Causes of px arising from shoulder:

- Rotator cuff disorder
- Frozen shoulder
- Instability disorder
- AC joint disorder
- GH joint osteoarthritis
- Inflammatory arthritis
- Septic arthritis

Causes of px which arise from elsewhere:

- Malignancy
- Referred pain from the neck, heart, or lungs
- Polymyalgia rheumatica

Red flags on Hx or examination:

- Trauma, pain & weakness, or sudden loss of ability to actively raise the arm (with or without trauma): suspect acute rotator cuff tear
- Any shoulder mass or swelling: suspect malignancy
- Red skin, painful joint, fever, or the person is systemically unwell: suspect septic arthritis
- Trauma leading to loss of rotation & abnormal shape: possible shoulder dislocation
- New Ssx of inflammation in several joint: suspect inflammatory arthritis

Further investigations:

- **Blood tests:** if malignancy, poly myalgia rheumatica, or inflammatory arthritis are suspected
- Testing for **diabetes** considered if pt with frozen shoulder
- **X-rays:** if Hx of trauma; little improvement with conservative care; Ssx last more than 4 weeks; severe pain or restriction of movement; arthritis suspected

Treatments:

- Initial management: explanation & education on diagnosis; analgesia if appropriate; MSK treatment
- Corticosteroid injections may be considered, depending on the suspected cause & Ssx severity
- If orthopaedic referral is indicated (suspected septic arthritis / dislocation), should not be delayed
- Referral to 2° care considered if pain & function are not improving following conservative treatment for 3 months
- Earlier referral considered if: pain is having significant impact on ADLs; recurrent shoulder instability; severe post-traumatic pain

Adhesive capsulitis

- **GREEN or YELLOW** - Condition gradually develops
- **Intro:**
  - Also known as *Frozen shoulder*
  - Inflammatory condition causing stiffness & px in the shoulder joint
  - Dx emphasises the gradual development of global limitation of shoulder motion
  - Significant radiographic findings may not be present
  - Assessing passive ROM crucial for Dx

Adhesive capsulitis (cont)

• **Aetiology (risk factors):**	- Prevalence 2-5% in general pop - Common in people starting 55yrs - F>M (1.4:1) - Non-dominant hand often affected - Associated w/ autoimmune comorbidities: thyroid disorders, DM (poorer treatment outcomes) **Form classification:** - **1°:** idiopathic, gradual onset, associated w/ other conditions (e.g. DM, thyroid disease, drugs, hypertriglyceridemia, or Cx spondylosis) - **2°:** result of shoulder trauma (e.g. rotator cuff tears, #, surgery, or prolonged immobilisation)
• **Pathophysiology:**	- Not fully understood - Leading hypothesis: inflammation begins in joint capsule & synovial fluid - Subsequent reactive fibrosis & adhesions in synovial lining - Initial inflammation causes px - Fibrosis & adhesions limit ROM
• **Clinical presentation:**	- Gradual onset of shoulder px - Worsens over weeks - months - Followed w/ significant limitation in shoulder ROM
• **Physical examination:**	- Reduced AROM & PROM - Specifically affected movements: EXT rotation → ABD → INT rotation → forward FX - Severe cases may loose natural arm swing during walking & muscular dystrophy - TTP around the joint - Distal neurology MUST remain intact - RROM elicits px & marked limitation, resembling rotator cuff tear - Apley scratch test: measure INT rotation - +ve special tests: Need, Hawkins, & Speed's (indicating impingement or biceps tendinopathy)
• **Diagnosis:**	- Dx based mainly on clinical & physical findings - X-rays considered for alternative Dx or underlying pathology - Injection test can help differentiate adhesive capsulitis from other shoulder pathologies - MRI may reveal characteristic findings: rotator interval synovitis, coracohumeral lig hypertrophy, loss of subcoracoid fat triangle, & thickening of the GH capsule (they're not specific)

Adhesive capsulitis (cont)

• **Staging:**	**3 clinical phases:** - **Phase 1:** painful phase is characterised by diffuse & disabling shoulder px, initially worse at night, along w/ increasing stiffness, can last 2-9 months - **Phase 2:** frozen or adhesive phase involves a progressive limitation in ROM in all shoulder planes, the intensity of px gradually diminishes during this phase, typically lasts from 4-12 months - **Phase 3:** thawing or regression phase is marked by gradual return of the ROM, recovery of ROM may take 12-24 months for complete restoration
• **Complications:**	- Residual shoulder px &/or stiffness - Humeral fracture - Rupture of the biceps & subscapularis tendons - Labral tears - GH dislocation - Rotator cuff tear
• **Management:**	- SMT & STW - IASTM/TFM - Spencer technique - NSAIDs - Corticosteroids / steroid injections - Arthroscopic capsular release - Exercises phase 1: Codman pendulum, Cane - FX & ABD, Cross body stretch, Shoulder INT rotation - towel, EXT rotation doorway stretch - Exercises phase 2: Side lying horizontal ABD, resisted shoulder EXT prone, Resisted shoulder FX **Indication for surgery:** - Pt fails a trial of steroids or NSAIDs - No response to GH or SC injections - No response respond to PT **Contraindications for surgery:** - Pt has had an inadequate course of steroids or NSAIDs - Pt has not had any attempt at conservative therapy - Acute infection - Pt has a concomitant malignancy in the shoulder - Pt has a neurological deficit or nerve complaint originating from the Cx spine

Adhesive capsulitis (cont)

• **Ddx:**	- Cx radiculopathy - AC arthrosis - Bicep tendinopathy - GH arthritis - Fracture - Calcifying tendinitis/synovitis - Malignancy - Polymyalgia rheumatica - Shoulder impingement s.

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AC joint injury

• **GREEN to RED**	- Sprain degree to torn degree
• **Intro:**	- Common among athletes & adolescents - Around 40% of all shoulder injuries - Mild injuries usually don't cause significant morbidity - Severe injuries can result in substantial strength loss & function of shoulder - AC injuries may be linked to clavicular # - They can lead to impingement s. - Neurovascular insults are a rare complication associated w/ AC injuries
• **Aetiology (risk factors):**	- Commonly occur after sporting events, car accidents, & falls - Make up about 40% of all shoulder injuries - Up to 10% of all injuries in collision sports (e.g. football, lacrosse, & ice hockey) - AC joint: lateral process of clavicle meets the acromion process projecting off the scapula - Stabilised by AC lig (anterior, posterior, superior, & inferior portion), where superior portion crucial for stability - Mild injuries don't cause significant issues, but severe can lead to substantial strength & function loss - Linked to clavicular #, impingement s., & occasionally neurovascular problems
• **Pathophysiology:**	- Most common: direct trauma to lateral aspect of the shoulder or acromion process w/ the arm in ADD - Falling on an outstretched hand or elbow may also lead to AC separation

AC joint injury (cont)

• **Clinical presentation:**	- Antero-superior shoulder px **Mechanism of injury:** - Blunt trauma to ABD shoulder - Landing on outstretched arm **Px description:** - Radiating to neck or shoulder - Aggravated by movement - Worse when sleeping on affected shoulder
• **Physical examination:**	- Swelling, bruising, or deformity of AC - TTP - Restricted A&PROM due to px - "Piano key sign": palpation of the distal clavicle demonstrates a feeling of "giving way" - Cross-body ADD test - BvR test - Paxino's test - AC differential test
• **Diagnosis:**	- X-rays are 1° for Dx - US & MRI may be considered if Dx remains uncertain
• **Staging:**	**Rockwood classification** (gold standard)**:** **I:** AC ligament sprain; CC ligament intact; no radiographic abnormalities **II:** AC ligament is torn; CC ligament sprain; clavicle has elevated but is not superior to the border of the acromion, or exhibits a less than 25% increase in the CC interspace compared to the contralateral **III:** AC & CC ligaments are torn; clavicle has elevated above the border of the acromion, or there is an increase of 25-100% in the CC interspace compared to the contralateral **IV:** AC & CC ligaments are torn; posterior displacement of the distal clavicle into the trapezius **V:** AC & CC ligaments are torn; superior displacement of the distal clavicle by more than 100% in the CC interspace compared to the contralateral **VI:** AC & CC ligaments are torn; inferolateral displacement in a subacromial or subcoracoid displacement behind the coracobrachialis or biceps tendon

