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Management of recurrent shoulder instability in patients with epilepsy

Tanujan Thangarajah, MB, ChB (Hons), MRCS, MSc*, Simon M. Lambert, BSc, FRCS, FRCSEd (Orth)

The Shoulder and Elbow Service, The Royal National Orthopaedic Hospital, Stanmore, Middlesex, UK

Epileptic seizures can cause dislocation of the shoulder. Recurrent instability is common and occurs soon after the first dislocation. Significant bone loss from the humeral head and glenoid is thought to be responsible for this, and therefore the majority of surgical approaches focus on bone augmentation of the glenohumeral joint. Understanding of the current management strategies and the anatomic lesions associated with seizure-related shoulder instability will guide clinical decision-making. The purpose of this article was to review the pathoanatomy, treatment options, and clinical outcomes of seizure-related shoulder instability.

Level of evidence: Narrative Review

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Epilepsy and shoulder instability

Epilepsy is the most common serious neurologic condition in adults and affects up to 60 million people worldwide.[47] More than 30% of patients are unable to control their seizures with medical treatment and develop musculoskeletal injuries.[36,42] The incidence of shoulder dislocation during a seizure is approximately 0.6%, but this may be an underestimation because many go undetected.[16,61] Patients are typically young, active men between the ages of 20 and 30 years.[67] Index dislocation often coincides with a seizure in the absence of direct trauma, with subsequent episodes occurring during normal daily activities and further seizures.[67] Recurrent instability is common and occurs in more than two-thirds of cases, with the incidence of anterior and posterior instability differing between published reports.[10,67]

Treatment protocols vary and encompass a wide range of soft tissue (Putti-Platt, Bankart repair) and bone augmentation strategies (Eden-Hybinette, coracoid transfer, glenoid neck/humeral osteotomy, arthrodesis).[10] Despite several technically satisfactory procedures, some patients experience persistent instability and are unable to cope with the disabling symptoms.[67] After surgery, the recurrence rate in the epileptic population is higher than in nonepileptic groups (69% vs. 10%).[33,67] This is due to ongoing seizure activity, poor compliance with anticonvulsant therapy, significant combined bone loss from the glenoid and humeral head, and the young age at which surgery is undertaken.[8,42,67]

The purpose of this article was to describe the pathoanatomy, treatment options, and clinical outcomes of seizure-related shoulder instability to provide evidence-based guidelines for its management.

*Reprint requests: Tanujan Thangarajah, MB, ChB (Hons), MRCS, MSc, The Shoulder and Elbow Service, The Royal National Orthopaedic Hospital, Stanmore, HA7 4LP, UK.

E-mail address: tanujan1@hotmail.com (T. Thangarajah).

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Pathoanatomy

Relatively little is known about the relationship between the type of instability and the pattern of structural lesions in patients with epilepsy. Bone loss from the humeral head, glenoid, or both is common and occurs from both the force of the dislocation and underlying metabolic bone disorders associated with anticonvulsive therapy.[42,67] Bone defects occur more frequently in the epileptic population compared with non–seizure-related cases of recurrent shoulder instability.[10,48,65,67]

Posterior instability is a common finding in the epileptic population, but there are few reports describing associated bone defects.[10,40] In a systematic review, Longo et al[40] demonstrated that in cases of posterior instability, glenoid bone loss was found in 9%, humeral head bone loss in 39%, and bipolar bone loss in 2% of shoulders. Early identification, characterization, and targeted management of these lesions are imperative as they have been recognized as independent risk factors for failure of surgical treatment.[7,11] Few studies have examined bone loss specifically in an epileptic population, and so the basic principles of treatment need to be extrapolated from the non–seizure-related instability population.[10,67]

