

Association Between Osteochondritis Dissecans of the Humeral Capitellum and Medial Epicondyle Lesion in Baseball Players

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Background: Osteochondritis dissecans (OCD) of the humeral capitellum occurs in adolescent overhead athletes, and medial epicondyle (ME) lesions are also common in this population.

Purpose: To evaluate the association between elbow OCD and ME lesions in adolescent baseball players.

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

Methods: This study retrospectively evaluated adolescent baseball players with unstable elbow OCD who underwent surgery between January 2000 and February 2020. Patients were excluded if they had osteoarthritis of the elbow. A total of 139 elbows were included in this study (138 male and 1 female athlete; mean \pm SD age, 13.6 \pm 1.5 years). The patients were first divided into 2 groups based on OCD location: a central lesion group (72 elbows) and a lateral group (67 elbows). Next, patients were divided according to OCD size into a localized group (56 elbows) and a widespread group (83 elbows). Finally, OCD lesions that were both lateral and widespread were defined as lateral-widespread (60 elbows), resulting in 5 groups. ME apophyseal fragmentation and elongation were evaluated and defined as ME lesions. We then compared the relationship between OCD and ME lesions.

Results: Of the 139 elbows, 63 (45.3%) had ME lesions. The prevalence of ME lesion was higher in the lateral group than the central group (56.7% vs 34.7%; $P = .009$) and higher in the widespread group than the localized group (55.4% vs 30.4%; $P = .004$). Furthermore, the prevalence ratio of ME lesion was significantly higher in the lateral-widespread group than for other lesions (58.3% vs 35.4%; $P = .007$).

Conclusion: In patients undergoing surgery for capitellar OCD, the presence of ME lesions was more commonly associated with lateral and widespread capitellar lesions when compared with central and localized lesions.

Keywords: osteochondritis dissecans (OCD); elbow; medial epicondyle lesion

Osteochondritis dissecans (OCD) of the humeral capitellum (elbow OCD) frequently occurs in adolescent baseball players and gymnasts.³⁶ There are various possible causes, such as ischemia,¹¹ genetic factors,^{25,32} and micro-trauma.^{1,31} Of these, repetitive valgus stress during throwing motion is a significant etiologic factor.^{4,6,17} Meanwhile, medial epicondyle (ME) lesions, such as epiphyseal fragmentation and elongation of the ME, are also common in adolescent baseball players. One ultrasound (US) study reported a prevalence of 52.9% for ME lesions in baseball players aged 6 to 17 years.²⁶ The combination of repetitive traction stress on the ulnar collateral ligament (UCL) and

contraction of the flexor-pronator muscle is considered the main mechanism of various types of morphologic changes of the ME.^{9,26,28}

For early stage elbow OCD, nonoperative treatment is generally selected. Matsuura et al¹⁹ reported that nonoperative treatment for elbow OCD allowed healing in 90.5% of patients with radiolucent lesions and in 52.9% of patients with nondisplaced fragment lesions. Advanced-stage elbow OCD, such as International Cartilage Research Society (ICRS) classification grades 3 and 4,⁵ is treated using surgical procedures including drilling, fragment fixation, free body resection, and osteochondral autograft transfer (OAT).³³ Several authors have indicated that OAT provides satisfactory results.^{3,13,30,38} However, numerous authors have also stated that treatment for lateral OCD lesions provides worse outcomes as compared with treatment for

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central lesions^{15,19} and that widespread OCD lesions have the potential to have worse outcomes compared with localized lesions.³⁶ Furthermore, radiographic outcomes of OAT are supposedly poorer in patients with lateral lesions than in those with central lesions.^{20,21} However, most medial elbow injuries of juvenile athletes are treated nonoperatively. Such patients are usually prohibited from pitching for at least 4 weeks until the medial elbow pain is gone. Throwing would then start while the pitching strength is gradually increased.² Although OCD lesions and ME lesions are both related to overhead sports, the relationship between OCD and ME lesions remains unknown.

