has been found that intense training loads in a short amount of time and specific training that involves repetitive movement patterns that cause high inertial loads have a significant effect.

Also, this finding shows that high tension in the biceps femoris, rectus, soleus, and gastrocnemius muscles could be a factor in developing pathophysiological conditions. In this study, we want to determine how a particular drug affects people with a condition. When the knee bends from 30 to 60 degrees, the contraction of these muscle groups may change the length of the lever arm, the force applied, and the compression on the patella (13). The person didn't give us any text to change. This may help explain the link between how well your quads work and OSD. Also, sports like kicking a ball may increase the risk of OSD, a disease that causes the center of gravity to be in the wrong place because the pelvis is turned backwards. This causes the quads to work harder to compensate for the muscle problems in both joints that come with

OSD. The user's text is academic, so it shouldn't be changed (6).

### **Morphological Factor**

Weight, height, body mass index (BMI), and the height of the internal longitudinal arch of the support foot (risk increases with a more prominent angle) are the critical risk factors for the support leg (14), a decrease in ankle dorsal flexion of 10 or less, a lack of flexibility in both legs, tibial rotations (an increase inside the tibial external rotation condyle-malleolar angle), lateral patellofemoral maltracking, and concurrent genu valgum and pronated foot (15). That morphology, position, and stability may significantly impact the moment of force generated at the patellar tendon insertion (16,17).

### **Environmental Factor**

Critical risk factors for the support leg include weight, height, body mass index (BMI), the height of the internal longitudinal arch of the support foot (risk increases with a more prominent angle) (18) lack of flexibility in both legs, tibial rotations (an increase in the tibial external rotation condyle-malleolar angle), lateral patellofemoral maltracking, genu valgum, and a pronated foot (15). The patellar tendon insertion's morphology, position, and stability may substantially affect the moment of force generated there (16,17).

### **Psychological Factor**

Education and reassurance for the family are the most critical aspects of OSD management, treatment, and recovery. A prolonged duration of OSD symptoms, sometimes exceeding a year, may affect how patients perceive their injury and recovery. Most sports are associated with long-term injuries in which the patient

experiences discomfort, elevated anxiety, worsened sleep, heightened catastrophizing, and a depressed mood, especially in children and young athletes. Therefore, the support of medical professionals and family members who treat a patient with OSD is indispensable (19,20,21).

### **Diagnosis**

OSD is characterized by multiple-intensity pain that worsens when suppressed, especially when kneeling (22). Hypersensitivity and inflammation are common in the anterior tibial tuberosity, where the patellar tendon inserts. A limp during sports practice or physical activity may indicate that. Acute symptoms usually increase from intermittent and light to constant and severe pain (23,24,9). Palpation may reveal patellar tendon insertion thickening and discomfort, especially during counter-resisted flexions or knee extensions (21). Pain is usually linked to increased blood flow, which may cause neovascularization over time (25). Guldhammer et al. (26) found that 42.9% of OSD patients had daily pain, and the median duration was 90 months (interquartile range, 24–150 months). According to Kaya et al. (27), 50% of patients healed within two years of diagnosis; however, strength remained lower. Patient age at the time of the event, sport played, and many cultural and environmental factors may explain the discrepancies between studies. OSD is diagnosed clinically based on symptoms. Additionally, supplementary radiological studies (X-ray, ultrasound, or MRI) must be performed to distinguish OSD from tumors, fractures, and infections (22,25,26,27).

## **X-Ray**

To exclude alternative pathologies, an X-ray is the complementary radiological test of first preference (28). Particularly in the early grades, a sagittal plane of the knee with thigh rotation of 10–20°C enables straightforward identification of separations and irregularities of the apophysis from the tibial tuberosity. It allows for detecting and identifying bone fragmentation in more difficult grades (29). In general, X-ray imaging enables the differentiation of three distinct degrees of involvement: grade (I), discernible tuberosity fragmentation; grade (II), marginal tuberosity elevation; and grade (III), radio lucidity of the tuberosity. Conversely, several instances remain asymptomatic, notwithstanding the identification of structural modifications through radiological assessment (30).

## **Ultrasonography (USG)**

Ultrasound is employed for OSD monitoring and diagnosis due to its non-invasive nature, rapidity, dependability, and affordability. It enables the visualization of the fragmentation resulting from patellar tendon injuries, the ossification center, the presence of edema, and the potential development of reactive bursitis (13,18).

