



**AALBORG UNIVERSITY**  
DENMARK

**Aalborg Universitet**

## **A systematic review of imaging findings in patients with Osgood-Schlatter Disease**

Sørensen, Line Bay; Rathleff, Michael Skovdal; Dean, Benjamin John Floyd; Oei, Edwin; Magnussen, Stig Peter; Olesen, Jens Lykkegaard; Holden, Sinéad

*Published in:*  
Translational Sports Medicine

*DOI (link to publication from Publisher):*  
[10.1002/tsm2.281](https://doi.org/10.1002/tsm2.281)

*Publication date:*  
2021

*Document Version*  
Accepted author manuscript, peer reviewed version

[Link to publication from Aalborg University](#)

*Citation for published version (APA):*  
Sørensen, L. B., Rathleff, M. S., Dean, B. J. F., Oei, E., Magnussen, S. P., Olesen, J. L., & Holden, S. (2021). A systematic review of imaging findings in patients with Osgood-Schlatter Disease. *Translational Sports Medicine*, 4(6), 772-787. <https://doi.org/10.1002/tsm2.281>

### **General rights**

Copyright and moral rights for the publications made accessible in the public portal are retained by the authors and/or other copyright owners and it is a condition of accessing publications that users recognise and abide by the legal requirements associated with these rights.

- Users may download and print one copy of any publication from the public portal for the purpose of private study or research.
- You may not further distribute the material or use it for any profit-making activity or commercial gain
- You may freely distribute the URL identifying the publication in the public portal -

### **Take down policy**

If you believe that this document breaches copyright please contact us at [vbn@aub.aau.dk](mailto:vbn@aub.aau.dk) providing details, and we will remove access to the work immediately and investigate your claim.

Article type : Review

## A systematic review of imaging findings in patients with Osgood-Schlatter Disease

Line Bay Sørensen\*<sup>1</sup>, Michael Skovdal Rathleff<sup>1,2,6</sup>, Benjamin John Floyd Dean<sup>3</sup>, Edwin Oei<sup>4</sup>, Stig Peter Magnussen<sup>5</sup>, Jens Lykkegaard Olesen<sup>2</sup>, Sinéad Holden<sup>1,2</sup>

<sup>1</sup>Department of Health Science and Technology, Aalborg University, Aalborg Denmark, <sup>2</sup>Center for General Practice at Aalborg University, Aalborg, Denmark, <sup>3</sup>Nuffield Department of Orthopaedics, Rheumatology and Musculoskeletal Science (NDORMS), Botnar Research Centre, University of Oxford, UK, <sup>4</sup>Department of Radiology & Nuclear Medicine of Erasmus MC, University Medical Center, Rotterdam, the Netherlands, <sup>5</sup>Department of Orthopaedic Surgery M, Institute of Sports Medicine, Bispebjerg Hospital, Copenhagen, Denmark, <sup>6</sup>Department of Occupational Therapy and Physiotherapy, Department of Clinical Medicine, Aalborg University Hospital, Aalborg, Denmark

Original Research Article for *Translational Sports Medicine (TSM)*

### \*Corresponding author

Line Bay Sørensen, Post.doc

Department of Health Science and Technology

Faculty of Medicine, Aalborg University

Fredrik Bajers Vej 7, DK-9220 Aalborg, Denmark

Phone: +45 52373229,

E-mail: lbs@hst.aau.dk

This article has been accepted for publication and undergone full peer review but has not been through the copyediting, typesetting, pagination and proofreading process, which may lead to differences between this version and the [Version of Record](#). Please cite this article as [doi: 10.1002/TSM2.281](https://doi.org/10.1002/TSM2.281)

This article is protected by copyright. All rights reserved

## **Conflict of interest**

The authors have no conflict of interest to declare

## **ABSTRACT**

This systematic review aimed to describe the imaging characteristics of Osgood-Schlatter (OSD) compared with controls and imaging findings over time. A systematic search was conducted in Embase, CINAHL, and PubMed from inception until July 2021. Forty studies were eligible and included based on inclusion criteria on OSD diagnosis, the number of patients, and imaging outcomes. In patients with OSD, but not controls, findings were soft-tissue swelling of the cartilage and infrapatellar bursa, tendon changes, increased Doppler flow, and fragmentation of the secondary ossification center. Follow-up studies reported improvements over time, but some identified persistent tendon thickening and/or ossicles. Adults with OSD generally present with free ossicles. Findings were inconsistent on whether different morphometric features were altered in OSD compared to controls. OSD patients were classified within the early stages of tibial tuberosity maturation. This review documents that OSD presents with tissue alterations that do not appear in controls or the patient's asymptomatic knee. Notably, a large portion had tendon involvement, and ossicles seem to be associated with residual symptoms after maturation. Standard imaging of adolescents with OSD needs to be carefully considered when determining if tissue alterations are related to disease progression or part of the normal maturation.

**Keywords:** Apophysis; imaging; tibial tuberosity; growth; overuse; patellar tendon

## **Introduction**

Osgood Schlatter disease (OSD) is a common knee pain condition that affects 10% of adolescents between 9 and 15 years of age<sup>33</sup>. The key symptom is anterior knee pain below the kneecap, where the patellar tendon inserts onto the developing part of the tibial tuberosity. As up to one in five cases is observed in highly active adolescents, OSD is believed to be a sport-related pain complaint during maturation of the tibial tuberosity<sup>33,44</sup>. Although a clinical exam often

determines the diagnosis, routine imaging is usually done in adolescents with this condition <sup>38</sup>. However, little is still known about disease etiology and the involvement of different tissues over time as the disease progresses. Studies suggest that OSD pain symptoms may be driven primarily by avulsions of the secondary ossification center <sup>34,45</sup>. In contrast, others have suggested that OSD may have traits similar to patellar tendinopathy rather than a tibial tuberosity lesion <sup>45</sup>. Previous imaging studies have evaluated the tibial tuberosity's developmental stages using different imaging modalities <sup>3,16,34</sup>. However, it is unclear which tissue alterations are associated with OSD or part of the normal development of the apophysis during adolescence <sup>7</sup>. Hence, understanding the features related to OSD compared to normal skeletal development is essential for interpreting imaging findings.

Classifications such as that reported by De Flaviis et al. <sup>16</sup> describe OSD stages based on alterations of the secondary ossification center versus the soft tissues such as the patellar tendon or bursitis. However, imaging findings may depend on the severity of the condition and the imaging modality being used <sup>24,45</sup>. To date, there has been no systematic synthesis detailing the potential tissue characteristics or maturation status of the tibial tuberosity in patients with OSD. Therefore, this study aims to systematically review imaging findings on radiographs, magnetic resonance imaging (MRI), computed tomography (CT), or ultrasound (US) related to soft tissue changes, bone alterations, tibial tuberosity maturation, and morphometric features in patients diagnosed with OSD. Where possible, we intended to compare the imaging findings to controls without OSD, on follow-up, or to patient's asymptomatic knees in patients with unilateral OSD. A secondary aim was to evaluate the association between imaging findings and pain outcomes.

## **Methods**

The protocol for this systematic review was prospectively uploaded to the Open Science Framework prior to initiating the search (*OSF, registration: <https://osf.io/96wmn>*). This study reporting follows the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement <sup>40</sup>.

### **Search strategy**

A systematic search was carried out in PubMed, Embase, and the Cumulative Index to Nursing and Allied Health Literature (CINAHL) from database inception until July 2021. Additionally, the search was supplemented by a manual search, authors' own work, citation search of relevant articles, and forward citation tracking to identify items that were not found by the first search. There were no restrictions on date or language. Articles in English, German, Dutch, Scandinavian languages, and French were considered. The researchers (LBS, SH, and MSR) developed the search strategy and consulted with a research librarian (full details included in the *supplemental material*).