The purpose of this study was to evaluate the prevalence of ME lesions in patients with elbow OCD and to clarify the association between the existence of ME lesions and the severity of elbow OCD. We hypothesized that patients with more severe OCD would have a higher rate of ME lesions.

METHODS

Patients

The study design and methods were approved by the ethics committee of our institution. We retrospectively assessed 213 adolescent patients who underwent surgery for elbow OCD in our institution between January 2000 and February 2020. Surgical procedures included drilling, fragment fixation, free body resection, and OAT. The exclusion criteria were previous surgery, elbow osteoarthritis, and incomplete data. A total of 139 adolescent baseball players were analyzed in this study (Figure 1). There were 138 male and 1 female athlete. Descriptive data were collected from preoperative information. Participants had a mean \pm SD age at the time of surgery of 13.6 ± 1.5 years (range, 9-19 years), height of 162.3 ± 8.8 cm, and weight of 57.1 ± 11.2 kg. Regarding their positions, there were 62 pitchers (44.6%), 20 catchers (14.4%), 40 infield players (28.8%), and 17 outfield players (12.2%) (Table 1). All patients underwent a physical examination, and no valgus laxity was detected. The image evaluation was performed using bilateral elbow plain radiography, involved side elbow computed tomography (CT), and magnetic resonance imaging (MRI). The plain radiography included the anteroposterior planes at 0° and 45° of elbow flexion and a lateral plane. If the patient had images taken at different ages, the preoperative images closest to the date of surgery were used for this study. MRI findings were used to determine the surgical indication, which was classified into ICRS grades 3 and 4 as unstable OCD.⁵

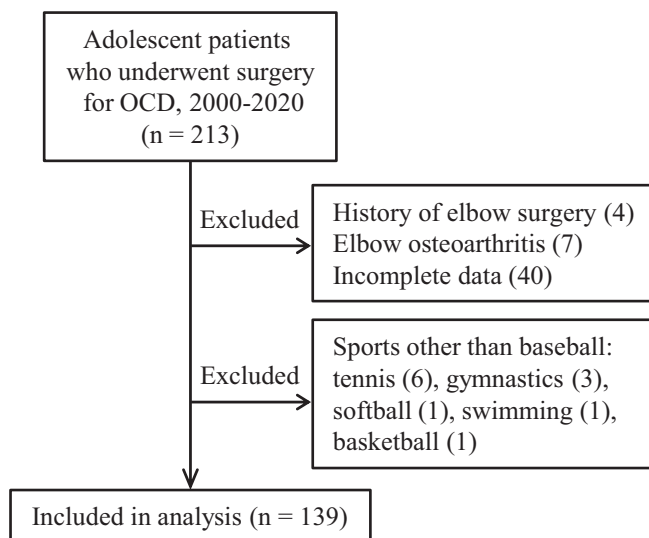


Figure 1. Flowchart of study patients. OCD, osteochondritis dissecans.

OCD Lesion Classification

OCD lesions were grouped by location and size on plain radiographs and CT scans. All patients had bilateral radiographic data and affected-side CT data. The image evaluation and grading were performed by a fellowship-trained orthopaedic surgeon (K.K.). OCD location was classified as central for lesions located within the articular surface of the capitellum and lateral if lesions extended to the lateral cortex of the capitellum (Figure 2, A and B).^{15,21} Regarding size, OCD lesions were classified as follows: localized, in which the width of the OCD lesion was less than half of the humeral capitellum width, as described by Takahara et al³⁵ (Figure 2C); widespread, in which the width of the OCD lesion was more than half of the capitellum width (Figure 2D). Patients with lesions that were lateral and widespread were categorized separately into a lateral-widespread group (Figure 2E).

