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"Weak Links" of the Pediatric Skeleton: Common Foci for Disease and Trauma. Part 2—Areas of Weakness Beyond Bone and Cartilage Transitions

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Abstract In this article, we review recent updates in the radiologic imaging of injuries to the pediatric skeleton. While part 1 focused on areas of weakness at cartilage–bone interfaces, part 2 focuses on other areas of the developing skeleton that are uniquely susceptible to injury. Over the past 5 years new and interesting research has helped us better understand the disease processes and how to image these "weak links." These cartilage–bone interfaces are unique to the pediatric skeleton, and an understanding of this anatomy is critical in the performance and interpretation of pediatric musculoskeletal imaging examinations.

Keywords Imaging · Pediatrics · Skeleton · Radiology

Introduction

The "weak links" in the pediatric skeleton are primarily found at transitions in tissue types (Table 1). Both acute and chronic (e.g., repetitive motion) injuries to the pediatric skeleton tend to occur at the boundaries between bone, cartilage, and fibrous tissue. However, these interfaces can also serve as barriers to disease proliferation. Recent work has further investigated the roles of these tissue boundaries in pediatric diseases unique to the developing skeleton. This current review will summarize and highlight the more common pediatric skeletal diseases involving weak links.

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Transition from Tendon/Ligament to Cartilage/Bone

Osgood–Schlatter Disease (OSD)

Avulsion fractures occur when acute pulling force on ligament, tendon, or joint capsule overcomes the stress capacity of the attached bone and results in detachment of the bone fragment. They represent an example of fractures at the bone and ligament/tendon interface. Adolescents are most vulnerable to avulsion fractures because of strong muscles and relatively weak apophysis [1]. The extensor compartment of the knee is a common site for avulsion fractures [1], and OSD is a frequent cause of anterior knee pain, which is a common musculoskeletal complaint in adolescent and preadolescent athletes [2].

OSD is a chronic avulsion injury at tibial tuberosity due to repeated traction by the patellar tendon without a precise known etiology [1]. The disease is bilateral in about 50 % of the cases and is rarely associated with damage to other ligaments and tendons of the knee. Diagnosis is almost always made clinically [1, 3]. Patients with OSD mostly present with tenderness and swelling at the tibial tuberosity where pain is reproduced with sporting activity and extension of knee against resistance.

It is debated whether tendinitis of patellar tendon or avulsion of portion of tibial tuberosity is the true cause of OSD. After bone and/or cartilage is pulled away from tuberosity, it continues to grow and ossify. The fragment may later unite with the tubercle or stay as a separate ossicle [3]. Rosenberg and colleagues [4] followed 28 cases

Table 1 Lesions in children that occur at the interface of different skeletal tissues

Tissue transition	Pathology
Bone and cartilage	
Acute	Salter–Harris
	SCFE
	Patellar sleeve avulsion
	Transitional fractures (tri-plane, Tillaux)
Chronic	Osteochondritis dissecans
	Physeal injury from repetitive stress
	Little league shoulder
	Little league elbow
	Gymnast wrist and knee
Bone and synchondrosis	Apophyseal avulsion
	Traction apophysitis
	Osgood–Schlatter disease
Tendon/ligament to cartilage/ bone	Osgood–Schlatter disease
	Sinding-Larsen–Johansson disease
Transitions in bone	Bowing fractures
Periosteum and bone	Osteomyelitis
	Tumor
	Hemorrhage
	Trauma

of OSD and found that bony ossicle was seen in only nine cases, however soft tissue changes consistent with tendonitis (CT finding of increased width and decreased attenuation of the tendon, and MR finding of enlargement and increased signal intensity of the tendon) was seen in all 28 cases. Furthermore, in three of the nine cases, the ossicle remained non-united with the tubercle despite symptom relief. These findings suggest that soft tissue changes including damage to tendon correlates with OSD compared to the presence of bony ossicle.

