EASILY MISSED?

Posterior shoulder dislocations

Robert C Jacobs, Nicole A Meredyth, James D Michelson

After being tackled earlier that day, a 33 year old rugby player presented to a walk in clinic with diffuse left shoulder pain, limited abduction, and arm held in slight internal rotation, with a flexed, adducted, and internally rotated position (fig 1, see thebmj.com). He was discharged and diagnosed as having a spontaneously reduced anterior shoulder dislocation after an anterioposterior view of the shoulder was read as normal (fig 2).

One week later he presented to the emergency department with continued symptoms and crepitus in his shoulder with attempted movement. A posterior shoulder dislocation was diagnosed when an orthogonal view (axillary) of the shoulder was taken (fig 3). After conscious sedation, the dislocation was reduced (fig 4).

What is a posterior shoulder dislocation?

A posterior shoulder or glenohumeral dislocation is the posterior displacement of the humeral head relative to the gelenoid. The anterior humeral head is typically impacted on to the gelenoid rim, so patients may present with limited range of shoulder motion. Posterior shoulder dislocations are classified as acute if identified within three weeks of injury and chronic afterwards. Chronic posterior shoulder dislocations are often less painful and have a greater range of movement than acute posterior dislocations.

How common are posterior shoulder dislocations?
The glenohumeral joint is the most commonly dislocated joint in the body, and posterior shoulder dislocation comprises 2-4% of all shoulder dislocations. In an audit of 120 dislocations, posterior shoulder dislocations were seen most often in men aged 20-49 years, and the most common causes were traumatic events (67%) and seizures (31%).

Why is it missed?
The diagnosis is often missed initially—delayed diagnosis occurs in 50-79% of patients. Delay can result from the clinician or patient (or both) deeming the mechanism of injury insufficient to cause dislocation or subtle clinical examination findings relative to anterior dislocation. Patients often have enough motion in the joint and minimal palpable humeral displacement to confound a diagnosis of dislocation. Inadequate initial imaging increases the chances of missing the diagnosis. Often, only an anteroposterior view of the shoulder is ordered, without an orthogonal one; in such circumstances a posterior dislocation can look normal (fig 2). A dedicated protocol for shoulder injury that includes an orthogonal view significantly reduces the rate of missed diagnoses (fig 3).

Why does it matter?
A delay in diagnosis can result in serious morbidity because posterior dislocation often produces an impression fracture on the anterior aspect of the humeral head (reverse Hill-Sachs lesion; fig 5). The fracture can enlarge and propagate with prolonged dislocation while damaging the articular cartilage. This may lead to osteoarthritis and eventual avascular necrosis from impaired blood flow to the humeral head. Early diagnosis reduces the risk of these complications and decreases the likelihood of needing a subsequent shoulder arthroplasty.

How is it diagnosed?
Clinical diagnosis
The key to accurate and timely diagnosis is to maintain a high index of suspicion, based on mechanism of injury, and to perform appropriate diagnostic imaging. Clinical diagnosis is challenging, although specific history and physical examination findings increase the likelihood of identifying this injury. The most common mechanisms are indirect trauma, with the shoulder in a position of flexion; adduction and internal rotation, with a coaxial force applied on the arm; or extreme muscular contraction, such as seizure or electrocution. Patients typically hold the affected shoulder in adduction internal rotation. They may have a mechanical block to external rotation, often with severe pain if the injury is acute. An abnormal shoulder contour with a prominent coracoid process anteriorly and a palpable posterior positioned humeral head may be present, but the findings are often subtle.

Posterior dislocations are often associated with other shoulder injuries, including fractures (34%), reverse...
Once identified, refer these dislocations to an emergency department for prompt reduction under adequate muscular relaxation. Deep procedural sedation, with airway monitoring, may be needed. Computed tomography may be needed in the emergency department to diagnose non-displaced fractures or further define fractures that could become greatly displaced with attempted closed reduction. Prevention of further injury helps reduce morbidity from these injuries—many will not require operative fixation and will be able to be managed with sling immobilization.

Hill-Sachs injuries (29%), and rotator cuff tears (2–13%). At least two orthogonal views (anterioposterior plus axillary, Velpeau, or scapular Y) are needed to exclude a posterior dislocation. The axillary view may require painful positioning, and antecedent analgesia or use of a curved film cassette may help. The combination of anterioposterior and Velpeau views as part of a shoulder trauma series results in an 89% detection rate of traumatic posterior shoulder dislocation. Point of care ultrasound can also diagnose these injuries at the bedside, although extra training is needed and results are operator dependent.