Evaluation of the ME Lesion

ME lesions were evaluated using plain radiographs and CT scans. Apophyseal fragmentation and elongation of the elbow ME indicated an ME lesion (Figure 3).²⁹ ME elongation was defined as enlargement of the ME without fragmentations when compared with the ME of the uninvolved elbow. If the ME lesion could not be clearly identified on radiographs because of incorrect rotation or radiographic

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Ethical approval for this study was obtained from the Hirosaki University Graduate School of Medicine (No. 2011-199).

TABLE 1
Patient Characteristics^a

	Total (N = 139)	ME Lesion		OCD Location		OCD Lesion Size		Lateral-Widespread (n = 60)
		Positive (n = 63)	Negative (n = 76)	Central (n = 72)	Lateral (n = 67)	Localized (n = 56)	Widespread (n = 83)	
Age, y	13.6 ± 1.5	13.3 ± 1.3	13.9 ± 1.6	13.8 ± 1.5	13.3 ± 1.4	14.0 ± 1.6	13.3 ± 1.4	13.2 ± 1.4
Male:female, No.	138:1	63:0	75:1	71:1	67:0	55:1	83:0	60:0
Height, cm	162.3 ± 8.8	160.5 ± 8.7	163.8 ± 8.6	162.9 ± 8.3	161.7 ± 9.3	162.7 ± 8.2	162.1 ± 9.2	161.3 ± 9.4
Weight, kg	57.1 ± 11.2	52.2 ± 11.0	55.7 ± 11.3	54.7 ± 11.3	53.4 ± 11.2	55.3 ± 10.9	53.2 ± 11.4	53.2 ± 11.5
BMI	20.3 ± 2.9	20.1 ± 3.1	20.6 ± 2.8	20.4 ± 3.0	20.3 ± 2.9	20.8 ± 2.8	20.1 ± 3.0	20.3 ± 3.0
Competitive play duration, mo	57.9 ± 18.8	54.3 ± 18.0	60.9 ± 19.0	61.4 ± 18.6	54.1 ± 18.4	61.1 ± 20.1	55.7 ± 17.6	53.3 ± 17.8
Position played								
Pitcher	62 (44.6)	24 (38.1)	38 (50.0)	36 (50.0)	26 (38.8)	30 (53.6)	32 (38.6)	24 (40.0)
Catcher	20 (14.4)	12 (19.0)	8 (10.5)	7 (9.7)	13 (19.4)	5 (8.9)	15 (18.1)	12 (20.0)
Infield	40 (28.8)	19 (30.2)	21 (27.6)	21 (29.2)	19 (28.4)	12 (21.4)	28 (33.7)	18 (30.0)
Outfield	17 (12.2)	8 (12.7)	9 (11.8)	8 (11.1)	9 (13.4)	9 (16.1)	8 (9.6)	6 (10.0)

^aData are expressed as mean ± SD or No. (%) unless otherwise indicated. BMI, body mass index; ME, medial epicondyle; OCD, osteochondritis dissecans.