Four radiological stages are identified for maturation of tibial apophysis [5] and are commonly used in radiological evaluation: the cartilaginous stage (age 0–11 years where tibial tuberosity is cartilaginous), the apophyseal stage (age 11–14 years where an ossification center develops on tibial tuberosity and is distinct from ossification center in epiphysis), the epiphyseal stage (age 14–18 years where the apophyseal and epiphyseal ossification centers fuse into a single large ossification center), and the bony stage (>18 years where physis of tibial tuberosity is closed/fused). Most OSD cases occur during the apophyseal stage [3]. Similarly, on ultrasound, tibial tuberosity maturation can be classified into three stages [6]: sonolucent (presence of apophyseal cartilage), individual (presence of apophyseal cartilage with its own secondary ossification center), and connective (union of tibial epiphysis with secondary ossification center of tibial tuberosity).

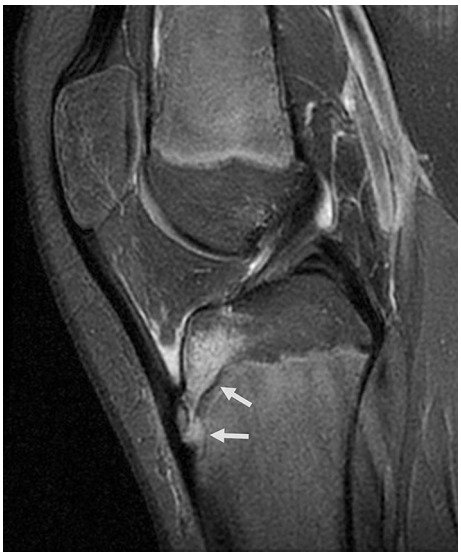

Fig. 1 MRI of OSD. Bone marrow edema in the apophysis of the tibial tuberosity (*arrows*) on sagittal T2-weighted fat saturated MRI

In OSD, radiographic finding may be normal or show irregularity in early stages, and fragmentation in later stages, at the level anterior to the tibial tubercle. Similarly, soft tissue swelling and obliteration of inferior angle of infrapatellar fat pad can also be seen [1, 3].

Based on MR findings, the OSD is classified into five stages [7]: normal (symptomatic patient with normal MRI), early (symptomatic patient with edema like changes around tibial tuberosity), progressive (symptomatic patient with cartilaginous damage of tibial tuberosity and tear of secondary ossification center), terminal (Fig. 1) (symptomatic patient with ossicle being completely separated from tibial tuberosity and thickening of patellar tendon at insertion site), and healing (asymptomatic patient with no separation of ossicle). OCD does not necessarily progress sequentially from the normal stage, to the early stage, to the progressive stage, and so on. Sometimes, normal, early, and progressive stages may progress directly to the healing stage. Additional MRI findings include a distended deep infrapatellar bursa, bone marrow edema in the tibia, and thickened cartilage adjacent to the tibial tubercle [1]. Radiologic findings at the normal, early, and healing stages are normal. The progressive stage shows radiolucency in avulsed parts of the secondary ossification center, and the terminal stage shows ossicle formation on radiograph [7].

Ultrasound is effective for visualization of soft tissues and therefore has high utility in early-stage OSD diagnosis as well as follow-up [6, 8]. Ultrasound can detect bursitis located in the deep and superficial infrapatellar bursae, synovial edema, soft tissue inflammation, and status of

cartilage around tibial tuberosity, fibrosis, and vascularity at insertion of patellar ligament. Follow-up is, however, usually made clinically without the need for further imaging.

Transitions in Bone

Two areas of the bone are unique in the pediatric skeleton and are sites affected by trauma: (1) the transition from diaphyseal to metaphyseal cortex and (2) the immature region of the metaphysis.

