Achilles Tendinopathy

• GREEN	
• **Intro:**	- The achilles tendon (aka triceps surae) is the strongest & largest tendon in the body - Connects aponeuroses of the gastroc, soleus, & plantaris m. to the calcaneus bone - Crucial foe enabling calf muscles to exert force on the heel, necessary for walking & running - Various factors can contribute to achilles tendon injuries, affecting specific locations such as insertional (damage at insertion on the post calcaneus) & non-insertional (inv. "watershed area", 2-6cm proximal to the calcaneal insertion) tendonitis, paratenonitis, & tendon rupture -
• **Aetiology (risk factors):**	- Lifetime incidence of 24% in athletes - Common in runner - M>F (3.5:1) - **Intrinsic factors:** anatomic factors, age, sex, metabolic dysfunction, foot cavity, dysmetria, muscle weakness, imbalance, gastroc dysfunction, anatomical variation of the plantaris m., tendon vascularisation, torsion of the achilles tendons, slippage of the fascicle, & lateral instability of the ankle - **Extrinsic factors:** mechanical overload, constant effort, inadequate equipment, obesity, medications (corticosteroids, anabolic steroids, etc), improper footwear (arch support), insufficient warming or stretching, hard training surfaces, & direct trauma, etc - **Systemic risk factors:** diabetes, hypertension, inflammatory arthropathy, gout, & corticosteroids

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Achilles Tendinopathy (cont)

• **Pathophysiology:**	- Mechanical tension concentrated at medial/central paratenon & middle segment, common site of injury (sports-related) - Tendon twists counterclockwise on the R & clockwise on the L, rotating 90° during descent - Soleus fibres insert anteromedially, while larger gastroc fibres insert posterolaterally - Configuratio may influence biomechanics & contribute to achilles tendinopathies - Insertional achilles tendinopathy characterised by degeneration: loss of parallel collagen I fibres, fatty infiltration, & capillary proliferation - Degeneration leads to thickening of the tendon in advanced imaging - No evidence of acute or recent inflammatory process
• **Clinical presentation:**	- Pts may present w/ Ssx from acute strain or gradual onset repetitive irritation - Complaints inc. px or tenderness in the tendon or heel, intensifying w/ activity, esp. walking or running - Difficulty standing on toes or walking downstairs, morning px, & stiffness are common - Warmth & swelling increases throughout the day, related to activity - Symptoms can be tracked using the VISA-A Questionnaire - Shoe insole assessment: may reveal wear patterns indicating hallux limits (disproportionate wear under the 2-5th metatarsal heads & the pad of the great toe)

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Achilles Tendinopathy (cont)

• **Physical examination:**	**Localisation:** - Palpation helps localise the injury to the "water-shed area" or calcaneal insertion - Mid-tendon px suggests non-insertional tendonitis; posterior calcaneal px suggests insertional tendinitis - Chronic tendinopathy may show fusiform swelling & tendinous or bony enlargement - ROM reveals passive dorsiFX deficits w/ px on resisted plantarFX **Special tests:** - Silverskiold test: differentiates achilles vs gastroc tightness - Thompson test (calf squeeze test) excludes tendon rupture - Motion palp of subtler joint assesses mobility & identifies restrictions **Functional deficits throughout kinetic chain:** - Non-insertional tendinopathy in runners linked to foot hyperpronation (subtalar eversion) - Assessments inc.: loss of medial longitudinal arch, forefoot abduction, calcaneal eversion, & navicular drop - Check posterior tibialis strength (calcaneal eversion during heel raises), gastroc/soleus flexibility, knee flexor/hamstring strength, & hip abductor (glute med) strength - Glute medius is associated w/ ankle dysfunction **Hallux limitus & foot functional stability:** - Limitation in passive dorsiFX of the 1st MTP joint associated w/ achilles tendon px - Functional assessment inv. simulating a ground reaction force & checking for fluid dorsiFX & concurrent plantar FX of the 1st metatarsal head
• **Diagnosis:**	- Radiographs often unnecessary - Ottawa ankle rules for ankle or mid-foot px post-trauma - No defined rules for imaging non-traumatic heel px, consideration may given in cases of significant trauma w/ altered gait or to rule out other pathology - Radiographs of achilles tendinopathy: tendon calcification & spurs/enthesophytes on the posterior calcaneus - US or MRI to help identify & define tendon pathology

