Background: Osgood-Schlatter disease (OSD) is characterized by traction apophysitis of the tibial tuberosity. Few studies on symptomatic and asymptomatic OSD have correlated findings with clinical examination.

Purpose: To investigate the relationship between lower limb tightness and practice time among adolescent baseball players with symptomatic OSD.

Study Design: Cross-sectional study; Level of evidence, 3.

Methods: The study participants were 402 male baseball players (N = 804 knees) with a mean ± SD age of 10.9 ± 1.5 years (range, 7-14 years). Participant age, height, body weight, body mass index, practice time per week, range of motion of the hip and ankle joints, heel-buttock distance (HBD), and straight-legged raise angle were evaluated. Tibial tuberosity development, as assessed with ultrasonography, was classified into 4 stages: cartilaginous, apophyseal, epiphyseal, and bony. Knees that were classified as apophyseal or epiphyseal were investigated. Participants were divided into 3 groups: non-OSD, asymptomatic OSD, and symptomatic OSD.

Results: Of 400 knees in the apophyseal and epiphyseal stages, 23 knees had asymptomatic OSD, and 11 had symptomatic OSD. Players with symptomatic OSD practiced for significantly longer than the non-OSD group (P = .001) and asymptomatic OSD group (P = .001). Players with symptomatic OSD also had a larger HBD (P = .006) and smaller range of motion regarding internal rotation of hip (P = .023) and dorsiflexion of the ankle (P = .013) than the non-OSD group.

Conclusion: Players with symptomatic OSD had longer practice times than the non-OSD and asymptomatic OSD players. Symptomatic OSD was also associated with tightness of the lower limbs as assessed by the HBD and range of motion in the hip and ankle joints.

Keywords: ultrasonography; tibial tuberosity development; overuse; adolescent baseball players
reports that tightness of the hamstring and triceps surae muscle is associated with the onset of OSD, although the details are unclear. Irregular imaging findings of the tibial tuberosity have also been suggested as a preliminary step of OSD, but few studies have examined the relationship between such irregular findings and symptomatic OSD.

The purpose of this study was to investigate the relationship between lower limb tightness and practice time among adolescent baseball players with symptomatic OSD.

METHODS

Baseball teams registered with the Junior Baseball Federation of Gunma Prefecture in Japan participated in this study. A total of 419 male baseball players initially participated, and the 402 players (N = 804 knees) who were able to undergo US evaluations were included (mean ± SD age, 10.9 ± 1.5 years; range, 7-14 years). This study was reviewed and approved by our university's ethics committee.

Ultrasound Evaluation

The skeletal maturation of the distal attachment of the patellar tendon was examined with a LOGIQ e instrument (GE Healthcare) with high-resolution linear-array probes at 12 MHz. The US evaluations were performed with both knees in 90° of flexion in the supine position. Two orthopaedic surgeons (T. Omodaka, T. Ohsawa) performed US and the evaluations. The tibial tuberosity was examined on longitudinal US images at the site at which the tuberosity was most clearly visualized. Tibial tuberosity development as seen on US was divided into the 4 stages mentioned by Ehrenborg,®7,13,26: cartilaginous (stage C), apophyseal (stage A), epiphyseal (stage E), and bony (stage B). Stage C is characterized by the presence of a large amount of apophyseal cartilage. Stage A is characterized by the patellar tendon attaching to apophyseal cartilage, and the secondary ossification center is in the apophysis. Stage E is characterized by the connection of the secondary ossification and bony epiphysis. Stage B is completely ossified, and the patellar tendon is attached to a rough tibial tuberosity (Figure 1). Knees that were classified as stage A or E were investigated.

OSD Diagnosis

We defined patients as being symptomatic when they reported pressure on the tibial tuberosities as painful. An OSD diagnosis was made per the findings of fragmentation of the bone and irregularity of the ossification center on US (Figure 2). Patients were divided into 3 groups: non-OSD (no pain, no fragmentation on US), asymptomatic OSD (no pain, but fragmentation seen on US), and symptomatic OSD (pain, fragmentation on US).

