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Case Report

Glenoid hypoplasia

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ABSTRACT

Glenoid hypoplasia, also known as glenoid dysplasia and dysplasia of the scapular neck, is a failure of ossification of the posteroinferior two-thirds of the glenoid. Once thought to be a rare condition, more recent studies have shown that the incidence of glenoid hypoplasia ranges from 18% to 35%. This case report and literature review highlights the typical clinical presentation, the radiologic findings, and the management options for patients with glenoid hypoplasia.

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Case report

A 43-year-old Caucasian man presented for evaluation of a multiple year history of shoulder pain and stiffness of insidious onset with no known traumatic event. His active range of motion was reduced on the affected shoulder with some posterior instability noted. The unaffected shoulder demonstrated a normal range of motion with no pain or stiffness. A magnetic resonance imaging (MRI) of the affected shoulder was obtained. Axial imaging shows the deficient posterior glenoid, retroversion and concomitant posterior labral tear, detachment and hypertrophy (Figs. 1A and B). The hypoplastic glenoid may also demonstrate rounding of the posteroinferior glenoid rim and cartilage, labral hypertrophy,

and widening of the inferior glenohumeral joint space. [1,2] Retroversion is assessed by obtaining the angle between the glenoid articular surface and a line extending through the long axis of the scapula on axial view (Fig. 1C). Humeral head subluxation often accompanies glenoid hypoplasia and is assessed by calculating the percentage of the humeral head that lies posterior to the long axis of the scapula (Fig. 1D). Additional imaging features may include hypoplasia of the humeral head, hyperplasia of the coracoid process and acromion, and hooking of the distal clavicle [3]. Osteophyte formation along the posteroinferior glenoid rim may be present, indicating accelerated degenerative joint disease [4]. The findings of glenoid hypoplasia can be contrasted to normal glenoid anatomy demonstrated in Figure 1B. The

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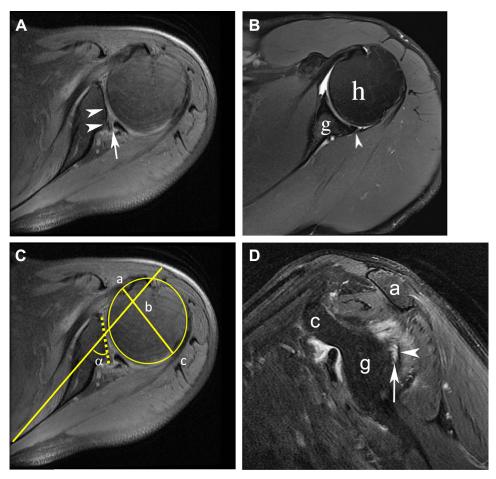


Fig. 1 – MRI of glenoid hypoplasia in a 43-year-old man. (A) Axial fast spin echo (FSE) proton density (PD) fat-suppressed (FS) image shows hypoplasia of the posterior glenoid (arrowheads), retroversion of the glenohumeral joint, and mild posterior humeral head subluxation. Note the concomitant posterior labral tear, detachment (arrow), and enlargement. (B) Normal shoulder in 40-year-old man. Axial FSE PD fat-suppressed image through the midglenohumeral joint shows the normal glenoid version, posterior labrum (arrowhead), and glenohumeral relationship (g = glenoid, h = humeral head). (C) Retroversion is assessed by obtaining the angle (α) between the glenoid articular surface (dotted line) and a line extending through the long axis of the scapula on axial view. Humeral head subluxation is assessed by calculating (ab/ac) × 100%. The conventional method of assessing glenoid version on cross-sectional imaging was described by Friedman et al [5]. Glenoid version is the angle between the glenoid line (the axis along the anterior and posterior glenoid rim) and the line perpendicular to the scapular axis (along the root of the scapular spine and center of the glenoid line). (D) Sagittal FSE T2 FS image shows the deficient portion of the posterior glenoid with complex joint fluid (arrow) extending between the glenoid (g) and imaged portion of the posterior labrum (arrowhead). c = coracoid, a = acromion.

patient's initial radiographs demonstrating glenoid hypoplasia are demonstrated in Figure 2 and are compared with the radiograph findings in an individual with normal glenoid anatomy.

Discussion

Glenoid hypoplasia, also known as glenoid dysplasia and dysplasia of the scapular neck, is a failure of ossification of the posteroinferior two-thirds of the glenoid. The scapula develops through intramembranous ossification at 8 ossification centers, two of which compose the glenoid. Aberrant formation of the inferior glenoid ossification center results in the characteristic findings of hypoplasia of the inferior

glenoid promontory with marked hypertrophy of the articular cartilage [1,2]. It was first described by Giongo in 1927 [6]. Multiple etiologies have been described in the literature including idiopathic, familial, and as a part of a clinical syndrome, such as Apert syndrome, Holt-Oram syndrome, and others [2]. Once thought to be a rare condition, more recent studies have shown that the incidence of glenoid hypoplasia ranges from 18% to 35%, depending on the stringency of diagnostic criteria [1,7].

Glenoid hypoplasia is most commonly bilateral and asymptomatic, with the majority of cases discovered incidentally on chest x-ray [8,9]. Symptomatic patients often present with glenohumeral joint instability, pain, stiffness, and restricted range of motion, with abduction most significantly affected [1,3]. In a 2011 literature review, Suryawanshi

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