### **Eligibility criteria**

The studies had to include: 1)  $\geq 10$  patients with OSD; 2) patients diagnosed with OSD (of any age or sex); and 3) OSD-related imaging findings, tibial tuberosity maturation, or morphological abnormalities (morphometric measures) using radiographs, magnetic resonance imaging (MRI), computed tomography (CT), or ultrasonography (US). The OSD diagnosis was required to include pain localized to the tibial tuberosity, regardless of whether imaging findings confirmed the diagnosis. Original research articles were included, and there were no restrictions on follow-ups for cohort studies. Studies were excluded if they reported on imaging findings related to other growth-related conditions (e.g., apophyseal injuries) or any traumatic knee pain conditions without explicitly reporting separately on OSD cases. Studies reporting on the tibial tuberosity maturation without any cases of OSD were not considered.

### **Selection of studies**

All citations identified by the search were exported, and duplicates were removed. The relevant studies were imported into Covidence for the initial screening, and full-text was retrieved and saved before the data extraction. Two reviewers (LBS, SH) independently screened titles and abstracts. Relevant full-text articles were retrieved, and the same reviewers completed a full-text evaluation. Any disagreements were discussed and resolved through consensus. In case consensus was not reached, a third reviewer (MSR) adjudicated cases of disagreement.

### **Data extraction**

Two of three reviewers extracted all data (LBS, SH, and/or BD) independently. Data on study design and aims, population, setting, participant characteristics, diagnosis, follow-up, and pain characteristics were extracted, and all imaging outcomes for both OSD and controls, or the asymptomatic side in participants with unilateral pain at baseline and follow-up assessment(s).

### **Outcome**

The main outcomes of interest from the different imaging techniques were any tissue characteristics associated with OSD. Secondary outcomes included morphometric features related to OSD and tibial tuberosity maturation and any associations between imaging findings and pain outcomes.

### **Quality assessment**

A methodological quality assessment was performed on all included studies by two independent reviewers (LBS, MSR) using the Scottish Intercollegiate Guidelines Network (SIGN) checklist.

Although the included studies had varied study designs, all were evaluated using the SIGN Quality checklist for cohort studies.

### **Data synthesis**

Data were synthesized descriptively. The primary synthesis compared patients with OSD to control subjects without OSD. In addition, a comparison to contralateral asymptomatic limb for those without a control group was included - finally, changes in imaging characteristics over time for patients with OSD were evaluated. A sub-group analysis investigated the association between imaging findings and pain.

## **Results**

### **Study selection**

The initial search identified 5323 studies. One study was identified from the authors' own work, and two articles were found from the subsequent hand search of citations, resulting in 5326 identified studies. After the removal of duplicates (n=885), 4441 studies were screened by title and abstract. One hundred and twelve were screened by full-text, with 40 of these meeting the

inclusion criteria. In Supplemental material, an overview of study identification, screening, and inclusion is presented in (Fig. S1 PRISMA Flowchart).

### **Characteristics of included studies**

Study designs were cross-sectional and case-controls (n=21), retrospective cohorts (n=12), prospective cohort studies (n=5) and prospective case series (n=2). The overall number of participants included with OSD was 1955. Of the studies that reported sex and age range, 70% were male, and 30% were female, with adolescents aged from 9 to 17 years. Five studies included adults with unresolved OSD aged 18-39 years <sup>11,29,36,42,52</sup>. Full study characteristics are summarized in *Supplemental Material, Table S1*.

### **Quality assessment**

The methodological quality of the studies assessed by SIGN is shown in *Supplemental Material, Table S1*. Four studies were rated as high quality. Most studies were deemed unacceptable quality and only fulfilled a few criteria (n=19) or acceptable quality (n=17). Twelve of the included studies were retrospective studies, and on this basis, they could not be rated higher than acceptable. Fourteen studies did not have a comparison group. Six studies used asymptomatic knees as controls, one study used other apophyseal injuries, and two studies used patients with tibial tubercle fractures control group. Two studies used both a healthy control group and the asymptomatic knee to relate to OSD imaging findings. Four out of 40 studies used a blinded outcome assessor, while 21/40 studies reported who interpreted the images.

### **Imaging findings**

Of the 40 studies included, twenty-five studies reported on OSD-related imaging findings <sup>3,4,9-12,14,16,21,23,24,27,29,31,32,34-36,39,42,45,47,48,50,52</sup>. Most studies used radiography (n=10) or US (n=6), with the remaining studies using radiography and US (n=4), radiography and MRI (n=3), MRI only (n=1) and one study using both CT, MRI, radiography, and bone scintigraphy (n=1). The baseline imaging characteristics are summarised in Table 1. Studies reported imaging findings such as cartilage swelling, bone fragmentation, and separated ossicles, patellar tendon thickening, and vascularisation demonstrated by US Doppler activity, as well as infrapatellar bursitis and

tendinitis (full details outlined in Table 1). Four studies used DeFlaviis classification of the 'Type' (i.e., tissue involvement), while five used a staged rating related to severity. Most studies primarily used qualitative assessments (n=16) regardless of whether a scoring system was used or not. Further details on classification and type/stage related to OSD severity are presented in *Supplemental Material Table S2*.

\*\*\*\*\* Insert **Table 1. Baseline imaging findings.** \*\*\*\*\*

#### *Comparison with control subjects without OSD and asymptomatic knees*

Eight cross-sectional <sup>14,31,34,39,47,48,50,52</sup> and two case-control studies <sup>3,16</sup> reported on OSD imaging findings compared to controls. In the studies that included a control group, the imaging findings described above (Table 1) were not observed in the controls without OSD <sup>16,34,39,52</sup>. One of the studies that included the asymptomatic knee of OSD patients described these findings as absent <sup>14</sup>. The findings are presented in *Supplemental Material Table S3*

#### *Changes in OSD imaging characteristics over time*

Seven prospective studies <sup>4,12,21,24,27,35,45</sup> and six retrospective <sup>9-11,22,32,42</sup> reported on imaging findings at follow-up. Follow-up time points ranged from five months to ten years from the initial assessment. As observed on imaging, improvements of OSD severity included decreased tendon thickness, reduced bursitis, and union of ossicles <sup>4,24,45</sup>.

However, no study reported complete recovery in all participants. The presence of ossicles and cases with 'worsening' of soft tissue findings were also described at follow-up. Some studies



noted 'deterioration' and/or cases of abnormal tibial profiles such as roughening or prominence of the tuberosity. One study reported that patients with OSD were associated with lower grade Ogden fracture. Full details are reported in *Supplemental material section 2.3.2 and Table S4*

#### *Imaging characteristics in young adults with unresolved OSD*

Five studies reported explicitly on imaging findings in an adult population with unresolved OSD symptoms<sup>11,29,36,42,52</sup> (Table 1). Two retrospective studies with no control<sup>11,42</sup> reported that 93% and 100% of OSD had separate ossicles on pre-operative radiographs. Only Pihlajamäki et al.<sup>42</sup> obtained postoperative images after ten years (6-19 years) showing new ossicles in 44 knees (38%, Table S4). Another retrospective study by Kamel et al.<sup>29</sup> found that 67% of those with residual OSD (aged 20 – 50 years) had a single ossified fragment. The remaining either demonstrated multiple ossified fragments (21%) or fragments that had fused with the tibial tuberosity. Moreover, a cross-sectional evaluation of OSD that had been operated on for painful and prominent tibial tuberosity<sup>52</sup> showed that 92.7% cases had permanent ossicles and six cases had a distinct tibial tuberosity prominence as compared to a control group, in which their images were defined as normal (Table S3). Lastly, Lee et al.<sup>36</sup> observed patellar tendinopathy and bone marrow edema in 43% of cases in a sample with symptomatic OSD, which was not present in a matched control group.

#### **Morphometric measures**

Sixteen studies quantified anatomical and morphometric features proposed to be related to OSD<sup>1,2,6,13,14,17,18,28,29,31,36,42,49–52</sup>. Full details are outlined in Table 2 (patellofemoral characteristics) and Table 3 (tendon, attachment and tibial characteristics).