Evaluation Items

We also recorded the age, height, body weight, body mass index (BMI), and weekly practice duration of the team to which the athlete belonged. The examination was

Figure 1. Ultrasound images of knees that are representative of the 4 stages of the maturation process of patellar tendon attachment. (A) The cartilaginous stage is characterized by a large amount of apophyseal cartilage. (B) The apophyseal stage is characterized by apophyseal cartilage. (C) The epiphyseal stage is characterized by no detectable signs of apophyseal cartilage. (D) The bony stage shows mature attachment.

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The authors declared that there are no conflicts of interest in the authorship and publication of this contribution. AOSSM checks author disclosures against the Open Payments Database (OPD). AOSSM has not conducted an independent investigation on the OPD and disclaims any liability or responsibility relating thereto.

Ethical approval for this study was obtained from the Gunma University Graduate School of Medicine.
performed by 2 orthopaedic surgeons (T. Omodaka, T. Ohsawa), with 1 assistant helping each. The heel-buttock distance (HBD) was measured in the prone position, and the straight-legged raise angle was measured in the supine position and knee extension position. The range of motion was stabilized such that when one of the examiners felt resistance, the second examiner measured the HBD at the distance between the rib and buttocks. The straight-legged raise angle, defined as the angle between the floor and the thigh was measured with a goniometer and a ruler. Hip flexion was measured in the supine position with knee flexion at 90°, and internal and external rotation was measured in the prone position. One examiner stabilized the hip and then passively rotated it into flexion, internal rotation, and external rotation until an end feel was detected. The second examiner aligned the movable arm of the goniometer along the tibial border and recorded the angle in degrees. Plantar flexion and dorsiflexion of the ankle joint were measured in the supine position, while the hip joint and knee flexion were both at 90°. Plantar flexion and dorsiflexion were passively applied to the ankle joint until one examiner detected a final feel. The second examiner aligned the movable arm of the goniometer along the border of the ribs and the border of the fifth metatarsal and recorded the angle. The same examiner recorded all of the measurements of lower limb muscle tightness.

Statistical Analysis

Analysis of variance was used for comparisons among the 3 groups, and the Tukey-Kramer method was used as a post hoc test. All statistical procedures were conducted with the SPSS (v 25; IBM Japan). P values <.05 were considered statistically significant.

RESULTS

Tibial tuberosities were classified as stages C, A, E, and B for 381, 195, 205, and 23 knees, respectively. Among those

with stages A and E, asymptomatic OSD was observed among 23 knees (17 players), while symptomatic OSD was observed among 11 knees (10 players) (Table 1). Height (P = .005), body weight (P < .001), BMI (P < .001), and weekly practice duration (P = .001) were significantly higher in the symptomatic versus the asymptomatic OSD group (Table 2). HBD (P = .002), internal rotation of the hip joint (P = .024), and dorsiflexion of the ankle joint (P = .006) were significantly less in the symptomatic versus the asymptomatic OSD group (Table 3). In the symptomatic OSD group, the total weekly practice duration was 23.0 ± 9.0 hours, which was significantly greater than in the non-OSD group (16.5 ± 5.7 hours, P = .001) and asymptomatic OSD group (15.3 ± 3.6 hours, P = .001). The HBD of the symptomatic OSD group was 10.5 ± 4.8 cm, while that of the non-OSD group was 6.0 ± 4.9 cm, which was significantly lower (P = .006). In the symptomatic OSD group, the hip internal rotation was 37.4° ± 8.6° and the ankle dorsiflexion was 15.1° ± 6.1°; in the non-OSD group, the hip internal rotation was 47.0° ± 12.1° and the ankle dorsiflexion was 21.4° ± 7.1°, which was significantly higher (Figure 3).

DISCUSSION

In this study, the patients with symptomatic OSD had significantly greater height, weight, BMI, and weekly practice

### Table 1

| Stage | Non-OSD (366 knees) | aOSD (23 knees) | sOSD (11 knees) | Total | Age, y |
|-------|---------------------|----------------|----------------|-------|--------|
| C     | 381                 | 0              | 0              | 381   | 10.0 ± 1.3 |
| A     | 179                 | 15             | 1              | 195   | 11.0 ± 0.9  |
| E     | 187                 | 8              | 10             | 205   | 12.2 ± 1.1  |
| B     | 20                  | 1              | 2              | 23    | 13.5 ± 0.7  |

Values are presented as No. or as mean ± SD. A, apophyseal; aOSD, asymptomatic Osgood-Schlatter disease; B, bony; C, cartilaginous; E, epiphyseal; OSD, Osgood-Schlatter disease; sOSD, symptomatic Osgood-Schlatter disease.

