CASE REPORT

Bilateral posterior four-part fracture—dislocations of the shoulders following electric shock
A case report and literature review

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Introduction

Of all shoulder dislocations, 4% are posterior and 1% are associated with fractures. However, when one considers bilateral dislocation, 45% are posterior. This is due to the markedly different aetiologies between unilateral and bilateral dislocation. The so-called ‘triple E syndrome’ of epilepsy, extreme trauma and electric shock has been coined. Approximately 50% of bilateral posterior dislocations are due to convulsive seizures. If the dislocations are associated with fractures this rises to over 90%, leading some authors to suggest that this injury is pathognomonic of a seizure. Electric shock accounts for <5% of these injuries. Diagnosis can be difficult and is often delayed and many treatments have been carried out, from conservative management to total shoulder replacement.

Bilateral posterior fracture—dislocation is a very rare injury. A literature search has revealed only 30 reported cases. The case below is, we believe, the first presented of bilateral posterior four-part fracture—dislocations of the shoulders due to electric shock.

Case report

A 63-year-old male, retired electrician, presented to accident and emergency following electric shock. He had bilateral burns to the hands, full thickness in places and painful, stiff shoulders. There was no other injury noted at that time and no neurovascular deficit was documented. X-rays (Fig. 1) of the shoulders showed bilateral, comminuted, displaced proximal humeral fractures.

He was referred to the plastic surgeons regarding the burns and he was admitted to an orthopaedic ward for management of the shoulder injuries. He had a CT scan of both shoulders (Fig. 2) which confirmed bilateral posterior four-part fracture—dislocations. The plastic surgeons elected to treat his hand wounds conservatively by way of dressings and observation. It was not thought to present a serious infection risk.

Five days following admission he underwent bilateral shoulder hemiarthroplasties (Neer prosthesis). Post-operatively he was immobilized in poly-slings for 1 week after which passive mobilisation with the physiotherapists was commenced. At 4 weeks he started assisted-active mobilisation progressing to active after 2 months. Post-operative X-rays are shown in Fig. 3.
Figure 1  Plain anteroposterior radiographs of right (a) and left (b) shoulders at presentation.
At 3-month follow-up his pain had significantly decreased. Active abduction was to $60^\circ$ on the right and $80^\circ$ on the left. He was having difficulty in operating above shoulder height. Physiotherapy was continuing and it was advised to increase exercised to maximise strength and range of movement. He also mentioned a sensation of pins and needles, particularly along the ulnar borders of both hands. There was no loss of power on examination. A referral to neurophysiology for nerve conduction studies was made.

At 6 months he was pain free and although his range of movements were still limited his activities of daily living were unhindered and he was able to drive his automatic car. He still complained of bilateral paraesthesiae in the hands, and, on examination some wasting of the right hypothenar eminence compared to the left was noted. The nerve conduction studies reported a partial right brachial plexus injury affecting the ulnar nerve fibres. There was no distal lesion and the left side was normal. The patient was not unduly concerned regarding these symptoms.

He was reviewed again 1 year post injury. He remained pain free at rest and his paraesthesiae were improving. His Constant scores were 49 on the right and 57 on the left. He has been discharged from follow-up.

Discussion

This case demonstrates a particularly severe presentation of bilateral posterior fracture dislocations of the shoulders treated successfully by hemiarthroplasties. Given the very small numbers of similar cases and the complexity of injury, there is no accepted protocol for their management.

The proposed mechanism of shoulder injury during convulsive seizures has been well described. It is likely that electric shock, causing violent muscular contractions, is similar. The entry point of electricity in this case was the right hand, exiting via the left. This may explain why injury was limited to the upper limbs and why the fractures were so severe. The normal muscle tone around the shoulder is largely responsible for joint congruity. Tonic contraction of all the muscles allows the stronger flexors, adductors and internal rotators to predominate, dislocating the shoulder posteriorly. Continued forceful contraction pushes the internally rotated humeral head into the posterior aspect of the glenoid causing a compression fracture (reversed Hill–Sachs lesion).9 Avulsion fractures of muscular origins lead to greater and lesser tuberosity fractures and pull of the strong adductors against deltoid and supraspinatus leads to fractures of the humeral neck. In a study of 73 posterior fracture—dislocations in 66 patients, only 3 were found to be four-part fractures.5