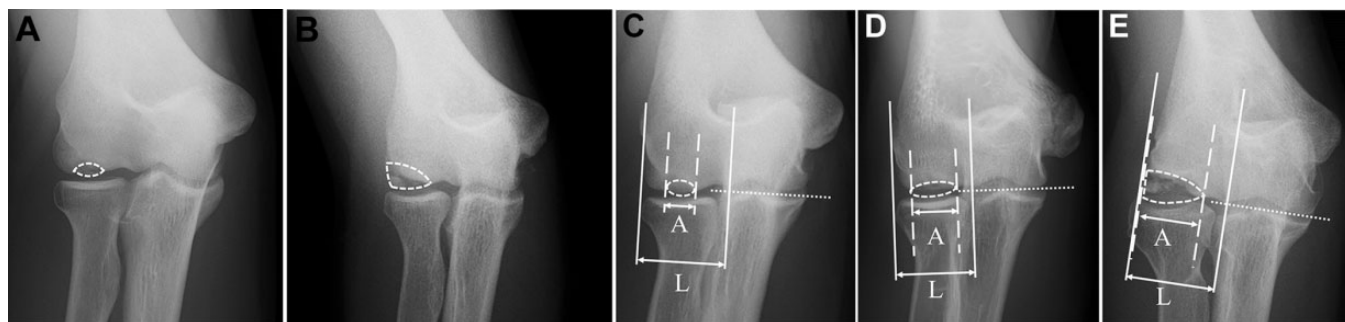


Figure 2. Osteochondritis dissecans (OCD) lesion classification on radiographs. OCD location was classified as (A) central if the lesion (dotted/circles lines) did not extend to the lateral cortex and (B) lateral if the lesion extended to the lateral cortex. To calculate OCD size, the width of the OCD lesion, A, was compared with half of the humeral capitellum width, L. The size was classified as (C) localized if the width of the lesion was less than half of the humeral capitellum width (mean A/L <50%) and (D) widespread if the width of the lesion was more than half of the humeral capitellum width (mean A/L >50%). (E) Lesions that were lateral and widespread were classified into a separate lateral-widespread group.

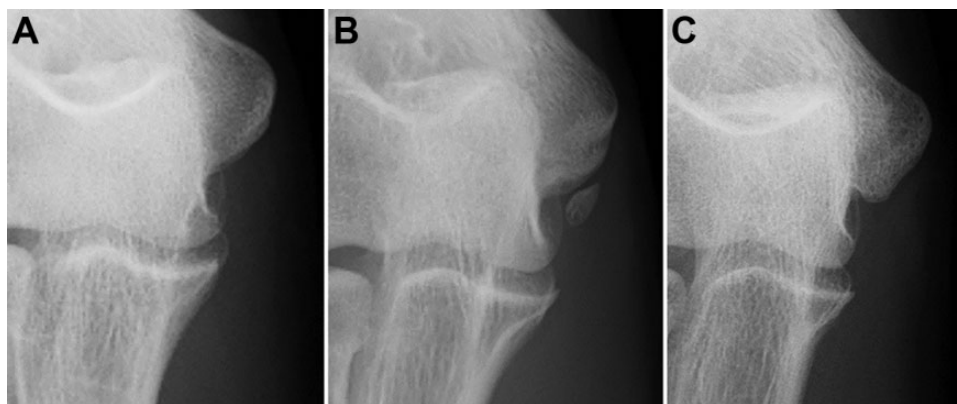


Figure 3. Medial epicondyle lesion: (A) normal, (B) apophyseal fragmentation, and (C) elongation. Apophyseal fragmentation and elongation are indicative of a medial epicondyle lesion.