AC joint injury (cont)

• **Complications:**	- Residual joint px (30-50% of pts) - AC arthritis (more common in surgical management) - Following fixation: hardware irritation, infection, adhesive capsulitis, coracoid, & clavicular # - Hook plate: acromion irritation, subacromial impingement, & osteolysis
• **Management:**	- Generally favourable prognosis - Functional motion regain by 6 weeks & return to normal activity by 12 weeks - Non-operative grade 1, 2 & 3; 3 operative if athlete / > displacement - Acute (within 6 weeks): stabilisation & reduction of Ssx - STW - SMT (not shoulder) - IASTM / TFM - Exercises phase 1: scapular clocks & protraction / retraction - Exercises phase 2: resisted shoulder EXT rotation, cane - FX, low row
• **Ddx:**	- AC distal clavicle osteolysis - AC arthritis - Acromion # - Adhesive capsulitis - Anterior humerus subluxation - Complex pain s. - Erb-Duchenne injury - Glenoid labrum tear - Os acromiale - Rotator cuff injury - Superior tabral tear - Septic arthritis - Shoulder dislocation

link text

AC osteoarthritis

• **GREEN**

AC osteoarthritis (cont)

• **Intro:**	- Common (spec. in 40 & older) & causes anterior / superior shoulder px - Px exacerbated during overhead & cross-body activities - 1° affects middle-aged pts due to degeneration of the fibrocartilaginous disc - Many pts are asymptomatic, w/ findings often discovered incidentally on shoulder x-ray / MRI
• **Aetiology (risk factors):**	- Less common than knee / hip OA, but more common than GH OA - Approx 54-57% of elderly pts exhibit x-ray evidence of degenerative changes in AC, though clinically relevant AC OA is less common - Approx 20% of all shoulder px - Common in 40 & older pts **Types of AC arthritis:** - **1° OA:** articular degeneration w/o an apparent underlying cause, often occurring due to constant stress from repeated overhead lifting activities - **2° OA:** resulting from associated causes such as post-trauma (prevalent) or underlying disease (e.g. RA) - Arthritic Ssx have been observed in Grade I & II sprains of the AC
• **Pathophysiology:**	- AC is a synovial joint connecting the axial skeleton & scapula - Limited ROM characterises the AC - Articular connection involves the distal clavicle's convex surface & the acromial facet's slight convex surface - Fibrocartilage disc exists between the hyaline cartilage covered facets (akin to knee meniscus) - Degenerative changes are part of the natural process - In early adulthood, the fibrocartilage disc undergoes degeneration, leaving behind fibrous remnants

AC osteoarthritis (cont)

• **Clinical presentation:**	- Hx of trauma, e.g. direct impact on the joint or a FOOSH injury - Occupational Hx, e.g. occupation that requires repeated overhead lifting activities - Participation in sports that stress / injure AC, e.g. weightlifting, rugby - Complaints of px at night during sleeping on affected shoulder - Pt may experience *popping*, *clicking*, *grinding*, or *catching* sensation w/ movement of the shoulder - Functional limitations ACJ px include difficulty w/ resisted-training activities that place the GH in an extended position, common in weightlifters AKA *Weightlifter's Shoulder* - Damage to AC can be synchronous w/ damage to the supraspinatus tendon & osteophytes from the arthritic AC joint may contribute to *subacromial impingement* exacerbating & producing further shoulder px
• **Physical examination:**	- Pts typically maintain intact ROM, EXCEPT for specific movements: cross-body ADD, behind the back (scratch back), & overhead reaching, which exacerbate px - Localised superior shoulder px is common - TTP, possible accompanied w/ swelling due to distal clavicle osteolysis - Px can be induced in deltoid area through certain movements: forward FX to 90° w/ horizontal ADD (Cross-body test) or straight-ahead pushing (e.g. bench press) - Most sensitive tests: TTP over Acromioclavicular point & Paxino's test, & AC resisted EXT test
• **Diagnosis:**	- Dx relies on Hx, physical exam, imaging (x-ray, MRI), & diagnostic local anaesthetic injection **Imaging:** - Plain film & Dx local anaesthetic local injections are essential Dx tools - X-ray & MRI provide comprehensive imaging of AC joint pathology - US is effective in detecting signs of AC OA & is commonly used for imaging - US-guided injections: +ve if Ssx reduction; -ve if persistent px post-injection suggesting alt shoulder pathologies (commonly rotator cuff injury)

AC osteoarthritis (cont)

• **Management:**	- Activity modification (avoid repetitive & overhead movements), NSAIDs, PT modalities, corticosteroid & local anaesthetic injections - Surgery **Physical therapy:** - Px management using electro-modalities, SMT/STW - Maintaining active ROM & strengthening scapular stabiliser muscles - Rotator cuff strengthening exercises - Postural correction - pec muscle stretching & retractors strengthening
• **Ddx:**	- Calcific tendonitis - GH arthritis - Adhesive capsulitis - Rotator cuff impingement s.

link text

Tendinopathies*

"**Tendinopathy** is an umbrella term to decribe the tendon px, w/ an unknown cause"

"**Tendinitis** describes a tendon in which inflammatory processes are present. However, studies show that tendons are rather in a degenerative state than in an inflammatory state."

"**Tendinosis** describes the degenerative state of tendons & therefore, this term is more applicable"

• Eccentric exercises major role in treatment
- Promote cross-linking of collagen fibres
- Promote tendon remodelling

• Tendinosis can be described on a continuum

Calcific tendonitis

• **GREEN**

• **Intro:**	- Self-limiting disorder, identified by calcium deposits in rotator cuff tendons (esp. infra & supraspinatus) - Common & painful condition, that decreases ROM - Visible signs of calcium deposits overlying rotator cuff insertion on shoulder x-rays
• **Aetiology (risk factors):**	- Up to 20% of pts are asymptomatic - 40-60% of shoulder pts - 30-60yrs - F>M **Localisation:** - Supraspinatus tendon (80%): critical zone (most common) - Infraspinatus tendon (15%): lower 1/3 - Subscapularis tendon (5%): pre-insertional fibres

Calcific tendonitis (cont)

• **Pathophysiology:**	- Unclear **Hypothesis include:** - Repetitive trauma of tendon → tendon degeneration → calcification - Tendon necrosis → intracellular calcium accumulation - Active process mediated by chondrocytes arising from metaplasia → calcium deposition - Phagocytosis of metaplastic areas reforms normal tendon
• **Clinical presentation:**	- Night px, causing loss of sleep - Constant dull ache - Px increases considerably w/ AROM - Decrease in ROM, or complaint of stiffness - Radiating px up into suboccipital region, or down into the fingers
• **Physical examination:**	**Cluster (+ve):** - Neer's test - Hawkins-Kennedy test - Drop arm test - Jobe's test
• **Staging:**	**Chronic (silent) phase:** presence of the calcific deposit is asymptomatic & may be so for years **Acute painful phase:** severe px, disability, & frequently nocturnal discomfort **Mechanical phase:** tendon impingement being a prominent finding; px of less severe nature than the acute phase
• **Diagnosis:**	- Diagnosed through x-rays
• **Complications:**	- Adhesive capsulitis - Rotator cuff tear - Ossifying tendinitis
• **Management:**	- NSAIDs, PT, stretching & strengthening, steroid injections - ESWT (most useful in refractory calcific tendonitis in the formative & resting phase) - US-guided needle lavage - Surgery (surgical decompression of calcium deposit) **Physical therapy:** - Mobs / drops - ROM exercises to avoid articular stiffness - Strength exercises to restore normal mechanics - Commonly scapular dyskinesia needs to be treated at the same time