Fig 3 | Pre-reduction axillary radiograph of left posterior shoulder dislocation. The humeral head (H) is clearly seen posterior and impacted on the glenoid (G). The scapular body (S) and coracoid process (C) can also be identified. The lateral aspect of the clavicle (CL) and acromion process (A) are immediately anterior to the humeral head.

How is it managed?
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Fig 4 (left) | Post-reduction anterioposterior (top panel) and axillary (bottom panel) radiographs of the left shoulder showing concentrically located humeral head (H) within the glenoid (G). The anterioposterior views are similar before (fig 2) and after reduction, whereas the axillary views are very different (fig 3). The clavicle (CL) and acromion process (A) are superimposed on the humeral head. S=scapular body, C=coracoid process. Top of each image is anterior, bottom is posterior.

Fig 5 (above) | Pre-reduction axial computed tomogram of the left shoulder. Note the reverse Hill-Sachs impaction lesion (HS) to the anteromedial humeral head (H). G=glenoid, C=coracoid process, S=scapula.

**ANSWERS TO ENDGAMES, p 35** For long answers go to the Education channel on thebmj.com

**STATISTICAL QUESTION**
Intention to treat analysis versus per protocol analysis of trial data
Statements b, c, and d are true, whereas a is false.

**ANATOMY QUIZ**
Coronal proton density weighted magnetic resonance image of a 9 year old child’s left ankle and foot
A: Tibialis posterior tendon
B: Flexor digitorum longus tendon
C: Subtalar joint
D: Fibula (lateral malleolus)
E: Peroneus brevis
F: Calcaneus

**CASE REPORT**
An adolescent with an altered state of mind
1. Acute intoxication by an emerging drug of misuse, probably a synthetic cannabinoid. Clues to the diagnosis include acute onset, otherwise unexplained, central nervous system and autonomic disturbances in a healthy young person with negative drug screening tests and a history of smoking herbal incense.
2. Synthetic cannabinoids can cause severe neurological and psychiatric toxic effects as well as injury to other organ systems, including myocardial infarction, cardiac dysrhythmia, hypokalaemia, hyperthermia, and acute kidney injury. A small number of deaths have also been reported.
3. Synthetic cannabinoids are not usually detected by conventional drug screening tests. In the absence of a positive history, advanced tests like liquid chromatography-tandem mass spectrometry may identify the substance, but these are not generally available in the acute setting.
4. Intoxicated patients should be observed until recovery and should undergo further evaluations, including basic blood tests and electrocardiography. Treatment is mainly supportive.
UNCERTAINTIES

Should vitamin D supplements be recommended to prevent chronic diseases?

Haakon E Meyer,1 2 Kristin Holvik,1 Paul Lips3

Vitamin D has gained much attention in research and clinical practice as a possible preventive factor for a wide array of chronic diseases, including cardiovascular disease, various cancers, type 2 diabetes, autoimmune diseases, and chronic obstructive pulmonary disease. Vitamin D3 (cholecalciferol) is a steroid hormone precursor and is synthesised when skin is exposed to ultraviolet B radiation. It is also found in a limited number of foods, especially oily fish. The other form of the vitamin, vitamin D2 (ergocalciferol), is found in dietary plant sterols exposed to ultraviolet B radiation and is somewhat less effective than vitamin D3. Vitamin D has well known effects on calcium metabolism and is traditionally linked to the prevention of rickets in children. It is also now clear that vitamin D deficiency causes bone loss through secondary hyperparathyroidism.2

Because vitamin D receptors are present in many organs and tissues, vitamin D may have extraskeletal effects.2 In addition, many observational studies have shown associations between 25-hydroxyvitamin D3 (25(OH)D3), the major circulating form of vitamin D, and the risk of chronic diseases.3 As a consequence of increased popularity, measurement of 25(OH)D3 has become common, and vitamin D supplements, at doses far exceeding the recommended daily allowances or dietary reference values,4 6 are often given, despite limited evidence of an effect.

The Bottom Line

• Do not recommend vitamin D supplements to prevent chronic disease because clear evidence of benefit does not currently exist and adverse effects cannot be excluded.
• Vitamin D supplements in doses of 600-800 IU (15-20 µg) per day combined with calcium (0-1000 mg/day, depending on current dairy intake) may be recommended to prevent fractures in elderly people.

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THE BOTTOM LINE

What is the evidence of the uncertainty?