## **Magnetic Resonance Imaging (MRI)**

Even before an ossification center rupture occurs, MRI enables the visualization of cartilage and the detection of edema, making it arguably the most sensitive technique for monitoring and diagnosis (30). Furthermore, it is critical for timely pathology identification (29,30,18,31). However, its application is frequently restricted to circumstances where preceding methods have proven inadequate due to its exorbitant price.

Hirano et al. utilized MRI to establish five classifications for OSD: A grade of 0-MRI is considered normal, regardless of the patient's potential manifestation of specific symptoms. Grade 1 or early stage—radiological examination reveals no discernible indications of inflammation; Grade 2 or progressive stage—the presence of a defined torn secondary ossification center; Grade 3 or terminal stage—complete sequestration of the ossicle accompanied by tendon thickening; and Grade 4 or healing proliferation—the definition of newly formed bone tissue (7).

## **Treatment**

A variety of treatment approaches have been suggested, including reducing physical activity (reduced activity, cold application, physical therapy), utilising knee orthoses that apply pressure to the patellar tendon to mitigate the tractional load on the insertion, (32) incorporating warm-up and cool-down exercises before and following physical exercise, (33) and performing leg extensor musculature stretches to alleviate tension generated by the extensor apparatus (34). Additionally, it is critical to avoid applying undue force during the insertion process. Cast immobilisation was contemplated for severe cases despite its potential to induce structural disarray and quadriceps musculature atrophy (32,33).

Jumping, running, and changing directions should be avoided until symptoms improve. Swimming and cycling add little tendon load and could replace those activities (34). Since poorer core stability is linked to higher knee flexion peak torque during the stance phase of running, numerous writers recommend core stabilisation activities (35). Increased core

stability improves jumping knee capability. However, analgesics, including ibuprofen, paracetamol, flurbiprofen, naproxen, and ketoprofen, were prescribed to alleviate pain, increase prostaglandin synthesis, and reduce inflammation (34). NSAIDs relieve symptoms but do not cure OSD (32). Low success rates were also found with NSAID infiltration. NSAIDs may cause subcutaneous adipose tissue atrophy, skin striae, or tendon ruptures (36,37). Decreased blood flow and collagen production are the leading causes of tendon deterioration. Thus, corticosteroids and NSAIDs should never be prescribed for this condition. Dextrose injections and saline are viable OSD treatments (38). Other recommended therapies include manual and electro-acupuncture, magnetic field therapy, and extracorporeal shock wave therapy. Surgery was indicated only if other treatments failed and bone fragments (intra- or extra-tendon) remained following ossification. Surgery is needed for non-displacement fractures. Other methods and procedures include ossification centre extraction, percutaneous tibial tuberosity fixation, arthroscopic contouring, and tendon debridement. Otherwise, tibial tuberoplasty or bone transplants may be considered (39).

For alternative medical procedures, ACP has been suggested (8). ACP is a concentrated extract of platelets derived from autologous blood, containing their secreted growth factors, potentially contributing to the healing process. Soft tissue disorders in which the healing response has failed and the inflammation has developed into a chronic condition may be amenable to ACP treatment. An autologous preparation injected into the tendinopathy site is hypothesised to induce

a therapeutic response (40). ACP injections used to treat musculoskeletal disorders are increasing (41). ACP's growth factors stimulate tissue-improving mechanisms, including matrix synthesis and differentiation, modulation of inflammatory processes, chemotaxis, cell proliferation, and migration. The therapeutic applications of these substances are safe and minimally invasive. In addition, the process of producing conditioned plasma using autologous blood is uncomplicated (8).

#### **Autologous-Conditioned Plasma Substance**

ACP supplies utilize the Arthrex ACP system in certain locations. The Arthrex ACP system will deliver leukocyte-free PRP (42). Platelet density in advanced circulation plasma is greater than twofold that of whole blood. Compared to whole blood, the concentration levels of growth factors in ACP are significantly elevated. For instance, platelet-derived growth factor AB experiences a 25-fold increase in concentration. In contrast, epidermal growth factor, vascular epidermal growth factor, and platelet-derived growth factor-BB increase 5 to 11 times. Insulin-like growth factor-1 and remodeling growth factor-b increase concentration by up to a 5-factor (43).