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Achilles Tendinopathy (cont)

• **Complications:**	**Major complications:** - tendon avulsion or rupture, any reoperation, DVT, reflex dystrophy, persistent neuralgia, deep infections, deep suture reactions, & major wound problems - **Minor complications:** discomfort, superficial infections, minor wound problems, scar sensitivity, hypertrophy, mild paresthesia, prolonged hospitalisation
• **Management:**	- Non-operative treatment is the 1° approach - Best proven care: rest, eccentric rehab, & correcting mechanical faults - Eccentric exercise programs, e.g. Alfredson's heel drops, are effective - Soft tissue therapy, stretching, & myofascial release are necessary for flexibility - Slowly progressive loading programs are favoured over complete rest - Return-to-play criteria inc. ankle dorsiFX, calf circumference, & heel raises - Referral suggested for pts failing conservative care, w/ limited proven alternatives - Supplements like Boswellia serrata & curcuminoids may improve Ssx
• **Ddx:**	- In children & adolescents, the epiphyseal growth plate is weaker, more prone to *Sever's disease* (calcaneal apophysitis) from stressors that would cause achilles tendinopathy in adults - Achilles tendon rupture - Retrocalcaneal bursitis - Plantaris tendinopathy - Dislocation of plantar flexor tendons - Posterior ankle impingement - Os trigonum syndrome - Fascial tears - Calcaneal fracture - Irritation/neuroma of sural n. - Fat pad irritation - Systemic inflammatory disease

link text; VISA-A Questionnaire; Ottawa Rules

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Achilles Tendon Rupture

• **YELLOW or RED**	Partial or complete rupture
• **Intro:**	- Most common tendon rupture of the LL - Common in 30-40 yo, esp. "weekend warriors" - Acute ruptures, often w/ sudden onset of px, accompanied w/ a "snapping" sound or audible "pop" at the injury site - Pts may describe the sensation as being kicked - Injury leads to significant px & disability - Often associated w/ soccer, racket games, or basketball - MisDx as ankle sprains in 20-25% - Risk factors: prior intratendinous degeneration (tendinosis), steroid use, & inflammatory arthritides
• **Aetiology (risk factors):**	- 40/100,000/year - M>F (75% of recreational sports) - Runners (7-18%) - Dancers (9%) - Gymnasts (5%) - Tennis players (2%) - American football players (<1%) - Causes inc. sudden forced plantarFX, direct trauma, & long-standing tendinopathy or intratendinous degenerative conditions - **Systemic factors:** chronic renal failure, collagen deficiency, diabetes, gout, infections, lupus, parathyroid disorders, RA, thyroid disorders - **Foot problems:** cavus foot, insufficient gastroc-soleus flexibility & strength, limited dorsiFX, tibia vara, varus alignment w/ functional hyperpronation

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Achilles Tendon Rupture (cont)

