The case:

A 43-year-old woman presented with left wrist pain for 6 months not helped by splinting.

Figure: Anteroposterior (A) and lateral (B) radiographs of the left wrist.

Your diagnosis?

For answer see page 637
A 43-year-old woman with diabetic peripheral neuropathy and a history of remote carpal tunnel release and dorsal wrist ganglion excision 7 years earlier presented to an orthopedic surgeon for a second opinion on her left wrist pain, which had persisted for 6 months. The patient reported intermittent throbbing pain on the dorsum of her left wrist that occurred several times a day and was worse with exercise and usual daily activities. She rated this 7 out of 10 on the pain scale. She also noticed decreased range of motion in her left wrist. She had consulted 3 other physicians and had tried splinting, which provided no relief of her pain.

**Physical Examination**

Range of motion was reduced by approximately 30° on left wrist dorsiflexion compared with the contralateral wrist. Wrist palmar flexion, pronation, and supination were intact. There was moderate to severe tenderness directly over the lunate dorsally, along with minimal tenderness over the anatomic snuffbox; no other areas of tenderness existed. Digital motion was good, but sensation was blunted bilaterally as would be expected with peripheral neuropathy.

**Imaging**

The wrist radiographs were interpreted as demonstrating both Kienbock’s disease and scapholunate advanced collapse (SLAC wrist). Diffusely sclerotic collapsed lunate and negative ulnar variance are 2 features of Kienbock’s disease. Widening of the scapholunate interval and proximal migration of the capitate with dorsal tilting of the scaphoid are characteristic of a SLAC wrist.

**Discussion**

Kienbock’s disease is osteonecrosis of the lunate bone, also known as lunatomalacia in the older literature. It is often unilateral and usually seen in 20- to 40-year-old men who perform manual labor and have activity-related dorsal wrist pain.1 Although its exact etiology is unclear, Kienbock’s disease is believed to be related most frequently to trauma and sometimes to negative ulnar variance.2,3 In early Kienbock’s disease, injury-induced vascular compromise in the proximal hand leads to osteonecrosis of the lunate, resulting in pain and stiffness in the wrist. In late stages, the osteonecrotic lunate collapses and leads to further degenerative changes around the PRC. In practice, radiographs are the basis for diagnosing Kienbock’s disease. Lichtman’s classification divides Kienbock’s disease into 5 stages. Stage I has normal radiographic appearance. Stage II demonstrates sclerosis of the lunate. Stage IIIA shows a collapsed lunate without volar tilting of the scaphoid. Stage IIIB displays fixed volar subluxation.

**Diagnosis:**

Kienbock’s Disease and Scapholunate Advanced Collapse

I-Yuan Joseph Chang, MD; Amar Mutnal, MD; Peter J. Evans, MD; Murali Sundaram, MD

**Answer to Radiologic Case Study**

Case facts appear on page 578
of the scaphoid, indicating carpal instability. Perilunate degenerative changes are present in Stage IV. Computed tomography is sometimes used to evaluate for subtle fracture, loose fragments, and early perilunate osteoarthritis in staging Kienbock’s disease. Magnetic resonance imaging (MRI) may demonstrate marrow edema (bright T2 signal) and osteonecrosis (low T1 and low T2 signal) of the lunate. When radiographs yield normal findings in a suspected case of Kienbock’s disease, MRI should be considered to evaluate for marrow edema, which is seen in Stage I. In some centers, perfusion MRI is used to determine marrow viability of the lunate.

Scapholunate advanced collapse of the wrist is the most common degenerative arthritis in the wrist. Unlike Kienbock’s disease, SLAC wrist is seen in slightly older patients, with most being in their 40s. Scapholunate advanced collapse of the wrist is most frequently caused by partial or complete tear of the scapholunate ligament allowing the scaphoid and lunate bones to dissociate. Scaphoid nonunion advanced collapse (SNAC) from chronic nonunited scaphoid fracture is another common cause of SLAC wrist. Radiographic characteristics of a SLAC wrist include radioscaphoid joint degenerative changes, widening of the scapholunate interval, proximal migration of the capitate between the scaphoid and the lunate, and late-stage lunate dorsal tilt resulting in dorsal intercalated segmental instability. Magnetic resonance imaging is highly accurate (90%) in evaluating the degree of scapholunate ligament injury, scapholunate dissociation, and rotatory subluxation of the scaphoid.

Concomitant Kienbock’s disease and SLAC wrist is a rarely reported entity. The authors’ literature search yielded only one 1991 article in which Bourne et al reported 6 such cases. The etiology of such concomitant findings is unclear, and a potential causal relationship between Kienbock’s disease and concurrent SLAC wrist has been in dispute. While many believe that Kienbock’s disease is a cause of SLAC wrist, Taniguchi et al argued that advanced Kienbock’s disease does not cause SLAC wrist because 16 patients (mean age, 30 years) they studied with chronic Stage IV Kienbock’s disease did not develop SLAC wrist. Most recently at the 2012 American Society for Surgery of the Hand annual meeting, Bain proposed Kienbock’s disease advanced collapse as an end stage of Kienbock’s disease that has imaging features of SLAC wrist but that is considered distinct from SLAC wrist due to its unique etiology. Additional studies are needed to improve understanding of the etiology and pathophysiology of Kienbock’s disease before appropriate classification of concomitant Kienbock’s disease and SLAC wrist can occur.

The rarity of the concomitant findings of Kienbock’s disease and SLAC wrist confounds management in a symptomatic patient with restricted movement.

Surgical Intervention

Literature regarding the surgical management of concomitant Kienbock’s disease and SLAC wrist is somewhat sparse. One study suggested that in advanced Kienbock’s disease (Stages IIIA or IIIB), rotatory subluxation of the scaphoid would inevitably lead to SLAC wrist. These authors advocated for scaphotrapeziotrapezoid (STT) fusion to avoid this. However, more recent studies did not find a correlation between the 2 disease processes. In addition, Taniguchi et al found that despite chronic scaphoid subluxation in those with Stage IV Kienbock’s disease, the radioscaphoid joint space was maintained. For this reason, they recommended against STT fusion for Kienbock’s disease. Furthermore, a recent observational study comparing STT with PRC for Stage IIIB Kienbock’s disease revealed that those receiving the latter treatment fared slightly better at 1 year. Although reported in the literature as valid treatment options for Stage IV Kienbock’s disease, some argue that PRC and STT fusion are not beneficial in the presence of wrist arthritis (radiolunate or capitulate). Accordingly, during PRC, mild arthritic changes of the capitate can be addressed with interposition arthroplasty; wrist arthrodesis could be considered as a salvage procedure and wrist denervation could be performed alone or in combination with either intervention.

Conclusion

In SLAC wrist, surgical treatment includes SLAC re-reconstruction (scaphoid excision and arthrodesis of the capitate, lunate, hamate, and triquetrum) for early disease and is precluded by radiolunate arthritis. Proximal row carpectomy is indicated in more advanced stages of SLAC wrist as long as the articular surfaces of the proximal capitate and lunate fossa are free of significant pathology.

References