PRP was the first autologous orthobiologic to achieve widespread application in contemporary orthopaedics. It elicits a supraphysiological response called biological augmentation, which accounts for its therapeutic properties. Pro-chondrogenic growth factors comprise the preponderance of the components found in PRP. The PRP supplied by ACP is devoid of leukocytes. ACP is an additional blood-

derived orthobiologic that utilizes the anti-inflammatory properties of substances derived from blood. IL-1 receptor antagonist (IL-1Ra), an inhibitor of IL-1 derived from a natural source, is synthesized by macrophages. By binding to the IL-1 receptor, it inhibits the biological activity of IL-1. As it is hypothesized that IL-1 is implicated in developing inflammatory symptoms, inhibiting its activity should alleviate these symptoms. Through incubation, a high concentration of IL-1Ra can be produced (44).

While ACP comprises IL-1Ra and anti-inflammatory cytokines (IL-4, IL-10, and IL-13), PRP comprises VEGF, IGF-1, IGF-2, FGF, and HGF (45). We contend that the enhanced outcomes of implementing PRP are predominantly attributable to VEGF (46). VEGF, a crucial component in the angiogenesis process during the early phases of tendon regeneration, is especially influential in a region with limited blood flow, such as the Achilles tendon. According to Lyras et al., the administration of PRP accelerated Achilles tendon recovery by promoting angiogenesis via its high VEGF concentration (47).

ACP comprises many biological components that establish an ideal milieu for restoring OA. The essential GFs and cytokines present in ACP are outlined in Figure 1. Numerous studies have demonstrated the significance of these cytokines and GFs in the pathogenesis and potential resolution of OA. These cytokines and growth factors, particularly ACP, have been identified in PRP (48).

Factor	Name	Effects/Activities
PDGF-AB	Platelet-derived growth factor AB	Cell proliferation and mitogenesis
TGF-β1	Transforming growth factor-beta1	Extracellular matrix (ECM) synthesis, cell proliferation
VEGF	Vascular endothelial growth factor	Angiogenesis, cartilage metabolism
IL-10	Interleukin-10	Prevention of IL-1β and TNF-α activity
IL-1ra	Interleukin-1 receptor antagonist	Prevention of IL-1β and TNF-α activity
IL-1β	Interleukin-1 beta	Cartilage degradation and inflammation
TNF-α	Tumor necrosis factor-alpha	Cartilage degradation and inflammation

**Figure 1.** Biological factors in ACP

### **Autologous-Conditioned Plasma Mechanism and Effectiveness to Treat Osgood-Schlatter Disease**

One biological therapeutic approach uses autologous growth factors in ACP, which provoke collagen regeneration for well-ordered angiogenesis (43,49). Degranulating the alpha granules within the platelets liberates some dissimilar growth factors in high numbers. Autologous growth factors are delivered locally via platelet-rich plasma (PRP) by injection to the tendon repair site. PRP has acquired popularity as a potentially worthwhile regenerative therapy to cope with many musculoskeletal injuries. Hence, most growth factors are short-lived, so repeated administration is required. Repeated injection of ACP is a promising technique and has already guaranteed improved tendon healing and pain relief (43,50).

The rationale for using PRP for tendon disorders is that alpha granules in the platelets liberate various growth factors, which provoke collagen regeneration for tendon healing and well-ordered angiogenesis. Therefore, using ACP in treating tendon lesions over several years

has led to a meaningful improvement in healing. Commercial preparations of ACP vary in their ability to concentrate platelets and leukocytes. McCarrel and Fortier showed that leukocyte concentration is certainly associated with catabolic gene expression in ligaments and tendons, which suggested that transmission of concentrated leukocytes to a site of injury might not provide a favorable environment for tissue healing (43).