Briefly, results were conflicting for patellar features, particularly for patella height, shape, and angle. One study<sup>29</sup> found greater patellar maltracking in OSD compared to controls, whereas another<sup>18</sup> found no difference in tibial-tuberosity to trochlear groove distance between OSD and controls.

Regarding tendon characteristics, two studies found enlarged tendons and that the patellar attachment was broader and more proximal compared to controls<sup>6,36</sup>. Two studies by Enomoto reported increased tendon stiffness compared to controls<sup>13,14</sup>.

Two studies found increased posterior slope in OSD compared with controls (without knee pain<sup>19</sup> or those with other knee pain<sup>51</sup>), while one study found a difference in the medial proximal tibial angle<sup>52</sup>. One study identified differences in tibial torsion angle between OSD and controls<sup>18,51</sup>.

Pihlajamäki et al.<sup>42</sup> and Visuri et al.<sup>52</sup> reported that the tibial tuberosity index was significantly greater in adults with residual OSD than in the controls. Other anatomical characteristics (remote from the knee) are presented in *Table S5*, and further detail of all findings can be found in *Supplemental Material section 2.4.1*

### **Maturation**

Ten studies reported on tibial tuberosity maturation<sup>4,10,12,15,23,27,30,37,41,47</sup> and six of these did not include controls<sup>10,12,15,23,30,37</sup>. Most studies (n=6) used the Ehrenborg classification or a modified version ranging from the cartilaginous stage to a bony/mature attachment. One study used a modified Nakase classification<sup>27</sup>, whereas another study used a 4-point scale to classify the tibial maturation status<sup>47</sup>. Full results on the maturation of the tibial tuberosity are outlined in Table 4.

\*\*\*\*\**Insert Table 2. Patellofemoral characteristics and morphometric features.*\*\*\*\*\*

\*\*\**Insert Table 3. Patellar tendon, attachment & tibial characteristics and morphometric features.*\*\*\*

\*\*\*\*\*Insert Table 4. Tibial tuberosity maturation.\*\*\*\*\*

### **Association between imaging findings and pain**

Three studies evaluated the association between imaging and pain <sup>27,32,47</sup>. One prospective study found an association between US findings at first assessment and the presence of pain at follow-up <sup>27</sup>. Those classified as 'Normal' were less likely to report pain (0%), while compared to the other groups, Type 4 De Flaviis 'associated bursitis' was associated with a significantly increased proportion of participants reporting pain (100%). One retrospective study <sup>32</sup> found that patients with symptoms at nine-year follow-up (average) were those with distorted tibial tuberosity associated with initial fragmentation at baseline. In a retrospective study of adults undergoing surgery, Pihlajamäki et al. <sup>43</sup> found no association between reduced tuberosity size and outcomes following surgery. Saily and colleagues reported a cross-sectional association between Doppler US and pain, which found that pain on palpation ( $47.0 \pm 24.5$  vs.  $18.0 \pm 11.4$ ) and resisted contraction ( $59.0 \pm 20.2$  vs.  $17.0 \pm 12.5$ ) was significantly higher in OSD with a positive TT Doppler activity than those with a negative Doppler activity <sup>47</sup>.

### **Discussion**

This systematic review shows that patients with OSD present with tissue alterations such as soft-tissue swelling, fragmentations of the bone, and patellar tendon changes that are not found in controls or patients' asymptomatic knees. Longitudinal studies indicate continued involvement of the patellar tendon, ossicles, and elevated tuberosity persist in some cases at follow-up. Observations of persistent ossicles have been reported in the adult with symptomatic OSD. Improvements in OSD severity seemed to be characterized by decreased soft tissue-swelling in the tendon and bursae. Contrary to popular beliefs surrounding high patella and predisposition to OSD, there were inconsistent findings between studies on patella height, morphology, and angle compared to controls. Two studies indicated differences in the patellar tendon attachment to the tuberosity in OSD patients compared to controls, with the tendon inserting more proximally and broader in OSD and others found increased posterior tibial slope and tuberosity

thickness in OSD. Patients with OSD symptoms appear to be at the early stages of tibial tuberosity maturation generally.

### **Controversy surrounding OSD aetiology and tissue involvement?**

Despite the debates surrounding this condition <sup>26</sup>, OSD was generally thought to be due to partial avulsions of the secondary ossification center. Since then, the involvement of soft tissues and particularly changes within the distal portion of the tendon has been acknowledged <sup>17</sup>. More recent studies indicate that fragmentation may occur with normal maturation <sup>4,7,45</sup> and is therefore not specific for OSD.

The identified findings in this study might depend on the imaging modalities. Studies using radiography tended to report changes observed in the bone, such as fragmentation and irregularities of the apophyseal surface <sup>4,23,32</sup>. Tendon involvement assessed by US in OSD includes increased patellar tendon thickening and increased Doppler flow, indicating neovascularization present in both the tendon and tibial attachment <sup>27</sup>. MR images demonstrated an increase in signal intensity at the distal tendon sites on T1-weighted images indicative of local tendinitis <sup>45</sup>. Additionally, there appear to be different presentations/involvement of OSD, and only four studies used the De Flaviis classification to characterize the tissue involvement <sup>3,16,31</sup>. There may be different sub-groups or changes over time with severity, however, this is unclear. Both studies by Hirano <sup>25</sup> and Duperron <sup>9</sup> described 'severity' based on the extent of tissue involvement, ranging from, e.g., cartilage swelling only to swelling, plus fragmentation plus soft tissue involvement. As neither included controls, the validity of these scales is lacking. Further, they did not associate the groups with clinical outcomes.

### **Morphology and anatomical risk factors of OSD**

Morphological and morphometric features have been considered potential risk factors in the development of OSD <sup>17,52</sup> and have been suggested as a mechanical etiology of OSD <sup>28</sup>. This review generally demonstrates differences surrounding patellar tendon attachment, the posterior tibial slope, and other tibial characteristics such as the tibial torsion angle and proximal

medial tibial angle may indicate that such anatomical features may predispose individuals to develop OSD. Interestingly, most of the identified differences were related to the tibial and not patellar characteristics (such as patellar height), which have traditionally been thought to be associated with OSD. Four studies showed the patellar height to be close to normal with findings of both patellar alta and baja in OSD and controls, although different standards were used to determine the patella's positioning <sup>2,36,43,50</sup>. Lee et al.<sup>36</sup> and Demirag et al.<sup>6</sup> found that the patellar tendon attaches more proximally and in a broader area onto the tibial tuberosity than controls. This could theoretically increase stress on the tuberosity and be linked to the development. However, determining causality is difficult due to its cross-sectional nature and requires further investigation with more prospective and long-term evaluations.

Increased patellar and tuberosity thickness were also identified, although it is more likely these occur secondary to OSD rather than predisposing factors. Mechanical properties of the tendon (strain and stiffness) were also different from controls <sup>13,14</sup>. Kaya et al.<sup>31</sup> found that OSD patellar tendon length and thickness were increased relative to controls but normalized after two years.

#### **When does OSD present during maturation?**

Most patients with OSD appeared to be categorized within the early stages of maturation before the tuberosity has ossified. This could be when adolescents are most susceptible to overuse injuries due to the weak apophyseal cartilage <sup>5,17</sup>, since the cellular layer of the tibial tuberosity is an active zone that changes up until maturation, making it the weakest structure of the tuberosity during growth <sup>5</sup>. Relatively few of the studies included in this review take into account findings of healthy appropriate controls. Still, others have demonstrated that multiple fragmentations of the secondary ossification center merely reflect the normal variation of the developing tibial tuberosity rather than OSD <sup>4,7,8</sup>.