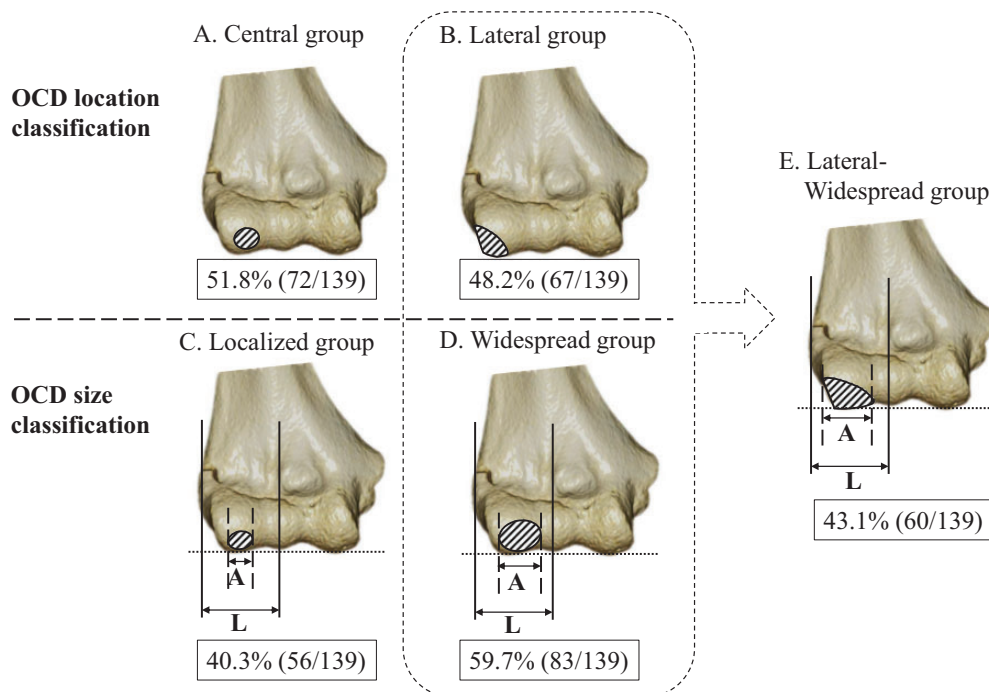


Figure 4. Prevalence of osteochondritis dissecans (OCD) lesions by group: (A) central group, (B) lateral group, (C) localized group, (D) widespread group, and (E) lateral-widespread group. The number of lesions and percentage of the total are shown in boxes. A, the width of osteochondral defect; L, the length of the capitellum.

density, CT data were used for a detailed evaluation of the ME lesion.

Statistical Analysis

The prevalence of ME lesions in 5 groups was assessed. The ratio of fragmentation and elongation of ME lesions in each group was compared using a chi-square test. We also compared the prevalence of ME lesions in each OCD location classification using a chi-square test. In addition, the prevalence of ME lesions for each OCD lesion classification in the lateral-widespread group and in the other groups was compared using a chi-square test. The level of significance was set at $P < .05$. All statistical data were analyzed using SPSS Version 22 (IBM Corp).

RESULTS

In terms of OCD location, 72 of 139 elbows (51.8%) were in the central group, and 67 (48.2%) were in the lateral group. Regarding OCD size, 56 elbows (40.3%) were in the localized group, and 83 (59.7%) were in the widespread group. Of 139 elbows, 60 (43.2%) were classified into the lateral-widespread group (Figure 4).

ME lesions were present in 63 of 139 elbows (45.3%). Among those, ME apophyseal fragmentation was found in 29 elbows (46.0%), and ME elongation was found in 34 (54.0%). There was no significant difference in the ratio of fragmentation and elongation for each OCD group (Table 2).

The prevalences of ME lesion were as follows: 34.7% (25/72) in the central group, 56.7% (38/67) in the lateral group, 30.4% (17/56) in the localized group, and 55.4% (46/83) in the widespread group. The prevalence of ME lesion in the lateral-widespread group was 58.3% (35/60), and that in others was 35.4% (28/79). There was a significantly higher prevalence of ME lesions in the lateral group than the central group ($P = .009$) and in the widespread group than the localized group ($P = .004$), and the lateral-widespread group had a significantly higher prevalence of ME lesion than did the others ($P = .007$) (Table 2).

DISCUSSION

This study revealed the prevalence of ME lesions in patients with unstable OCD. Other studies have shown a relationship between elbow OCD and ME lesions. Serita et al²⁹ reported a relationship between abnormality of the ME lesion and OCD lesion size. Temporin et al³⁷ demonstrated that enlargement of the ME increased the incidence of OCD (odds ratio, 3.3). However, few studies have shown a relationship between the existence of an ME lesion and the OCD location and size.

