Pediatric Osteonecrosis of the Capitate

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Capitate bone avascular necrosis is a rare disease in pediatric age group. A twelve-year-old male was diagnosed as pediatric osteonecrosis of the capitate. The patient was managed conservatively with limited immobilization and observation. At one-year follow-up, the patient recovered clinically and radiographically.

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Osteonecrosis results from the temporary or permanent loss of blood flow to a particular bone. The term osteochondrosis is used more often to describe a similar phenomenon when the condition affects one or more ossification centers in children. Osteochondrosis is characterized by a transient disruption of the blood supply to a previously normal endochondral growth region, leading to a disturbance in endochondral ossification, including chondrogenesis and osteogenesis. In children, the majority of osteochondrosis is self-limiting and will respond to symptomatic, non-operative treatment with complete resolution[1]. In contrast, the prognosis for adults with advanced osteonecrosis is not that favorable[2-3].

Most of the injuries in the wrist occur in the scaphoid[4]. The capitate is less affected and uncommon, partly due to the fact it is in the center of the carpal bones, and that its force of injury is usually transmitted at the radial and ulnar sides of the wrist.

However, idiopathic (atraumatic) osteonecrosis of the capitate is rare. In addition, it can be missed or overlooked and could heal uneventfully as it is usually undisplaced which makes it rare to report[9-11].

The capitate is well protected in the center of the carpus and is rarely injured. Fractures are uncommon and usually heal uneventfully unless the injury is part of the naviculo-capitate fracture syndrome in which the proximal pole rotates through 180 degrees[12]. In these instances, the displaced head frequently becomes avascular[13].

The aim of this presentation is to report a case of capitate bone avascular necrosis, which is a rare disease in pediatric age group.

THE CASE

A twelve-year-old male presented in August of 2010 with a history of mild pain on and off of the right wrist for two years. The patient was active; he fell while playing hockey in June 2010, after which, he started experienced the pain in the wrist. Before the fall, X-ray reported negative findings. A bone scan was done on 23 July 2009, which showed mild to moderate uptake, see figure 1. The patient was referred to with wrist pain and limited range of motion. He attended physiotherapy without improvement. Past medical and surgical history was unremarkable.

On examination, no swelling, bruises, ecchymosis or crepitation of the right wrist was found. Tenderness over the dorsal aspect of the wrist at radiocarpal joint and mild tenderness over the anatomical snuff box with restricted ROM revealed. The patient was treated with a wrist splint and last seen on 12 May 2011. Increased density and sclerotic change of the right capitate bone indicated avascular necrosis of the capitate bone, see figure 2. At three months, bone marrow edema in capitate bone was seen with adjacent minimal joint effusion, see figure 3. At eight months, the lucency seen in the capitate was less evident although still present along with sclerosis in the capitate, see figure 4. At nine months, the amount of bone marrow edema seen in the capitate bone was significantly decreased with only minimal low T1 signal in the capitate, see figure 5.

Clinically, the patient had full ROM and no discomfort. The patient was contacted by phone in July 2011, and his mother reported that he had no discomfort and was back to full physical activity.

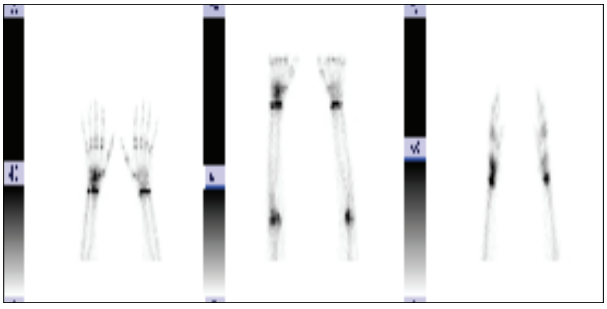

Figure 1: Bone Scan Showing High Uptakes in the Capitate Bone in the Right Wrist

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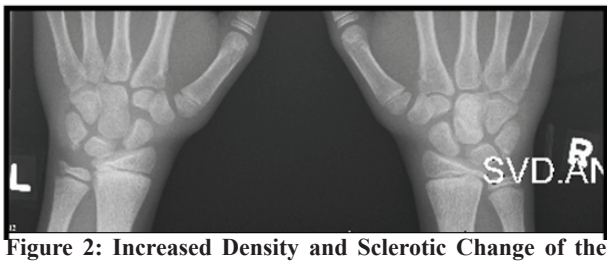

Figure 2: Increased Density and Sclerotic Change of the Right Capitate Bone Indicating Avascular Necrosis of the Capitate Bone

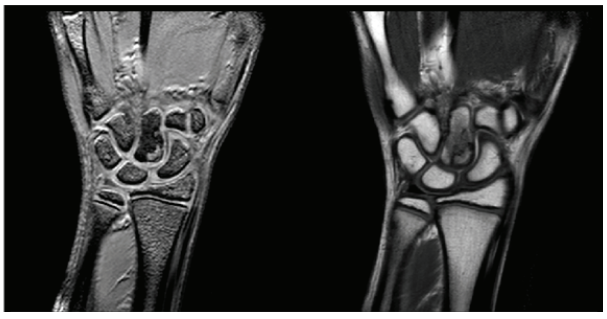

Figure 3: At Three Months – Bone Marrow Edema in Capitate Bone with Adjacent Minimal Joint Effusion