#### **Future directions and clinical implications**

This current review has focused on the imaging findings related to OSD and tissue involvement. Further longitudinal studies should be conducted to show a temporal perspective on the disease progression and further shed light on whether the different tissues involvement represents different grades of severity or disease progression. Although studies demonstrated improvements in OSD on imaging findings, no study reported a complete recovery. Ossicles were documented as sequela associated with pain in young adults with OSD history, and few studies have now demonstrated that a smaller part of OSD patients still suffer from ongoing pain locally at the tibial tuberosity upon maturation or have experienced continued OSD-related symptoms<sup>20,32</sup> and restrictions on activity<sup>46</sup>. Observations of separated ossicles were also described in the follow-ups in adolescents, although it was less clear if these were associated with pain. It is often cited that OSD is self-limiting<sup>53</sup>. However, findings of this review indicate there may be, in some cases, be continued imaging findings associated with OSD in the medium to longer term. Additionally, routine imaging must be considered carefully as some findings may not necessarily be associated with OSD.

Preliminary evidence indicates that pain and imaging may be associated. Saily et al.<sup>47</sup> suggested that increased Doppler signal and maturation status were associated with more severe pain on palpation. Holden et al.<sup>27</sup> found that those with less tissue involvement on baseline US were less likely to report pain at a 24-month follow-up. Similarly, in a retrospective study on immobilization, the presence of ossicles was associated with a significant delay in return to sports<sup>9</sup>. This, taken together with findings that ossicles are related to persistent pain beyond maturation of the tibial tuberosity, indicates that may therefore be a promising way to characterize severity on imaging and those at greater risk of a poor prognosis. Indeed, this is critical to inform the role of imaging in the management of this condition.

Lastly, the studies investigating anatomic and morphometric variations in OSD suggest that these may be associated with OSD, especially how the patellar tendon inserts onto the tibial tuberosity could be considered a predisposing factor as many active kids will never develop OSD. Hence, looking into such factors could potentially advance our knowledge of this common condition and provides a context for understanding standard imaging done in adolescents with OSD.

## Limitations

Although an extensive systematic search was undertaken, the language of this review was limited to include articles in English, German, Dutch, Scandinavian languages, and French only.

Additionally, for the inclusion of OSD, a diagnosis including localized pain at the tibial tuberosity was required. However, this was poorly described, and many studies used imaging to confirm the diagnosis – making it difficult to infer from these.

## Conclusion

This review is the first to systematically show that imaging characteristics of tissue involvement in OSD patients are altered compared with controls. It appears that findings are not only limited to the apophysis or secondary ossification center but extend to include soft tissues such as the tendon and bursa. There is preliminary evidence to indicate that imaging may be used to grade severity and/or those at risk of a worse prognosis. Further research is needed to validate the clinical utility of this. There were inconsistent findings on whether patella height and angle were altered in OSD. In contrast, morphometric features such as alterations in patella tendon attachment and tibial characteristics appeared different compared to controls. Patients with OSD seemed to be categorized as being in the early stages of tibial tuberosity maturation.

## Reference

1. Andrisano, A., Mignani, G., Mazzetti M. Long term results in Osgood-Schlatters Disease. *Ital J Orthop Traumatol.* 1985;11(4):483-486.
2. Aparicio, G. Abril, J. Calvo, E. Alvarez L. Radiologic Study of Patellar Height in Osgood-Schlatter Disease. *J Pediatr Orthop.* 1997;17(1):63-66.
3. Blankstein, A.; Cohen, I.; Heim, M.; Diamant, L.; Salai, M.; Chechick, A.; Ganel A. Ultrasonography as a diagnostic modality in Osgood-Schlatter disease A clinical study and review of the literature. *Arch Orthop Trauma Surg.* 2001;121(9):536.
4. Cohen B, Wilkinson RW. The Osgood-Schlatter lesion. A radiological and histological study. *Am J Surg.* 1958;95(5):731-742. doi:10.1016/0002-9610(58)90622-6
5. Czynny Z. Osgood-Schlatter disease in ultrasound diagnostics--a pictorial essay. *Med*

- Ultrason*. 2010;12(4):323-335.
6. Demirag B, Ozturk C, Yazici Z, Sarisozen B. The pathophysiology of Osgood-Schlatter disease: a magnetic resonance investigation. *J Pediatr Orthop B*. 2004;13(6):379-382. doi:10.1097/01202412-200411000-00006
  7. Ducher G, Cook J, Lammers G, et al. The ultrasound appearance of the patellar tendon attachment to the tibia in young athletes is conditional on gender and pubertal stage. *J Sci Med Sport*. 2010;13(1):20-23. doi:10.1016/j.jsams.2009.03.003
  8. Ducher G, Cook J, Spurrier D, et al. Ultrasound imaging of the patellar tendon attachment to the tibia during puberty: a 12-month follow-up in tennis players. *Scand J Med Sci Sports*. 2010;20(1):e35-40. doi:10.1111/j.1600-0838.2009.00889.x
  9. Duperron L, Haquin A, Berthiller J, Chotel F, Pialat JB, Luciani JF. Étude d'une cohorte de 30 patients immobilisés avec une résine cruro-malléolaire pour une maladie d'Osgood-Schlatter. *Sci Sport*. 2016;31(6):323-335. doi:10.1016/j.scispo.2016.04.014
  10. Ehrenborg, G. Lagergren C. Roentgenologic Changes in the Osgood-Schlatter Lesion. *Acta Chir Scand*. 1961;(121):315-327.
  11. El-Husseini TF, Abdelgawad AA. Results of surgical treatment of unresolved Osgood-Schlatter disease in adults. *J Knee Surg*. 2010;23(2):103-107. doi:10.1055/s-0030-1267474
  12. Engel, A. Windhager R. Der Stellenwert des Ossikels und der Therapie bei M. Osgood-Schlatter. *Sportverletz Sportschaden*. 1987;1(2):100-1.
  13. Enomoto S, Tsushima A, Oda T, Kaga M. The characteristics of the muscle-tendon unit in children affected by Osgood-Schlatter disease. *Transl Sport Med*. 2019;2(4):196-202. doi:10.1002/tsm2.79
  14. Enomoto S, Tsushima A, Oda T, Kaga M. The Passive Mechanical Properties of Muscles and Tendons in Children Affected by Osgood-Schlatter Disease. *J Pediatr Orthop*. 2020;40(4):e243-e247. doi:10.1097/BPO.0000000000001426
  15. Falciglia F, Giordano M, Aulisa AG, Poggiaroni A, Guzzanti V. Osgood Schlatter lesion: histologic features of slipped anterior tibial tubercle. *Int J Immunopathol Pharmacol*. 2011;24(1 Suppl 2):25-28. doi:10.1177/03946320110241s206
  16. De Flaviis, L.; Nessi, R.; Scaglione, P.; Balconi, G.; Albisetti, W.; Derchi LE. Ultrasonic diagnosis of Osgood-Schlatter and Sinding-Larsen-Johansson diseases of the knee. *Skeletal*