On the basis of the existing evidence, we can conclude that vitamin D supplements combined with calcium decrease the incidence of fractures in elderly people.7 8 However, there is insufficient evidence that, in the doses tested, vitamin D supplements alone prevent fracture.1 7

It is biologically plausible that vitamin D can help prevent various chronic diseases, and this seems to be supported by observational studies.2 However, observational data might be biased by confounding, and diseases might also lead to low vitamin D rather than the other way around. According to meta-analyses and systematic reviews, clinical trials have shown a consistent effect of vitamin D supplements on the incidence of cardiovascular disease, cancer, chronic obstructive lung disease, or diabetes.3 5 9 A Cochrane review concluded that vitamin D supplementation for an average of 4.4 years slightly decreased all cause mortality (from 11.4% to 11.0%; relative risk 0.94, 95% confidence interval 0.91 to 0.98), and the result was also significant in those given less than 800 IU a day. However, these results were not considered to be robust enough to recommend widespread supplementation.1 2

What dosage?

The dose consistent with fracture prevention in randomised controlled trials (RCTs) was in general not higher than 800 IU (20 µg) a day when combined with calcium (1000-1200 mg/day in most trials).7 8 Updated dietary reference values for vitamin D from Europe and the United States, mainly based on its benefits for bone health, are 400-600 IU (10-15 µg) per day for adults and 800 IU (20 µg) per day for elderly people.4 6

Who would benefit?

It is plausible that most gain can be obtained by increasing concentrations in people with the lowest baseline values. In a recent meta-analysis of observational studies, the inverse association between 25(OH)D, and total mortality was non-linear, with the largest difference in mortality between the groups with the lowest and second lowest 25(OH)D values, a finding that concurs with several other disease outcomes, such as cardiovascular disease and colon cancer.7 Whether optimal serum concentrations of 25(OH)D should be 50 nmol/L7 or 75 nmol/L is under debate,11 and this is not helped by standardisation of laboratory assays for 25(OH)D, being less than ideal.7

Most studies on the health effects of vitamin D have been performed in white populations. The evidence is even more scant in other ethnic groups. Non-Western immigrants living in Western countries often have widespread vitamin D deficiency (serum 25(OH)D, <25 nmol/L), and their 25(OH)D concentration is lower than that of people in their country of origin.12 In the Women’s Health Initiative Observational Study, low 25(OH)D, was associated with an increased risk of
Ongoing and planned randomised controlled trials with at least 1000 participants that will study the effect of vitamin D on the incidence of chronic diseases, fractures, and mortality in adults

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<th>Intervention (IU‡)</th>
<th>Results expected</th>
<th>Main endpoints§</th>
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VITAL=Vitamin D, marine omega-3 fatty acids and placebo in a 2×2 factorial design; CAPS=Calcium to intervention and control group; DO-HEALTH=Vitamin D, Omega 3 fatty acids, exercise program and placebo in a 2×2 factorial design; TIPS-3=Vitamin D, Polycap DS (thiazide, atenolol, ramipril, simvastatin), aspirin and placebo in a 2×2×2 factorial design. †F=female; M=male. ‡Dose of vitamin D3. §Some trials are also studying other endpoints not mentioned here. CVD=cardiovascular disease. ¶INTERHEART risk score of ≥10.

**RECOMMENDATIONS FOR FURTHER RESEARCH**

Population: People over 60 years with low vitamin D status (serum 25-hydroxyvitamin D <50 nmol/L)

Intervention: Vitamin D, 800 IU/day or vitamin D, 2000 IU/day.

Control: Placebo

Outcome: Cardiovascular disease, type 2 diabetes, respiratory diseases, cancer, mortality

without concomitant calcium can prevent chronic disease in healthy populations. However, these trials may not completely answer this question. For example, the VITAL trial (table) was planned with a statistical power to detect risk reductions of 15-40% (depending on the outcome), but the true preventive effect of vitamin D on chronic diseases could be less than 15%. The suggested effect of vitamin D supplements on total mortality was small according to the Cochrane review (relative risk 0.94). An effect of this size might be regarded as negligible at an individual patient level but might be relevant at the population level. In addition, owing to the inclusion criteria, a large proportion of participants in new trials will probably not have a low initial 25(OH)D concentration, and the statistical power to study subgroups with a low vitamin D status at baseline will be smaller. As well as the ongoing studies in the table, our suggestions for further trials are in the recommendations box.

**What should we do in the light of the uncertainty?**

We need a balanced view on vitamin D, with not too little and not too much. Because clear evidence of benefit over harm for vitamin D has not been proved, we should not recommend vitamin D supplements for prevention of chronic diseases (such as cardiovascular disease, cancer, chronic obstructive lung disease, or diabetes) until more definitive further research evidence is available.

Vitamin D deficiency (25(OH)D <30 nmol/L) should of course be treated to prevent skeletal complications. Vitamin D supplementation at doses of 600-800 IU (15-20 µg) per day combined with calcium may be recommended to prevent fractures in elderly people, according to evidence from a Cochrane review and updated nutritional recommendations for vitamin D intake in the general population.¹ ³