Calcific tendonitis (cont)

- **Ddx:**
  - Incidental calcification: found in 2.5-20% of 'normal' healthy shoulders
  - Degenerative calcification: found tendons w/ tear Hx; generally smaller; slightly older individuals
  - Loose bodies: associated chondral defect; associated 2° OA

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GH dislocation

- **RED**
- **Intro:**
  - Separation of the humerus from glenoid of scapula at the GH joint
  - 50% of all joint dislocations
  - Anterior dislocation most common
  - Shoulder is an unstable joint due to a shallow glenoid that only articulates w/ a small part of humeral head
- **Aetiology (risk factors):**
  - Directions: anterior, posterior, inferior, or anterior-superior
  - **Risk factors:** Hx of shoulder dislocation, RC tear, Hx of glenoid fracture
  - M>F
  - Younger individuals, likely due to higher activity levels, more prone to redislocation
  - Dislocation occurs due to a strong force or extreme rotation, e.g. blow to the shoulder or trauma from contact sports, motor vehicle accidents, or falls
  - Fibrous tissue connecting the shoulder bones can be stretched or torn during dislocation, complicating injury

GH dislocation (cont)

• Pathophysiology:	**Anterior dislocation:** - Up to 97% of shoulder dislocations - Mechanism: typically a blow + ABD + EXT rot + EXT - Exam findings: ABD + EXT rot arm, prominent acromion - Associated injuries: nerve damage, labrum tears, glenoid fossa or humeral head fractures (up to 40%) **Posterior dislocation:** - 2-4% of shoulder dislocations - Mechanism: hit to the anterior shoulder, axial loading of ADD + INT rot arm - Exam findings: arm held in ADD + INT rot, inability to EXT rot - Higher risk of associated injuries: surgical neck or tuberosity #, reverse Hill-Sachs lesions, labrum or rotator cuff injuries **Inferior dislocation (laxation erecta):** - Least common type (less than 1%) - Mechanism: hyperABD or axial loading on the ABD arm - Exam findings: arm held above & behind the head, inability to ADD the arm - Often associated w/: nerve injury, rotator cuff injury, tears in the internal capsule, highest incidence of axillary nerve & artery injury among shoulder injuries
• Clinical presentation:	- Pts may report: popping sensation, sudden onset of px w/ decreased ROM, sensation of joint rolling out of the socket - Ask about PREVIOUS dislocations - Nerves can get stretched out during shoulder dislocation, some pts may report *stinging* & *numbness* in the arm at the time of dislocation

GH dislocation (cont)

• **Physical examination:**	- ROM diminished & painfull - **Anterior dislocation:** arm ABD & EXT rot; in thin pts potentially prominent funeral head felt anteriorly, & void can be seen posteriorly - **Posterior dislocation:** easy to miss (pt appears to only guard the extremity) because arm is in INT rot & ADD; in thin pts potentially prominent head can be palpated posteriorly - Neurovascular exam (IMPORTANT): axillary nerve injury (40%) - **Special tests:** apprehension test (anterior & posterior), sulcus sign (inferior instability), load & shift test (anterior & posterior), anterior & posterior drawer test
• **Diagnosis:**	- Assess for axillary nerve injury: innervates deltoid & teres minor, & sensation to lateral shoulder - Fractures of tuberosity & surgical neck may occur - *Bankart lesion:* disruption of glenoid labrum, w/ or w/o avulsed bone fragment - *Hill-Sachs deformity:* compression # of postern-lateral humeral head 1° w/ anterior dislocations - *Reverse Hill-Sachs deformity:* impaction # of antero-medial aspect of humeral head in posterior dislocations
• **Management:**	- Posterior shoulder reduction **Anterior shoulder reduction:** - Scapular manipulation: highest success rate - EXT rotation technique - Cunningham technique - Milch technique - Stimson technique - Traction-countertraction technique - Spaso technique - FARES technique - Fulcrum technique
• **Ddx:**	- AC injury - Bicipital tendonitis - Clavicle fracture - RC injury - Shoulder dislocation - Swimmer's shoulder

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GH instability*

• **YELLOW**

• **Intro:**	- Includes dislocation & subluxation events - Approx 1-2% of general population experience GH dislocation in their lifetime - Shoulder instability events are common among young, active, athletic population - Anterior shoulder instability accounts for over 95%
• **Aetiology (risk factors):**	**Classification criteria:** - Uni- or multidirectional instability - Traumatic or atraumatic - Presence or absence of accompanying soft-tissue hyperlaxity - M>F - Rugby & football have particularly high incidence rates - Anterior labral tears & Hill-Sachs lesions are frequently observed

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GH instability* (cont)

• Pathophysiology:

GH anatomy:

- Complex, mobile, multiracial ball-and-socket articulation
- Allows motion in frontal, transverse, & sagittal planes
- Glenoid fossa articulates w/ humeral head, allowing 360° circumduction
- Movements at 4 distinct joints: SC, AC, GH, & scapuloTx

Stabilisers:

- **Static:** GH articulation, labrum, ligaments, RC interval structures, intra-articular pressure
- **Dynamic:** RC muscles, deltoid, scapular & periscapular stabilisers

Shoulder instability cascade:

- Excessive translation of humeral head on glenoid leads to px, weakness, dysfunction
- Anatomic risk factors identified
- Differentiation between joint laxity & instability crucial

Unidirectional instability:

- May result from acute trauma or low-energy instability events
- Soft tissue hyperlaxity may accompany
- Hill-Sachs lesion on humeral side common
- Glenoid bone loss prevalent, detected via CT scans
- Blunted osseous defects due to acute or chronic/recurrent processes

Multidirectional instability:

- Definition not precise; involves instability in multiple directions
- Often accompanied by capsulolabral injuries
- Soft tissue hyperlaxity associated w/ generalised hyperlaxity

Long-term implications:

- Altered biomechanics due to glenoid bone loss
- Scapular dyskinesia common, predisposing to instability
- Recurrent instability possible post non-operative/operative management
- Dislocation arthropathy: degenerative changes following instability events, possibly leading to GH arthritis

GH instability* (cont)

• Clinical presentation:	**1st time dislocations:** - Recent high-energy trauma or collision is often reported as the cause - Ask about: degree of trauma, sports activities & positions, discernment between true dislocation & subluxation, & the need for manual reduction **Chronic cases:** - Pts often present after ROM limitations impact daily activities significantly - Detailed Hx of inciting instability events should be gathered - Initial injury may be overlooked, leading to chronic instability/recurrence - Heightened clinical suspicion is warranted in cases of seizures, polytrauma, or low-energy, recurrent subluxation

GH instability* (cont)

