



Is Attention Deficit Hyperactivity Disorder a Risk for Kohler's Disease? Osteonecrosis of Navicular Bone of Foot

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Abstract

We present a pediatric case report of foot pain due to Kohler's disease. We discussed the etiology and treatment of Kohler's Disease of a 5-year-old boy who had Attention Deficit Hyperactivity Disorder in this case report. Our goal is elucidate the etiology of these cases and their relationship with behavioral disorders.

Key Words: Osteonecrosis of Navicula; Kohler Disease; Attention Deficit Hyperactivity Disorder (ADHD), Trauma

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Introduction

Osteonecrosis, also referred to as avascular necrosis, refers to the death of cells within bone caused by a lack of circulation. It has been documented in many bones throughout the body. In the foot, osteonecrosis is most commonly seen in the talus, the first and second metatarsals and the navicular. Although uncommon, osteonecrosis has been documented in almost every bone of the foot and therefore should be considered in the differential diagnosis when evaluating both adult and pediatric foot pain. Kohler's disease is a rare, self-limiting, avascular necrosis of the navicular bone, first described in 1908. It is usually unilateral and most often affects boys. Its usual onset is between 4 to 5 years of age but can present as early as 2 years of age. Girls with this condition are often younger than boys, probably owing to earlier onset of ossification. The behavioural problems accompanying ADHD and comorbid psychiatric disorders are important in terms of unintentional injuries and trauma. Recurrent childhood traumas may lead osteonecrosis on late ossified and load-bearing bones like navicular bone of foot.

Case

A 5-year-old boy presented with painful left foot pain for 1 month. Pain had worsened the previous day after playing outside, and he was now refusing to bear weight on the right foot. He had untreated ADHD and got minor polytrauma or accidental injuries every day.

On examination, he had pain and tenderness over his left dorsomedial mid foot with no local skin changes. He walked with an antalgic gait with weight bearing on the lateral side of the foot. Blood investigations has shown no elevation of the white cell count or other markers such as the erythrocyte sedimentation rate and C-reactive protein (CRP). His bilateral both side foot radiographies obtained and diagnosed as osteonecrosis (Image I-II). We confirmed the diagnosis with an ankle Magnetic Resonance Imaging (MRI) (Image III). It's reported as navicular bone osteonecrosis of left foot. We applied a weight-bearing, below-the-knee cast during 4 weeks. At the end of 4 weeks, there were no pain and antalgic walk. The pathophysiology of this condition is best explained by a mechanical cause associated with a delayed ossification.

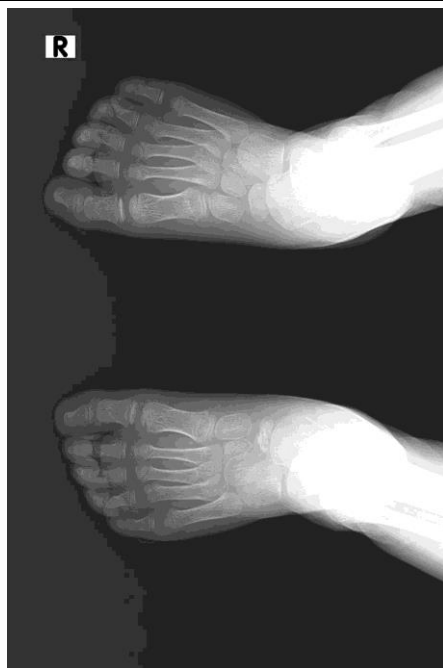


Image I-II: Anteroposterior and lateral radiographic views of foot



Image III-IV: MRI views of left foot (Navicular osteochondrosis)

Discussion

Navicular is the last tarsal bone to ossify and can get compressed between the already ossified talus and cuneiforms when the child becomes heavier. Vascularization of the navicula occurs in two ways and is identical in adults and in children. These blood vessels create a dense network around the bone and come from the perichondrium toward the center of the cartilage [1]. This in turn compresses the navicular bone's perichondral ring of blood vessels, producing ischemia of the central spongy bone and avascular necrosis. The prognosis remains excellent owing to this radial arrangement of blood supply [2]. Radiologic findings show patchy areas of navicular with sclerosis and rarefaction with loss of normal trabecular pattern. Sometimes the navicular may appear collapsed or may be normal in shape with a uniform increase in density with minimal fragmentation. Treatment includes pain control and using soft arch supports or medial heel wedge. Patients with worse symptoms may benefit from a short leg walking cast for 4 to 6 weeks. Symptoms in untreated patients last longer than in treated patients (15 months vs 3 months) [3-4]. Patients with persistent pain should be examined for other conditions such as talar coalition.

Loder et al explored the behavioral characteristics of 24 children with Perthes' disease. One third (33%) of the children had abnormally high scores in profiles associated with ADHD (impulsive, hyperactive, and psychosomatic categories), much higher than the 3-5% incidence of ADHD in the general population [5]. These findings have shown that the ADHD can be associated with avascular necrosis. On the other hand, some studies suggest that there exists a genetic influence on the association between childhood trauma and the severity of inhibitory deficits in children with ADHD [6]. The etiology of this condition is not well known, but vascular accidents, coagulation anomalies and heredity have been implicated [7].

In conclusion, we believe that the ADHD can be accepted risk factor for Navicular osteonecrosis in the background. We discussed the etiology of a disease not been fully clarified in this regard. Childhood trauma with ADHD may be leading to this situation at the age of games. Erdogan et al found statistically significant difference between the rates of ADHD diagnosed children with proximal humerus fractures and ADHD diagnoses in normal population ($p < 0.01$). There was also statistically significant difference between operation rates of children with or without ADHD diagnosis (55 % vs. 15 %) ($p < 0.01$) [8]. These

findings support that ADHD associated trauma has shown higher risk than normal population. Also we know that the children with ADHD have a higher risk of experiencing bone fracture accidents than without ADHD [9]. There can be significant interaction between ADHD and Navicular osteonecrosis. These patients in the outpatient clinic with a diagnosis of ADHD should be carefully examined or Kohler disease cases should be questioned in terms of ADHD.

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