• **Pathophysiology:**	- Involves a combination of mechanical, structural, & biomechanical factors - **Mechanical factors:** AT descends from its origin, twists counterclockwise on R & clockwise on L, rotating 90°, leading to its strength but can rupture if suddenly exposed to excessive tensile loads - **Structural factors:** as people age, parallel collagen fibres become less organised & more prone to degeneration, additionally, certain conditions (e.g. diabetes or chronic kidney disease) can compromise the tendon's structural integrity & increase risk of rupture - **Biomechanical:** stiffness is associated w/ potential risk factors, while high foot arches decrease the risk of injury; when the tendon is exposed to chronic stress or repeated microtrauma, biomechanical factors combined w/ a compromised blood supply can lead to the degeneration of tendon fibres & potential rupture
• **Clinical presentation:**	- Acute, sharp px in the achilles region, typically following a sport - Often accompanied by audible sound - Hx of tendinopathy
• **Physical examination:**	- Inability to stand on toes - Weakness in ankle plantarFX - Tendon discontinuity or bruising around the posterior ankle may be palpable - +ve Thompson test (calf squeeze test)
• **Diagnosis:**	- Imaging tests used to confirm Dx & rule out other injuries - Plain radiographs for fractures - MRI or US for confirming achilles tendon rupture - MRI should be reserved for ambiguous presentations or chronic injuries due to cost & time concerns, & to avoid delaying surgical treatment
• **Complications:**	- Re-rupture - Wound healing complications - Surgical nerves injury

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Achilles Tendon Rupture (cont)

• **Management:**	- Excellent prognosis, but some pts may have residual deficits (e.g. reduced ROM) - Good results from both, surgical & conservative treatment - Higher re-rupture rate in non-surgical - Conservative treatment will be prolonged - RICE, px control, & functional bracing - Muscle strengthening & ROM
• **Ddx:**	- Achilles bursitis - Fractures - Impingement syndrome - OA - Sprain - Calf injuries - Calcaneofibular ligament injury - DVT - Talofibular ligament injury

link text

Ankle dislocations

• **YELLOW or RED**

•	- Common in A&E & come in two forms: *tru dislocations w/o fracture* & *fracture - dislocation* (more common)
Intro:	- Ankle joint complex: subtalar, talocalcaneonavicular, & talocrural joint - True ankle is the talocrural joint, functioning as a hinge joint for PLANTAR & DORSI - Subtalar joint is for IN & Eversion (frontal plane) - Talocaclaneonavicular joint & subtalar together for IN & Eversion - Joint stability maintained by 3 ligament groups: tibiofibular syndesmosis (limits motion between tibia & fibula), deltoid lig. (supports the medial ankle & resist Eversion), & lateral collateral ligament (resists INversion) - Most cases, ligaments are strong enough to cause bones to give way, causing fracture-dislocation

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Ankle dislocations (cont)

• **Aetiology (risk factors):**	- Pure ankle dislocations w/o fracture is rare (0.065% of all ankle injuries) - Talocrural dislocations accompany 21-36% of ankle fractures - Most cases occur in M (72%) due to sports (31%) or motor vehicle accidents (30%) - Common dislocation direction: postero-medial (46%) - Irreducible ankle fracture-dislocation may occur, e.g *"Bosworth Fracture"* where fibula locks behind the tibia
• **Pathophysiology:**	**Mechanism varies:** - Pure ligamentous dislocation can occur in multiple directions & mechanisms - Common mechanism involves maximal PLANTAR w/ axial load & forced IN of the foot - This mechanism damages anterior talofibular & calcaneofibular ligaments, leading to postero-medial dislocations - Superior dislocation happen when EVERTED foot is DORSI, leading to rupture of the tibiofibular syndesmosis - Predisposing factors: peroneal muscle weakness, ligamentous laxity, & previous strains - Common ankle fracture-dislocations occur via similar mechanics as non-dislocated ankle fractures - Sometimes dislocations spontaneously reduce, leaving a malleolus fracture
• **Clinical presentation:**	- Pts typically present w. dislocated foot relative to the tibia - Urgent need for appropriate analgesia & rapid realignment of foot & ankle to proper anatomical position - Delay can lead to skin breakdown & formation of fracture blisters, potentially resulting in permanent disability - Severe pain

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Ankle dislocations (cont)