• **Physical examination:**	**Cx exam:** - Rule out Cx radiculopathy in neck or shoulder pathology - Evaluate neck posturing, muscular symmetry, palpable tenderness, & ROM - Conduct *Spurling manoeuvre*, myelopathies testing, reflex testing, & neurovascular exam **Shoulder exam:** - Compare B shoulder girdles for asymmetry, muscle bulk, or atrophic changes - Check for anterior fullness in chronic anterior instability - Assess scapulothoracic motion & scapular winging - AROM & PROM, noting limitation in complex instability cases - Assess axillary nerve function, supraspinatus muscle, & sensory examination **Provocative tests:** - Assess global tissue laxity, GH translation, & hypermobility - Anterior apprehension test: reproduce Ssx of anterior instability - Jobe relocation test: alleviate Ssx - Load & shift test: assess humeral head translation (Grade 1, 2, or 3) **Other exam considerations:** - Check for posterior & multidirectional instability - Expect associated shoulder pathologies based on age (e.g. RC injuries in older pts) - Note weakness or px of specific shoulder injuries (e.g. RC tears or Bankart lesions)
• **Diagnosis:**	- X-rays for comprehensive evaluation - MRI & CT for advanced imaging
• **Complications:**	- Redislocation following surgical fixation - Nerve injuries (esp. axillary n.) - Infection (surgery) - Implant-related problems

GH instability* (cont)

• Prognosis:

- Depends on various factors
- Instability severity index score (ISIS) to guide shoulder instability management
- Risk factors for recurrence: age, gender, joint hyperlaxity, sport participation level/type, Hx of instability, & osseous lesions (10-point scoring of ISIS)

5-year overall success rates:

- 94% w/ 1-2 risk factors (ISIS score ≤ 3)
- 85% w/ ISIS score of 4-6
- 55% w/ ISIS score >6

• Management:

- Rehab program aim: enhance scapular stability; correct postural or functional deficits; increase RC function; improve proprioception
- Closed-chain exercises help stability w/o increasing shear force
- **Phase 1 (rehab):** decrease px, regain ROM, improve functional coordination
- **Phase 2 (exercises):** improve strength, coordination, proprioception
- Derby shoulder instability programme for recurrent *posterior* instability: stepwise exercise progression
- Scapular stability exercises focus on improving retraction & EXT rotation
- RC deficits, especially subscapularis, are crucial to address
- *Forward shoulder posture* may benefit from SMT in EXT rotation
- *Controllable* functional instability usually managed conservatively; *non-controllable* cases may need surgical repair
- Six-month conservative care trial appropriate before surgical intervention for non-traumatic posterior instability

GH instability* (cont)

- **Ddx:**
  - Labral defect
  - SLAP lesion
  - Bankart lesion
  - Hill-Sachs lesion
  - Fracture
  - Inflammatory arthropathy
  - Shoulder impingement
  - RC tendinopathy
  - Biceps tendinopathy
  - Suprascapular n. entrapment
  - Quadrilateral space s.
  - Cx spine referral
  - Radiculopathy

link text

GH internal rotation deficit (GIRD)

- **GREEN**
- **Intro:**
  - Commonly results from repetitive over-head throwing
  - Results in loss of IR
  - Functional deficit, not a specific injury
- **Aetiology (risk factors):**
  - Throwing motion ABD + ER + EXT w/ high velocities
  - High amount of stress on static & dynamic stabilisers of shoulder
  - Throwers often have a component of pathologic laxity or micro-instability (deposition for injuries)
- **Pathophysiology:**
  - Chronic tensile loading of posterior capsule leads to micro-tears & scarring
  - Resultant tissue changes contribute to loss of INT GH rotation
  - Limitation contributes to various shoulder, elbow, & wrist conditions
  - Sequellae inc: scapular dyskinesia, anterior shoulder impingement, RC s., & labral lesions
  - Limited shoulder ROM can also result from these conditions
- **Clinical presentation:**
  - Vague posterior shoulder px
  - Need for prolonged warm-up due to shoulder stiffness
  - Loss of throwing velocity, described as dead arm
  - Ssx exacerbated in the late cocking phase of throwing, typically localised to the posterior shoulder
  - Rare radiation of discomfort extending into the arm

GH internal rotation deficit (GIRD) (cont)

• **Physical examination:**	- Increased EXT rotation & decreased INT rotation - NOT related to MSK injuries or px in overhead throwing athletes - TrPs: infraspinatus & teres minor **GIRD Dx criteria:** - At least 20* deficit of IR in dominant arm (compared B) - TTP in posterior shoulder musculature
• **Management:**	- Target improving shoulder ROM (early focus), reduce muscle stiffness, & increase flexibility - Stretching targets tightness in posterior capsule & INT rotators - pecs, biceps, subscapularis, infraspinatus, teres minor, & levator - Crossbody stretching may be beneficial - After pain-free ROM, follow w/ incremental strengthening of GH & scapular stabilisers - TrPs like infraspinatus & teres minor (EXT rotators) - SMT - IR & inferior glide - Rest from throwing & physical therapy for 6 months
• **Ddx:**	- Shoulder Impingement s. - RC s. - Biceps tendinopathy - Labral lesion

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Glenolabral articular disruption (GLAD)*

• **YELLOW or RED**

• **Intro:**	- Soft tissue shoulder injury subtype - Involves a tear to anterior-inferior labrum & adjacent glenoid articular cartilage damage - Uncommon but established post-trauma cause of shoulder px - Associated w/ stable GH joint; full ROM w/o apprehension or subluxation - GLAD lesions seen in isolated or recurrent dislocations, challenging clinical Dx - Imaging required for confirmation

Glenolabral articular disruption (GLAD)* (cont)

• **Aetiology (risk factors):**	- Rare condition - Est. 1.5-2.9% of cases of traumatic labral tears - Younger M, consistent w/ general traumatic labral pathology - Result from shoulder joint trauma, often involving forced ADD from a position of ABD + EXT rot, e.g. *FOOSH* - Injury mechanism also inv. forceful ADD from throwing - Anterior GH instability is a common injury mechanism associated w/ GLAD
• **Pathophysiology:**	- Affects the labrum & underlying glenoid cartilage in the GH joint - GH: synovial ball & socket joint formed by the humeral head & glenoid fossa of the scapula - Labrum function: adds depth to fossa & attachment point for long head of biceps tendon & GH ligaments - Anterior labroligamentous complex: anterior-inferior GH ligament & labrum - Function: prevents anterior dislocation & maintaining shoulder stability - Injury mechanism: forceful ADD of the humeral head against the glenoid fossa, potentially accompanied by shear force, resulting in tears to the labrum & varying degrees of cartilage damage - Despite the damage, the anterior labroligamentous complex often remains intact → shoulder joint remains stable in GLAD lesions - Association between GLAD lesions & anterior shoulder instability
• **Clinical presentation:**	- Younger male, w/ clear onset of px after the event - Potentially anteriorly, possibly diffusely - Pt may localise px to deep-seated anterior joint - Clear Hx of FOOSH, mechanism ADD force onto an ABD + EXT rot shoulder
• **Physical examination:**	- Px may be elicited on ABD & EXT rot - Force ADD may produce *'popping'* sensation - **Special tests:** Crank test; O'Briens test; Anterior Apprehension test; Passive Compression test - High association between GLAD & anterior shoulder instability

