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Upper extremity overuse injuries in pediatric athletes: clinical presentation, imaging findings, and treatment☆

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ABSTRACT

Given the frequency and severity of overuse injuries in pediatric athletes and the potential for long-term deleterious effects, it is important for radiologists to have a comprehensive understanding of these injuries and their imaging spectrum. This article addresses chronic overuse injuries involving the upper extremity in the pediatric athlete. Chronic upper extremity overuse injuries in competitive pediatric athletes yield imaging findings that can be subtle, obvious and characteristic, or atypical. Prompt application of the appropriate imaging modalities and their accurate interpretation expedites management, returning the pediatric athlete to the playing field while minimizing long-term adverse outcomes.

Summary statement: Proper modality selection and interpretation in the imaging evaluation of upper extremity overuse injuries in pediatric athletes include an understanding of skeletal development, mechanism of injury, and clinical presentation to provide accurate diagnoses and mitigate long-term adverse sequelae.

Learning objectives: After reading this article and taking the test, the reader will be able to:

- Describe the clinical presentation and imaging characteristics of a variety of pediatric overuse injuries in the upper extremity including little league shoulder, rotator cuff tendinosis, gymnast wrist, climber's finger, and myriad pathologies about the elbow.
- Explain how the physis plays a crucial role in pediatric overuse injury and how to evaluate physeal injury in a multimodal approach.
- Understand the clinical management for certain pediatric overuse injuries, especially those that have potential for long-term and/or permanent disability.

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1. Introduction

Approximately 1.5% of pediatric recreational athletes suffer some type of injury [1]. Skeletal immaturity inherent to pediatric athletes makes them prone to physeal/apophyseal injury for two reasons. [CME#1a-d] TP [First, epiphyseal plates and apophyses are the weakest components in the pediatric musculoskeletal system. Second, ligamentous structures are two to five times stronger than the open growth plate.] Proper imaging used to guide the management of pediatric athletes must consider patient age (i.e., variable secondary ossification centers), skeletal sites prone to injury, mechanism of injury varying by sport, and radiation exposure. Upper extremity overuse injuries can affect athletes playing numerous sports with variable activities and motions, from the shoulder to the finger [Table 1].

1.1. The importance of the physis

The crux of image interpretation and management of many pediatric injuries, both chronic overuse and acute trauma, lies in a proper understanding of the physis. Histologically, the physis consists of an arrangement of chondrocytes surrounded by a matrix of proteoglycan aggregates. During closure, the physis is most prone to injury, which can result in growth arrest, a potentially significant adverse outcome [2].

[CME #2 a-d] The physis consists of three chondrocyte zones: reserve, proliferative, and hypertrophic. The reserve zone (also called *resting*, *germinal* or *stem cell*) is located adjacent to the epiphysis, consists of irregularly stacked chondrocytes, demonstrates a low rate of proliferation, and provides cartilage used in unidirectional bone growth. The reserve zone is critical in the skeletally immature patient as injury can

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Table 1
Upper extremity overuse injuries in pediatric athletes

Anatomic location	Type or description of injury
Shoulder	LLS
	Shoulder impingement
	Rotator cuff tendinosis
	Swimmer's shoulder
Elbow	LLE
	Epiphysiolysis of olecranon
	MEA
	UCL injury
	Lateral epicondylitis
	ME
	OD of the capitellum
	Valgus extension overload syndrome
Wrist	Gymnast wrist
Fingers	Climber's finger

result in growth cessation. The stem cells provided by the reserve zone enter the proliferative zone, consisting of stacked columnar chondrocytes, and eventually produce the extracellular matrix for longitudinal growth via active cell division. The chondrocytes hypertrophy and travel further from the epiphysis into the hypertrophic zone which is not involved in active growth; rather, it is responsible for maturation, degeneration, and provisional calcification. The hypertrophic zone is the weakest zone in the physis and is the site often involved in fracture or alteration (e.g., widening). Physeal stresses in overuse injuries have several potential sequelae including physeal widening, early calcification, fracture, and premature closure (physeal bar formation) [3].

In addition to the physis, there are also two secondary ossification centers that may play a role in pediatric overuse injuries. The first is the epiphysis, which is the articular surface seen at the end of long bones and contributes to longitudinal growth. The apophysis, on the other hand, is a tendinous insertion site that is prone to muscular avulsion.