- Radiol.* 1989;18(3):193-197.
17. Gholve PA, Scher DM, Khakharia S, Widmann RF GD. Osgood Schlatter syndrome. *Curr Opin Pediatr.* 2007;19(1):44-45. doi:10.1185/030079905X46322
  18. Gigante, Antonio., Bevilacqua Claudio., Bonetti, Massimo & Greco F. Increased external tibial torsion in Osgood-Schlatter disease. *Acta Orthop Scand.* 2003;74(4):431-436. doi:10.1080/00016470310017749
  19. Green DW, Sidharthan S, Schlichte LM, Aitchison AH, Mintz DN. Increased Posterior Tibial Slope in Patients With Osgood-Schlatter Disease: A New Association. *Am J Sports Med.* 2020;48(3):642-646. doi:10.1177/0363546519899894
  20. Gulddammer C, Rathleff MS, Jensen HP, Holden S. Long-term Prognosis and Impact of Osgood-Schlatter Disease 4 Years After Diagnosis: A Retrospective Study. *Orthop J Sport Med.* 2019;7(10):1-6. doi:10.1177/2325967119878136
  21. Guzzanti, V., Pezzoli, F.M., Bergami, G., D'Arienzo M. Ultrasound in young athletes with osteochondrosis at the insertion of the patellar tendon. *J Sport Traumatol.* 1996;18(4).
  22. Haber DB, Tepolt FA, McClincy MP, Hussain ZB, Kalish LA, Kocher MS. Tibial tubercle fractures in children and adolescents: A large retrospective case series. *J Pediatr Orthop Part B.* Published online 2021:13-18. doi:10.1097/BPB.0000000000000756
  23. Hanada M, Takahashi, Matsuyama. Relationship between the clinical findings and radiographic severity in Osgood&ndash;Schlatter disease. *Open Access J Sport Med.* Published online 2012:17. doi:10.2147/oajsm.s29115
  24. Hirano A, Fukubayashi T, Ishii T, et al. Magnetic resonance imaging of Osgood-Schlatter disease: The course of the disease. *Skeletal Radiol.* 2002;31(6):334-342. doi:10.1007/s00256-002-0486-z
  25. Hirano A, Fukubayashi T, Ishii T, Ochiai N. Relationship between the patellar height and the disorder of the knee extensor mechanism in immature athletes. *J Pediatr Orthop.* 2001;21(4):541-544. doi:10.1097/00004694-200107000-00024
  26. Holden S, Rathleff MS. Separating the myths from facts: Time to take another look at osgoodschlatter "disease." *Br J Sports Med.* 2020;54(14):824-825. doi:10.1136/bjsports-2019-101888
  27. Holden Sinead, Olesen, Jens Lykkegaard, Winiarski M, Lukasz, Krommes, Kasper, Thorborg,

- Kristian, Hölmic, Per, Rathleff MS. Is the Prognosis of Osgood Schlatter- poorer than anticipated? A Prospective Cohort study with 24-Month Follow-up. *Orthop J Sport Med.* 2021;19;9(8).
28. Jakob, R. P.; von Gumppenberg, S.; Engelhardt P. Does Osgood-Schlatter disease influence the position of the patella? *Z Orthop Ihre Grenzgeb.* 1981;122(6):798-802. doi:10.1055/s-2008-1045069
29. Kamel SI, Kanesa-Thasan RM, Dave JK, et al. Prevalence of lateral patellofemoral maltracking and associated complications in patients with Osgood Schlatter disease. *Skeletal Radiol.* 2021;50(7):1399-1409. doi:10.1007/s00256-020-03684-6
30. Kaneuchi Y, Otoshi K, Hakozaiki M, et al. Bony Maturity of the Tibial Tuberosity With Regard to Age and Sex and Its Relationship to Pathogenesis of Osgood-Schlatter Disease: An Ultrasonographic Study. *Orthop J Sport Med.* 2018;6(1):1-7. doi:10.1177/2325967117749184
31. Kaya DO, Toprak U, Baltaci G, Yosmaoglu B, Ozer H. Long-term functional and sonographic outcomes in Osgood-Schlatter disease. *Knee Surgery, Sport Traumatol Arthrosc.* 2013;21(5):1131-1139. doi:10.1007/s00167-012-2116-1
32. Krause, B. L.; Williams, J. P.; Catterall A. Natural History of Osgood-Schlatter Disease. *J Pediatr Orthop.* 1990;10(1):65-68.
33. Kujala U, Kvist M, Heinonen O. Osgood-Schlatter ' s disease in adolescent athletes. Retrospective study of incidence and duration. *Am J Sports Med.* 1985;13:239:236-241.
34. Lanning P, Heikkinen E. Ultrasonic features of the osgood-schlatter lesion. *J Prosthetics Orthot.* 1991;11(4):538-540. doi:10.1097/01241398-199107000-00023
35. Lazović, D.; Wegner, U.; Peters, G.; Gossé F. Ultrasound for diagnosis of apophyseal injuries. *Knee Surgery, Sport Traumatol Arthrosc.* 1996;3(4):234-237. doi:10.1007/BF01466625
36. Lee DW, Kim MJ, Kim WJ, Ha JK, Kim JG. Correlation between Magnetic Resonance Imaging Characteristics of the Patellar Tendon and Clinical Scores in Osgood-Schlatter Disease. *Knee Surg Relat Res.* 2016;28(1):62-67. doi:10.5792/ksrr.2016.28.1.62
37. Lohrer H, Nauck T, Schöll J, Zwerver J, Malliaropoulos N. Einsatz der extrakorporalen Stoßwellentherapie bei therapieresistentem M. Schlatter. *Sportverletzung-Sportschaden.*

- 2012;26(4):218-222. doi:10.1055/s-0032-1325478
38. Lyng KD, Rathleff MS, Dean BJF, Kluzek S, Holden S. Current management strategies in Osgood Schlatter: A cross-sectional mixed-method study. *Scand J Med Sci Sport*. 2020;30(10):1985-1991. doi:10.1111/sms.13751
39. Mahlfield, K., Kayser, R., Franke, J., Merk H. Sonographische Diagnostik bei M. Osgood-Schlatter. *Ultraschall der Medizin*. 2001;22(4):182-185.
40. Moher D, Liberati A, Tetzlaff J, Altman DG. Preferred reporting items for systematic reviews and meta-analyses: The PRISMA statement. *BMJ*. 2009;339(7716):332-336. doi:10.1136/bmj.b2535
41. Omodaka, Takuya; Ohsawa, Takashi; Tajika, TsuyoOmodaka, Takuya; Ohsawa, Takashi; Tajika, Tsuyoshi; Shiozawa, Hiroyuki; Hashimoto, SyoOmodaka, Takuya; Ohsawa, Takashi; Tajika, Tsuyoshi; Shiozawa, Hiroyuki; Hashimoto, Syogo; Ohmae, Hiroaki; Shitara, Hitoshi H. Relationship Between Lower Limb Tightness and Practice Time Among Adolescent Baseball Players With Symptomatic Osgood-Schlatter Disease. *Orthop J Sport Med*. 2019;7(5):PAG-N.PAG. doi:10.1177/2325967119847978
42. Pihlajamäki HK, Mattila VM, Parviainen M, Kiuru MJ, Visuri TI. Long-term outcome after surgical treatment of unresolved Osgood-Schlatter disease in young men. *J Bone Jt Surg - Ser A*. 2009;91(10):2350-2358. doi:10.2106/JBJS.H.01796
43. Pihlajamäki HK, Visuri TI. Long-term outcome after surgical treatment of unresolved osgood-schlatter disease in young men: surgical technique. *J Bone Joint Surg Am*. 2010;92 Suppl 1:258-264. doi:10.2106/JBJS.J.00450
44. Rejeb A, Johnson A, Vaeyens R, Horobeanu C, Farooq A, Witvrouw E. Compelling overuse injury incidence in youth multisport athletes. *Eur J Sport Sci*. 2017;17(4):495-502. doi:10.1080/17461391.2016.1275820
45. Rosenberg ZS, Kawelblum M, Cheung YY, Beltran J, Lehman WB, Grant AD. Osgood-Schlatter lesion: Fracture or tendinitis? Scintigraphic, CT, and MR imaging features. *Radiology*. 1992;185(3):853-858. doi:10.1148/radiology.185.3.1438775
46. Ross MD, Villard D. Disability Levels of College-Aged Men with a History of Osgood-Schlatter Disease. *J Strength Cond Res*. 2003;17(4):659-663. doi:10.1519/1533-4287(2003)017<0659:DLOCMW>2.0.CO;2

