



POSNA

The Core Curriculum

Developmental dysplasia of the hip (older infant)

Objectives

1. Describe physical and radiographic findings in the older infant with developmental dysplasia of the hip
2. Describe the pathoanatomy of developmental dysplasia of the hip in the older infant
3. Describe the vascular supply of the infant's hip
4. Discuss treatment of the older infant with developmental dysplasia of the hip
5. Describe complications of treatment of the older infant with developmental dislocation of the hip

Discussion

If the unstable hip in the newborn does not spontaneously stabilize, it will become fixed in a dislocated position. From that point, secondary changes develop in response to the dislocation. The lateral acetabulum will become deficient, due to the lateral position of the femoral head, and the center will be more shallow due to the absence of the femoral head. The femoral neck will develop into a valgus and anteverted configuration. The iliopsoas and adductor muscle group will undergo adaptive shortening. Fibrofatty tissue will fill the void in the true acetabulum. The inferior capsule will become constricted, from the continuing pressure of the overlying iliopsoas tendon. The intracapsular blood vessels to the femoral head will gradually alter their course to accommodate the change in position. These changes occur concurrently, although at different rates in different hips. Some of the changes (fibrofatty tissue, iliopsoas shortening, and capsular constriction) will impede reduction, some will render the reduction more unstable (changes in configuration of the head and acetabulum) and some (changes in route of blood vessels) will jeopardize the safety of the process of reduction. Strategies to deal with these problems are constantly evolving. An acute reduction of the hip under anesthesia is not difficult, but it is risky for subsequent avascular necrosis, and is thus unwise. The Pavlik harness can still achieve a reduction, although much less reliably. Traction on the leg to gradually (and presumably more safely) elongate the soft tissues and vessels can reduce the rate of vascular complication. Open reduction, by virtue of eliminating barriers to reduction and tension on the vasculature by virtue of reducing soft tissue tension has become popular. Avascular necrosis has been reported in follow-up studies of treatment of DDH in the contralateral originally normal hip, presumably as a result of unwise positioning for immobilization.

Complications include failure of reduction, redislocation, and avascular necrosis of the proximal femoral epiphysis. The incidence of failure of reduction or redislocation can be reduced with the usage of postoperative CT scanning of the hip. The rate of avascular necrosis is variable from study to study with the same treatment, primary medial open reduction being the current example.

There is some debate as to whether the presence of the bony ossific nucleus (always delayed in appearance in a dislocated hip) has a salubrious effect in diminishing the rate of avascular necrosis resulting from treatment. Fittingly, the most recent two studies on this subject reached contrary conclusions.

Though the terminology for the vessels varies from author to author, the consensus is that there is some penetration of the physis by metaphyseal vessels in infancy, but the major source of vasculature to the femoral head is via intracapsular vessels that then penetrate the epiphysis. The terminal branch of the medial femoral circumflex, called the lateral ascending cervical artery is the major supply.

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