### Table 2

| Parameter                  | Non-OSD (366 knees) | aOSD (23 knees) | sOSD (11 knees) | P     |
|----------------------------|---------------------|----------------|----------------|-------|
| Age, y                     | 11.6 ± 1.2          | 11.8 ± 1.3     | 12.3 ± 1.0     | .163  |
| Height, cm                 | 150.9 ± 5.8         | 150.7 ± 11.8   | 157.2 ± 5.4    | .005  |
| Body weight, kg            | 44.1 ± 9.3          | 43.8 ± 10.8    | 52.0 ± 4.4     | <.001 |
| BMI, kg/m²                 | 19.2 ± 2.9          | 19.1 ± 2.6     | 21.0 ± 0.9     | <.001 |
| Practice time, h/wk        | 16.5 ± 5.7          | 15.3 ± 3.6     | 23.0 ± 9.0     | .001  |

Values are presented as mean ± SD unless noted otherwise. aOSD, asymptomatic Osgood-Schlatter disease; BMI, body mass index; OSD, Osgood-Schlatter disease; sOSD, symptomatic Osgood-Schlatter disease.

bP < .01, aOSD vs sOSD.
duration than those without OSD and those with asymptomatic OSD. In addition, the symptomatic OSD group had significantly restricted HBD and range of hip and ankle joint motion when compared with the non-OSD group.

A study by de Lucena et al\(^5\) reported that the incidence of OSD was 9.8\% with sport activities. Nakase et al\(^18\) reported that the OSD incidence was 12.9\% in a football study. In the present study, the incidence of OSD was 2.9\%. The reason for the lower prevalence of OSD in the present study may be that this study included a relatively young cohort and was conducted among baseball players—athletes engaged in a sport with relatively little jumping and cutting action.

In our study, the tightness of the quadriceps femoris, triceps truncus, and hip were significantly higher in the symptomatic OSD group than in the non-OSD group. Whether this was a secondary change could not be determined. The study by de Lucena et al\(^5\) reported that the regular practice of sports during the pubertal phase and the shortening of the rectus femoris muscle were the main factors associated with OSD among students. Nakase et al\(^18\) reported that quadriceps femoris muscle tightness and muscle strength during knee extension and flexibility of the hamstring muscles were increased in OSD.

### TABLE 3
One-way Analysis of Variance Results for Lower Leg Tightness

| Parameter      | Non-OSD (366 knees) | aOSD (23 knees) | sOSD (11 knees) | P   |
|----------------|---------------------|----------------|----------------|-----|
| Knees, n       | 366                 | 23             | 11             | .156|
| SLRA, deg      | 70.7 ± 9.9          | 73.6 ± 6.9     | 67.0 ± 8.9     | .149|
| HBD, cm        | 6.0 ± 4.9           | 7.9 ± 4.1      | 10.5 ± 4.8     | .002b|
| Hip, deg       |                     |                |                |     |
| Flexion        | 122.9 ± 10.8        | 119.2 ± 16.6   | 118.8 ± 13.9   | .149|
| IR             | 47.0 ± 12.1         | 44.9 ± 8.7     | 37.4 ± 8.6     | .024c|
| ER             | 51.9 ± 11.0         | 54.0 ± 9.6     | 56.2 ± 11.1    | .307|
| Ankle, deg     |                     |                |                |     |
| DF             | 21.4 ± 7.1          | 18.9 ± 8.4     | 15.1 ± 6.1     | .006b|
| PF             | 51.4 ± 7.6          | 51.7 ± 8.5     | 49.2 ± 5.5     | .617|

Values are presented as mean ± SD unless noted otherwise.

aOSD, asymptomatic Osgood-Schlatter disease; DF, dorsiflexion; ER, external rotation; HBD, heel-buttock distance; IR, internal rotation; OSD, Osgood-Schlatter disease; PF, plantar flexion; SLRA, straight-legged raise angle; sOSD, symptomatic Osgood-Schlatter disease.