• **Physical examination:**	- Note direction of foot relative to the ankle mortise - Assess presence/absence of dorsalis pedis & posterior tibial pulses - Check capillary refill of the distal foot - Evaluate for associated injuries of the foot - Identify localising areas of tenderness & swelling - Sensory exam should inc. dorm of the foot, lateral & medial aspects, & sensation proximal to 1st & 2nd MT (innervation od deep peroneal n.) - Assess ability to FX & EXT toes
• **Diagnosis:**	- Plain X-rays are crucial as 1st step (AP, lateral, Mortise) - CT after ortho surgeon recommendation
• **Complications:**	- Infection - Malunion/nonunion - Skin necrosis - Post-traumatic arthritis (PTOA) - Smokers have higher rates of post-surgical infections - Diabetics higher rate of complications e.g. malunion, wound healing issues, & deep infections
• **Prognosis:**	**Pure ankle dislocations:** - Generally favourable - Majority of pts become asymptomatic after proper treatmemt - Symptomatic cases, mainly F, report stiffness or PTOA - Closed dislocations lead to fewer Ssx compared to open dislocations - Prognostic factors for worse outcomes: advanced age, vascular injury, delay to reduction, & inferior tibiofibular ligament injury - Late complications: stiffness, degenerative changes, joint instability, & capsular calcification **Ankle fracture-dislocation:** - Prognosis varies - Worse outcomes compared to non-dislocated ankle fractures - Up to 63% of pts develop PTOA - Factors contributing to PTOA: type of #, pt's sex, & reduction accuracy - Study: 82% of pts had an "excellent" to "good" outcome after 2-6 yrs follow-up
• **Management:**	- PRICE & NSAIDs - Immediate referral

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Ankle dislocations (cont)

• Ddx:	- Subtalar dislocation may occur alone or w/ ankle dislocation / fracture-dislocation, potentially leading to misdiagnosis during physical exam - Plain films reveal reduced tibiotalar joint in isolated subtalar dislocations, aiding correct Dx - High-energy mechanisms may cause total talus extrusion, inv. both tibiotalar & subtalar dislocations

link text

Recurrent ankle sprain

•GREEN

•Intro:	- Common condition - About 40% can lead to chronic Ssx lasting at least 12 months post-injury - Roughly 20% progress to chronic instability - Both general public & athletes are susceptible - Impairment of proprioception may contribute to recurrence
•Aetiology (risk factors):	- Often caused by 1st-time ankle sprain - Most commonly due to INversion + ADduction, 1° affecting ATFL - **Associated factors:** diminished postural control, impaired proprioception, loss of muscle strength, ligamentous laxity (e.g. Ehlers-Danlos s., Marfan s., Turner's s.), decreased ankle joint ROM, cavus foot-type - 2.15 / 1000 in US - Peak incidence 10 & 19 yrs (younger pts have higher rates) - M 15-24yrs>F - F 30-99yrs>M - African American & Caucasians - Nearly 50% of sprains occur during sports (basketball 41.1%, football 9.3%, soccer 7.9%) - Military>civilians

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Recurrent ankle sprain (cont)

•Pathophysiology:	- Index ankle sprains result in microscopic tears & attenuation of ligaments - Attenuation can result in functional & mechanical instability - Most commonly injured: ATFL, CFL, PTFL - Lateral ankle instability: functional or mechanical - Proper Dx crucial for treatment - Functional instability: chronic, described subjectively by pts - No clinical or radiographic findings for functional instability - Proprioceptive deficits common - Mechanical instability: excessive motion in ankle joint - Clinically assessed w/ anterior drawer sign or radiographically
•Clinical presentation:	- Detailed Hx, inc. mechanism of injury - Consideration of previous ligamentous attenuation from index ankle sprain
•Physical presentation:	- Observe for dislocation or asymmetry - Palpate for tenderness, inc. medial ankle & fibula length - Assessment of edema & ecchymosis - ROM evaluation comparing contralateral side (normal: dorsi 10° w/ knee EXT, 20° knee FX) - Muscle strength tests: PLANTARfx, DORSIfx, INversion, & Eversion - DTR & sensation -**Special tests:** anterior drawer test (ATFL integrity), "dimple" sign (subtalar instability), Talar tilt test (CFL integrity & subtalar instability), Kleiger EXT rot test (deltoid ligament injury/ankle syndesmosis injury)