Glenolabral articular disruption (GLAD)* (cont)	
• **Diagnosis:**	- Imaging, especially MRA, crucial for Dx - Challenging to detect on non-contrast MRI or CTA - Findings: superficial tear to the anterior-inferior labrum w/ an underlying glenoid cartilage defect (from superficial to trans-chondral) - MRA demonstrates contrast tracking the labral tear & filling into the chondral defect or under a damaged articular flap
• **Complications:**	- Linked to episodes of anterior shoulder instability - Higher failure rates in arthroscopic Bankart repair w/ GLAD lesions - Correlation between GLAD lesions & reduced GH stability - GLAD lesions as biomechanical risk factor in shoulder instability by reducing joint concavity depth - Risk of OA following GLAD injury (hypothesis)
• **Management:**	- Conservative: time, NSAIDs, & PT (especially for older pts) - Incidental findings on imaging may complicate Dx in older pts due to common age-related cartilage & labral degeneration - Treatment approach depends on the size & nature of the chondral defect & labral injury
• **Ddx:**	- Common traumatic labral tears, tearing of the labrum & associated ligaments partially or completely off the glenoid, most commonly the anterior-inferior labrum *(Bankart lesions)* - Anterior-inferior instability lesions that include a glenoid rim # - *bony Bankart lesions* - *Perthes lesion*: labral complex injury, but the labrum is still attached to the glenoid via a periosteal sleeve - *Anterior ligamentous periosteal sleeve avulsion*: another labral injury, but it displaces medially on the glenoid neck - *(HAGL)* or *Bony HAGL*: this time, the anterior-inferior GH ligament is avulsed from the humeral rather than labral attachment

link text

Polymyalgia rheumatica (PMR)*	
• **YELLOW or RED**	- Red if signs of vascular arteritis
• **Intro:**	- Rheumatic disorder 1° affecting white adults >50 - Characterised by px in neck, shoulder, & hip areas - Inflammatory condition w/ elevated erythrocyte sedimentation rate (ESR) & C-reactive protein (CRP) - Coexistence w/ or development of Giant cell arteritis (GCA) possible - Dx challenges inc. distinguishing PMR from other conditions - Does not lead to RA development
• **Aetiology (risk factors):**	- 100,000 / year - White >50 - Second most common inflammatory autoimmune rheumatic disease (after RA) - Etiology not well understood - Some genetic predisposition - Infection contribute: mycoplasma pneumonia, parvovirus B19, & Epstein-Barr virus (EBV) - Some connection between PMR & diverticulitis, suggesting a role for changes in microbiota & chronic bowel inflammation
• **Pathophysiology:**	- Immune-mediated disorder - Elevated inflammatory markers are common - PMR pts have decreased number of circulating B cells (correlates w/ ESR & CRP) compared to healthy pts
• **Clinical presentation:**	- Symmetrical px & stiffness: affects shoulders, neck, & hip girdle - Morning stiffness: worst in the morning, worsens after rest or inactivity - Restricted shoulder ROM: common - Upper body complaints: px & stiffness in upper arms, hips, thighs, upper & lower back - Rapid onset: Ssx develop within day - 2 weeks - Impact on quality of life: px impairs sleep & ADLs, e.g. getting out of bed, showering, driving, etc - Inflammation sites: GH & hip joint, subacromial, subdeltoid, & trochanteric bursa - Systemic Ssx: fatigue, malaise, anorexia, weight loss, low-grade fever (in some cases) - Peripheral involvement: arthritis in 1/4 of pts, carpal tunnel s., distal extremity swelling w/ pitting edema, distal tenosynovitis

Polymyalgia rheumatica (PMR)* (cont)

• Physical examination:

- Diffuse tenderness over shoulder
- Restricted AROM
- Normal PROM
- Restricted Cx & hip movements
- Muscle tenderness: neck, arms, & thigh
- Intact muscle strength despite complaints of weakness
- Normal sensory & reflexes (helps rule out mimicking conditions, e.g. peripheral neuropathy)
- Gait changes due to px & stiffness, e.g. shortened stride length, slow gait speed, stiffness, difficulty initiating movement, antalgic gait, decreased arm swing, & trunk lean

• Diagnosis:

Labs:

- Elevated ESR (>40mm)
- Elevated CRP
- Liver enzymes, especially alkaline phosphate, occasional elevated
- Serologic test (ANA, RF, Anti-CCP AB) negative
- CPK value within normal range

Imaging:

- US: assess subacromial/subdeltoid bursitis, biceps tenosynovitis, & GH synovitis
- MRI: depicts bursitis, synovitis, & tenosynovitis, more sensitive for hip & pelvic girdle findings; pelvic MRI often shows B peri-t-endinous enhancement of pelvic girdle tendons & occasional low-grade hip synovitis
- PET: shows FDG uptake in shoulders, ischial tuberosities, greater trochanters, GH, & SC joints

Provisional classification criteria for PMR:

Age 50 or older w/ B shoulder aching & abnormal CRP/ESR, + specific points from:

- Morning stiffness >45 min duration
- Hip px or restricted ROM
- Absence of rheumatoid factor or anti-citrullinated protein antibodies
- Absence of other joint involvement
- US findings (if available)

Polymyalgia rheumatica (PMR)* (cont)	
• **Complications:**	- PMR pts have an increased risk of CV diseases - Premature arteriosclerosis due to chronic inflammation is the probable cause of premature coronary artery disease (CAD) - Some increased risk of lymphoplasmacytic lymphoma - Higher likelihood of developing inflammatory arthritis (factors: small joint synovitis, younger age, & +ve anti-CCP)
• **Management:**	- Excellent prognosis w/ prompt Dx & appropriate treatment - Medication - Vitamin D & calcium supplementation for long-term steroids - Pt should be educated on temporal/optic arteritis & how to act
• **Ddx:**	- RA - GCA - ANCA related vasculitis - Inflammatory myositis & statin-induced myopathy - Gout & CPPD - Fibromyalgia - Overuse or degenerative shoulder pathology (e.g. OA, RC tendinitis & tendon tear, adhesive capsulitis) - Cx spin disorders (e.g. OA, radiculopathy) - Crown dens s. - Hypothyroidism - Obstructive sleep apnea - Depression - Viral infections (e.g. EBV, hepatitis, HIV, parvovirus B19) - Systemic bacterial infections, septic arthritis - Cancer - Diabetes
• **Temporal arteritis (TA):**	- 1 in 5 pts develop TA - Systemic inflammatory vasculitis of arteries - Scalp is painful to touch (hair brushing) - Prominent, hardened & tender superficial temporal artery - HA - Claudication masticatoria - Preliminary stage to optic arteritis (threat to visual ability)

link text

Rotator cuff injury*	
• **GREEN to RED**	- Grade 1, 2, 3
• **Classification of strains:**	- **Grade 1 (green):** few torn/stretch muscle fibres w/ normal strength - **Grade 2 (yellow):** several injured muscle fibres w/ muscle px, tenderness, mild swelling, bruising & loss of strength - **Grade 3 (red):** complete tear of muscle w/ a possible audible sensation & a total loss of muscle function, severe px, bruising & swelling - Referral depending on grade
• **Intro:**	- RC injuries range from *tendinopathy* to *complete tears* - Rotator cuff: subscapularis (INT rotator), supraspinatus (ABductor), infraspinatus (EXT rotator), & teres minor (EXT rotator)
• **Aetiology (risk factors):**	- Most common tendon injury in adults - Approx 30% of adults >60 have a tear, 62% in those >80 - Age is 1° factor for RC disease, being degenerative & progressive - Risk factors: smoking (increases severity), family Hx, poor posture (kyphotic-lordotic, flat-back, swayback), trauma, hypercholesterolemia, & overhead activities - Partial tears are prone to further propagation, factors inc. tear size, Ssx, location, & age - Larger tears more likely to deteriorate structurally, w/ actively enlarging tears having higher likelihood of developing Ssx - Anterior tears are more likely to progress to cuff degeneration
• **Pathophysiology:**	- **Macro**-trauma leads to acute tears, commonly in younger pts, resulting in complete tears - **Micro**-trauma causes tendon degeneration, leading to degenerative tears - Acute tears are typical in younger pts, while degenerative tears occur in older pts - Sufficient tendon degeneration can make a complete tear possible w/ less force - Multiple possible mechanisms: chronic degenerative tear, chronic impingement, acute avulsion injuries, iatrogenic injuries

