

Distal Ulna Fracture With Delayed Ulnar Nerve Palsy in a Baseball Player

Charles B. Pasque, MD, Clark Pearson, ATC, Bradley Margo, MD, and Robert Ethel, BS

Abstract

We present a case report of a college baseball player who sustained a blunt-trauma, distal-third ulna fracture from a thrown ball with delayed presentation of ulnar nerve palsy. Even after his ulna fracture had healed, the nerve injury made it difficult for the athlete to control a baseball while throwing, resulting in a delayed return to full baseball activity for 3 to 4 months. He had almost complete nerve recovery by 6 months after his injury and complete nerve recovery by 1 year after his injury.

Ulnar nerve injury leads to clawing of the ulnar digits and loss of digital abduction and adduction because of paralysis of the ulnar innervated extrinsic and intrinsic muscles. Isolated motor paralysis without sensory deficit can occur from compression within the Guyon canal.¹ Cubital tunnel at the elbow is the most common site for ulnar nerve compression.² Compression at both levels can be encountered in sports-related activities. Nerve compression in the Guyon canal can occur with bicycling and is known as *cyclist's palsy*,³⁻⁶ but it can also develop from canoeing.⁷ Cubital tunnel syndrome is the most common neuropathy of the elbow among throwing athletes, especially in baseball pitchers and can result from nerve traction and compression within the fibro-osseous tunnel or subluxation out of the tunnel.² Both compression syndromes can develop from repetitive stress and/or pressure to the nerve in the retrocondylar groove.

Ulnar nerve palsy may be associated with forearm fractures, which is usually caused by simultaneous ulna and radius fractures, especially in children.⁸⁻¹² To our knowledge, there are no reports in the literature of an ulnar nerve palsy associated with an isolated ulnar shaft fracture in an adult. We report a case of delayed ulnar nerve palsy after an ulnar shaft fracture in a baseball player. The patient provided written informed consent for print and electronic publication of this case report.

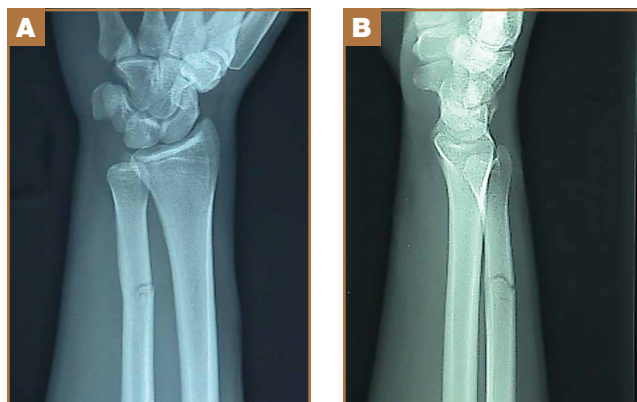
Case Report

A 19-year-old, right hand-dominant college baseball player was batting right-handed in an intrasquad scrimmage when

a high and inside pitched ball from a right-handed pitcher struck the volar-ulnar aspect of his right forearm. Examination in the training room and emergency department revealed moderate swelling and ecchymosis over the distal third of the ulna. He had a normal neurovascular examination, including normal sensation to light touch and normal finger abduction/adduction and wrist flexion/extension. He was otherwise healthy. Radiographs of the right forearm showed a minimally displaced transverse fracture of the distal third of the ulna (Figures 1A, 1B).

The patient was initially treated with a well-padded, removable, long-arm posterior splint for 2 weeks with serial examinations each day in the training room. At 2-week follow-up, he reported less pain and swelling but stated that his hand had "felt funny" the past several days. Examination revealed clawing of the ulnar digits with paresthesias in the ulnar nerve distribution (Figures 2A, 2B). His extrinsic muscle function was normal. Radiographs showed stable fracture alignment. Ulnar neuropathy was diagnosed, and treatment was observation with a plan for electromyography (EMG) at 6 weeks after injury if there were no signs of nerve recovery. Physical therapy was instituted and focused on improving intrinsic muscle and proprioceptive functions with the goal of an expeditious, but safe, return to playing baseball. Three weeks after his injury,

Figure 1. (A) Anteroposterior view and (B) lateral view of radiographs at time of injury, showing minimally displaced distal-third ulna shaft fracture.



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the patient had decreased tenderness at his fracture site and was given a forearm pad and sleeve for light, noncontact baseball activity (Figure 3). A long velcro wrist splint was used during conditioning and when not playing baseball. Forearm supination and pronation were limited initially because of patient discomfort and to prevent torsional fracture displacement or delayed healing. Six weeks after his injury, the patient returned

to hitting and was showing early signs of improved sensation and intrinsic hand strength. He had progressed to a light throwing program and reported difficult hand coordination, poor ball control, and overall difficulty in accurately throwing over the next 3 to 4 months. Because of his difficulty with ball control, the patient began a progressive return to full-game activity over 6 weeks, which initially included a return to batting only, then playing in the outfield, and, eventually, a return to his normal position in the infield. Serial radiographs continued to show good fracture alignment with appropriate new bone formation (Figures 4A, 4B). Normal motor strength was noted at 3 months after injury and normal sensation at 4 months after injury.

By the end of his summer league, 6 months after his injury, the patient was named Most Valuable Player and had a batting average over .400. He reported near-normal hand function. One year after injury, his examination revealed normal hand function (Figure 5), including normal sensation to light touch, 5/5 intrinsic hand function, and symmetric grip strength. Radiographs showed a healed fracture (Figures 6A, 6B). The patient has gone on to play more than 9 years of professional baseball.