In US evaluation, the prevalence of elbow OCD among juvenile baseball players has been reported to be between 1.3% and 3.4%.^{10,14,20,26} Matsuura et al²⁰ reported that the prevalence of OCD diagnosed using US was 2.1% in elementary school children aged 10 to 12 years who participated in regional baseball competitions. Kida et al¹⁴ stated that the prevalence of OCD on US examination was 3.4% in

TABLE 2
Comparison of ME Lesion Classification and Presence With OCD Classification^a

OCD Classification	ME Lesion Classification ^b		P Value	Presence of ME Lesion ^c		P Value ^d
	Fragmentation (n = 29)	Elongation (n = 34)		Positive (n = 63)	Negative (n = 76)	
Location			.799			.009
Central (n = 72)	12 (41.4)	13 (38.2)		25 (34.7)	47 (65.3)	
Lateral (n = 67)	17 (58.6)	21 (61.8)		38 (56.7)	29 (43.3)	
Size			.638			.004
Localized (n = 56)	7 (24.1)	10 (29.4)		17 (30.4)	39 (69.6)	
Widespread (n = 83)	22 (75.9)	24 (70.6)		46 (55.4)	37 (44.6)	
Location and size			.571			.007
Lateral-widespread (n = 60)	15 (51.7)	20 (58.8)		35 (58.3)	25 (41.7)	
Others (n = 79)	14 (48.3)	14 (41.2)		28 (35.4)	51 (64.6)	

^aData are expressed as No. (%). ME, medial epicondyle; OCD, osteochondritis dissecans.

^bThe number of fragmentations and elongations between the OCD groups was compared using a chi-square test.

^cThe presence of ME lesions in the OCD groups was compared using a chi-square test.

^dBolded *P* values indicate statistically significant difference between the groups ($P < .05$).

junior high school and high school baseball players aged 12 to 18 years who participated in baseball skill training camps. In these 2 studies, the baseball players underwent radiographic evaluation after US evaluation. OCD lesions are classified into 3 stages using Minami radiographic classification.²⁴ Stage I is characterized by the existence of radiolucent areas; stage II is defined by nondisplaced fragments; and stage III has loose bodies and sclerotic change.¹⁹ While Matsuura et al²⁰ indicated that 90.9% of elbow OCD was stage I, Kida et al reported that stage I accounted for only 14.7% and that stages II and III occurred in 38.2% and 13.2% of patients, respectively. However, the ages of the patients were different in these 2 studies. Therefore, early diagnosis and treatment may reduce the rate of advanced OCD.

OCD location and size affect the results of treatment. Regarding the OCD location, Matsuura et al¹⁸ divided patients who underwent OAT for symptomatic OCD into central lesion and lateral lesion groups. The outcomes of the surgery were worse in patients with lateral lesions than in those with central lesions. The lateral lesion group showed poor postoperative range of motion, lower postoperative Timmerman and Andrews scores, lower incidence of return to sports, higher incidence of radial head subluxation, and higher incidence of osteoarthritis than did the central group. As for OCD sizes, Takahara et al³⁴ published the clinical results of OCD fragment removal and stated that small defects that were less than half of the capitellar articular width showed better outcomes than did large defects. Kosaka et al¹⁶ reported a reoperation rate >50% after fixation of lateral widespread lesions. Based on these studies, lateral lesions and the widespread lesions of OCD are associated with poor clinical outcome. Our study showed that the prevalence of an ME lesion might pose a risk of greater lateral OCD extension and increased OCD size. In other words, treatment for OCD with an ME lesion might be complicated and associated with a poor outcome.

ME lesions are associated with repetitive valgus stress of the elbow during throwing motion. In highly skilled adult baseball players, the maximum valgus torque of the elbow has been reported to be 64 N·m during pitching.⁷ In skeletally immature juvenile players, the traction force of the UCL causes ME apophysitis. Hang et al⁹ evaluated plain radiographs of Little League baseball players who participated in regional competitions, and the prevalence ratio of ME fragmentation was 19%. Otoshi et al²⁶ performed the medical checkups of 4249 baseball players aged 6 to 17 years using US evaluation, showing that the prevalence of ME lesions was 52.9% and increased with age. In addition, they found that 93 players (2.2%) had capitellar OCD and 44 players also had ME lesions. These players had elbow pain more frequently than did players with only OCD or an ME lesion.