Rotator cuff injury* (cont)

• **Clinical presentation:**	- Typically begins w/ px, which can be acute or gradual - Athletes often adapt biomechanics until they can no longer do their sport w/o px - Pts may experience increasing px & difficulty w/ overhead activities & lifting heavy objects - Px can radiate into the deltoid muscle area & may be felt when lying on the affected side - Younger pts often have overuse tendinopathy - Older pts may have OA contributing to the condition
• **Physical examination:**	- Tenderness at muscle insertion - Muscle atrophy - Abnormal scapular motion - **Special tests:** Jobe (empty can) test, resisted EXT rotation, belly press test, drop arm test, & EXT & INT rotation lag sign
• **Diagnosis:**	- Plain radiography - US - good for evaluating RC - MRI - gold standard
• **Complications:**	- Retearing the cuff repair - Adhesive capsulitis - Inability to regain motion - Cuff strength
• **Management:**	- Surgical & conservative treatment largest improvement at 12 months - Surgery generally recommended for complete tears in pts <40, followed by rehab - Conservative: PT, NSAIDs, subacromial corticosteroid injections - STW - SMT Cx & Tx - GH mobs - Nerve floss - brachial plexus - Exercise phase 1: Codman pendulum, YTWL scapular depression, GH INT rotation, corner pectoral stretch - Exercises phase 2: low row, eccentric supraspinatus, eccentric scapular stabilisers, eccentric shoulder ER's

Rotator cuff injury* (cont)

- **Ddx:**
  - SLAP or other labral tears
  - Subacromial impingement from bursitis, os acromiale, bone spurs
  - AC OA
  - Biceps tendinitis
  - Cx radiculopathy

link text

Rotator cuff tendinopathy*

- **YELLOW**
- **Intro:**
  - RC injuries vary from minor contusions & tendonitis to chronic tendinopathy, partial tears (PTTs), & full-thickness tears (FTTs)
  - They can impact diverse pt groups, from recreational athletes (weekend warriors) to elite athletes
  - RC pathology is observed across all age demographics
- **Aetiology (risk factors):**
  - Subacromial impingement s. (SIS) is the most common cause of shoulder px, RC tendonitis is often seen associated
  - Occur acutely due to trauma or chronically from repetitive overuse activities
  - 5-10% in pts <20, & over 60% in pts >80
  - **Acute RC tendonitis** often affects athletes due to direct trauma, poor throwing mechanics, or FOOSH
  - **Chronic RC tendinopathy** can result from *extrinsic* compression (mechanical impingement) or *intrinsic* mechanisms (cuff degeneration)
  - Extrinsic compression can be caused by degenerative bursa, acromial spurring, or presupposing acromial morphologies
  - Intrinsic degenerative theories suggests cuff degeneration compromises joint stability, making the cuff susceptible to extrinsic compressive forces
  - Risk factors: vascular changes, age, sex, & genetics

Rotator cuff tendinopathy* (cont)

• **Pathophysiology:**	**Acute RC tendonitis can be caused by:** - Direct blows to the shoulder - Poor throwing mechanics in overhead sports - FOOSH **Tendinopathy develops from repetitive RC injury, leading to:** - Recurrent pathological cycle - Acute or chronic tendonitis - Increasing levels of tendinopathy & tendinosis - Potential progression to PTTs &/or FTTs **Exact pathogenesis of RC tears is controversial, but likely involves:** - Extrinsic impingement from surrounding structures - Intrinsic degeneration within the tendon itself
• **Clinical presentation:**	- Acute RC tendonitis: Hx of trauma or acute exacerbation on a chronic condition - Chronic RC tendinopathy: either acute on chronic Hx/mechanism or a gradual, atraumatic onset - Ssx may worsen w/ overhead activities - Px, especially at night, is common **Thorough exam includes:** - Sports participation (including specific position played) - Occupational Hx & current status - Hand dominance - Hx of shoulder &/or neck injury/trauma - Relevant surgical Hx

Rotator cuff tendinopathy* (cont)

• Physical examination:

Cx exam:

- Rule out Cx radiculopathy (Spurling's test)
- Evaluate neck posturing, muscular symmetry, tenderness, & ROM
- **Special tests:** Spurling's, sensation testing, reflex testing, & neurovascular exam (7 P's)

Shoulder exam:

- Shoulder girdle symmetry, posturing, & muscle bulk
- Check for scapular winging & skin abnormalities
- Palpate for tenderness
- AROM & PROM
- Consider *RC tendonitis* w/ anterolateral tenderness
- Test motor strength C5-T1

Special tests:

- **Supraspinatus (SS):** Jobe's & drop arm test
- **Infraspinatus (IS):** Strength test & EXT rotation lag sign
- **Teres minor (TM):** strength test & Hornblower's sign
- **Subscapularis (SubSc):** IR lag sign, passive ER ROM, lift-off test, & belly press
- **EXT / subacromial impingement:** Neer impingement sign, Near impingement test, & Hawkin-Kennedy test
- **Internal impingement:** pt supine, shoulder brought into terminal ABD & EXT rotation; +ve if px reproduced

• Diagnosis:

- Imaging should be obtained in all pts w/ acute or chronic shoulder px
- Plain radiographs
- US (should be used more due to their specificity)
- MRI (provides more accurate tear details)

Rotator cuff tendinopathy* (cont)

• Complications:

Non-operative:

- Persistent px / recurrent Ssx
- Setting of PTTs: risk of tear propagation, lack of healing, fatty infiltration, atrophy, & retraction
- Risks for tear progression: initial presence of FTT, medium-sized cuff tears (1-3cm), smoking
- Setting of chronic/atrophic tears: DJD & RC atrophy

Surgical:

- Most effective for pts who failed 4-6 months of conservative care
- Risks of surgery: recurrent px/Ssx, infection, stiffness, neurovascular injury, & risks associated w/ anaesthetic use
- Subacromial decompression/acromioplasty: deltoid dysfunction or anterosuperior escape

• Management:

- Majority of pts w/o FTTs improve w/ non-operative management
- NSAIDs, rest/activity modification, cortisone injections
- STW RC muscles
- Cx & Tx SMT
- GH mobs
- Nerve floss - brachial plexus
- Exercises Phase 1: Codman pendulum, YTWL scapular depression, GH IR, & Corner pec stretch
- Exercises Phase 2: low row, eccentric supraspinatus, eccentric scapular stabilisers, eccentric shoulder ER's
- Surgery

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Rotator cuff tendinopathy* (cont)

• **Ddx:**

Impingement:
- External / subacromial
- Subcoracoid
- Calcific tendonitis
- Internal (inc. SLAP, GIRD, little League shoulder, posterior labral tears)

RC pathology:
- Tendonitis (acute), Tendinopathy (chronic or acute on chronic)
- PTTs vs FTTs
- RC arthropathy

Degenerative:
- Advanced DJD (often associated w/ RC arthropathy)
- GH arthritis
- Adhesive capsulitis
- AVN
- Scapulothoracic crepitus

Proximal biceps:
- Subluxation (associated w/ subscapularis injuries)
- Tendonitis & tendinopathy

AC joint conditions:
- AC separation
- Distal clavicle osteolysis
- AC arthritis

Instability:
- Unidirectional instability - seen in association w/ an inciting event/dislocation (anterior, posterior, inferior)
- Multidirectional instability (MDI)
- Associated labral injuries/pathology

Neurovascular conditions:
- Suprascapular neuropathy (can be associated w/ paralabral cyst at the spinoglenoid notch)
- Scapular wining (medial or lateral)
- TOS
- Quadrilateral space s.