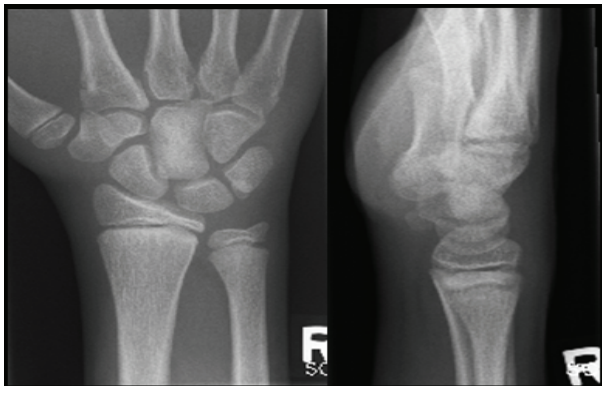

Figure 4: At Eight Months – The Lucency Seen in the Capitate is Less Evident Although Still Present along with Sclerosis Elsewhere in the Capitate

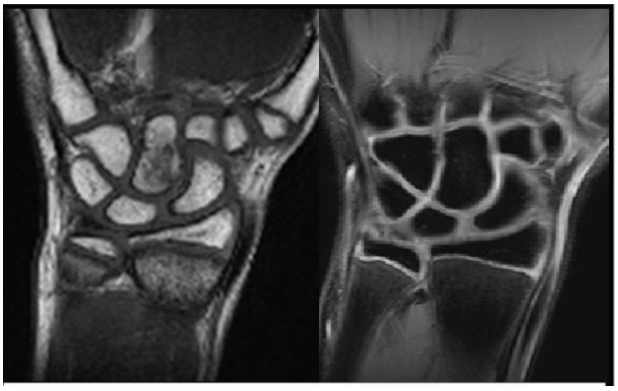

Figure 5: At Nine Months – The Amount of Bone Marrow Edema Seen in Capitate Bone is Significantly Decreased with Only Minimal Low T1 Signal Seen in Capitate

DISCUSSION

Osteonecrosis of the capitate is a rare disorder, which was described in 1942 by Jonsson[14]. Five published studies were found regarding Avascular Necrosis of the Capitate in a pediatric group of 0-18 years in skeletally immature patients. The youngest patient previously reported to have this condition was five years. Osteonecrosis of the capitate has been attributed to several factors, the most common of which was an episode of severe trauma. The typical injury may result in fracture of the capitate bone alone or in association with other broken carpal bones. Osteonecrosis without a history of trauma is much less common compared to the traumatic cases[1,5,6,13].

Osteonecrosis of the carpal bones can be seen easily on MRI; it has been recommended as the preferred imaging modality for all chronic unexplained wrist pain[7]. Kutty and Curtain found that standard radiologic examinations initially were inconclusive[15]. Milliez et al proposed a radiographic classification system for this condition. Their system categorizes osteonecrosis based on the location of involvement in the capitate: **Type I** osteonecrosis, the involvement is limited to the head (proximal pole). **Type II** involves the body. **Type III** involves the entire capitate. This classification system does not serve as a guide for treatment and management.

Three mechanisms may cause fracture of the capitate[1]. First is the direct trauma to the dorsal surface of the bone. Second is a fall on the palm with the wrist in forced extension, which is the most frequent. Third, is a fall on the forcefully flexed hand, which is rare.

In the cases described by Adler and Shaftan, the mechanism was a fall on the palm in eleven cases, on the dorsum in six and a direct blow in two[12]. Rand et al reported a dorsiflexion mechanism in six patients, direct dorsal trauma in one and an unknown mechanism in six[16].

Several occurrences have been documented in patients who did not have a specific traumatic event, but participated in sports such as gymnastics, ice hockey, climbing, and weight lifting[1,6]. It is thought that the repetitive wrist motions necessary for these physical activities may cause microfractures leading to osteonecrosis[8]. There have been three previously reported gymnasts who developed osteonecrosis of the capitate. Other medical conditions have been associated with osteonecrosis of the capitate, which include gout, Gaucher disease and systemic lupus erythematosus (SLE)[8,17]. Osteonecrosis of the capitate also has been associated with steroid therapy and repetitive mechanical vibration[17]. Dorsal instability of the carpus with wrist extension also has been blamed for osteonecrosis of the capitate[6]. Only one occurrence of bilateral osteonecrosis of the capitate has been reported[6]. It occurred in a patient with an anomalous blood supply, carpal instability and trauma[6].

Some authors have attempted immobilization as an initial step in the treatment of osteonecrosis of the capitate. Several different surgical treatments also have been attempted. These include excision of the proximal pole of the capitate with tendon interposition, midcarpal arthrodesis, curettage and bone grafting, partial resection and drilling, posterior interosseous denervectomy, and arthroplasty with silicone prosthesis[16].

CONCLUSION

The plain radiographs, in the presented case were suggestive of osteonecrosis, which was confirmed by MRI. The child was treated conservatively with observation and limited immobilization. Unlike adults with this diagnosis, our pediatric patient achieved full clinical and radiographic recovery after 1 year.

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