Glenoid and humeral head bone loss is the most common cause of failure after surgical shoulder instability stabilization. Large Hill-Sachs lesions involving >30% of the articular surface of the humeral head typically occur after neglected or locked shoulder dislocations. Such large lesions may require osteochondral allograft reconstruction or prosthetic replacement to regain shoulder stability and function.

Previous reports of osteochondral allograft reconstruction have focused on adults. This article describes a case of a 16-year-old autistic boy who sustained an anterior dislocation of the right shoulder following a seizure episode. The dislocation was diagnosed 12 days later as a progressive deformity, and his parents noticed his inability to use his upper extremity. The patient had a large (30%) humeral head Hill-Sachs lesion and persistent anterior shoulder instability after initial closed reduction. He underwent an open osteochondral allograft reconstruction for the restoration of the humeral head articular surface. The sizing of the defect, matching harvest of the allograft, and perfect fit of the allograft to the defect are critical steps to ensure congruent restoration of the humeral head. The allograft was stabilized in the defect of the humeral head using cancellous screws placed from below the articular surface. Radiographs 20 months postoperatively showed complete incorporation of the osteochondral allograft. At 30 months postoperatively, his shoulder was stable and functional.
In 1940, Hill and Sachs described an impaction fracture of the posteroslateral humeral head following traumatic anterior shoulder dislocation. Since then, studies have shown such lesions to exist in 80% of acute shoulder dislocations and almost 100% of recurrent shoulder dislocations. Although smaller Hill-Sachs lesions can be ignored during the management of anterior shoulder dislocation, larger lesions should be addressed to prevent recurrent shoulder instability. Larger Hill-Sachs lesions typically exist after neglected, missed, or recurrent anterior shoulder dislocation. Large lesion reconstruction using humeral head osteoarticular allografts has been reported in adults but has not been reported in an adolescent patient.

**CASE REPORT**

A 16-year-old autistic boy presented to the emergency department with his parents for an increasing deformity over his right dominant shoulder and restricted right upper extremity use following a seizure episode approximately 12 days previously. Physical examination showed obvious shoulder deformity, anterior position of the humeral head on palpation, and restricted upper limb mobility. Neurovascular examination of the right upper extremity was normal. Radiographs revealed an anterior dislocation of the humeral head and a Hill-Sachs lesion. Due to the inability to reduce the shoulder in the emergency department, he underwent closed reduction under anesthesia (Figure 1).

Intraoperatively, the shoulder was unstable and dislocated in 30° to 40° of external rotation with the arm in adduction. Postreduction magnetic resonance arthrogram revealed a large Hill-Sachs lesion affecting approximately 30% of the articular surface of the humeral head (Figures 2A, B). Other findings included an intra-articular loose body in the posterior aspect of the humeral head, a partial tear of the biceps tendon, and injury to the anterior capsulolabral structures. The glenoid showed no significant bony erosion on the anterior aspect. The patient underwent elective reconstruction for the right shoulder based on the availability of fresh osteochondral humeral head allograft.

The standard deltopectoral approach to the shoulder was performed under general anesthesia in the beach chair position. The subscapularis tendon and underlying capsule was cut approximately 5 mm from its insertion on the lesser tuberosity. The humeral head was dislocated anteriorly, and the posteroslateral impaction fracture defect was visualized (Figure 3A). Because initial attempts at osteoplasty failed, osteoarticular allograft reconstruction was performed. The articular-arc deficit was approximately 30%, and the humeral head was dislocated in approximately 30° to 40° of external rotation. The posteroslateral defect was prepared with a microsagittal saw to have straight sides and a wedge shape for adequate allograft placement. The defect size, including its length, width, and depth, were measured with a ruler, and a suture pack (foil) was used as a template to estimate the shape of the defect. The shape and dimensions were marked on the fresh osteoarticular humeral head allograft corresponding with the site and area of the defect.

A wedge was harvested using a saw approximately 2 mm larger than the measured size (Figure 3B). The wedge was trimmed to press-fit into the defect (Figure 3C). Three 4.0-mm partially threaded cancellous screws were inserted from below the lesser tuberosity into the osteoarticular wedge to achieve adequate graft fixation (Figure 3D). Once the humeral head was restored, joint stability was satisfactory in all planes of motion. The anterior capsulolabral structures were
then repaired using 3-0-mm Bio-FASTak suture anchors (Arthrex, Naples, Florida) inserted at the 3-o’clock and 5-o’clock positions. The humeral head was kept moist throughout the procedure. The subscapularis tendon was repaired, with the arm held in approximately 15° of abduction and 15° of external rotation. Routine closure was performed over a drain, which was removed on postoperative day 3.

Postoperatively, the patient’s arm was placed in a sling for approximately 3 weeks, followed by gradual progressive range of motion exercises and shoulder strengthening. Physical therapy continued for 6 months. Radiographs and clinical follow-up 20 months postoperatively showed complete osteochondral allograft incorporation and a functional, stable shoulder. Thirty months postoperatively, the patient’s family was contacted over the phone, and they reported that the patient’s shoulder had satisfactory function.