Other conditions:
- Scapulothoracic dyskinesia
- Os acromiale
- Muscle ruptures (pec major, deltoid, lat dorsi)
- Fracture (acute injury or px resulting from long-standing deformity, malunion, or nonunion)

link text

Scapulothoracic dyskinesis*

• **GREEN**

• **Intro:**	- Altered position & motion of the scapula - Also known as *dysrhythmia*, *dyskinesia*,or *SICK scapula syndrome* - Scapular wining, exists but denoted a distinct condition typically following Tx or spinal accessory n. injury - Observed in overhead athletes & pts w/ shoulder issues like RC disease, GH instability, impingement s., & labral tears, as well as in healthy pts - No clear relationship between SD & shoulder px, even though some pts present w/ shoulder px - Theory: SD might predict future shoulders even in the absence of current Ssx
• **Aetiology (risk factors):**	**Shoulder-related:** - Shoulder pathologies associated w/ SD *(AC instability, shoulder impingement, RC injuries, glenoid labrum injuries, clavicle #)* - Inflexibility of the pec minor & short head of biceps - Stiffness of posterior GH capsule **Neck-related:** - Mechanical neck px s. - Cx n. root-related s. **Posture-related:** - Excessive Tx kyphosis & Cx lordosis - Athletes show these are more related causes SD

Scapulothoracic dyskinesis* (cont)

• Pathophysiology:

Scapular motions:
- Upward/downward rotation
- Internal/external rotation
- Anterior/posterior tilt

Scapular translation:
- Upward/downward sliding on the Tx
- Medial/lateral sliding around the curvature of Tx

Common scapular patterns:
- Scapular retraction: EXT rot + posterior tilt + upward rot + medial translation
- Protraction: INT rot + anterior tilt + downward rot + lateral translation
- Shrug: upward translation + anterior tilt + INT rot

Normal overhead elevation:
- Minimal INT/EXT rot until 100°
- 1° scapular motion: upward rot
- 2° scapular motion: posterior tilt

Scapulohumeral rhythm:
- Coordinated movement between scapula & humerus for efficient arm movement
- 2:1 ratio between GH elevation & scapular upward rot
- Consistent pattern during scapular plane elevation: upward rot + posterior tilt + EXT rot + clavicular elevation + retraction

Altered mechanics in SD:
- Increased scapular anterior tilt
- Increased scapular INT rot
- Altered scapular upward rot

• Clinical presentation:

- Pts w/ SD can be symptomatic or asymptomatic

Ssx can be one or combination of the following:

- Anterior shoulder px
- Posterosuperior scapular px (may radiate into ipsilateral para spinous Cx region or radicular/thoracic outlet-type Ssx in the affected UL)
- Superior shoulder px
- Proximal lateral arm px

Scapulothoracic dyskinesis* (cont)

• **Physical examination:**	- Assess AC & SC for instability - Infraspinatus strength test - Manual resistance of the arm at 130° of FX (for serratus anterior) - Manual resistance of the arm at 130-150° of ABD (for lower & middle traps) - Extension of the arm at the side (for rhomboids) - Low row test - Scapulohumeral rhythm test - Quadruped rock - Lateral scapular slide test - Scapular dyskinesia test - SICK scapula sign
• **Diagnosis:**	**Classification of dyskinesia types:** - **Type 1:** inferior angle prominence (i.e. anterior tilt of scapula) - **Type 2:** medial border prominence (i.e. winging of the scapula) - **Type 3:** early scapular elevation or excessive/insufficient upward rot during arm elevation
• **Complications:**	- SD diminishes subacromial space & leads to decreased RC strength, impingement Ssx, & eventual RC damage - 100% of pts w/ shoulder impingement demonstrate dyskinesia - 5% of pts w/ dyskinesia have neurologic injury/damage (spinal accessory, long Tx, suprascapular) - SD can occur from core & hip ABD weakness - SD becomes more apparent w/ dynamic testing, particularly during the lowering phase of arm movement - Recognition & rehab should begin independent of (generally absent) Ssx
• **Management:**	- STW: upper traps, pec minor, biceps - SMT: Cx & Tx - Scapular mobs - Treatment aims at restoration of scapular retraction, posterior tilt & EXT rot - Exercises Phase 1: trap stretch - sitting, YTWL scapular depression - Exercises Phase 2: low row, burger w/ band

link text

Subacromial bursitis

• YELLOW	
• **Intro:**	- Bursa is a fluid-filled sac - Lubricatesjoints & body surfaces prone to wear & friction - Subacromial bursa is surrounded by the acromion, coracoid, coracoacromial ligament, & deltoid muscle fibres - Inflammation of this bursa can lead to subacromial bursitis
• **Aetiology (risk factors):**	- Around 0.4% of primary care visits - F=M **Common aetiologies:** - Subacromial impingement (especially in older pts) - Repetitive overhead activities / overuse (athletes, factory workers, manual labourers) - Direct trauma - Crystal deposition - Subacromial hemmorhage - Infection - Autoimmune disease (e.g. RA)
• **Pathophysiology:**	- Aetiologies can cause inflammation of the subacromial bursa, leading to increased fluid & collagen formation - Fluid is often rich in fibrin & can become hemorrhagic **Bursitis has 3 phases:** - **Acute:** marked by local inflammation w/ thickened synovial fluid, resulting in painful movement, especially w/ overhead activities - **Chronic:** constant px due to a chronic inflammatory process, which can weaken & eventually rupture surrounding ligaments & tendons. Require attention to tendinitis as they may coexist - **Recurring:** can result from repetitive trauma or routine overhead activities, & it may also be seen in pts w/ inflammatory conditions (e.g. RA)

Subacromial bursitis (cont)

• **Clinical presentation:**	- Px in the anterolateral aspect of the shoulder - Possible causes: trauma (fall w/ direct impact), repetitive overhead activities (sports, lifting) - Impingement s. as a common cause - Mechanism: decreased subacromial space due to overhead activities - Effect of arm ABD: brings humerus closer to acromion, reducing subacromial space - Function of subacromial bursa: protects supraspinatus muscle from wear between humeral head & acromion - Result of repetitive activity: irritation & inflammation of the bursa - Consideration of tendon pathology: supraspinatus tendinitis or tear may coexist w/ impingement s.
• **Physical examination:**	- TTP at anterolateral aspect of shoulder below acromion - Localised px, doesn't usually radiate (if it does, consider Cx pathology) - Warm or boggy skin at site, but no erythema typically - Px on resisted ABD of arm beyond 75-80° - Compression of subacromial bursa at undersurface of acromion during motion
• **Diagnosis:**	- X-rays may be used to rule out other pathologies (e.g. fractures, dislocations, OA, etc) - MRI: burial fluid accumulation visible - US: evaluates the thickness of the bursa
• **Complications:**	- Not associated w/ many complications - Repeated steroid injections: theoretical risk of introducing an infection into skin/joint - Risk of damaging RC muscles w/ recurrent injections
• **Management:**	- Good prognosis for pts w/ conservative care, even w/ surgery - Rest, NSAIDs, PT, & corticosteroid injections - Surgery for pts non responsive to conservative care
• **Ddx:**	- Impingement syndrome - RC tendinitis/tear - Biceps tendinitis - Adhesive capsulitis - AC joint OA

link text

Subacromial impingement syndrome (SIS)*

• GREEN

• **Intro:**
- SIS is the inflammation, irritation, degradation in subacromial space structures
- Shoulder impingement s. is considered most common cause of shoulder px
- Shoulder px often persists or recurs
- 54% of pts experience persistent Ssx after 3 years