Overuse or fatigue of the elbow may cause joint laxity that may increase the stress on the elbow joint. Hattori et al¹² reported US evaluation of medial elbow stability in high school baseball players. Medial elbow joint space gapping under valgus stress significantly increased after 60 pitches as compared with before pitching. Millard et al²³ found that repeated wrist flexion exercise decreased the stability of the medial elbow. These 2 studies stated that fatigue of the forearm flexor muscle decreased the power of stabilization for valgus stress of the medial elbow. However, details of the association between elbow medial joint laxity and the development of OCD lesion or the presence of ME lesion are not understood.

Some studies have examined the relationship between medial elbow joint laxity and stress of the radiocapitellar joint. Ruchelsman et al²⁷ stated that overuse and fatigue led to increased compression and shear forces during pitching motion across the radiocapitellar joint. Funakoshi et al⁸ examined the stress distribution of the elbow joint associated with UCL insufficiency using CT osteoabsorptiometry, which assesses long-term stress distribution by measuring

subchondral bone density. They evaluated nonthrowing volunteers, asymptomatic baseball pitchers, and baseball pitchers with symptomatic valgus instability. Their results indicated that symptomatic UCL insufficiency produced excessive stress on the anterolateral part of the capitellum. However, Mihata et al²² studied the contact pressure of the radiocapitellar joint in a cadaveric study, mimicking an advanced OCD lesion. While larger and more lateral capitellar defects increased the stress on the capitellum, an intact UCL could not decrease contact pressure caused by the capitellar osteochondral defects. Therefore, an advanced OCD lesion itself increased the stress of the capitellum under valgus stress without dysfunction of the UCL. It is important to avoid excessive practice to prevent medial elbow joint laxity caused by overuse. Alternatively, it is possible that larger OCD lesions could cause greater valgus angulation in the elbow, resulting in increased UCL stress. Mihata et al demonstrated that elbow valgus laxity increased as the size of the osteochondral defect increased. However, the same article showed that the size of the osteochondral defect did not alter UCL strain at any degrees of elbow flexion. Thus, there is currently no evidence that larger OCD lesions result in increased UCL stress.

Based on these results, ME lesions are more commonly seen in more lateral and more widespread OCD lesions. Children with OCD frequently do not present at a medical clinic until the disease has progressed to the advanced stages. However, if those with pain from ME lesion were carefully evaluated and followed up, it might be possible to detect and treat more OCD cases in the early stages. Previous studies have reported that US screening is valuable for the diagnosis of elbow OCD and that US is less expensive than is MRI.^{10,14} Therefore, medical checkups for juvenile baseball players are recommended to identify patients in the early stages of OCD via US. As a future plan, we should investigate the treatment results of nonoperative and operative cases and the prognosis of ME lesions. Furthermore, given that there are few opinions on how past overuse affects nonoperative therapy, it is necessary to evaluate whether an ME lesion itself poses a risk of medial elbow instability.

This study had some limitations. First, it was a retrospective and cross-sectional study of OCD among patients who underwent surgery. Second, this study did not evaluate the clinical findings and outcome of these patients. For these reasons, the result of this study could not evaluate longitudinal findings or address whether the ME lesion could be used to predict the progression of OCD. Third, this series did not include the early stage of OCD and nonoperative treatment. Because our institute is a university hospital, most patients had been referred for surgery. Fourth, this study performed only a radiographic evaluation of the ME lesions. Therefore, some lesions that could be found via US may have gone undetected. The symptoms of a medial elbow injury, such as ME tenderness or valgus instability, were not considered in this study. However, no patients had valgus instability under clinical examination. Fifth, image evaluation was performed only once by a single orthopaedic surgeon. Sixth, this study did not investigate the details of

sports activities, such as practice time per week, amount of throwing, off-season duration, and so on.

CONCLUSION

ME lesions were present in 45.3% of patients with elbow OCD who underwent surgery. OCD in the lateral group, widespread group, and lateral-widespread group had a significantly higher prevalence of ME lesion.

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