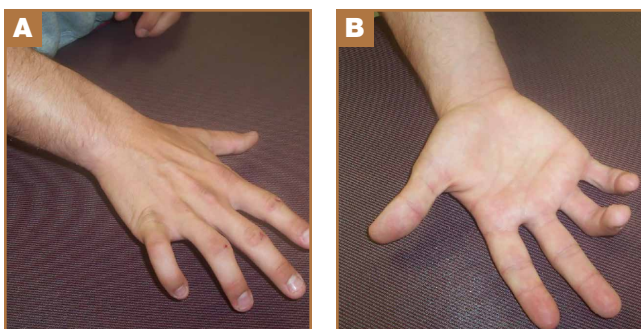


Figure 2. (A, B) Two-week follow-up examination, showing ulnar digit clawing.



Figure 3. Custom forearm pad and sleeve for return to baseball activity.

Discussion

The ulnar nerve has a course that runs down the volar compartment of the distal forearm. The flexor carpi ulnaris provides coverage to the nerve in this area. Proximal to the wrist, the



Figure 5. One-year follow-up examination, showing normal forearm and hand appearance, emphasizing change from ulnar digit clawing during initial examination in Figure 2. Pencil tip is on fracture site.



Figure 4. (A) Anteroposterior view and (B) lateral view of 6-week follow-up radiographs, showing hypertrophic callus formation.

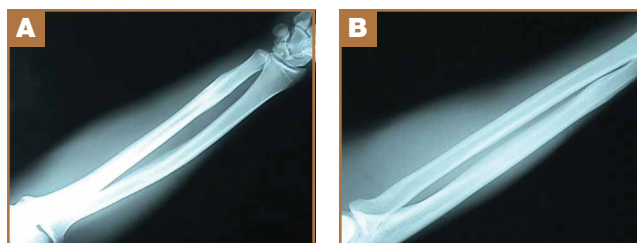


Figure 6. (A) Anteroposterior view and (B) lateral view of 1-year follow-up radiographs, showing complete fracture healing and remodeling.

nerve emerges from under the flexor carpi ulnaris tendon and passes deep to the flexor retinaculum, which is the distal extension of the antebrachial fascia and blends distally into the palmar carpal ligament.¹³ In our patient, the most likely cause of this presentation of ulnar neuropathy was the direct blow to the nerve from the high-intensity impact of a thrown baseball to this superficial and exposed area of the forearm. Since the patient presented with delayed paresthesias and ulnar clawing 2 weeks after injury, possible contributing causes could be evolving pressure or nerve damage from a perineural hematoma and/or intraneural hematoma or increased local pressure from intramuscular hemorrhage.¹⁴ There are both acute and chronic cases of ulnar nerve entrapment by bone or scar tissue that resolved by surgical decompression.⁸⁻¹² Surgical exploration was not deemed necessary in our case because the fracture was minimally displaced, and the patient regained sensation and motor function over the course of 3 to 4 months.

Nerve injuries can be classified as neurapraxia, axonotmesis, or neurotmesis. Neurapraxia is the mildest form of nerve injury and neurotmesis the most severe. Neurapraxia may be associated with a temporary block to conduction or nerve demyelination without axonal disruption. Spontaneous recovery takes 2 weeks to 2 months. Axonotmesis involves an actual loss of axonal continuity; however, connective tissue supporting structures remain intact and allow axonal regeneration. Finally, neurotmesis is transection of the peripheral nerve, and spontaneous regeneration is not possible. The mechanism of injury in our patient suggests that the pathology was neurapraxia.^{1,15}

Management of these injuries should proceed according to basic extremity injury–care practices. Initial care should include thorough neurovascular and radiographic evaluations. If nerve deficits are present with a closed injury and minimal fracture displacement, treatment can include observation and serial examinations with a baseline EMG, or waiting until 4 to 6 weeks after injury to obtain an EMG if there are no signs of nerve recovery. Early EMG testing and surgical exploration may be warranted if there is a concern for nerve disruption or entrapment, such as marked fracture displacement or an open injury. Additional early-care measures should include swelling control modalities and immobilization based on the type of fracture. Ultrasound was not readily available at the time of our patient's injury, but it may be a helpful adjunct in guiding decision-making regarding whether to perform early surgical exploration for hematoma evacuation or nerve injury.¹⁶⁻¹⁸

Our case report was intended to provide an awareness of the unusual association between an isolated ulnar shaft fracture and a delayed ulnar nerve palsy in an athlete. Nerve injuries may be unrecognized in some patients in a trauma situation, since the focus is usually on the fracture and the typical patient does not have to return to high-demand, coordinated athletic activity, such as throwing a ball. Because of the possible delayed presentation of these nerve injuries, close observation of nerve function after ulna fractures from blunt trauma is warranted.

Dr. Pasque is Professor, Program Director, and Team Physician, Department of Orthopaedic Surgery and Rehabilitation, University of Oklahoma College of Medicine, Oklahoma City, Oklahoma. Mr. Pearson is an Athletic Trainer, Department of Athletics, Auburn University, Auburn, Alabama. Dr. Margo is in private practice, McBride Clinic, Oklahoma City, Oklahoma. Mr. Ethel is Medical Student, Department of Orthopaedic Surgery and Rehabilitation, University of Oklahoma College of Medicine, Oklahoma City, Oklahoma.

Address correspondence to: Charles B. Pasque, MD, Department of Orthopaedic Surgery and Rehabilitation, University of Oklahoma College of Medicine, P.O. Box 26901, Suite WP-1380, Oklahoma City, OK 73190 (tel, 405- 271-4426; fax, 405-271-3461; email, charles-pasque@ouhsc.edu).

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