The case:

A 21-year-old man presented with multidirectional instability in his left shoulder and reported his left shoulder had been “popping out” and painful ever since an opposing player had blocked his shot while playing basketball. The patient had a history of recurrent dislocations and previous surgery to both shoulders. On physical examination, he could sublux both shoulders posteriorly, the left more than the right. On the left, he demonstrated a 1 plus sulcus sign and a positive Cofield test but negative apprehension and relocation tests. Radiograph (Figure 1) and magnetic resonance imaging (Figure 2) was obtained following an arthrogram.

Figure 1: AP radiograph of the left shoulder in external rotation following an arthrogram. Figure 2: Axial T1-fat saturation sequence (A), T2-parasagittal sequence (B), and paracoronal T2-fat saturation (C).

Your diagnosis?

For answer see page 1315
A

lthough glenoid hypo-
plasia or dysplasia is
thought to be rare, recent liter-
ature suggests it is more com-
mon than originally believed.
In fact, within 4 weeks of this
patient’s presentation, a sec-
ond patient with the same dis-
order presented to our institu-
tion. A plain radiograph of a
different patient with glenoid
dysplasia shows the character-
istic shallow glenoid fossa and
rather ovoid appearance of the
humeral head (Figure 3).

Glenoid dysplasia is usually
a solitary abnormality and
often bilateral. However, it also
has been described in associa-
tion with a number of other
entities including multiple epi-
physeal dysplasia (Figure 4)
and deficiency of either vita-
min C (scurvy) or vitamin D
(rickets). It also can be seen in
association with various mus-
cular dystrophies. In their
excellent paper on glenoid dys-
plasia, Currarino et al1 added at
least another 10 rare congenital
anomalies with which glenoid
dysplasia may be associated.
These include Apert’s syn-
drome and Holt-Oram syn-
drome as well as Hurler’s syn-
drome and some of the other
mucopolysaccharidoses.

Answer to Radiologic Case Study
Case facts appear on page 1216

Figure 1: AP radiograph of the left shoulder in external rotation following an
arthrogram shows the very thick superior labrum (arrow), flattened glenoid,
and shift of the axillary pouch laterally (arrow heads). Note that the glenoid
neck is absent. Figure 2: MRI of the same patient following arthrography.
Axial T1-fat saturation sequence. Note the shallow glenoid with an enlarged
anterior labrum and thickening of the middle glenohumeral ligament (A). T2-
parasagittal sequence shows both the thick middle glenohumeral ligament
and thickening of the anterior and inferior labrum (B). Paracoronal T2-fat
saturation sequence confirms these findings but also shows a somewhat oval
humeral head (C).
PATHOGENESIS

The pathogenesis of glenoid dysplasia appears to be failure of the inferior glenoid precartilage to ossify; although the inferior glenoid precartilage is in fact present, it remains unossified. The scapula normally has 10 growth centers: 3 for the acromion, 2 for the coracoid, and 3 for the scapular blade as well as the 2 growth centers for the glenoid.\(^1\)

The upper glenoid growth center is responsible for the growth of the base of the coracoid and the upper one third of the glenoid itself. The lower glenoid center is horseshoe-shaped and is responsible for the lower two thirds of the glenoid fossa.\(^1\)

In their series of 12 patients, Smith and Bunker\(^2\) reported a 25% incidence of instability. However, this incidence was based on patients’ symptoms only and not on clinical examination.

In a review article, Wirth et al\(^1\) divided their patients into three groups. They reported that generally <2% of patients with glenoid dysplasia have symptomatic instability, although in fact, 5 (31%) of their 16 patients demonstrated instability. In an overview of the more recent literature on glenoid dysplasia, the incidence of posterior instability appears to be approximately 30%, although none of the authors reported frank dislocations as observed in our two cases.