OSD commonly settles with conservative therapy (restricted physical activity and mild analgesia) or age, although it could be treatment-resistant or reoccurring (4,13). Additionally, Danneberg reported two patients with OSD at his clinic who were effectively treated with ACP simultaneously (8). Both patients were 14 and 23 years old. They were energetic and intensive tennis players, suitable for the typical profile of OSD (6,51). For both patients, the novel ACP treatment came after the failure of conventional therapies. For three to five injections, patients received once-weekly subcutaneous injections of one mL ACP on either side of the palpable Osgood–Schlatter lesion/swelling. The first patient was treated in both knees for four weeks, and the second in the right knee for three weeks. The knee was extended throughout the injection to relax the tendon fibers, facilitating ACP diffusion into the injured area. Local anesthesia and corticosteroid were not used. There were no post-injection complications. The first patient experienced pain-free after six weeks and could return to sport. The second patient experienced pain subtraction of 50% after the first injection. After three weeks, the patient was pain-free and could return to sports. Danneberg reported the successful novel use of ACP

therapy for OSD, providing the first insight into a selection therapy for patients with standard-treatment failure or recurrent OSD (8).

## CONCLUSION

Based on the analysis and synthesis of problems described in the literature review, ACP is the appropriate alternative therapy for OSD. Successful OSD treatment can be achieved by injecting ACP into the problem area. The effectiveness of ACP for OSD and other musculoskeletal injuries will make it a great therapy for orthopaedics and traumatology in the future. Further research on ACP therapy for OSD in a larger and wider patient group is indicated to optimize the treatment protocol.

## REFERENCES

1. Vaishya R, Azizi AT, Agarwal AK V V. Apophysitis of the Tibial Tuberosity (Osgood-Schlatter Disease): A Review. *Cureus*. 2016;8(9).
2. Bezuglov EN, Tikhonova AA, Chubarovskiy PV, Repetyuk AD, Khaitin VY, Lazarev AM UE. Conservative treatment of Osgood-Schlatter disease among young professional soccer players. *Int Orthop*. 2020;44(9):1737–43.
3. Holden S, Olesen JL, Winiarski LM, Krommes K, Thorborg K, Hölmich P RM. Is the Prognosis of Osgood-Schlatter Poorer Than Anticipated? A Prospective Cohort Study With 24-Month Follow-up. *Orthop J Sport Med*. 2021;9(8).
4. Pascarella F, Ziranu A MG. Tibial tubercle fracture in a 14-year-old athlete with bilateral lower pole bipartite patella and osgood-schlatter disease. *Case Rep Orthop*. 2015;2015.
5. Nkaoui M EAE. Osgood-schlatter disease: risk of a disease deemed



6. banal. Pan Afr Med J. 2017;28(56). Zonfrillo MR, Spicer RS, Lawrence BA MT. Incidence and costs of injuries to children and adults in the United States. *Inj Epidemiology*. 2018;5(1):37.
7. Corbi F, Matas S, Alvarez-Herms J, Sitko S, Baiget E, Reverter-Masia J L-LI. Osgood-Schlatter Disease: Appearance, Diagnosis and Treatment: A Narrative Review. *Healthc*. 2022;10(6):1011.
8. Danneberg DJ. Successful Treatment of Osgood-Schlatter Disease with Autologous-Conditioned Plasma in Two Patients. *Joints*. 2017;5(3):191–4.
9. Halilbašić A, Kreso A, Klepić M, Jaganjac A AD. The Algorithm for overload syndrome prevention: Osgood-Schlatter's syndrome (OSD) as an overload syndrome caused by early inclusion of children in sports and excessive physical activity (sports and recreation). *J Heal Sci*. 2019;9(3).
10. Enomoto S, Tsushima A, Oda T KM. The Passive Mechanical Properties of Muscles and Tendons in Children Affected by Osgood-Schlatter Disease. *J Pediatr Orthop*. 2020;40(4):e243–7.
11. Lyng KD, Rathleff MS, Dean BJF, Kluzek S HS. Current management strategies in Osgood Schlatter: A cross-sectional mixed-method study. *Scand J Med Sci Sport*. 2020;30(10):1985–91.
12. Nakase J, Aiba T, Goshima K, Takahashi R, Toratani T, Kosaka M, Ohashi Y TH. Relationship between the skeletal maturation of the distal attachment of the patellar tendon and physical features in preadolescent male football players. *Knee Surg Sport Traumatol Arthrosc*. 2014;22(1):195–9.
13. Yanagisawa S, Osawa T, Saito K, Kobayashi T, Tajika T, Yamamoto A, Iizuka H TK. Assessment of Osgood-Schlatter Disease and the Skeletal Maturation of the Distal Attachment of the Patellar Tendon in Preadolescent Males. *Orthop J Sport Med*. 2014;2(7).
14. Watanabe H, Fujii M, Yoshimoto M, Abe H, Toda N, Higashiyama R TN. No Title. Pathog Factors Assoc With Osgood-Schlatter Dis Adolesc Male Soccer Play A Prospect Cohort Study. 2018;6(8).
15. Jibri Z, Jamieson P, Rakhra KS, Sampaio ML DG. Patellar maltracking: an update on the diagnosis and treatment strategies. *Insights Imaging*. 2019;10(1):65.
16. Gaulrapp H NC. The Osgood-Schlatter disease: a large clinical series with evaluation of risk factors, natural course, and outcomes. *Int Orthop*. 2022;46(2):197–204.
17. Green DW, Sidharthan S, Schlichte LM, Aitchison AH MD. Increased Posterior Tibial Slope in Patients With Osgood-Schlatter Disease: A New Association. 2020;48(3):642–6.
18. Mebis W, Jager T VHE. Intratendinous Patellar Ganglion Cyst with Coexistent Osgood Schlatter Disease. *J Belgian Soc Radiol*. 2016;100(1):86.
19. Hall R, Barber Foss K, Hewett TE MG. Sport specialization's association with an increased risk of developing anterior knee pain in adolescent female athletes. *J Sport Rehabil*. 2015;24(1):31–5.
20. Cahalan R, Purtill H, O'Sullivan P OK. A cross-sectional study of elite adult Irish dancers: biopsychosocial traits, pain, and injury. *J Danc Med Sci*. 2015;19(1):31–43.
21. Cahalan R, Bargary N OK. Pain and Injury in Elite Adolescent Irish Dancers: A Cross-Sectional Study. *J Danc Med Sci*. 2018;22(2):91–9.
22. Nur Suhaila I, Siti Suhaila MY WAW. An active boy with bilateral knee pain. *Malaysia Fam Phys*.