Bowing/Torus/Greenstick Fracture

The cortex of long bones becomes thin as it transitions from the diaphysis to metaphysis. This area of bone is relatively susceptible to bowing/torus and greenstick fractures. Of note, pediatric bones are more susceptible to bending and bowing before fracture due to their inherently softer composition. Torus fractures can be subtle (Fig. 2) and unique to the pediatric skeleton [9].

Classic Metaphyseal Lesion

The classic metaphyseal lesion (CML) occurs in the long bones of infants and is a highly specific marker of infant abuse (<1 year age) [10•, 11]. Common sites for CML include the distal femur, proximal and distal tibia, and proximal humerus [12]. CML is a planar fracture that passes through the most immature region of metaphysis—the trabecular transition zone (the region between primary and secondary spongiosa)—and extends out through the sub-periosteal bone collar [10•, 12] (Fig. 3). Primary spongiosa is part of the metaphysis adjacent to the physis that is made of newly ossified region of hypertrophic chondrocytes. It consists of longitudinally oriented struts of cartilage that are covered with bone by newly formed surface osteoblasts [10•]. Secondary spongiosa is made from remodeling of primary spongiosa. It has thicker and fewer bone trabeculae with a high volume of marrow space compared to primary spongiosa. Thus, CML represents a fracture at tissue transition zone in metaphysis.

Kleinman et al. [11] used single-emulsion, single-screen imaging mammographic system to look at 31 infants who died due to inflicted skeletal injury at an average age of 3 months and found that 64 (89 %) of 72 long bone fractures were CML. However, long bone shaft fractures are more common than CML in the abused older infant and child [13]. Similarly, a 10-year retrospective study used high-detail mammographic screen-film and high-resolution dual-side-read computer radiographic system to compare CML prevalence in high-risk and low-risk infants (<1 year age) for abuse. They found that at least one CML was identified in 50 % of infants in high-risk group, while none were identified in low-risk group [14]. These studies indicate CML as a high-yield marker of infant abuse.

Radiographs are most commonly used in the diagnosis of CML [15]. Classic radiologic appearance of CML include 'bucket handle' and 'corner fracture' patterns

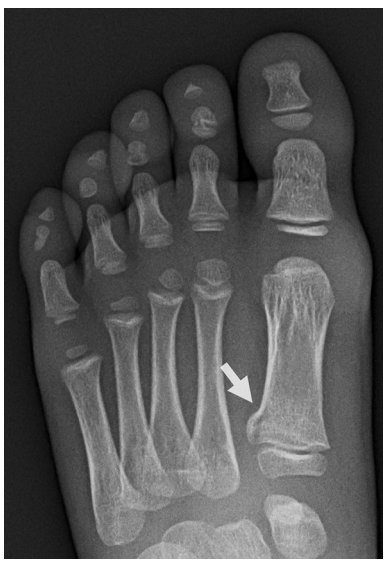

Fig. 2 Pediatric bed bunk fracture. Subtle bowing fracture at the proximal metaphysis of the great toe

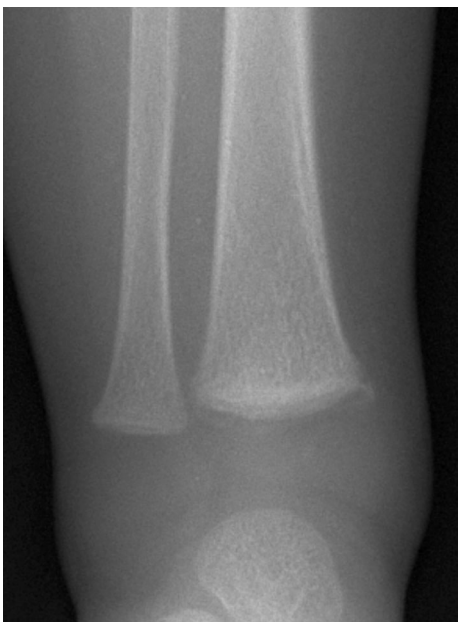

Fig. 3 Classic metaphyseal lesion (CML) at the distal tibial. The CML is a high-specific marker of non-accidental trauma in infants. The fracture occurs at the trabecular transition zone, the most immature region of metaphysis

based on the plane of radiographic imaging. Corner fracture is seen when long axis of metaphysis and radiographic projection are perpendicular. Similarly, bucket handle is seen when physis is viewed with beam angulation [12].

