

Bilateral Posterior Fracture-Dislocation Of The Shoulder As A Presentation Of An Intracranial Tumor

P Rhee, C Main

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Abstract

Bilateral posterior-fracture dislocations of the shoulder are pathognomonic for seizure induced injuries. Patients that have not been diagnosed with a seizure disorder will often present with nocturnal injuries and no history of trauma. The allusive nature of this injury is compounded by mild presenting signs and symptoms and possible autoreduction with subsequent seizures. Therefore, a high degree of suspicion must be maintained for a seizure disorder as the cause for nocturnal injuries or posterior dislocations at the glenohumeral joint. Among the various causes of seizure disorders, we present a case in which a bilateral posterior-fracture dislocation served as a presenting sign of an intracranial tumor.

INTRODUCTION

Bilateral posterior fracture-dislocations of the shoulder are uncommon orthopedic injuries virtually pathognomonic for convulsion related trauma. The mechanism of trauma can be attributed to an adducted and internally rotated state.

Convulsions due to seizures or electrocutions exploit the inherent muscular imbalance between the strong internal rotators (teres major, subscapularis, and pectoralis muscles) and the relatively weaker external rotators (infraspinatus and teres minor muscles).

A few cases have been reported in which a nocturnal fracture has been the presenting feature of unrecognized epileptic seizures. One such case, by Aboukasm et al, illustrates a patient who awoke with severe midback pain. The work-up revealed an undiagnosed epilepsy as the cause of the fracture.¹ Similarly, another case by Rupprecht et al, depicted a patient with a double thoracic, humerus, and scapula fracture with an unrecognized cerebral astrocytoma which caused a tonic-clonic seizure.² Therefore, it is imperative to assess the possibility of an epileptic seizure as the cause of trauma in nocturnal idiopathic injuries.

Patients who present with a nocturnal occurrence of posterior fracture-dislocation will not recall any recent trauma and the mild physical findings can make this diagnosis allusive. The importance of an axillary view or a scapular Y view is stressed to assess the glenohumeral joint when a seizure-induced injury is suspected and when there is marked limitation in external rotation and forward flexion at

the glenohumeral joint. As suggested, a high degree of suspicion must be present when dealing with an injury related to a nocturnal convulsive episode.

CASE REPORT

A 62-year-old right-handed man presented to his primary care physician with complaints of right shoulder pain after awaking with no known injuries. He denied any recent falls, traumas, and lifting injuries. Initial exam demonstrated a displacement of the biceps muscle and ecchymosis in the axillary fold. Initially, he had noticed quite a bit of pain and swelling which progressed to weakness, limited ROM, and ecchymosis overlying the anterior and axillary region of his right upper extremity.

On examination, there was a considerable loss of passive ROM and pain in forward flexion and abduction past 30° at the right glenohumeral joint. His right elbow flexion/extension arc was also limited and painful. He complained of pain with passive pronation and restricted supination. Grossly, pulses were 2+ and there were no neurologic deficits noted. Radiographs of the right shoulder, AP (Figure 1) and axillary (Figure 2), revealed a fracture-dislocation through the anatomical neck of the proximal humerus as well as comminution around the proximal metaphyseal region. Due to the non-traumatic nature of this injury, an MRI was scheduled to determine the possibility of a tumor and to assess the biceps muscle. The MRI displayed the biceps tendon to be medially subluxated and the marrow pattern did not appear to be consistent with necrosis or

tumor.

Figure 1

Figure 1: AP view of right glenohumeral joint. This view illustrates a fracture through the anatomical neck of the proximal humerus. (RheeFigure 1)



Figure 2

Figure 2: Axillary view of the right glenohumeral joint. This view captures the posterior dislocation of the humeral head. (RheeFigure 2)



At 15 days post-injury, the patient underwent a right shoulder hemiarthroplasty at which point it was noted that the displaced humeral head was dislocated in the

retroglenoid position. Articular head fragments were removed and the biceps tendon was noted to be medially subluxated and intact. The rotator cuff tendons were repaired to the implant. Following the repair, the biceps tendon was then replaced in the bicipital groove and secured with a soft tissue leaflet. Satisfactory passive ROM was noted in all planes. The patient was discharged at post-op day 3 and scheduled for physical therapy.

