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Review article

Management of growth arrest: Current practice and future directions

Sherif Dabash[a,*], Gautham Prabhakar[b], Eric Potter[b], Ahmed M. Thabet[b], Amr Abdelgawad[b], Stephen Heinrich[c]

[a] Department of Orthopaedic Surgery, University of Texas Health Science Center at Houston, United States
[b] Department of Orthopaedic Surgery & Rehabilitation/Paul L. Foster School of Medicine, Texas Tech University Health Sciences Center, El Paso, TX, United States
[c] Department of Orthopaedics, Tulane University, New Orleans, LA, United States

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ABSTRACT

Introduction: Premature growth arrest can pose a challenge to the orthopedic surgeon. Various options for treating physeal arrest exist.
Methods: Systematic searches were conducted on PubMed/Medline, ScienceDirect, OVID, and Cochrane Library. Secondary searching was performed, where certain articles from reference lists of the selected studies were reviewed that were not found in the primary search.
Results: This review article discusses the different methods of management for premature growth arrest.
Conclusions: The use of mesenchymal stem cells provides a promising alternative treatment modality.

1. Introduction

Physeal injuries complicate 18–30% of pediatric fractures. Growth arrest occurs in 5–10% of cases in those with physeal fractures. The incidence of growth arrest is quite variable depending on physeal location, type of injury, and treatment received.[1–2] Premature growth arrest is characterized by an unexpected discontinuation of longitudinal and/or appositional bone growth secondary to an insult to the growth plate prior to skeletal maturity. Growth arrest is frequently posttraumatic; however, other etiologies include congenital conditions (e.g. Blount's disease), infection, neoplasm, irradiation, metabolic/hematologic abnormalities, ischemia, disuse, and iatrogenic injury.[3] The most frequently injured physis is the distal radius.[1] According to Mizuta, the physis of upper limb is more frequently injured than those of lower limb in a ratio of 3:1.[1] The outcome of premature growth arrest is based on skeletal age, location and extent of the associated physeal bar. A central physeal bar results in cessation of longitudinal growth, and subsequently a limb length discrepancy (LLD). A peripheral physeal bar may result in both a limb length discrepancy as well as angular deformity.

There are many current treatment strategies that focus on either restoring the propensity for growth, such as surgical removal of physeal bars with or without placement of interpositional material, or mitigating the resulting complications of the arrest, encompassing deformity correction.[4] The goal of this review is to present a concise summary of the current practice and future directions in management of premature growth arrest.

2. Epidemiology of growth arrest

Injury to the reserve or proliferative zone of the physis may produce irreversible damage to the growing cells, resulting in growth disturbance. Physeal cartilage is exceptionally susceptible to injury for many reasons. Compared to adult articular cartilage, physeal cartilage is more susceptible to stress forces. The physis may be 2–5 times weaker than the surrounding fibrous tissue, so growth plates fail before ligaments, opposite to adult fracture patterns. Physeal cartilage is also more prone to tension and shear forces than adjacent bone.[5–7] Fracture involving the growth plate was first described in 1963 by Salter and Harris, and the described Salter-Harris classifications system is the most widely used.[8] Fig. 1.

3. Anatomy of physeal injury

Type 1 fracture involves extension through the hypertrophic zone of the physis Fig. 2. In these fractures, the physeal germinal

* Corresponding author.
E-mail address: sherif.a.dabash@uth.tmc.edu (S. Dabash).

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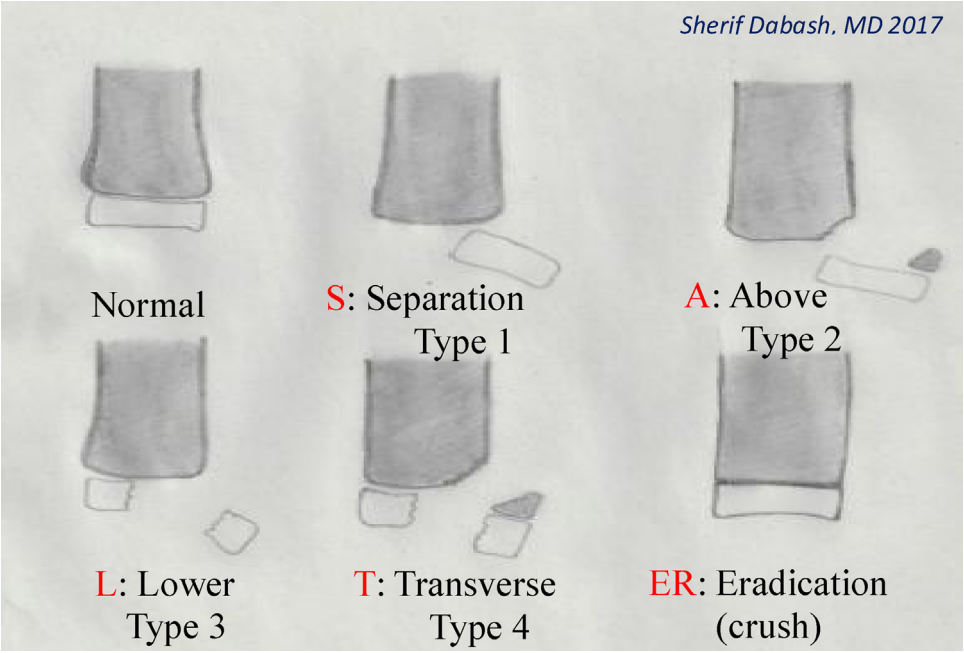