- 2019;14(1):26–8.
23. Moy A, Song E, Wallace SJ, Teixeira R TD. Simultaneous Bilateral Patellar Tendon Rupture in a Young Adult Male: A Case Report and Review of the Literature. *Cureus*. 2020;129.
24. Kartini C W-S DI. Osgood-Schlatter disease: A review of current diagnosis and management. *Cureus*. 2022;33:294–8.
25. Saily M, Whiteley R JA. Doppler ultrasound and tibial tuberosity maturation status predicts pain in adolescent male athletes with Osgood-Schlatter's disease: a case series with comparison group and clinical interpretation. *Br J Sports Med*. 2013;47(2):93–7.
26. Guldhammer C, Rathleff MS, Jensen HP HS. Long-term Prognosis and Impact of Osgood-Schlatter Disease 4 Years After Diagnosis: A Retrospective Study. *Orthop J Sport Med*. 2019;7(10).
27. Kaya DO, Toprak U, Baltaci G, Yosmaoglu B OH. Long-term functional and sonographic outcomes in Osgood-Schlatter disease. *Knee Surg Sport Traumatol Arthrosc*. 2013;21(5):1131–9.
28. Ohtaka M, Hiramoto I, Minagawa H, Matsuzaki M KH. Screening of the Maturity Status of the Tibial Tuberosity by Ultrasonography in Higher Elementary School Grade Schoolchildren. *Int J Environ Res Public Health*. 2019;16(12):2138.
29. Hart E, Meehan WP 3rd, Bae DS, d'Hemecourt P SA. The Young Injured Gymnast: A Literature Review and Discussion. *Curr Sport Med Reports*. 2018;17(11):366–75.
30. Circi E, Atalay Y BT. Treatment of Osgood-Schlatter disease: review of the literature. *Musculoskelet Surg*. 2017;101(3):195–200.
31. Circi E BT. Results of arthroscopic treatment in unresolved Osgood-Schlatter disease in athletes. *Int Orthop*. 2017;41(2):351–6.
32. Yen YM. Assessment and treatment of knee pain in the child and adolescent athlete. *Pediatr Clin North Am*. 2014;61(6):1155–73.
33. Herrero-Morín JD, Fernández González N, Gutiérrez Díez C, Pérez Menéndez MT FFE. Enfermedad de Osgood-Schlatter en un adolescente deportista. Caso clínico [Osgood-Schlatter disease in adolescent athlete. *Arch Argent Pediatr*. 2017;115(6):445–8.
34. Morris E. Acupuncture in Osgood-Schlatter disease. *BMJ Case Rep*. 2016;
35. Ladenhauf HN, Seitlinger G GD. Osgood-Schlatter disease: a 2020 update of a common knee condition in children. *Curr Opin Pediatr*. 2020;32(1):107–12.
36. Neuhaus C, Appenzeller-Herzog C FO. A systematic review on conservative treatment options for OSGOOD-Schlatter disease. 2021;49:178–87.
37. Wise K, Warren D DL. Unilateral striae distensae of the knee after a steroid injection for the treatment of Osgood-Schlatter disease. *Dermatol Online J*. 2017;23(31).
38. Weiler R, Ingram M WR. 10-Minute Consultation. Osgood-Schlatter disease. *BMJ*. 2011;343.
39. Narayan N, Mitchell PD LM. Complete resolution of the symptoms of refractory Osgood-Schlatter disease following percutaneous fixation of the tibial tuberosity. *BMJ Case Rep*. 2015;
40. Lebiecziński R, Synder M, Buchcic P, Polgaj M, Grzegorzewski A SM. A randomized study of autologous conditioned plasma and steroid injections in the treatment of lateral epicondylitis. *Int Orthop*. 2015;39(11):2199–203.
41. Omodaka T, Ohsawa T, Tajika T, Shiozawa H, Hasimoto S, Ohmae H, Shitara H, Ichinose T, Sasaki T,

