Loss of Hand Function in a Collegiate Baseball Player
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A 19-year-old male baseball catcher sustained an injury to his right forearm. He reported to the athletic training room prior to practice complaining of a strange sensation in the posterior upper arm combined with throbbing (4/10) throughout the day. No mechanism of previous history of injury was reported. He also reported a decreased ability to grip a baseball and inability to hold a pencil. Observation showed no abnormalities. No swelling, ecchymosis, or obvious deformities were present. Palpation of the humerus, radius, ulna, carpal bones, phalanges, and associated muscles appeared WNL and no area of palpation produced pain. Upper extremity dermatomes were WNL. Shoulder, elbow, and cervical ROM were WNL. Resistive wrist flexion was 4+/5. First and second phalanges showed decreases in active distal interphalangeal joint (DIP) flexion. All passive phalangeal ROM were WNL. Resistive ROM into IP flexion of the thumb was 1/5. Valgus stress and Allen’s were negative, while the “O” test was positive. Upper extremity myotomes showed decreased strength in nerve roots C7 & C8. The athlete was removed from play and referred to the team physician. The differential diagnosis at this time included Flexor Pollicis Longus (FPL) strain, Flexor Digitorum Profundus (FDP) strain, C8 pathology, brachial plexus, cervical nerve root compression, carpal tunnel syndrome. Three days post onset the athlete underwent an MRI of the right distal arm, which identified no significant findings. At 7 days he was seen by the team physician and diagnosed with anterior interosseous nerve (AIN) palsy. A conditioning program was implemented as well as light upper extremity strengthening, emphasizing the biceps and triceps with avoidance of gripping. The MD referred the athlete to a neurologist for EMG testing. The EMG showed isolated denervation of the FDP along with the FPL. The findings were consistent with AIN syndrome (Kiloh-Nevin Syndrome), along with FPL and FDP weakness (3/5). The neurologist prescribed an antiinflammatory and vitamin B complex. At this time (4 weeks) the athlete was cleared to hit only, first off a tee and then against live pitching. The neurologist also prescribed occupational therapy (OT) over the summer months. Due to scheduling conflicts the athlete did not begin OT until approximately 2 months post onset. OT consisted of muscle re-education via electrical stimulation, as well as strengthening activities. No gains were noted after one month, and the athlete ceased OT and sought a third opinion. At 4 months he visited a hand specialist who confirmed AIN. This physician established the injury to be the result of post viral neuropathy, an inflammatory immune mediated response that left the nerves vulnerable to damage. The final proposed cause was friction caused by repetitive throwing. The MD cleared the athlete for full RTP. At 6 months post the athlete still lacks strength and active ROM in his first and second DIP joints and still produces a positive “O” sign. AIN syndrome is a rare occurrence that comprises less than 1% of all upper extremity nerve palsies. The most common mechanisms of AIN are compression by the pronator quadratus or other local muscles, or a traumatic fracture, which can result in damage to the AIN. The mechanism for this particular injury is one that does not occur often. The uniqueness of this injury coupled with its abnormal mechanism is important to understand. It is unlikely that every athlete who suffers a viral infection will acquire some type of neuropathy; however, it is important to keep this injury in mind when evaluating athletes with loss of sensation.