Fig. 1. Salter Harris classification.

cells remain with the epiphysis and the calcified layer with the metaphysis. Type II injuries account for more than half of all physeal fractures and are the most common physeal injuries. Type II injuries extend through the hypertrophic zone and exit through a portion of the metaphysis, generating the characteristic triangular metaphyseal Thurston-Holland fragment.[9] As long as no vascular compromise exists and the germinal cells are retained with the epiphysis, the prognosis for the first two types of fractures is often good, with minimal incidence of growth disturbance.[10–16]

Unlike Type I and II fractures, type III and IV fractures involve the reserve zone and as a result present with an increased risk of irreversible growth arrest. This may be a result both of injury to the germinal cells of the physis as well as resulting physeal bar formation, tethering the epiphysis to the metaphysis. Physeal injuries are more common with increasing age, and it is more pronounced in boys,[1] due to increased vigor of activities, and relative weakness of the physis near puberty. The peak incidence of physeal fractures is 11 years for girls, and 12 years for boys. Type III injuries are more common in older children and generally convey a moderate prognosis if the vascular supply to the epiphyseal fragment is not compromised, there is no displacement of the fracture, and there is no injury to the germinal center. While the displacement may be evaluated on radiographs, the impact of the degree of displacement on prognosis is not well-understood. Furthermore, integrity of the vascular supply and ultimate injury to the germinal center cannot be easily assessed. While type III fractures do not always necessitate surgery, restoration of the articular surface is imperative. Type IV injuries inevitably damage the germinal layer and require surgical intervention to align the physis as well as the articular surface. Type V fractures are the most severe class and occur due to a crush injury to the germinal physeal layer. This commonly results in growth arrest; however, type V fractures are perhaps the most difficult to diagnose acutely and may only be evident once a patient has been followed longitudinally. Any insult to the reserve zone and germinal chondrocytes may result in premature growth arrest. Trauma, congenital conditions (e.g. Blount's disease), infection, neoplasm, irradiation, metabolic/hematologic abnormalities, ischemia, disuse, and iatrogenic injury have all been described as etiologies of growth arrest.[17] Prolonged ischemia such as in septic shock or vascular injury to the limb may result in damage to or loss of the reserve layer of the physis.[18] Infection of the spongiosa often spares the reserve zone[19]; however, septic arthritis or subperiosteal abscess can directly compress the epiphyseal vasculature resulting in ischemic necrosis of the germinal chondrocytes of the reserve zone.[20] Iatrogenic injury due to trans-physeal fixation techniques is well described.[21]

4. Presentation and evaluation

A thorough history and physical examination is critical. In the setting of trauma, poor prognostic indicators include high-grade Salter-Harris fractures (Type III or IV), high-energy trauma, numerous reduction attempts, poor final reduction, characteristically distal femur and tibial fractures, and greater initial displacement.[22]

Radiography remains the best method to evaluate physeal injuries.[23] After physeal injury has been established, repeat radiography is recommended every 3 months until normal growth has been documented for at least 6 months. CT and MRI can elucidate more information concerning size, location, and shape of associated physeal bars[23,24]; however, CT is not a first-line tool due to its limited effectiveness in the evaluation of soft tissues and concern of radiation exposure.[25] Although both CT and MRI are useful for detecting and determining size of bony bridge, MRI has the added benefit of providing various sequences allowing for additional information concerning abnormalities in cartilage (ex. non-ossified cartilage bar requiring further intervention) and surrounding soft tissue injury.[25] MRI has no risk of radiation exposure. Three-dimensional MRI may also be used for evaluating growth plate injury and allows for accurate measurement of the physeal bar.[24] The best sequence for preoperative physeal bridge imaging is thought to be fat-suppressed 3D spoiled gradient echo-weighted MRI.[26] Recently, the use of intraoperative computer-assisted navigation has been used to adequately identify the bony bar.[27]