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Recurrent ankle sprain (cont)

•Diagnosis:	**Ankle sprain classification:** - Grade 1: mild stretching of lateral ligament complex w/ microscopic tearing, no joint instability, mild edema, no functional loss - Grade 2: ligament tear or partial rupture (usually ATFL), moderate-severe edema & ecchymosis, moderate functional loss, mild-moderate joint instability - Grade 3: complete disruption/rupture of ligament w/ moderate-seve ankle joint instability, immediate edema & ecchymosis, moderate-severe joint instability **Imaging:** - Plain films to rule out fractures (present in 15% of ankle sprains) - MRI for soft tissue assessment, reserved for ligamentous surgical planning - Ottawa ankle rules **Dx criteria for acute ligament injury:** - Healthy ligaments: thin, linear, low-signal intensity - Acute injury: intrasubstance edema seen as increased signal intensity - Chronic injury: thickening, elongation, irregular contouring w/o significant soft tissue changes
•Complications:	- Prone to reinjuring the same ankle - 20-50% of cases of recurrent injuries lead to chronic px & instability (CAI) - CAI stems from proprioceptive deficits & increased ligament laxity due to repeated sprains - Pts w/ CAI usually have a Hx of multiple ankle sprains & severe INversion injuries
•Management:	- Up to 85% of injuries is treated conservatively - PRICE & NSAIDs - Neuromuscular training therapy (proprioception tasks & balance exercises) crucial for reducing recurrence rates - Immobilisation recommended for up to 10 days - After 10 days progress to bracing & taping - SMT/STW - IASTM/TFM - Mobs - Exercises phase 1: single leg stance, ankle alphabet, standing gastroc stretch, standing soleus stretch - Exericses phase 2: resisted ankle dorsifx w/ band, resisted ankle EVersion w/ band, wobble board

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Recurrent ankle sprain (cont)

•Ddx:	- Ankle fracture - Posterior tibial tendonitis - Neuromuscular disorder - Superficial peroneal nerve neuralgia - Peroneal tendon tears - Anterior process of the calcaneus fracture - Base of 5th MT fracture

link text; link text

Calcaneofibular ligament (CFL) sprain

Grade 1 to 3:	- Mild stretching of the ligament complex w/o joint instability - Partial rupture of the ligament complex w/o joint instability - Complete rupture of the ligament complex w/ instability of the joint
•Intro:	- Common A&E visit (7-10%) - 40% of sports injuries (usually affect the lateral ankle compartment) - Lateral ankle inc: ATFL (2/3rds of lateral ankle injuries), CFL, PTFL - Hard to differentiate between ATFL-superimposed CFL injuries & isolated CFL injuries
•Aetiology (risk factors):	- Large % of lateral ankle injuries are sports related, esp. indoor & court sports - Isolated CFL injuries are rare (usually classified under lateral lig. injury) - 30,000 ankle sprains occur / day - 25-40% of sports injuries - Lateral ligament compartment is inv. in 85% of ankle injuries (10,000 / day)
•Pathophysiology:	- Origin & insertion: anterior lateral malleolus - posterior lateral tubercle of the calcaneus - Crossed over by fibulas brevis & longus tendons - Resists INversion during PLANTARfx & DORSIfx, stabilises the subtalar joint during PLANTARfx - Mechanism of injury: results from combined INversion & supination, but can also occur from INversion in extreme DORSIfx
•Clinical presentation:	- Pt may report cracking sound - Swelling, redness & px - Inability to continue activities

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Calcaneofibular ligament (CFL) sprain (cont)