IMAGING

The pattern of abnormalities in isolated glenoid dysplasia is consistent with deficient ossification of the horseshoe-shaped inferior secondary ossification center. This results in an osseous defect along the anteroinferior glenoid fossa that has a congruent enlargement of the articular cartilage. The combined glenoid volume, including the osseous and articular cartilage components, is virtually normal, but produces the radiographic effect of widening the inferior glenohumoral space between the osseous margins. The contiguous scapular neck is often hypoplastic.

Magnetic resonance imag-
ing (MRI) has shown the osseous defect to additionally involve the posterior margin of the glenoid fossa. Hypertrophy of other shoulder components including the acromion, coracoid, and clavicle is seen. Flattening of the humeral head with a varus deformity occasionally also has been reported (Figure 3).

The radiographic findings of glenoid dysplasia are somewhat variable, although most patients have a flattened, shallow glenoid fossa. The neck of the scapula often is foreshortened or absent. Related findings include a prominent coracoid process, a large and elongated acromion, hooking of the distal aspect of the clavicle, hypoplasia of the upper ribs, and flattening of the humeral head.

Smith and Bunker noted 75% of their 12 patients had hooking or bossing of the distal clavicle and 50% of their patients had a prominent coracoid process. However, none of their patients had any abnormalities of the humeral head or neck. In contrast, the patient described by Collins et al had flattening of his glenoid with a notch inferiorly and slight flattening of the humeral head. Munshi and Davidson found similar changes. Their first patient was a 41-year-old man with pain and weakness in his right shoulder. The MRI arthographic findings included complete osseous hypoplasia of the entire posterior glenoid and scapular neck, and hypertrophy of the inferior glenoid labrum (Figures 1 and 2). Their second patient was a 67-year-old woman who had pain and decreased range of motion of the right shoulder. The MRI arthographic findings included osseous hypoplasia of the posterior and anteroinferior bony glenoid. The authors also noted intra-articular contrast material diffused into the abnormal tissue, consistent with fissuring of what was probably unossified cartilage.

In our patient, the right posterior, superior, and anterior labra were fixed laparoscopically. However, 2 weeks postoperatively, he continued to complain of and easily demonstrated voluntary left shoulder subluxation. Further physical examination revealed type 3 posterior instability of the left shoulder with spontaneous reduction, a positive Cofield test, and 1 plus sulcus sign. He could internally rotate to T6 on the left, but then only to L4 with the operated right shoulder.

**Complications**

Although the true incidence is unknown, by far the most frequently noted complication of glenoid dysplasia is osteoarthritis. Sperling et al reviewed six patients with degenerative arthritis secondary to glenoid dysplasia who were treated at the Mayo Clinic. All of the patients reported moderate to severe shoulder pain, which was confirmed radiographically. Four hemiarthroplasties and three total shoulder replacements were performed in these six patients. With follow-up ranging from 1.3 to 16 years, there were four unsatisfactory results including one patient who became infected with *Pseudomonas*. All of these patients underwent revision procedures, and three of the hemiarthroplasties were converted to total arthroplasties.

Smith and Bunker divided their 12 patients into two groups. All of the patients in the older group (>40 years) exhibited findings of degenerative arthritis. One 48-year-old patient progressed from relatively normal radiographic findings bilaterally to severe degenerative arthritis in both shoulders during a 6-year period. Three of their patients also underwent shoulder arthroplasty with mixed results.

Smith and Bunker commented on the fact that the lack of glenoid bone stock made access to and the insertion of a glenoid component difficult. The Mayo Clinic authors went further and stated glenoid deficiency and cartilage wear should be addressed at initial shoulder arthroplasty in patients with glenoid dysplasia.

On the other hand, Wirth et al reviewed 16 patients with glenoid dysplasia who were divided into three groups: those with bilateral changes without instability (8 patients), those with bilateral disease with symptomatic instability (5 patients), and those with unilateral glenoid hypoplasia (3 patients). Only two of their older patients had any evidence of degenerative change, and it was decided to treat the patients conservatively.

**References**