

MR Imaging of Cartilage in the Pediatric Patient

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- I. Hyaline cartilage of the growing epiphysis
 - a. Randomly organized cartilage cells and matrix
 - i. Development and changes of the unossified epiphysis during infancy and childhood.
Types in the distal femur:
 1. Infancy (< 1 year of age): homogenous signal intensity
 2. Early walking years (1-3 years of age): decrease signal intensity along weight-bearing region (cartilage response to the pressure of walking)
 3. Preschool years (3-5 years of age): increasing signal intensity of posterior condyles, primarily stippled (advancing ossification and vascularity)
 4. Early school age: ill-defined increased signal intensity in posterior condyles
 5. School age: well-defined increased signal intensity in posterior condyles
 - ii. Difference in signal intensities likely due to noncollagen portions of matrix (water, proteins, proteoglycans)
 - iii. Relatively decreased signal intensity in weight-bearing cartilage relative to physis possibly due to larger size of aggregates and greater concentration of proteoglycans, and more tightly bound water
 - iv. Vascularity of unossified epiphysis
 1. Nutrient vessels course through the epiphysis in nonanastomic channels
 2. Channels composed of arterioles and venules within loose connective tissue
 3. Vessels contribute to nutrition and osteogenesis
 4. Configuration changes with age
 - a. Infancy: parallel array with penetration across cartilaginous physis
 - b. With development of ossification center: convergence into a radial pattern
 - b. Secondary physis around the secondary center of ossification:
 - i. Normal structure similar to the primary physis that is responsible for longitudinal bone growth by endochondral ossification
 - ii. Composed of resting, germinal, proliferating, and hypertrophic cartilage cells in an organized pattern
 - iii. Original spherical configuration becomes hemispherical as the secondary center of ossification grows
 - c. Abnormal hyaline cartilage of the epiphyses
 - i. Infection: osteomyelitis or "chondro-osteomyelitis?"
 1. Acute or subacute
 2. Underestimated by conventional radiographs
 3. Predilection for rapidly growing end of long bones (70% of growth of the lower extremity is in the knee)
 4. Trauma precedes up to half of children with acute osteomyelitis
 5. MRI used for diagnosis, monitoring therapy, need for additional intervention
 6. Monitor for growth arrest within the physis
 - ii. Osteochondritis dissecans (OCD):
 1. Juvenile form (5-15 years of age, with open physis, M >>F)
 2. Adult form: older adolescents and adults with fused physes
 3. 75% of children have knee involvement
 4. Etiology remains controversial

- a. Indirect or repetitive trauma?
 - i. Repetitive impingement of tibial spine on lateral aspect of medial femoral condyle during internal rotation
 - ii. Contribution of ligamentous laxity
 - b. Ischemia: abnormal ischemic subchondral bone prone to fracture?
 - c. Defect of ossification that fails to heal?
 - d. Genetic subgroup prone to epiphyseal disturbances
 - e. Must differentiate from acute osteochondral fracture
5. Treatment
- a. Closed therapy preferred if physes still patent
 - b. Open therapy: drilling, fixation, osteochondral grafts, removal of loose bodies
 - c. Goal is to restore congruence of joint surface
 - d. Protected weight-bearing with early post-operative joint motion
6. Juvenile form tends to have a better prognosis: healing with mesenchymal tissue that becomes less resilient fibrocartilage, but does not become hyaline cartilage
7. Normal development versus OCD possibly differentiated by:
- a. Lack of surrounding edema
 - b. Accessory ossification centers
 - c. Does this explain the better prognosis of outcomes of juvenile versus adult OCD?
8. Association of chondro-osseous irregularity, increased vascularity, disruption of secondary physis around ossification center and development of OCD
- iii. Other osteochondral trauma
 - 1. Acute examples
 - a. Patellar sleeve fracture
 - b. Lateral condylar fractures of the elbow
 - 2. Chronic examples
 - a. Osgood-Schlatter disease
 - b. Sinding-Larsen-Johannson disease
 - c. Slipped capital femoral epiphysis

II. Physeal Cartilage

- a. Physeal thickness reflects rate of growth
- b. With age, physeal contour becomes more undulating (contribution to Salter Harris II injuries?)
- c. Vascular supply from the epiphyseal, peripheral metaphyseal and central diaphyseal blood vessels
- d. Disruption or alterations in cartilage or metaphyseal blood supply can result in interruption of normal endochondral ossification
 - i. Physeal bone bridges
 - 1. Normal to see small areas of fibrous bridging across the physis
 - 2. Result from breach in integrity of the peri-epiphyseal cells of the physis
 - 3. MR imaging used to define size (percentage of physis) and location of bony bridge, pre-operative planning
 - ii. Cartilaginous tongues or bands
 - 1. Normal to see small cartilaginous “tongues”
 - 2. Larger areas result from chronic metaphyseal stress (relative physeal ischemia)
 - a. Infection and inflammation
 - b. Trauma (acute and chronic)

- c. With rest: resumption of normal endochondral ossification
 - d. With continued stress: growth and alignment abnormalities
 - iii. "Dislocation of physeal cartilage"
 - 1. Into the metaphysis: development of enchondroma
 - 2. Perpendicular to the physis: development of osteochondroma
 - 3. Abnormal course of physeal cartilage can result in a "longitudinal epiphyseal bracket" or "delta phalanx"
 - a. Involves the phalanges, metacarpals, or metatarsals
 - b. Bones are "bracketed" by an epiphyseal or secondary ossification center
- III. Articular Cartilage
- a. Organized structure of articular cartilage is similar to that of adults
 - b. Juvenile rheumatoid arthritis (JRA)
 - i. T2 relaxation times in children with juvenile rheumatoid arthritis differ significantly from normal controls
 - ii. T2 relaxation time maps likely reflect alterations in cartilage microstructure
 - iii. Changes in T2 relaxation times are seen before changes in cartilage morphology are detected with conventional MR imaging
 - iv. Possible use as an objective method to monitor disease activity
 - c. Other synovial disorders that result in articular cartilage erosion
 - i. Hemophilia
 - ii. Synovial venous malformation
 - iii. Pigmented villonodular synovitis
 - iv. Septic arthritis

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