High-energy Pediatric Pelvic and Acetabular Fractures

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KEYWORDS

• High-energy fracture • Pelvis • Acetabulum • Pediatric

KEY POINTS

- Pediatric pelvic fractures are markers of high-energy injuries and are unlikely to be the cause of death, but are a sign of other associated life-threatening injuries such as head or thoracoabdominal trauma.
- Pelvic hemorrhage may go unrecognized in pediatric patients because of a more effective vasoconstriction mechanism and the ability of the immature periosteum to tamponade bleeding.
- Operative indications for pediatric pelvic fractures are based on amount of displacement and degree of instability and are indicated to prevent deformity and long-term disability and pain.
- Pediatric posterior wall acetabular fractures may be misinterpreted as small fragments when magnetic resonance imaging (MRI) reveals a large osteocartilaginous wall fragment requiring operative fixation. Most pediatric acetabular fractures involving the posterior wall require MRI to better judge the size of the fracture fragment(s).
- There is little evidence for the optimal treatment of triradiate acetabular fractures, and outcomes are poor for the most severe crush injuries.

INTRODUCTION

Pediatric pelvic and acetabular fractures are rare, with a reported incidence of 1 per 100,000 children per year.[1,2] Pediatric pelvic fractures caused by high-energy injury mechanisms are a marker of impending death because of associated head and other injuries.[3] Long-term outcome studies of pediatric pelvic fractures are lacking because of their rarity. Treatment can differ significantly between skeletally immature and skeletally mature pelvic fractures. Depending on the injury, the potential exists for long-term disability from residual deformity, growth disturbance, compensatory scoliosis, and pain. Literature specifically on pediatric acetabular fractures is even more scarce than literature on pediatric pelvic fractures. This article presents the anatomy, general considerations, classification systems, and treatment strategies for high-energy pelvic and acetabular fractures in skeletally immature patients based on the best available evidence and our own experience in treating these patients. Pediatric pelvic avulsion fractures, which are usually caused by low-energy mechanisms such as athletic events, are not addressed in this article.

The authors have nothing to disclose.

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ANATOMY

Pelvis

The pelvis begins to form during the first 2 months of embryologic development. The acetabulum, formed by the convergence of the ilium, ischium, and pubis, begins to cavitate and by the eighth week a fully developed hip joint exists. By 9 weeks, endochondral ossification of the ilium begins, with the ischium beginning to ossify at about 16 weeks, and the pubis a few weeks following this. The epiphyseal centers of the pelvis, including the triradiate cartilage, are visible at this time.[4]

The pubic symphysis, composed of thick cartilaginous endplates, has a variable rate of ossification until about age 10 years. The iliac crest and spines remain cartilaginous until adolescence, at which time secondary ossification centers develop along the anterolateral iliac crest at 13 to 15 years of age. As ossification of the wing advances posteriorly toward the posterior superior iliac spine, fusion to the rest of the ilium occurs from 15 to 17 years, with complete fusion often not occurring until age 25 years. The ilium is thought to develop normally based partly on the pull of the gluteal muscles on its outer wing.[4,5]

Acetabulum

The first ossification center of the acetabulum begins in infancy as woven bone at the level of the acetabular fossa. The triradiate cartilage is composed of 3 secondary ossification centers at the periphery of the acetabulum, with the pubis, the ilium, and ischium each contributing to an arm of growth (Fig. 1). These 3 secondary ossification centers begin to develop within the acetabular cartilage in the prepubescent period. The os acetabuli, part of the pubis, appears at 8 years of age, and develops into the anterior acetabular wall, with fusion normally by age 15 years. The acetabular epiphysis, the secondary ossification center of the ilium, appears between 8 and 9 years, and contributes to most of the roof of the acetabulum, and fully fuses by 18 years. The ischial epiphysis appears between 9 and 10 years with the secondary ossification center in the ischium continuing to develop through adolescence.[5] Before the visualization of secondary ossification centers during adolescence, injuries to this region can be missed with radiographs.

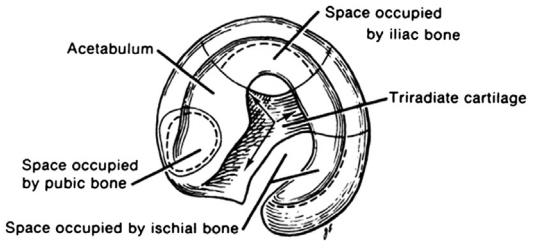

Fig. 1. The triradiate cartilage of the acetabulum, lateral view. Growth occurs outward from the triradiate cartilage until fusion occurs with the secondary ossification centers of the pubis, ilium, and ischium at skeletal maturity, and the acetabulum is fully ossified. (*From* Ponseti IV. Growth and development of the acetabulum in the normal child. Anatomical, histological, and roentgenographic studies. J Bone Joint Surg Am 1978;60(5):575–85; with permission.)

The triradiate cartilage and other ossification centers contribute to the widening of both the pelvic ring and the acetabulum. Interstitial growth in the triradiate cartilage contributes to the height and width of the acetabulum, but much of the growth as well as its depth are stimulated by mechanical stimuli. The acetabulum cavitates as a result of the pressure of the femoral head and also symbiotically contributes to the normal development of the femoral head. As the triradiate cartilage grows and develops into the acetabulum it also grows outward, contributing to the length of the pelvic bones.[4,5]

PATTERN OF TRAUMATIC ACETABULAR DYSPLASIA

Dysplasia caused by injury is distinct from developmental dysplasia of the hip (DDH). In DDH, the femoral neck is typically anteverted and the femoral head subluxes laterally and anteriorly while the pelvic ring remains symmetric and relatively unaffected. The acetabulum has a decreased lateral and anterior center edge angle. In posttraumatic hip dysplasia, proximal femoral anatomy is normal, but the true pelvis deforms and appears lengthened on the side of the injured acetabulum. The teardrop and inner wall of the acetabulum appear increased in size and the femoral head lateralizes. Patients usually have symmetric leg lengths but the leg can appear shortened because of the upward migration of the femoral head into the growth-arrested acetabulum. A positive Trendelenburg sign toward the uninjured side may be present because of an increased lever arm of the abductor muscle tendon-tendon unit on the injured side.[6]

CAUSE OF TRAUMATIC ACETABULAR DYSPLASIA

The exact cause of premature closure of the triradiate cartilage has yet to be elucidated. Rodrigues[7] suggested that the hematoma that forms after injury ossifies creating a bony bridge