At 7 days post-op from the right shoulder hemiarthroplasty, the patient was found in his recliner by his wife and presented to the emergency room with a comminuted fracture-dislocation of the left proximal humerus (Figure 3). A CAT scan was obtained of his head to rule out any cause of seizures, which revealed a 1.5 cm x 2mm intracranial tumor overlying the posterior right frontal lobe with surrounding edema (Figure 4). He was referred to neurosurgery for further evaluation.

Figure 3

Figure 3: Axillary view of the left glenohumeral joint. This view illustrates a fracture through the left proximal humeral and the humeral head in a retroglenoid position. (RheeFigure 3)

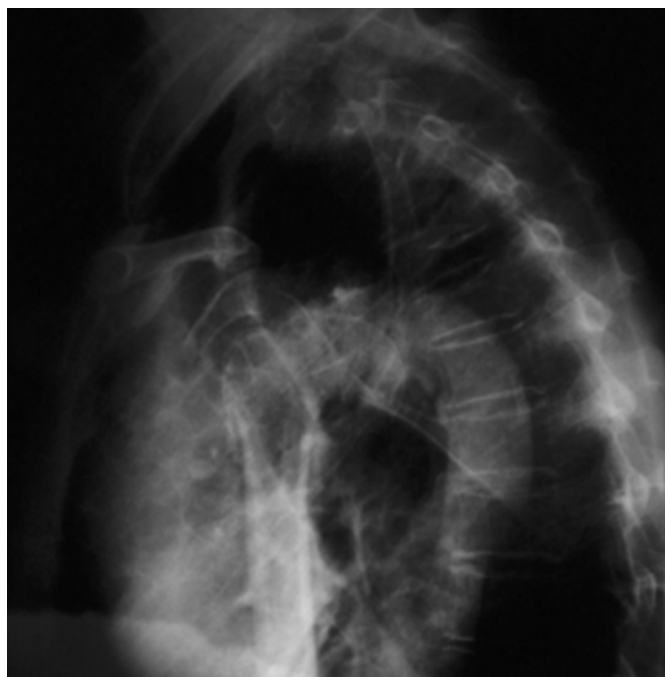
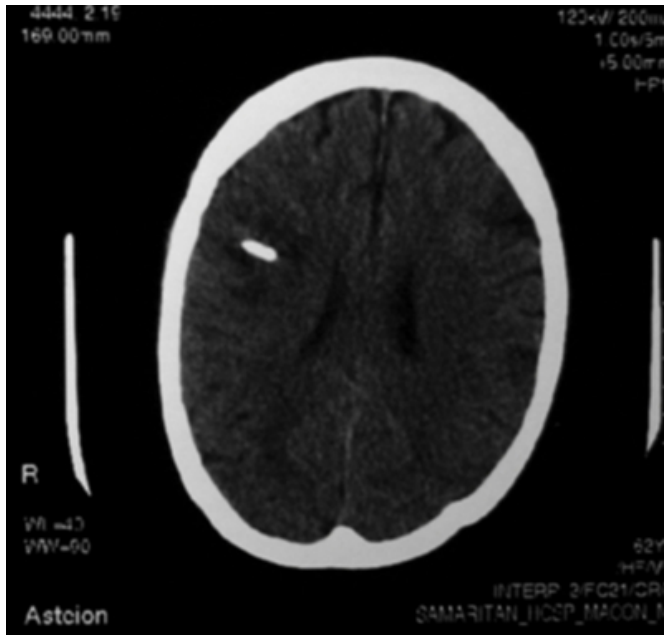


Figure 4

Figure 4: CAT scan of head. An area of calcification is shown in the posterior right frontal lobe with surrounding edema. (RheeFigure 4)



A MRI of the head was obtained at 11 days post-fracture-dislocation of the left shoulder, revealing a right perisylvian T2 signal abnormality with mildly effaced sulci. An additional CT of the head indicated a right frontoparietal region of linear calcification with minimal mass effect suggestive of an infarction or malignancy. Stereotactic brain biopsy was unsatisfactory as there appeared to be an area of fibrous tissue through which the needle could not be advanced. Adequate specimens sent to pathology indicated no tumor and the suggested the presence of a blood clot. The patient was cleared to undergo a left shoulder hemiarthroplasty at 20 days post-injury with further neurosurgical work-up post-op.