\(^b\)P < .01, aOSD vs Non-OSD.

\(^c\)P < .05, aOSD vs Non-OSD.

**Figure 3.** Tukey-Kramer post hoc test results for practice time and lower leg tightness. Values are presented as mean ± SD. aOSD, asymptomatic Osgood-Schlatter disease; sOSD, symptomatic Osgood-Schlatter disease. *P < .05. **P < .01.
Limited dorsiflexion in the ankle joint is associated with increased knee flexion, tibial internal rotation, and foot pronation during the stance phase of running.\(^1\) Sarcević\(^{21}\) reported that a high number of patients with OSD had limited ankle dorsiflexion. When the triceps surae is hypertonic, the forward tilt of the lower leg is reduced, moving the center of gravity backward. In addition, the torque of the quadriceps muscle is strengthened, and traction to the tibial tuberosity is increased, presumably leading to OSD.\(^5\)

To our knowledge, there have been no reports on the relationship between OSD and hip tightness. In this study, we recognized an association between the internal rotation of the hip and symptomatic OSD. Limited internal rotation of the hip may adversely affect the pelvis and body trunk forward rotation during a throwing motion, and posteriorization of the center of gravity may be involved in the onset of symptomatic OSD. Harding et al\(^{11}\) noted that internal rotation of the hip in the lead leg is important for absorbing force as the pitcher pivots the body forward.

Adolescents reportedly tend to experience overuse injuries, including OSD,\(^{23,24}\) and young athletes are thought to disproportionately incur injuries attributed to the overuse of their bodies.\(^{10,15}\) In the present study, only the symptomatic OSD group had a longer practice time per week.

This study has some limitations. First, this was a cross-sectional study conducted among preadolescent baseball players, and it is possible that lower limb tightness attributed to the influence of pain occurred in the symptomatic OSD group. To investigate the reasons for the onset of OSD, a longitudinal study will be necessary in the future. Second, there were relatively few cases of OSD in the present study. This is presumed to be because baseball is a sport with relatively little jumping and cutting action. Studies with a larger population should be conducted in the future. Third, because this study was based on the findings of a preparticipation physical examination of preadolescent baseball players, radiographic imaging was not performed. Furthermore, we did not perform a multivariate analysis given the small number of cases in this study. However, there were weak correlations among physique, lower limb tightness, and practice time.

CONCLUSION

The present study showed that the symptomatic OSD group had a significantly greater height, weight, BMI, and weekly practice duration than did the non-OSD and asymptomatic groups. In addition, the symptomatic OSD group had a significantly restricted HBD and range of hip and ankle joint motion when compared with the non-OSD group. The weekly practice duration was longer for the symptomatic OSD group, suggesting that this factor may be associated with symptomatic OSD.

REFERENCES

1. Almeida MO, Davis IS, Lopes AD. Biomechanical differences of foot-strike patterns during running: a systematic review with meta-analysis. \(J\) Orthop Sports Phys Ther. 2015;45:738-755.

2. Blankstein A, Cohen I, Heim M, et al. Ultrasonography as a diagnostic modality in Osgood-Schlatter disease: a clinical study and review of the literature. \(Arch\ Orthop Trauma Surg.,\) 2001;121:536-539.

3. Bloom OJ, Mackler L, Barbee J. Clinical inquiries: what is the best treatment for Osgood-Schlatter disease? \(J\) Fam Pract. 2004;53:153-156.

4. Czyzny Z. Osgood-Schlatter disease: a clinical study and review of the literature. \(Med\ Ultrason.\) 2010;12:323-335.

5. de Lucena GL, dos Santos Gomes C, Guerra RO. Prevalence and associated factors of Osgood-Schlatter syndrome in a population-based sample of Brazilian adolescents. \(Am\ J\ Sports Med.\) 2011;39:415-420.

6. Donatelli R, Wooden M, Ekedahl SR, Wilkes JS, Cooper J, Bush AJ. Relationship between static and dynamic foot postures in professional baseball players. \(J Orthop Sports Phys Ther.\) 1999;29:316-325.