•**Physical examination:**	- **Special tests:** anterior drawer test +ve; Talar tilt test +ve **Subsequent physical findings:** - Ecchymosis w/ localised px on palpation 4-5 days post-trauma, indicates 90% chance of *lateral ligament rupture* - TTP over the CFL suggests 72% risk of *ligament injury*
•**Diagnosis:**	**Ottawa ankle rule:** - Palpation of 4 px locations - Ability to bear weight **Imaging:** - US offers dynamic imgaging - MRI useful for cases w/ high suspicion of ligament injury **Lateral ankle injury classification:** - Grade 1: ligament stretchuing - Grade 2: moderate sprain - Grade 3: severe sprain w/ full ligament lesions
•**Complications:**	- Re-injury of the lateral compartment is a common occurrence in low-grade ankle sprains - Potential feeling of instability & px which inhibits functional mobility - Chronic joint instability can progress to post-traumatic ankle joint OA
•**Management:**	- Education: 74% of pts experience chronic Ssx 4 yrs after injury, potential instability or px, 32% pts report Ssx of original injury 7 yrs after - RICE (4-5 days) & NSAIDs - Immobilising w/ cast or boots ONLY in 1st week - 3 phases of healing: inflammatory (1-10 days), proliferative (4-8 weeks), & remodelling (up to one year) - Bracing or taping aids in return to activity after the initial immobilisation phase - Conservative & surgical approach has similar outcomes
•**Ddx:**	- ATFL - Osteochondral injury - Fibularis tendon injury - Ankle frcatures - Achilles rupture - Tendon dislocation - Subtalar joint injury

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Gout

• YELLOW	
• **Intro:**	- Also known as "disease of kings & king of diseases" - One of the most common causes of chronic inflammatory arthritis - Characterised by the deposition of monosodium urate (MSU) monohydrate crystals in tissues - Well-understood & manageable among rheumatic diseases
• **Aetiology (risk factors):**	- Older age & males (20:1) - >40 yrs - Purine diet & alcohol - **Comorbidites:** hypertension, diabetes, hyperlipidemia, & metabolic syndrome
• **Pathophysiology:**	- Genetic, metabolic factors can influence hyperuricemia (key factor) - Monosodium urate crystal deposition in periarticular soft tissue - Inflammatory response: macrophages phagocytise monosodium urate crystals → vasodilation → inflammation

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Gout (cont)

• **Clinical & physical findings:**	- Four distinct stages: asymptomatic hyperuricemia, acute gout attacks, inter-critical period, & chronic tophaceous gout **Asymptomatic hyperuricemia** - Many pts w/ this stage don't develop gout - Risk of gout increases w/ serum urate levels - This stage ends w/ the first gout attack **Acute gout attack** - Sudden, severe px & swelling - Common in LL, especially 1st MCP - Can also affect other joints, tendons, & bursa - Px is severe & may not respond to home remedies - Subsequent attacks can be prolonged - Certain factors like trauma, alcohol, diet, & medications can trigger attacks - Physical exam shows red, swollen, warm, & tender joints - Tophi, urate deposits, can occur in chronic cases **Intercritical gout** - Follows resolution of acute attack - Hyperuricemia persists, & subclinical inflammation may be present **Chronic tophaceous gout** - Tophi, granulomas around MSU crystal deposits develop - Appears as chalk-like nodules under the skin - Develops years after initial attack - Can lead to destructive arthritis & deformities - Top can appear in various sites, including digits, knees, & olecranon bursa - Deposits also reported in cornea & heart valves
• **Diagnosis:**	- Synovial fluid analysis - Labs inc. WBC, ESR CRP, still don't confirm gout - Imaging (DECT) not commonly used
• **Complications:**	- Tophi - Joint defomrity - OA - Bone loss - Urate nephropathy - Nephrolithiasis - May also cause ocular complications, eg. conjunctivitis or uveitis

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Gout (cont)