47. Saily M, Whiteley R, Johnson A. 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):93-97. doi:10.1136/bjsports-2012-091471
48. Scotti DM, Sadhu VK, Heimberg F, Edward O'hara A. Osgood-Schlatter's disease, an emphasis on soft tissue changes in roentgen diagnosis. *Skeletal Radiol.* 1979;4(1):21-25. doi:10.1007/BF00350589
49. Sen RK, Sharma LR, Thakur SR, Lakhanpal VP. Patellar angle in osgood-schlatter disease. *Acta Orthop.* 1989;60(1):26-27. doi:10.3109/17453678909150085
50. Seyfettinoğlu F, Oğur HU, Tuhanoğlu Ü, Acar B, Köse Ö, Çiçek H. Is There a Relationship between Patellofemoral Alignment and Osgood-Schlatter Disease? A Case-Control Study. *J Knee Surg.* 2020;33(1):67-72. doi:10.1055/s-0038-1676523
51. Sheppard ED, Ramamurti P, Stake S, et al. Posterior Tibial Slope is Increased in Patients with Tibial Tubercle Fractures and Osgood-Schlatter Disease. *J Pediatr Orthop.* 2021;41(6):e411-e416. doi:10.1097/BPO.0000000000001818
52. T, Visuri; H.K, Pihlajamäki; V.M, Mattila; M K. Elongated patellae at the final stage of Osgood-Schlatter disease: a radiographic study. *Knee.* 2007;14(3):198-203. doi:10.1016/j.knee.2007.03.003
53. Wall EJ. Osgood-Schlatter Disease Practical Treatment for a Self-Limiting Condition. *Phys Sportsmed.* 1998;26(3):29-34. doi:10.1080/00913847.1998.11440345

## Tables

**Table 1. Baseline imaging findings**

Study (year)	Modality	Scoring	Cartilage changes	Bony characteristics		Tendon changes	Other soft tissue changes
				System	Soft tissue swelling		
Blankstein (2001)	US	DeFlaviis	✓	✓ n=5 'type 3' ✓ n=3 'type 4'	✓	✓ Diffuse PT thickening	✓ Fluid (infrapatellar bursitis)
Cohen (1958)	Radiography	None	✓ n=7	✓ n=3	✓ n=1	NR	✓
DeFlaviis (1989)	Radiography US	DeFlaviis	✓	NR	✓	✓ 'Associated tendinitis' (diffuse thickening)	✓ 'Associated infrapatellar bursitis'
Duperron (2016)	Radiography	3 stages	✓	✓ n=14	✓	NR	NR
Ehrenborg and Lagergren (1961)	Radiography	None	✓ varying degrees	✓ detached calcified fragments	✓	NR	NR
El-Husseini (2010)	Radiography	None	NR	✓ n=35 (100%)	NR	✓	NR
Engel (1987)	Radiography	None	✓ n=28	✓ n=6	✓ n=7	NR	NR
Enomoto (2020)	US	None	✓	✓ free bone fragments	NR	NR	NR
Guzzanti (1996)	Radiography US	None	NR	NR	✓ Irregularity (75% of cases)	✓ Widespread thickening and tendinitis (22% of cases)	✓ Infrapatellar bursitis (11 % of cases)
Hanada (2012)	Radiography	3 stages	NR	✓	✓ Radiolucency of TT, Fragmentation	NR	NR
Hirano (2002)	Radiography MRI	5 stages	NR	✓	✓ T1: (low signal on secondary OC) / T2: (high signal on secondary OC)	✓ PT swelling and thickening at insertion site on TT	NR
Holden (2021)	US	DeFlaviis	✓	NR	✓ + Doppler signal on TT	✓ 'Associated tendinitis' ✓ + Doppler signal on tendon	✓ 'Associated infrapatellar bursitis'
Kamel (2021)	MR Imaging	None	NR	✓ n=115 (67%) single ossicle ✓ n=35 (21%) multiple ossicles	✓ n=21 (12%) had fused fragments to the tibial tuberosity	✓ PT thickening with edema (n=81, 47%)	✓ Deep infrapatellar bursitis (n=64, 37%) and sup. infrapatellar bursitis (n=21, 12%)
Kaya (2013)	US	DeFlaviis	✓	NR	✓	✓ 'Associated tendinitis' ✓ PT lengthening	✓ 'Associated infrapatellar bursitis'
Krause (1990)	Radiography	None	✓ 44 knees	✓ 28 knees	✓ Fragmentation of the epiphysis (44 knees), abnormal shaped TT (20 knees)	NR	NR
Lanning (1991)	US	None	✓	NR	✓ Shell-like elevated fragment (4 knees) and	✓ Distal PT thickening	NR

					multiple fragments at TT (12 knees)		
<b>Lazović (1996)</b>	Radiography US	4 stages	NR	NR	✓Detection of bony changes on TT (n=38)	NR	NR
<b>Lee (2016)</b>	Radiography MRI	None	NR	✓	✓bone edema (n=6)	✓Tendinopathy (n=7)	✓Peritendinous edema (n=14)
<b>Mahlfeld (2001)</b>	Radiography US	3 stages	NR	NR	✓Fragmentation of the apophyseal (n=9)	✓Tendon widening >1mm (n=11), tendon widening <1mm (n=4)	✓bursitis (n=8)
<b>Pihlajamäki (2009)</b>	Radiography	None	NR	✓109 knees (93%)	NR	NR	NR
<b>Rosenberg (1992)</b>	CT, MRI, Radiograph, Scintigraphy	None	✓increase in radio trace flow (n=1)	✓Bony fragments anterior to TT (32%)	✓Change in bone marrow signal (marrow edema, 9 knees, MRI)	✓Tendon enlargement (CT), increased signal intensity on T1, T2 (MRI)	✓Deep bursa: 45% (CT), 71% (MRI). Sup. bursa: 53% (MRI)
<b>Sailly (2013)</b>	US	None	NR	✓	NR	✓(+) Doppler signal: (n=10, 50%), (-) Doppler signal: (n=10, 50%)	✓Bursitis
<b>Scotti (1979)</b>	Radiography	None	✓Varying degrees (100%)	NR	✓multiple bony masses	✓PT thickening, varying degrees (100%)	✓irregularity of soft tissue planes and increased thickness
<b>Seyfettinoğlu (2020)</b>	Radiography	None	✓	NR	✓	✓	NR
<b>Visuri (2007)</b>	Radiography MR Imaging	None	NR	✓76 cases	✓Distinct TT prominence (6 cases)	NR	NR

Table 1 presents the baseline imaging findings with alterations in the cartilage, bone, tendon, and soft tissues as observed by the different imaging modalities. The denotation: ✓ indicates if the imaging characteristics are present. Abbreviations: NR: not reported, Sup. bursa: superficial bursa, OC: ossification center, TT: tibial tuberosity. The findings were either rated by a scoring system or descriptive.