Injury to the vascularity on the metaphyseal side of the physis leads to disruption of the normal cycle of chondrocyte-programmed cell death and subsequent ossification. Physeal widening is evident on radiographic imaging with findings of demineralization and fragmentation adjacent to the physis. These findings are not permanent and resolve with cessation of activity [4]. On magnetic resonance imaging (MRI), the appearance of physeal widening, as evidenced by cartilage signal intensity, is due to disruption of endochondral bone formation as long columns of hypertrophic cartilage cells extend from the physis to the metaphysis in overuse syndromes. Injury to the epiphyseal vascularity in the area nearest to the resting zone causes ischemia of the resting and proliferative zones. Physeal bridging results as fresh chondrocytes are no longer available to supply the physis. Support for this theory has been shown in a study using MRI to identify patterns of growth arrest after physeal insult which showed that premature physeal bony bridging in children is most often posttraumatic and tends to develop at the sites of earliest physiologic closure. In addition, rest and immobilization have yielded improvement of symptoms and imaging findings in several athletes with physeal widening [5]. Unless otherwise specified below, conservative management to include rest, ice, nonsteroidal antiinflammatories, and physical therapy for a period of 6–12 weeks is usually the first line treatment for most of these conditions. However, it is especially important to remain aware of the physis as management may differ in the setting of chronic overuse without physeal widening. Physical therapy and strengthening may actually exacerbate physeal injury if left undiagnosed. Therefore, an understanding of physeal development is vital in ensuring prompt, appropriate imaging-based diagnosis in order to prevent improper therapy and adverse outcomes.

2. Shoulder

Shoulder overuse injuries in pediatric athletes are primarily related to overhead activities such as swimming, throwing, or racket sports. One study suggests that 58% of baseball pitchers complain of shoulder or elbow pain during a season [6]. Several pathologies can be seen in the same patient, which can make distinguishing etiologies and treatment options difficult. In addition, proper use of imaging can change treatment plans [7]. Four types of shoulder injury will be described: Little League Shoulder (LLS), shoulder impingement, swimmer's shoulder, and rotator cuff tendinosis.

The proximal humerus initially contains three separate ossification centers as it develops: humeral head at age 1 year, greater tuberosity at age 2–3 years, and lesser tuberosity at age 5–6 years. These coalesce around age 7 and fuse with the humeral metaphysis between age 16 and 20 years [8]. The accelerated growth that occurs during adolescence coupled with the fact that 80% of longitudinal growth of the humerus occurs at the proximal physis predisposes adolescents to proximal humerus physeal injury [9].

2.1. Little League Shoulder

Epiphysiolysis of the proximal humerus, also known as (aka) *Little League Shoulder*, has had several descriptive names in the past [10,4,11]. Recent data suggests that the mechanism of repetitive torsional and distractive stress at the proximal physis from overhead activity results in reversible metaphyseal–physeal blood supply disruption, yielding chondrocyte proliferation and physeal widening [12]. Torsional stress, greatest in the late cocking phase of the throwing motion when the shoulder is placed in hyper-external rotation, places eccentric stress upon the shoulder adductors (pectoralis major and subscapularis) and internal rotators (latissimus and teres major). Physeal distraction occurs with ball release due to forward arm motion and opposing proximally directed rotator cuff force [13]. [CME #3a-d] Thus, the current belief is that LLS represents a Salter–Harris Type I fracture [14].

Clinical presentation in LLS classically involves an early midteenager (~age 14) baseball pitcher with tenderness over the lateral portion of the proximal humeral physis. Delayed or underdiagnosis might occur due to continued pitching despite limitations because of pain or not seeking medical attention until deterioration in pitching control is observed with an average reported symptom duration of approximately 7 months [15]. LLS has also been described in pediatric athletes engaged in gymnastics, [16] badminton, [17] cricket, [18] volleyball, and swimming [19].

Radiography [Fig. 1] demonstrates widening of the normally cone-shaped proximal humeral physis relative to the contralateral shoulder, taking into account variability between internal and external rotation with greater widening seen on anterior–posterior (AP) internal rotation view even in normal shoulders. Lateral fragmentation, sclerosis, demineralization, and cystic change may also be seen. Of note, these radiographic findings may be present as early as 3 weeks after the initial complaint; however, radiographs may be normal within 10 days of symptom onset suggesting the chronic repetitive nature of the injury [14].

MRI evaluation may be necessary to exclude superimposed injury when additional patient or caregiver reassurance is needed. MRI [Fig. 2] reveals physeal widening and bone marrow edema on both sides of the physis. Adjacent periosteal edema [20] and subchondral cysts [21] have also been reported. Other study results demonstrate [CME #4b] focal widening of the physis on the T1-weighted and gradient echo (GRE) sequences with physeal signal intensity extending into the metaphysis. [CME #4a] Marrow edema was also demonstrated with abnormal bone marrow signal along the metaphyseal side of the physis on both the T1-weighted and fat-saturated (FS) T2-weighted sequences, possibly the effect of local traction. Some studies also suggest epiphyseal signal abnormalities; however, this can also be seen in normal shoulders due to marrow variation [12,20]. Animal studies suggest that gadolinium-enhanced images also demonstrate [CME #4c] fracture lines with lower signal intensity fluid than adjacent cartilage [5] above [CME #4d].