• **Aetiology (risk factors):**
- Common in overhead sports (handball, volleyball), & manual labourers
- Incidence rises w/ age (especially 60s)
- Shoulder *external* impingement distinguished from *internal* impingement by RC anatomy

Extrinsic risk factors:
- Heavy loads
- Infection
- Smoking

• **Pathophysiology:**
- Normal shoulder movement narrows subacromial space, causing px
- Unclear whether tendon damage or narrowed space causes impingement
- Described by location (external/internal) & cause (1°/2°)

Anatomic borders:
- Acromion
- Coracoacromial ligament
- AC joint
- Humeral head

External (subacromial) impingement:
- Mechanical encroachment of soft tissue in subacromial space
- **1° impingement:** structural narrowing (e.g. abnormal acromion)
- **2° impingement:** onset during motion due to RC weakness

• **Staging:**

Neer's classification:
- **Stage 1:** edema, haemorrhage from overuse
- **Stage 2:** fibrosis, irreversible tendon changes
- **Stage 3:** tendon rupture/tear due to chronic fibrosis

Subacromial impingement syndrome (SIS)* (cont)

• Clinical presentation:

- Px upon lifting the arm or lying on the affected side
- Pts may report loss of motion, nighttime px, weakness, & stiffness
- Onset is gradual over weeks to months, w/o a specific traumatic event
- Px is typically felt over the lateral acromion w/ radiation to the lateral mid-humerus
- Inquire: onset, quality, exacerbating factors, interventions tried, & prior injuries
- **Important:** overhead & repetitive activities
- Relief: rest, NSAIDs, ice
- Ssx often return w/ activity

• Physical examination:

- Inspection, palpation, A & PROM, & strength testing of neck & shoulder
- B comparison
- Common weakness: ABD &/or EXT rotation
- *Scapular dyskinesis* during arm forward elevation
- Tenderness over the coracoid process of affected arm

Special tests for shoulder impingement:

- Hawkins test - subacromial (external)
- Neer sign - anterior px = subacromial; posterior px = internal
- Jobe (empty can test)
- Painful arc of motion

Special tests for shoulder instability:

- Sulcus sign
- Anterior apprehension
- Relocation test - internal

• Diagnosis:

- Dx made from physical exam
- Imaging used to confirm & rule out other issues

• Complications:

- Due to structural damage within subacromial space
- Altered biomechanics
- Avoidance of use w/ subsequent atrophy
- Potential pathologies that may result: RC tendonitis/tear, bicipital tendonitis/tear, or adhesive capsulitis

Subacromial impingement syndrome (SIS)* (cont)

- **Management:**
  - Most pts resolve within 2 yrs w/ conservative care (initial approach before considering surgery)
  - Restoring ROM is crucial, avoid aggravating movements e.g. elevation & INT rotation
  - Tape used enhance recovery & decrease px
  - Steroid injections
  - Surgery
  - STW (RC), SMT (Cx/Tx), GH mobs, nerve floss (brachial plexus)
  - Exercises Phase 1: Codman pendulum, YTWL scapular depression, GH INT rotation, Corner pec stretch
  - Exercises Phase 2: low row, Brugger w/ band
- **Ddx:**
  - Adhesive capsulitis
  - RC tear
  - AC OA
  - AC sprain
  - Trapezius muscle spasm
  - Biceps tendonitis
  - Biceps tendon rupture
  - Calcific tendonitis
  - GH arthritis
  - Distal clavicle osteolysis
  - Cx radiculopathy
  - TOS

link text

Hypermobility syndromes (HMS)

- **GREEN**

- **Intro:**
  - Generalised articular hypermobility, w/ or w/o subluxation or dislocation
  - Also known as *joint hypermobility s.* & *benign hypermobility joint s.*
  - Primary Ssx: excessive laxity of multiple joints
  - Differs from localised joint hypermobility & other disorders e.g. Ehlers-Danlos s, RA, lupus, & Marfan s.
  - May occur in chromosomal & genetic disorders like Down syndrome, & metabolic disorders e.g. homocystinuria & hyperlysinemia
  - Lab tests used to exclude other systemic disorders when HMS is suspected

Hypermobility syndromes (HMS) (cont)

• **Aetiology (risk factors):**	- Most prevalent in children & tends to decrease w/ age - Joint mobility is at its highest at birth, decreasing in children around 9-12 yrs - Adolescent girls hypermobility peak at 15, decrease after, influenced by hormonal changes - F>M - More prevalent in ASIA, Africa, & Middle East
• **Pathophysiology:**	- Involves systemic collagen abnormality - Joint hypermobility & tissue laxity are linked to abnormal collagen ratios - Collagen types I, II, & III are decreased in the skin - Dx criteria include joint abnormality - Affects cardiac tissue, smooth muscle in female genital system, & GI system - Impairs joint position sense

Hypermobility syndromes (HMS) (cont)

• Clinical presentation:

Joint instability & trauma:
- Recurrent ankle sprains
- Meniscus tears
- Acute or recurrent dislocations or subluxations of various joints (shoulder, patella, MCP joints, TMJ)
- Traumatic arthritis
- Bruising
- Fractures (chronic or non-traumatic)
- Chondromalacia

Soft tissue disorders:
- Tendinitis
- Epicondylitis
- RC syndrome
- Synovitis
- Juvenile episodic synovitis
- Bursitis

MSK conditions:
- Scoiliosis
- OA
- Congenital hip dislocation
- Delayed motor development
- Flat feet & sequelae

Neurological Ssx:
- Nerve compression disorders - carpal tunnel, tarsal tunnel, TOS
- Raynaud s.
- Clumsiness
- Chronic HA

Px & sleep issues:
- Exercise-related / post-exercise-related px
- Nocturnal leg px
- Low nocturnal sleep quality
- Joint swelling
- Back px
- Unspecified arthralgia or effusion of affected joint

Other systemic effects:
- Fibromyalgia
- Chronic fatigue s.
- Functional GI disorders
- Immune system dysregulation
- Pelvic dysfunction
- CV dysautonomia
- Exocrine glands dysfunction
- Little changes of the skin
- Greater risk of failures in tendon, ligament, bone, skin, & cartilage
- Enhanced flexibility
- Ankylosing spondylitis (axial spondyloarthritis)

Hypermobility syndromes (HMS) (cont)

• **Physical examination:**	- ROM - End feel - Beighton score - Paradoxical breathing evaluation
• **Diagnosis:**	**Major criteria:** - Beighton score of ≧4/9 - Arthralgia for >3 months in >4 joints **Minor criteria:** - Beighton score of 1-3 - Arthralgia in 1-3 joints - Hx of joint dislocation - Soft tissue lesions >3 - Marfan-like habitus - Skin striae, hyperextensibilty or scarring - Eye signs, lid laxity - Hx of varicose veins, hernia, visceral prolapse **Requirement for Dx of HMS:** - 2 major criteria - 1 major criteria + 2 minor criteria - 4 minor criteria - 2 minor criteria & unequivocally affected 1st-degree relative in FHx
• **Complications:**	- Px & stiffness - Clicking - Dislocations - Recurrent injuries - Digestive problems - Dizziness & fainting - Fatigue
• **Management:**	- Education - Abdominal brace exercise - Active mobs exercises - Strengthening exercises - muscle surrounding the joint - Proprioceptive exercises - Control neutral joint position - Re-train dynamic control - Motion control - NSAIDs for px management

Hypermobility syndromes (HMS) (cont)

- **Ddx:**
  - Ehlers-Danlos syndrome
  - Fibromyalgia
  - Chronic fatigue syndrome
  - Depression

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