**DISCUSSION**

Open or arthroscopic anterior capsulolabral repair stabilizes the shoulder in most patients with anterior shoulder instability, despite the presence of a Hill-Sachs lesion. However, Hill-Sachs lesions have caused recurrent instability in patients who have had a failed anterior capsulolabral repair.\(^{12,13}\) In patients with chronic, locked, or neglected dislocations (eg, those related to seizures), the risk of recurrent dislocation and the presence of large Hill-Sachs lesions are higher.\(^8\)

Quantifying humeral head bone loss in Hill-Sachs lesions is important in the management of shoulder instability. Biomechanical studies suggest that humeral head lesions as small as 12.5% and 25% affect shoulder stability.\(^{14}\) Burkhart and Danaceau\(^7\) introduced the concept of articular-arc deficit and identified the mismatch between the articular arc length of the humeral head and the glenoid in the presence of bone defects, thus causing the sensation of subluxation or dislocation. The concept depends on the size of the defect and its location in the arc. Hill-Sachs lesions that engage the anterior glenoid with the shoulder in a functional position of 90° of abduction and external rotation are termed *engaging Hill-Sachs lesions*. They are in contrast to *nonengaging lesions*, which cause symptoms in nonfunctional shoulder positions, such as extension or <90° of abduction.

In a recent study, Kaar et al\(^\text{15}\) modeled the humeral head as a circle when shown on an axial magnetic resonance imaging (MRI) or a computed tomography (CT) scan, and the humeral head articular surface to be approximately 180°. Based on their study, a defect of five-eighths of the radius of humeral head, which correspond with a defect of approximately 38% of humeral head, would affect glenohumeral stability. In the current study, the patient’s MRI revealed a defect of approximately 30% of the articular surface (Figure 2A), which corresponded with a defect of three- to five-eighths of the radius of the humeral head (Figure 2B).

Various operative procedures have been described in the literature for the management of Hill-Sachs lesions based on the percentage of the articular surface defect and the presence of engaging Hill-Sachs lesions. For humeral head defects <20% of the articular surface, arthroscopic Bankart repair has been effective, with no need to address the humeral defect.\(^\text{16}\) Various options exist for humeral defects that are 20% to 30% of the articular surface with an engaging Hill-Sachs lesion. Remplissage is the open or arthroscopic transposition of the infraspinatus tendon into the humeral head defect.\(^\text{17,18}\) Humeral head osteoplasty has also been reported, but this method...
cannot be used in large humeral head defects or patients with osteopenia who lack subchondral bone support.

Sekiya et al\textsuperscript{14} used a bone tamp percutaneously to elevate the Hill-Sachs defect through an anterior cortical window, which they termed as \textit{percutaneous humeroplasty}. Based on cadaveric studies, they concluded that large defects, could only be reduced into smaller defects and complete correction of the defect was not possible. In 1954, Latarjet\textsuperscript{10} described the coracoid process transfer to the anterior glenoid, thereby extending the glenoid arch such that it prevents engagement of the Hill-Sachs lesion on the glenoid surface. Although the precise mechanism by which the Latarjet procedure provides stability is unknown, disadvantages include loss of external rotation, coracoid graft osteolysis, and a higher incidence of glenohumeral arthritis.

For humeral head defects >30\%, the options include humeral rotational osteotomy, osteochondral bone graft, and arthroplasty.\textsuperscript{13} In 1984, Weber et al\textsuperscript{20} described the rotational osteotomy of the proximal humeral shaft, which increases the retroversion of the proximal humerus and reduces the likelihood of recurrent instability by redirecting the defect more posteriorly. However, the risk exists of redislocation, hardware failure, nonunion, malunion, and internal rotation deficit. Shoulder arthroplasty and osteochondral humeral head allograft reconstruction have been described to treat adults with large head defects. Their use in an adolescent patient has not been described.

Miniaci and Gish\textsuperscript{11} reported the use of osteochondral allografts in Hill-Sachs lesion reconstructions in adults. At 2-year follow-up, no recurrent instability existed, but 2 patients underwent hardware removal due to partial graft collapse. Hart and Kelly\textsuperscript{4} reported improved clinical outcomes after humeral head osteochondral allograft reconstruction 18 months postoperatively in 7 patients with no associated complications. Advantages include restoration of the near congruent articular surface, a biomechanically stable joint without alteration of the joint kinematics, and the possibility of future prosthetic replacement with no bone stock compromise.

**CONCLUSION**

In the current study, considering the patient’s age, history of uncontrolled seizures, and defect size, the authors performed a fresh osteochondral allograft for humeral head reconstruction rather than prosthetic replacement. Compared with the surgical technique described in the literature,\textsuperscript{11} the allograft was press-fit into the defect, and further fixation was achieved by using screws directed into the allograft from the nonarticular humeral head surface. This technical modification prevents articular surface damage during screw insertion, as well as articular surface damage due to screw prominence in case of cartilage resorption or graft collapse. Radiographs at 20-month follow-up showed good bony graft integration and a congruent articular surface (Figure 4). Osteochondral allograft reconstruction is a viable option for humeral head defect restoration in adolescent patients.

**REFERENCES**