Osteochondritis dissecans is a condition affecting a joint in which a variably sized wedge of subchondral bone and its overlying cartilage dies and becomes detached from the articular surface. Although it occurs most often in the knee joint of young male athletes, other reported sites of involvement include the patella, the capitellum, the talar dome, and the femoral head. In contrast, the occurrence of avascular necrosis (AVN), the death of all or part of a bone, has been reported at many joint sites. Several authors have described AVN of the scaphoid, or Preiser's disease.

We report a case with clinical, radiographic, and pathologic features consistent with osteochondritis dissecans of the scaphoid and discuss its possible relationship to Preiser's disease.

CASE REPORT

A 31-year-old male laborer presented to our clinic with complaints of persistent and increasing pain in his left wrist 4 months after the reported onset of symptoms. The patient associated the onset of the pain with an altercation with police, during which he was placed in handcuffs. He denied any bending or twisting of the wrist, but attributed the pain to the tightness of the cuff. There was no other history of trauma or pain in the wrist. He was examined by a physician and radiographs were taken that day. Radiographs of the left scaphoid demonstrated an unusual lesion of indeterminate age, interpreted as a subacute or chronic fracture (Fig 1). The patient's wrist had been immobilized with a splint for 1 month, after which he resumed work.

Four months post injury, our examination revealed a swollen left wrist with tenderness present over the radial half of the carpus. Joint motion was limited. Extension and flexion were 60° and 25°, respectively, compared with 65° and 45° for the right wrist. Radial and ulnar deviation were equal to the contralateral side. Grip strength was 5 lbs, compared to 80 lbs for the right hand. Tomograms further clarified the distribution of the lesion and better characterized its osteochondritis appearance (Fig 2). An operation was recommended because of the persistent pain and disability. Due to the location and nature of the lesion, the scaphoid was considered unsalvageable. A proximal row carpectomy was performed and the scaphoid was removed intact.

Gross examination of the scaphoid showed an irregularly outlined area on the proximal articular surface well demarcated from the surrounding cartilage by a depressed groove (Fig 3). This area corresponded in size, shape, and location to the area of subchondral sclerosis and separation seen radiographically (Figs 1-2). Microscopic sections (made by transecting through the abnormal area of the scaphoid) showed that the lesion consisted of a wedge of necrotic bone containing empty lacunae underly- ing essentially normal articular cartilage (Fig 4). Deep to the dead bone was a zone of fibrinous necrosis similar to that seen with the...
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Fig 4: Microscopic section of articular cartilage and underlying bone from area indicated by arrows in Figure 3. Note dead subchondral bone (bottom) with empty lacunae underlying essentially normal appearing hyaline cartilage (top).

Fig 5: Microscopic section through zone of fibrous necrosis corresponding to lucent area seen on preoperative radiographs. Note disorganized necrotic tissue in marrow spaces as well as fragmented appearing trabeculae of dead bone.

Crescent sign in AVN of the hip (Fig 5). The portions deep to this necrotic area contained normal appearing bone with osteocytes in the lacunae and no necrotic or inflammatory tissue within the marrow spaces. The diagnosis made by the pathology department was osteochondritis dissecans of the scaphoid.

At 8 months follow up, the patient had no pain or tenderness and had returned to heavy labor. Motion was 40° dorsiflexion, 35° volar flexion, 30° ulnar, and 15° radial deviation. Grip strength was still improving and measured 50 lbs, compared to 70 lbs on the right side.

Discussion

In small bones there may not be a clear distinction between osteochondritis dissecans and AVN. The radiographic appearance of osteochondritis dissecans has been described as "a well-circumscribed area of subchondral bone separated from the remaining bone by a crescent-shaped, radiolucent line." This accurately describes the lesion seen in our case, which was pathologically diagnosed as osteochondritis dissecans. However, it also defines the radiographic findings in most of the cases previously reported as AVN of the scaphoid.

There are a limited number of reports in the literature regarding Preiser's disease, the epoynym applied to AVN of the scaphoid. The usual presentation is of gradually increasing pain and swelling in the wrist of a young person with no history of overt trauma, but frequently with a vocation or avocation involving loaded repetitive wrist motion. Radiographically, progressive sclerosis is followed by collapse of the proximal pole. Histologic examination shows localized sequestrum consistent with compromise of the blood supply.