***Table 2. Patellofemoral characteristics and morphometric features.***



Study	Modality	Outcome	Method	OSD	Controls
<b>Patellar height</b>					
Andrisano (1985)	Radiograph	Patella height	BP method	Alta: 38% Baja: 0%	NA
Aparicio (1997)	Radiograph	Patella height	CD index	CD index: 1.22 Alta: 58% OSD	CD index: 1.07* Alta: 17% CON
Jakob (1981)	Radiograph	Patella height	BP method	Mean: 0.99 ± 0.16	mean 0.84 ± 0.11*
Lee (2016)	MRI	Patella height	IS method	IS index: 0.97 ± 0.2	IS index: 0.96 ± 0.1 (ns)
Pihlajamäki (2009)	Radiograph	Patella height	BP method, IS method	Mean IS index, 1.0 Alta: 3% (IS), 9% (BP) Baja: 3% (IS), 1% (BP) IS pre-and post-surgery: 1.0 vs. 1.09, P=0.003	NA
Seyfettinoğlu (2020)	Radiograph	Patella height	IS method, CD index, BP method	IS index: 0.99 ± 0.13 CD Index: 0.94 ± 0.21 BP Index: 0.76 ± 0.10 Alta: 5% (IS), 7.5% (CD), 2.5% (BP) Baja: 5% (IS), 0% (CD), 0% (BP)	IS index: 0.97 ± 0.10 CD Index: 0.94 ± 0.16 BP Index: 0.76 ± 0.09 Alta: 0% (IS) 5% (CD), 0% (BP) Baja: 5% (IS), 5% (CD), 2.5% (BP)
Visuri (2007)	Radiograph	Patella height	IS method, BP method, CD index	IS: (0.99 ± 0.12) BP: (0.94 ± 0.14) CD: (1.00 ± 0.15)	IS: (1.03 ± 0.12) BP: (0.86 ± 0.11) CD: (0.88 ± 0.12)
Sheppard 2021	Radiograph	Patella height	CD index	OSD: 1.23 (0.17)	Tibia tubercle fractures: 1.28 (0.24) Other knee pain: 1.20 (0.18) (ns)
Kamel 2021	MRI	Patella height	IS index (>1.3 = patella alta)	53% (91/171 knees) had patella alta	NR
<b>Patella morphology and shape</b>					
Seyfettinoğlu (2020)	Radiograph	Patella morphology	Grelsamer's morphology classification Type I normal: ratio ≥1.2≤1.5 Type II elongated: ratio >1.5 Type III shortened: ratio <1.2	Type 1 normal: n=11 (27.5%) Type 2 elongated: n=22 (55%) Type 3 shortened: n=7 (17.5%)	Type I normal: n=17(42.5%) Type II elongated: n=12 (30%) Type III shortened: n=10 (25%)
Visuri (2007)	Radiograph	Patella morphology	Grelsamer's morphology classification  Type I normal: ratio ≥1.2≤1.5 Type II elongated: ratio >1.5 Type III shortened: ratio <1.2	Patellar morphology ratio: (1.44 ± 0.11).  Type I normal: n=59 (72%) Type II elongated: n=22 (22%) Type III shortened: n=1 (1%)	Patellar morphology ratio: 1.28 ± 0.07***  Type I normal: n=79 (91%) Type II elongated: n= 1 (1%)* Type III: n=7 (8%)
Sen (1989)	Radiograph	Patella angle	Patella angle (degrees)	33 degrees. M: (n=21), 34 (1.17 SEM). F: (n=5) 42 (3.10 SEM)	47 degrees***. M: (n=44) 46 (1.03 SEM). F: (n=27) 49 (1.23 SEM).
Gigante 2003	CT	Patella tilt angle	Not described	Patellar tilt angle: 21 (8)°	Patellar tilt angle: 17 (8)°
<b>Patellofemoral alignment</b>					
Kamel 2021	MRI	Lateral patellar tracking	Maltracking was defined as the presence of edema in superolateral Hoffa's fat pad. Maltracking was also defined as a TT-TG distance >20 mm	Maltracking prevalence: 59%  Active maltracking was associated with an increased	Maltracking prevalence: 15%***

			with the presence of either lateral patellar tilt or lateral translation of the patella	likelihood of active OSD [p < 0.001, OR 8.6, 95% CI (2.9–25.2)].	
<b>Gigante 2003</b>	CT	TT-TG Distance	TT-TG distance (mm)	TT-TG distance (mm): 14 (4)	TT-TG distance (mm): 12 (5)
<b>Sheppard 2021</b>	Radiographs	Anatomic lateral distal femoral angle	Defined by the angle between the femoral anatomic axis and the articular surface of the distal femur	OSD: 82.23 (1.7)	Tibia tubercle fractures: 82.18 (2.24) Other knee pain: 81.89 (2.09) (ns)
<b>Seyfettinoğlu (2020)</b>	Radiography	Patellofemoral alignment	Congruence angle, sulcus angle, Q angle	Congruence angle: OSD: -1.8±4.7; Sulcus angle: OSD: 135.8±4.6; Q angle: 15.6±2.2	Congruence angle: -1.9±4.7; Sulcus angle: 136.1±4.2; Q angle: 14.3±2.5*
<b>Gigante 2003</b>	CT	Patella congruence angle	Congruence angle (not described)	Congruence angle: 6 (15)°;	Congruence angle: 10 (26)°;
<b>Andrisano (1985)</b>	Radiography	Patellofemoral joint abnormalities	Trochlear angle (angle between lateral and medial articular facets), any hypoplasia of the lateral femoral condyle, malalignment of the femoropatellar joint.	Femoro-patellar dysplasia in 57% of cases. The trochlear angle was above normal limits in 33%, in 57% there were anomalies in the relative length of the articular facets, and in 29%, the lateral femoral condyle was hypoplastic.	NA

*Table 2 shows the patellofemoral characteristics and morphometric features. Unless otherwise stated, data are reported as mean ± standard deviation (SD) in the table. \* indicates significantly different from OSD at the p<0.05 level; \*\*\*indicates significantly different from OSD at the p<0.001 level, (ns) indicated not significantly different from OSD. Abbreviations: BP: Blackburn and Peel method, CD: The Caton-Deschamps index, IS: Insall-Salvati method, NA: not applicable, M: male, F: female, TT-TG distance: tibial-tuberosity to trochlear groove distance*

**Table 3. Patellar tendon, attachment & tibial characteristics and morphometric features**

Study	Modality	Outcome	Method	OSD	Control
<b>Patellar tendon size and attachment</b>					
Demirag (2004)	MRI	Patella tendon attachment	(1) (A:B) ratio of distance between the distal pole of the patella and the prox. margin of the PT attachment, to the distance between the distal pole of the patella and the TT epiphysis. (2) (C:D): ratio of distance between the prox. margin of the PT attachment point to the tibia and the TT epiphysis to the distance between the knee joint level and the TT epiphysis.	Ratio A:B=0.54 ± 0.09 Ratio C:D=0.63 ± 0.14	Ratio A:B= 0.770 ± 0.099** Ratio C:D=0.306 ± 0.074**
Kaya (2013)	US	Tendon length, thickness and area	Length, proximal and distal thickness and area	Length: 4.8 ± 0.7 Proximal diameter: 0.4 ± 0.8 Distal diameter: 0.4 ± 0.8 Proximal area: 1.1 ± 0.2 Medial area: 1.0 ± 0.2 Distal area: 1.2 ± 0.1	Length: 4.8 ± 0.7 (ns) Proximal diameter: 0.4 ± 0.1 (ns) Distal diameter: 0.7 ± 1.0 (ns) Proximal area: 1.1 ± 0.2 (ns) Medial area: 1.0 ± 0.2 (ns) Distal area: 1.2 ± 0.1 (ns)
Lee (2016)	MRI	Free patellar tendon proportion (FPFP), thickness, Tibial attachment portion (TAP),	(FPFP =(1)/(2): (1) the distance between the inf. patella and the most prox. attachment site of the PT onto the TT or ossicles (2) the distance between the inf. patella and the most distal attachment site of the PT onto the TT. (TAP =(3)/(4): the distance between the most prox. attachment of the PT onto the TT or the ossicle and the most distal region of the TT and (4) the distance between the tibial articular surface and the most distal region of the TT	FPTP: (1)/(2): 0.55 ± 0.09 TAP: (3)/(4): 0.67 ± 0.08 PT thickness on tibial attachment (mm): 7.2 ± 0.7	FPTP: (1)/(2): 0.77 ± 0.11*** TAP: (3)/(4): 0.29 ± 0.07*** PT thickness on tibial attachment (mm): 4. ± 0.2
<b>Mechanical properties of the tendon</b>					
Enomoto (2019)	US	Patellar tendon strain	Elongation was expressed as strain: Strain (%) = L · TL-1*100; L is the elongation of the tendon structure during ramp contraction and TL is the length of the tendon structure at rest.	Maximum strain (P = 0.0003, ES = 0.49, 95% CI: 0.83 to 2.68) were significantly lower in the OSD group than in the CON group.	
Enomoto (2019)	US	Patellar tendon stiffness	Stiffness of the tendon structure was calculated as the load of the force-elongation relationship within the range of 247 N-392 N, which corresponds to a tensile force of 70%-100% MVC in the subject whose MVC was the lowest.	Tendon stiffness was significantly higher in the OSD group than in the CON group (P = 0.0008, ES = 0.48, 95% CI: -302.88 to -88.59).	
Enomoto	US	Patellar	Strain ratio: tendon strain/ reference material strain	Strain ratio of the patellar tendon in the OSD group was	