The anterior capsule and subscapularis tendon were noted to be intact, however, there was an avulsion of the supraspinatus and the anterior half of the infraspinatus off of the greater tuberosity fragment. Once again, the displaced humeral head was dislocated in a retroglenoid position. All fragments were removed and the biceps tendon was noted to be intact. The rotator cuff tendons were repaired to the implant itself and to the remaining cuff of tissue off the margin of the greater tuberosity. Satisfactory passive ROM was noted in all planes. At 19 days post-op, radiographs obtained demonstrated the glenohumeral joints to be in good position bilaterally and no neurologic deficits were noted on physical exam.

On day 21 of the left shoulder hemiarthroplasty, the patient underwent a right frontotemporal craniotomy to obtain a soft lesion with significantly fibrotic portions for diagnostic work-up. Continuous motor mapping indicated that the areas associated with the lesion did not have any motor function. No sensation and movements were elicited to stimulation. The lesion was resected and sent to pathology, which revealed an oligodendroglioma (WHO Grade II).

The patient is currently undergoing physical therapy for the bilateral shoulder posterior fracture-dislocations and has not experienced any further seizures post-resection. He is medically maintained on Phenytoin and is expected to have a prolonged course of rehabilitation.

DISCUSSION

This case report illustrates a nocturnal, bilateral humeral posterior fracture-dislocation as a presenting feature of an intracranial tumor. Bilateral posterior fracture-dislocations are an uncommon injury that has been highly associated with a convulsive episode in 78% of the cases reported.³ A history that reveals a nocturnal injury should include a seizure disorder in the differential as a cause of the trauma.

Once a seizure-induced trauma is suspected, a search for the underlying cause of the convulsive disorder must be established. We present a case in which an intracranial tumor was the cause of a seizure-induced bilateral posterior fracture-dislocation. There has been only one other case reported in which a bilateral shoulder dislocation served as the initial manifestation of a brain tumor. Yet, in that case, the patient's shoulders were dislocated in the opposite direction and the posterior dislocation was recognized with a 16 day delay.⁴

However, other causes of seizures have been found to cause bilateral shoulder fracture-dislocations. One case was reported in which a bilateral shoulder fracture and unilateral posterior dislocation was due to a tonic-clonic seizure in an episode of alcohol withdrawal.⁵ Even more in parallel to the case presented can be other serious nocturnal musculoskeletal injuries associated with insulin-dependent diabetes mellitus (IDDM) patients and insulin-induced nocturnal hypoglycemia.⁶

Posterior shoulder dislocations are often missed with initial diagnosis due to relatively mild presenting signs and symptoms. In patients with a history of seizures or electrocution, who present with limited external rotation at the glenohumeral joint, an axillary view or modified axillary

view should be obtained in order to rule out a posterior dislocation.⁷ In a unilateral presentation of a possible seizure-induced shoulder trauma, it is extremely important to radiograph the contra-lateral shoulder as 30% of cases present bilaterally.⁸ These injuries can also be missed on initial diagnosis due to an auto-reduction of the posterior dislocation with subsequent seizures.⁹ Therefore, the importance of a high degree of suspicion is warranted.

Nocturnal injuries in which the patient cannot recall any trauma must raise the suspicion of a convulsive disorder as the cause of the injury. The inherent biomechanics of the glenohumeral joint gives the clinician an additional clue that an atraumatic bilateral posterior fracture-dislocation could be attributed to a seizure. However, once a convulsive injury has been determined, the source of the seizure must be assessed. Among the many causes of convulsions, we present an unusual presentation of a seizure induced injury due to an intracranial tumor.

CORRESPONDENCE TO

Peter Rhee, B.A. (MSIII)
5345 E. Van Buren St. #251
Phoenix, AZ 85008
E-mail: none
Phone: (480) 650-4318
Fax: (602) 728-0979

Chris Main, D.O 1706 Prospect Dr. Macon, MO 63552 E-mail: prhee@atsu.edu Business: (660) 385-1006 Fax: (660) 385-1028

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Author Information

Peter Rhee, B.A. (MSIII)

Midwest Bone & Joint Center PC

Chris Main, D.O.

Midwest Bone & Joint Center PC