The radiodense 'bucket handle' visible in radiographs is referred as radiologic zone of provisional calcification (ZPC). Tsai and colleagues [10•, 16] employed micro-CT, flat panel CT, and digital radiography to study and correlate radiological findings of CML with histopathological findings. They found that the radiologic ZPC is different from histologic ZPC. Histologic ZPC consists of terminal layers of hypertrophic chondrocytes in the physis, and comprises only a small component of radiologic ZPC. In contrast, radiologic ZPC consists of primary spongiosa of the metaphysis in addition to histologic ZPC. Similarly, 'corner fracture' appearance is seen due to separation between sub-periosteal bone collar (SPBC) and adjacent metaphyseal cortex. The SPBC is continuous with radiologic ZPC on micro-CT suggesting that their mineralized structure is linked [10•]. The presence of radiologic ZPC and relatively thicker SPBC in the fracture fragment gives the fragment a mineralized discoid appearance that is thin centrally and thicker peripherally.

Likely mechanism of CML is suggested to be the presence of excessive torsional or shearing forces, or flailing of extremities during violent shaking [12]. Thomspson and colleagues [15] experimentally generated CML in pelvic specimen of 3–7 day old pigs and used micro-computed tomography for pre- and post-imaging. They found that fractures could occur on medial, lateral, or both sides of distal femur and proximal tibia irrespective of whether varus or valgus load were applied. Majority of fractures went through the entire metaphysis, and when they did not go, more fractures were seen on the medial side. Kleinman and Marks [17, 18] have found reduced trabecular density and discontinuity between sub-periosteal bone collar and cortex in medial aspect of tibia and femur which can explain this finding.

The common indicator of healing of CML in radiography is focal extension of physeal lucency into the metaphysis [12]. The lucency corresponds to the presence of hypertrophic chondrocytes in the primary spongiosa. Additionally, even though sub-periosteal new bone formation occurs in healing of long bone fractures, this finding is not usually seen in CML. Most CML become inconspicuous in about 4 weeks and heal in about 6 weeks [12].

There are a few conditions where CML-like appearance can be seen in radiographs in absence of abuse. In some cases of rickets, discreet osseous fragment resembling corner fracture may be seen in absence of other findings [12]. Other conditions include osteogenesis imperfecta, metaphyseal chondrodysplasia, spondylometaphyseal dysplasia, obstetric injury, developmental variants, and iatrogenic cases [12]. Recently, Connell et al. [19] reported on three cases of CML-like fractures in distal femur in neonates delivered by elective and uncomplicated lower segment cesarean section. They reviewed an obstetric practice of 22 years with about 8500 births per year. Two of these had breech presentation; one mother had unstable diabetes. It is, therefore important to place the radiographic findings within the appropriate context of the overall clinical and laboratory findings.

Link Between Periosteum and Bone

The layer of periosteum that covers the cortical surface of bone is often disrupted by disease processes in the pediatric skeleton. The space between the periosteum and bone creates a space that contains infection, tumors, and blood. This space is readily seen on MRI and ultrasound (Figs. 4,