(2020)		tendon stiffness/elasticity		significantly lower than the CON group (P < 0.05; ES = 0.31; 95% CI, 0.04-0.39).	
<b>Tibial characteristics</b>					
Green 2020	Radiography	Posterior tibial slope	First, the proximal anatomic axis of the tibia was drawn by connecting the midcortical diameters of the tibia 5 and 15 cm distal to the knee joint line. Next, a reference line perpendicular to this axis was drawn at the level of the tibiofemoral joint. Tibial slope is defined as the angle between the reference line and a line drawn tangent to the uppermost anterior and posterior edges of the medial tibial plateau.	OSD: 12.2 ± 3.6 Male OSD: 12.8 ± 3.6 Female OSD: 11.7 ± 3.6	Controls: 8.8 ± 2.8*** 8.0 ± 3.0*** 9.6 ± 2.4*
Sheppard 2021	Radiography	Posterior tibial slope	Defined as 90 degrees minus the angle between a line drawn tangentially to the medial tibial plateau and the longitudinal axis of the tibia	OSD: 9.66 (3.11)  Linear regression was used to correct for multiple demographic variables & posterior slope was still increased in the OSD group compared with the control group ( $\beta=3.14$ , $P<0.001$ ).	Tibial tubercle fractures: 10.48 (4.21) Other knee pain: 6.62 (3.05)***
Sheppard 2021	Radiography	Anatomic medial proximal tibial angle	Defined by the angle between the tibial anatomic axis and the articular surface of the proximal tibia	OSD: 86.15 (2.11)  Remained significantly different from control after correction for multiple demographic variables	Tibial tubercle fractures: 86.32 (2.1) Other knee pain: 87.12 (2.06)**
Gigante 2003	CT	Tibial torsion angle	external tibial torsion angle, on superimposed sections D and F	Tibial torsion angle: 28 (8) <sup>o</sup> .	Tibial torsion angle: 12 (8)****
<b>Tuberosity size/prominence</b>					
Pihlajamäki (2009)	Radiography	Tibial tuberosity prominence	Ratio of the thickness of the tuberosity to the distance between the top of the tuberosity and the middle vertical line of the tibia	Tuberosity thickness decreased by 47% (range, 17% to 65%) after surgery	NA
Visuri (2007)	Radiography	Tibial tuberosity prominence	Tibial tuberosity index: the ratio of the height of the tuberosity and the distance between the top of the tuberosity to the middle vertical line of the tibia.	0.28 (range 0.15–0.36)	0.07 (range 0.00-0.18)***

Table 3 presents the findings of the Patellar tendon, attachment and tibial characteristics, and morphometric features. Unless otherwise stated, data are reported as mean ± standard deviation (SD) in the table. \* indicates significant difference from OSD at  $p<0.05$ ; \*\* indicates significant difference from OSD at  $p<0.01$ ; \*\*\* indicates significant difference from OSD at  $p<0.001$ ; (ns) indicated not significantly different from OSD. Abbreviations: PT: patellar tendon, TT: tibial tuberosity, prox.: proximal, inf.: inferior,

**Table 4. Tibial tuberosity maturation.**

Study (year)	Modality	Classification system		Type/stage	Descriptive findings	Controls	OSD pain
		Scoring system	Outcome	Affected knee (n, %)			
Cohen (1958)	Radiography	Descriptive	Descriptive	Descriptive	Multiple ossification centers were shown in 8.3% of cases indicating normal variant of ossification of the TT	Asymp. knee	✓
Ehrenborg and Lagergren (1961)	Radiography	Ehrenborg	<b>Stage A: The cartilaginous stage</b> , before ossification centers are discernible. <b>Stage B: The apophyseal stage</b> , in which ossification centers appear in the tongue of cartilage. <b>Stage C: The epiphyseal stage</b> , in which the centers have concealed and fused with the tibial epiphysis. <b>Stage D: The bony stage</b> , when the epiphyseal line has closed	Stage A (1 case) Stage B (14 cases, 5 bilateral) Stage C (22 cases, 3 bilateral) Stage D -	NR	None	NR
Engel (1987)	Radiography	Ehrenborg	Stages of TT development	Stage A: (n=0, 0%) Stage B: (n=8, 17%) Stage C: (n=31, 66%) Stage D: (n=8, 17%)	Apophyseal surface: smooth (n=17), irregular (n=24), fluffy (n=1), raised (n=2) dent formation (n=3)	None	NR
Falciglia (2011)	Radiography MR Imaging	Ehrenborg	Stages of TT development	Apophyseal stage: (n=4, 30.7%) Epiphyseal stage: (n=9, 69.3%)	NR	None	NR
Hanada (2012)	Radiography	Ehrenborg	Stages of TT development	Stage A: (0 knees) Stage B: (36 knees) Stage C: (51 knees) Stage D: (7 knees)	NR	None	✓
Holden (2020)	Ultrasonography	Nakase (modified)	<b>Stage 1: Sonolucent stage</b> <b>Stage 2: Individual stage</b> <b>Stage 3: Connective stage</b> <b>Stage 4 Fully Mature</b>	Stage 1: (n=10, 20.8%) Stage 2: (n=7, 14.6%) Stage 3: (n=20, 41.7%) Stage 4: (n=11, 22.9%)	NR	Asymp. Knee	✓
Kaneuchi (2018)	Ultrasonography	Ehrenborg (modified)	<b>Stage C:</b> the cartilaginous stage. <b>Stage A:</b> the apophyseal stage. <b>Stage E:</b> the epiphyseal stage. <b>Stage B:</b> the bony stage	Stage C: (F: n=72, M: n=234) Stage A: (F: n=79, M: n=163) Stage E: (F: n=398, M: n=283)	59.2% girls vs. 8.0% boys (10 yrs.) were stage E 98.8% of girls were stage E/B vs. 40.6% boys (12yrs.) were stage C/A. 47.4% girls vs. 13.8% boys	None	✓

				Stage B: (F: n=70, M: n=20)	(14yrs.) were skeletally mature (stage B) OSD prevalence: around 12 yrs. (boys: stage C/ girls: stage A). There was a significantly high risk of being diagnosed with OSD from stage C-A (OR, 9.48; 95% CI, 2.54-61.48) and from stage A-E (OR, 2.22; 95% CI, 1.19-4.45), but no significant increase in stage E-B (OR, 1.02; 95% CI, 0.44-2.12)		
Lohrer (2012)	Radiography	Ehrenborg	Stages of TT development	Stage A: (n=0) Stage B: (n=3) Stage C: (n=8) Stage D: (n=2)	NR	None	✓
Omodaka (2019)	Ultrasonography	Ehrenborg (modified)	<b>Stage C:</b> the cartilaginous stage. <b>Stage A:</b> the apophyseal stage. <b>Stage E:</b> the epiphyseal stage. <b>Stage B:</b> the bony stage	Stage C: (0 knees) Stage A: (1 knee) Stage E: (10 knees) Stage B: (2 knees)	NR	Asymp. Knee	✓
Sailly (2013)	Ultrasonography	Four point scale	<b>Stage 1:</b> cartilage attachment without ossicles. <b>Stage 2:</b> cartilage attachment with ossicles. <b>Stage 3:</b> insertional cartilage. <b>Stage 4:</b> mature attachment	Stage 1: (n=0, 0%) Stage 2: (n=10, 50%) Stage 3: (n=8, 40%) Stage 4: (n=2, 10%)	9/10 (90%) with positive Doppler were stage 2, and 1/10 (10%) was stage 3. 1/10 (10%) with negative Doppler was stage 2, 7/10 (70%) were stage 3, and 2/10 (20%) were stage 4	Healthy	✓

Table 4 shows the tibial tuberosity maturation in patients with Osgood-Schlatter. The imaging modality, classification, type/stage, and findings are presented in the table for each study as well as whether controls were included or not and if pain was reported in the study. Abbreviations: Asymp.: asymptomatic, TT: tibial tuberosity, ant.: anterior, NR: not reported, yrs.: years