Although in this case diagnosis was not difficult, one particular pitfall of this type of injury is that diagnosis is often delayed,4 up to 50% not being correctly identified at first presentation.16 The retrospective history is often of a previously undiagnosed epileptic who wakes up with stiff painful shoulders and no memory of a traumatic event.
He or she either doesn’t present immediately or is misdiagnosed as having a soft-tissue injury or a ‘frozen shoulder’. Abnormal movement at the fracture site may be mistaken for gleno-humeral movement on examination and simple AP radiographs of the shoulder can look surprisingly normal. This illustrates how accurate history taking and examination coupled with at least AP and axillary views of the shoulder is essential when assessing any shoulder complaint. It has been shown that delay in diagnosis and treatment has an adverse effect on outcome. Some authors suggest excellent or good results can be obtained if treatment occurs within 2 years of injury, others recommend definitive treatment within 6 months. This probably represents a continuum whereby scarring, muscle atrophy and capsulitis develop over time, progressively hindering efforts to surgically restore anatomy and function.

For complex, multi-part fractures, or if there is any doubt regarding the potential viability of the humeral head, CT scanning is extremely helpful. It aids not only in the diagnosis, but is invaluable in planning surgery. Fractures through the anatomical neck of humerus are associated with a high risk of avascular necrosis (AVN) and in Neer’s four-part fracture–dislocations, the risk can be as high as 90%. The treatment should be tailored to the configuration of the injury and adapted to the individual patient, dependent on age, occupation and desired levels of activity. Conservative management should always be considered and is likely to be appropriate in cases where closed reduction of the dislocation is
possible, the fracture is minimally displaced and the viability of the humeral head is not in doubt. There should be a period of immobilisation to allow the soft tissues to heal followed by progressive mobilisation from passive to assisted-active to active until no further gains are seen. Procedures to resolve any resultant instability can be performed as required. It has been shown that good results depend not only on early diagnosis and treatment but also on early and continued physiotherapy.

For displaced fractures especially in the younger patient, surgical intervention is indicated. Two-part fractures have been successfully treated in the acute setting by open reduction and internal fixation with cannulated screws, acute osteochondral grafting and hemiarthroplasty. Excision of the humeral head or arthrodesis have both been shown to have poorer outcomes and are not recommended. Altay et al, reported a series of 10 four-part fracture—dislocations of the shoulder treated by limited open reduction and percutaneous fixation. Good results were achieved in nine patients with one case of AVN of the humeral head leading to a poor outcome. This is in contrast to the reported 90% AVN rate in these injuries. All nine good results had at least 1 cm of the neck part of the head fragment intact and it is therefore inferred that the blood supply to the humeral head remains at least partially intact in these cases.

If, in addition to humeral head involvement, there is also damage to the glenoid, total shoulder arthroplasty may be considered. Cheng et al, reported a series of seven shoulders in which the fracture was minimally displaced and a viable humeral head remained at least partially intact. The acute treatment may be summarised thus.

1. **Reducible dislocation**, undisplaced fracture, minimal articular surface involvement and viable humeral head—treat conservatively.
2. **Non-reducible dislocation** or displaced fracture with minimal articular surface involvement and a viable humeral head—open reduction, internal fixation.
3. **Articular surface involvement or non-viable humeral head**—hemiarthroplasty.
4. **Involvement of humeral and glenoid articular surfaces**—total shoulder arthroplasty.

Although this is a rare injury, the consequences of both arms functioning poorly is devastating to the patient’s life and livelihood. A high clinical suspicion coupled with good examination and appropriate radiology should greatly increase the chance of early diagnosis and therefore treatment. Patients must be informed that the severity of this injury means their shoulders will never be normal again. However, timely and appropriate surgery carried out by a specialist surgeon followed by early mobilisation and physiotherapy should maximise their functional outcome.

References