7. Ehrenborg G. The Osgood-Schlatter lesion: a clinical study of 170 cases, \(Acta\ Chir\ Scand.\) 1982;124:89-105.

8. Eun SS, Lee SA, Kumar R, et al. Direct bursoscopic ossicle resection in young and active patients with unresolved Osgood-Schlatter disease. \(Arthroscopy.\) 2015;31:416-421.

9. Flowers MJ, Bhadreshwar DR. Tibial tuberosity excision for asymptomatic Osgood-Schlatter disease. \(J\ Pediatr Orthop.\) 1995;15:292-297.

10. Gerrard DF. Overuse injury and growing bones: the young athlete at risk. \(Br\ J Orthop.\) 1993;27:14-18.

11. Gholve PA, Scher DM, Khakharia S, Widmann RF, Green DW. Osgood-Schlatter syndrome. \(Curr\ Opin\ Pediatr.\) 2007;19:44-50.

12. Harding JL, Picha KJ, Bliven KCH. Pitch volume and glenohumeral and hip motion and strength in youth baseball pitchers. \(J Athl\ Train.\) 2018;53:60-65.

13. Kaneuchi Y, Otsuki K, Hakozaki 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. \(J Orthop Sports Med.\) 2018;6:2325967117749184.

14. Kaya DO, Toprak U, Baltaci G, Yosmaoglu B, Ozer H. Long-term functional and sonographic outcomes in Osgood-Schlatter disease. \(Knee\ Surg\ Sports\ Traumatol\ Arthrosc.\) 2013;21:1131-1139.

15. Kujala UM, Kivist M, Heinonen O. Osgood-Schlatter’s disease in adolescent athletes: retrospective study of incidence and duration. \(Am\ J\ Sports Med.\) 1985;13:236-241.

16. Lanning P, Heikkinen E. Ultrasonic features of the Osgood-Schlatter lesion. \(J\ Pediatr\ Orthop.\) 1991;11:538-540.

17. Nakase J, Aiba T, Goshima K, et al. Relationship between the skeletal maturation of the distal attachment of the patellar tendon and physical features in preadolescent male football players. \(Knee\ Surg\ Sports\ Traumatol\ Arthrosc.\) 2014;22:195-199.

18. Nakase J, Goshima K, Numata H, Oshima T, Takata Y, Tsuchiya H. Precise risk factors for Osgood-Schlatter disease. \(Arch\ Orthop\ Trauma\ Surg.\) 2014;135:1277-1281.

19. Osgood RB. Lesions of the tibial tubercle occurring during adolescence: 1903. \(Clin\ Orthop\ Relat\ Res.\) 1993;286:4-9.

20. Saily M, Whiteley R, Johnson A. Doppler ultrasound and tibial tuberosity with regard to age and sex and its relationship to pathogenesis of Osgood-Schlatter disease: a case series with comparison group and clinical interpretation. \(Br\ J\ Sports\ Med.\) 2013;47:93-97.

21. Sarcević Z. Limited ankle dorsiflexion: a predisposing factor to Morbus Osgood Schlatter? \(Knee\ Surg\ Sports\ Traumatol\ Arthrosc.\) 2008;16:726-728.

22. Schlatter C. Verletzungen des Schnabel-formigen Fortsatzes der abebrnen Tibiaepiphysye. \(Beiträge zur Klinischen Chirurgie.\) 1903;38:874-876.

23. Straccioli A, Casciano R, Levey Friedman H, Meehan WP, Micheli LJ. Pediatric sports injuries: an age comparison of children versus adolescents. \(Am\ J\ Sports Med.\) 2013;41:1922-1929.

24. Straccioli A, Casciano R, Levey Friedman H, Stein CJ, Meehan WP, Micheli LJ. Pediatric sports injuries: a comparison of males versus females. \(Am\ J\ Sports Med.\) 2014;42:965-972.

25. Weiss JM, Jordan SS, Andersen JS, Lee BM, Kocher M. Surgical treatment of unresolved Osgood-Schlatter disease: osseous resection with tibial tubercleplasty. \(J\ Pediatr\ Orthop.\) 2014;27:844-847.

26. Yanagisawa S, Osawa T, Saito K, et al. Assessment of Osgood-Schlatter disease and the skeletal maturation of the distal attachment of the patellar tendon in preadolescent males. \(Orthop\ J\ Sports Med.\) 2014;2:2325967114524084.