A review of the literature shows one report of bilateral osteochondritis dissecans of the scaphoid. Presentation and findings are virtually identical to those in the reported cases of Preiser's disease, again illustrating that there is not a clear difference between the two conditions.

Repetitive trauma has been suggested as the etiology of both osteochondritis dissecans and Preiser's disease. In a recent report of five cases of AVN of the scaphoid, Ferlic and Morin noted that Preiser's disease, as originally described, is a posttraumatic (overt or repetitive trauma) development, and idiopathic AVN may be a separate entity. Of the 12 cases of AVN of the scaphoid that they found in the English language literature, two were reported by Guelpa et al as osteochondritis dissecans of the scaphoid.

The clinical and pathological findings in the case presented are consistent with a diagnosis of osteochondritis dissecans of the scaphoid. However, they are not distinctly different from those in the previously reported cases of Preiser's disease, or AVN of the scaphoid. The proposed etiologies are often the same and the pathological findings are very similar. Preiser's disease is traditionally thought to be idiopathic AVN of the scaphoid. However, as noted by Ferlic and Morin, there was a traumatic etiology in Preiser's original series. The original radiographic appearance of the scaphoid in this report is not consistent with an acute fracture. Proxi...
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We believe this case represents proximal pole necrosis and separation due to repetitive trauma to the wrist from the patient's occupation as a logger. The presentation and findings are very similar to previously reported cases labeled as both Preiser's disease and osteochondritis dissecans. Based on the cases reported thus far, these diagnoses probably represent the same condition. Most cases of "idiopathic" AVN of the scaphoid are probably the result of unrecognized trauma.

It is important to distinguish this group of patients from those with an acute fracture. Acutely fractured scaphoids will benefit from immobilization, fixation, or excision of the fragment, depending on its size, location, and patient characteristics. None of these treatments, however, would be expected to benefit a scaphoid with diffuse necrosis of the proximal pole, in which only salvage procedures are indicated, such as in the present case.

In summary, for all practical purposes, AVN (Preiser's disease) and osteochondritis dissecans of the scaphoid represent the same condition and consist of necrosis of the proximal pole, with or without a separated fragment, most probably the result of previous repetitive or unrecognized trauma. Differentiation should be made from acute fracture, as the treatment differs.

REFERENCES


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PERONEAL NERVE PALSY SECONDARY TO COMPRESSION FROM AN OSTEochondroma

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Osteochondroma is the most common benign bone tumor. It usually presents as an asymptomatic, hard swelling in adolescence. It rarely will present with symptoms due to a neural deficit. The following case is the only known reported case of a common peroneal nerve palsy secondary to compression from an isolated osteochondroma of the proximal fibula.

CASE REPORT

A healthy 12-year-old boy was admitted to the hospital with a 3-month history of progressive right "ankle weakness" and inability to dorsiflex the foot. The patient denied any associated pain. There was no history of trauma. The weakness appeared to be insidious in onset and slowly progressive.

Medical attention was sought 1 month prior to hospitalization, and electromyographic (EMG) and nerve conduction velocity (NCV) studies revealed a right common peroneal nerve palsy. There were no motor unit action potentials for the anterior tibialis (AT), and 1+/4+ for the extensor hallucis longus (EHL) and extensor digitorum longus (EDL). The patient was initially treated with a rigid ankle foot orthosis (AFO) and electrical muscle stimulator. However, the patient's peroneal function continued to deteriorate.

Two weeks prior to hospitalization he could no longer dorsiflex his toes and had increasing numbness and tingling in his first web space. He was then transferred to the authors' institution, where the orthopedic surgery service was asked to see the patient in consultation.

Physical examination at that time revealed a healthy appearing 12-year-old boy in no distress. Spine exam was normal. A hard, fixed mass 2.5 cm × 1.5 cm was palpable over the right proximal fibula. The mass was nontender to palpation and without associated erythema or warmth. There was no apparent tone or function in the muscles of the anterior compartment (strength 0/5 for AT, EHL, EDL). There was a subjective decrease in light touch in the first web space, but pin prick was intact. Deep tendon reflexes were 2+ and symmetrical. There was no clonus or Babinski responses. He ambulated with a right, drop-foot gait.

Radiographs of the right femur and tibia showed an osteochondroma of the proximal fibula metaphysis (Fig 1). Repeat EMG and NCVs revealed a complete peroneal nerve palsy at the level of the proximal fibula. There

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