Glenoid bone loss

Identification of glenoid bone loss is essential in planning of any stabilization procedure. A "glenoid rim lesion" represents either an acute fracture or bone erosion due to recurrent instability.[6] Greater loss of bone disrupts the glenoid concavity and thereby reduces the effectiveness of the centralizing concavity-compression mechanism (a deeper concavity with a greater arc of stability can resist greater displacing forces) that governs stability.[41] Persistent tonic-clonic seizures predispose to glenoid bone damage through repetitive high-contact bone-on-bone trauma.[67] Bigliani et al[6] classified glenoid defects into 3 distinct types: type I, united fragment attached to separated labrum; type II, malunited fragment detached from labrum; type IIIa, <25% glenoid bone loss; and type IIIb, >25% glenoid bone loss. Isolated soft tissue repairs were recommended for types I, II, and IIIa, whereas bone augmentation was suggested for type IIIb defects. Burkhart and De Beer[11] examined the results of 194 consecutive arthroscopic Bankart repairs to determine risk factors for recurrent instability. Morphologic assessment of the glenoid revealed that an "inverted pear shape" was associated with a significantly higher recurrence rate compared with glenoids without the defect (67% vs. 4%). Lo et al[39] found that at least 25% of the glenoid must be absent for it to resemble an inverted pear and that a bone grafting procedure to restore the normal articular arc of the glenoid should be considered to restore stability.

These clinical observations have been supported by a number of biomechanical studies. Gerber and Nyffeler[23] demonstrated that lesions involving more than half of the anteroposterior diameter of the anteroinferior glenoid rim would result in at least a 30% reduction of the forces resisting dislocation. Using a cadaveric model, Itoi et al[30] showed that a bone defect of at least 21% of the glenoid anterior arc length significantly reduced the translational force required for dislocation and that stability decreased as the size of the osseous defect increased. Yamamoto et al[70] undertook a laboratory study to determine whether an anterior glenoid defect would have a similar effect on anterior shoulder stability as that of an anteroinferior defect. A lesion at the 3-o'clock position with a width of at least 20% of the glenoid length was found to significantly reduce anterior stability. The effect of posterior glenoid bone loss on stability has not been examined as comprehensively as anterior bone loss, but in a cadaveric study, as little as 5° of posterior glenoid bone loss was demonstrated to significantly increase posterior translation of the humeral head.[9]

Current evidence suggests that glenoid bone loss between 20% and 25% adversely affects stability of the shoulder and should be taken into consideration in planning surgery. More recently, though, subcritical levels of bone loss (≥13.5%) have been associated with poor functional outcomes in patients who did not experience recurrent instability, suggesting that the historic threshold of 20% to 25% glenoid bone loss representing the most important anatomic factor influencing the type of operative strategy employed should be re-evaluated using patient-reported outcome measures.[63] This may be pertinent to the epileptic population, who exhibit higher failure rates due to powerful uncontrolled seizures disrupting surgical repairs.[67]

Humeral bone loss

The occurrence of a humeral head impression fracture was first documented in 1940 by Hill and Sachs and subsequently named the Hill-Sachs lesion (Fig. 1).[27] These defects typically occur when the posterolateral aspect of the humeral

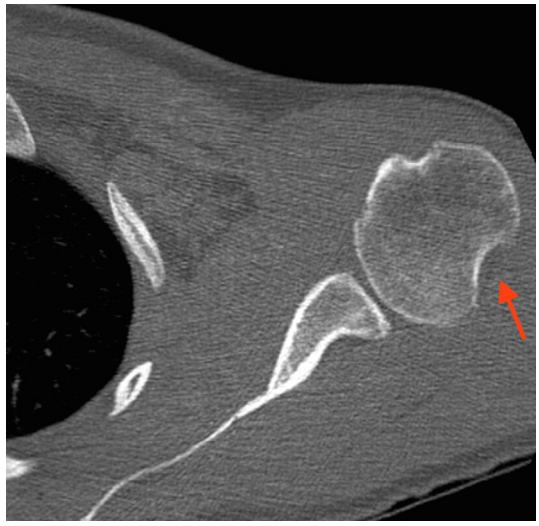

Figure 1 Hill-Sachs lesion seen on computed tomography in a 34-year-old man.

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