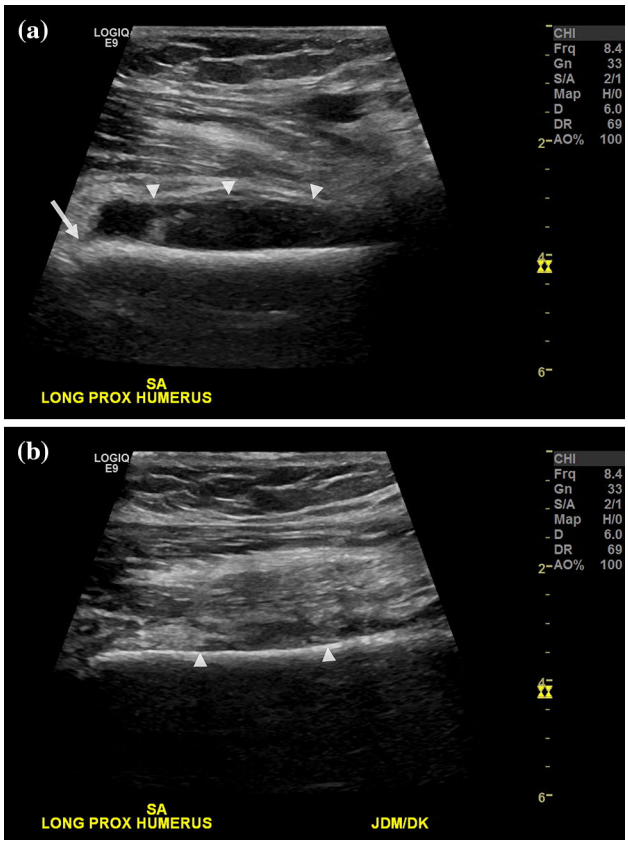

Fig. 4 A 13 year-old with sub-periosteal abscess involving the proximal metaphysis of the humerus. **a** The periosteum (*arrowheads*) is lifted away from the cortex by pus and creates an anechoic space between the periosteum and underlying cortex. The periosteum is attached at the periosteal bone collar (*arrow*) and prevents further spread of infection. The periosteum can detach from the cortical bone and create a space that contains pus, tumor, and blood. **b** After ultrasound-guided needle aspiration, the pocket of fluid has resolved and edematous soft tissue and periosteum again touch the underlying bone cortex (*arrowheads*)

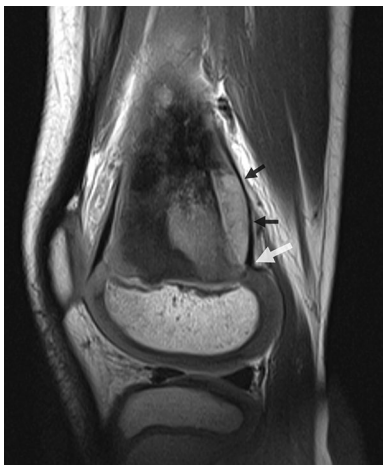

Fig. 5 Sub-periosteal bone collar (*white arrow*). Although easily detached along the cortical surface of the bone, the periosteum (*black arrows*) has a strong attachment near the physis at the sub-periosteal bone collar and limits the spread of disease, in this case, osteosarcoma

5). Although easily detached along the cortical surface of the bone, the periosteum has a strong attachment near the physis at the sub-periosteal bone collar (ring of Lacroix) [20]. This relative area of strength is a barrier to the spread of disease (Fig. 5).

Conclusion

Several unique tissue transitions occur in the pediatric skeleton that are at risk for injury. The transition from tendon/ligament to cartilage/bone, transitions in bone, and between bone and periosteum are foci for acute and chronic trauma. Injuries range from Osgood–Schlatter and Sinding-Larsen–Johansson disease at the knee to soft bones that bend before they break (e.g., torus fracture) and the classic metaphyseal fracture, a high specificity injury of non-accidental trauma. Although the periosteum is readily stripped from the underlying bone by processes such as infection and tumor, it also serves along with the sub-periosteal bone collar as a barrier for further spread of disease. Awareness of these areas of relative strength and weakness in the pediatric skeleton can aid in radiologists' ability to correctly interpret examinations in these patients.

Compliance with Ethical Standards

Conflict of Interest Sagar Wagle and Andrew S. Phelps declare no potential conflicts of interest. John D. MacKenzie reports Grants from GE Healthcare.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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