- Hamano N, Takagishi K CH. Relationship Between Lower Limb Tightness and Practice Time Among Adolescent Baseball Players With Symptomatic Osgood-Schlatter Disease. *Orthop J Sport Med.* 2019;7(5).
42. Dohan Ehrenfest DM, Bielecki T, Mishra A, Borzini P, Inchingolo F, Sammartino G, Rasmusson L EP. In search of a consensus terminology in the field of platelet concentrates for surgical use: platelet-rich plasma (PRP), platelet-rich fibrin (PRF), fibrin gel polymerization and leukocytes. *Curr Pharm Biotechnol.* 2012;13(7):1131–7.
  43. Charousset C, Zaoui A, Bellaiche L BB. Are multiple platelet-rich plasma injections useful for treatment of chronic patellar tendinopathy in athletes? a prospective study. *Am J Sports Med.* 2014;42(4):906–11.
  44. Arthrex Inc. Arthrex Double Syringe Ide-Pivotal. 2017;(January):1–98.
  45. Baria M, Vasileff WK, Miller M, Borchers J, Flanigan DC DS. Cellular Components and Growth Factor Content of Platelet-Rich Plasma With a Customizable Commercial System. 2019;47(5):1216–22.
  46. Genç E, Yüksel S, Çağlar A, Beytemur O GM. Comparison on effects of platelet-rich plasma versus autologous conditioned serum on Achilles tendon healing in a rat model. *Acta Orthop Traumatol Turc.* 2020;54(4):438–44.
  47. Khurana A, Goyal A, Kirubakaran P, Akhand G, Gupta R GN. Efficacy of Autologous Conditioned Serum (ACS), Platelet-Rich Plasma (PRP), Hyaluronic Acid (HA) and Steroid for Early Osteoarthritis Knee: A Comparative Analysis. *Indian J Orthop.* 2020;55.
  48. Lyras DN, Kazakos K, Verettas D, Polychronidis A, Tryfonidis M, Botaitis S, Agrogiannis G, Simopoulos C, Kokka A PE. The influence of platelet-rich plasma on angiogenesis during the early phase of tendon healing. *Foot Ankle Int.* 2009;30(11):1101–6.
  49. Mishra AK, Skrepnik NV, Edwards SG, Jones GL, Sampson S, Vermillion DA, Ramsey ML, Karli DC RA. Efficacy of platelet-rich plasma for chronic tennis elbow: a double-blind, prospective, multicenter, randomized controlled trial of 230 patients. *Am J Sports Med.* 2014;42(2):463–71.
  50. Vetrano M, Castorina A, Vulpiani MC, Baldini R, Pavan A FA. Platelet-rich plasma versus focused shock waves in the treatment of jumper's knee in athletes. *Br J Gen Pract.* 2013;41(4):795–803.
  51. G E. The Osgood-Schlatter lesion. A clinical and experimental study. *Acta Chir Scand Suppl.* 1962;288:1–36.