• **Management:**	- Prognosis depends on pts comorbidities - Mortality is higher in pts w/ CV disease - Medication - Rest & ice - Lifestyle modifications
• **Ddx:**	**Gout flare** - CPPD - Septic arthritis - OA - Psoriatic arthritis - Cellulitis - Trauma **Tophaceous gout** - Dactylitis - RA - Osteomyelitis

link text

Hallux rigidus & limitus

• **GREEN**

• **Intro:**	- Also known as *"turf toe"* - Sprain of the plantar capsule-ligament of the great toe MTP joint - Typically results from forceful hyperEXT of the 1st MTP, commonly experienced in sports - Injury to the plantar plate of the great toe causes px during push-off & decreases agility - Turf toe can severely impact elite athletes & cause inconvenience in the general pop
• **Aetiology (risk factors):**	- Often caused by forceful hyperEXT of the 1st MTP joint - Common in sports like basketball, soccer, & gymnastics, but particularly in football - Higher prevalence on artificial turf fields, especially older astroturf surfaces - Modern high-pile turf mimics natural grass better, reducing the risk of turf toe - Injury occurs due to the rigidity of the playing surface, placing strain on the feet

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Hallux rigidus & limitus (cont)

• **Pathophysiology:**	- The 1st MTP functions as both a hinge & sliding joint - Has shallow articulation between the convex MT head & the concave base of the proximal phalanx, resulting in little bony stability - Stability 1° relies on the complex attachments of the capsule, ligaments, & musculotendinous structures surrounding the joint - Strongest stabiliser of the 1st MTP is the plantar plate, which is a thickening of the joint capsule - Plantar plate attaches to the transverse head of the adductor hallucis, the flexor tendon sheath, & the deep, transverse intermetatarsal lig. Injuries to the plantar plate classification: - Grade 1: sprain of the plantar plate - Grade 2: partial tear of the plantar plate - Grade 3: complete tear of the plantar plate
• **Clinical presentation:**	- Pt will complain of px & swelling of 1st MTP - May also complain of antalgic gait & px, especially w/ foot flat to toe-off during gait cycle - May or may not describe an inciting event of acute forceful hyperEXT of the 1st MTP - Some reports of subacute to the chronic development of turf toe
• **Physical examination:**	**Inspection:** - Swelling & ecchymosis at the 1st MTP - Note antalgic gait, difficulty in toe raises, & joint deformities **Palpation:** - TPP over plantar aspect of 1st MTP - Tenderness over medial, lateral, or dorsal joint - Compare sesamoid bone position to assess proximal migration **ROM:** Passive & active ROM - Px w/ passive EXT & active FX of 1st MTP **Muscle strength:** - FX toes or EXT toe against resistance - Perform ABD **Special tests:** - Valgus & varus stress test: assesses medial & lateral stability - Vertical Lachman test: measure vertical translation of proximal phalanx compared to MT; compare B

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Hallux rigidus & limitus (cont)

• **Diagnosis:**	- Initial X-rays to assess for fracture or dislocation - MRI w/o contrast to assess for plantar plate or surrounding soft tissue injury **Anderson classification:** - Grade 1: acute sprain w/o bony pathology or joint instability, pt will have normal ROM & should be able to WB - Grade 2: partial tear of the plantar plate or joint capsule, the pt will have painful ROM, ecchymosis, swelling, & px w/ WB - Grade 3: complete tear w/ loss of continuity of the plantar plate or capsule, may not sesamoid bone migration, marked TTP, decreased ROM, swelling, ecchymosis, & difficulty WB
• **Complications:**	- Loss of push-off strength - Hallux rigidus - Cock-up deformity - Traumatic bunion deformity - Loose bodies in the joint space - Joint fibrosis - Acute complications: infections, scar formation 2° to hypertrophy, & plantar n. neuroma development

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Hallux rigidus & limitus (cont)

- **Management:**
  - Prognosis varies based on grade of the injury
  - Initial treatment: RICE, stiff sole/rocker bottom sole to limit motion
  - For severe injuries: CAM boot/walking cast to minimise motion & aid healing

  Progressive motion:
  - Start once injury stabilises
  - Grade 1: return to play in 1-2 weeks
  - Grade 2: recovery in 4-6 weeks; may require taping to resist hyperEXT of MTP joint
  - Corticosteroid/anestehtic injections not advised for grade 2 injuries
  - Grade 3: conservative treatment w/ immobilisation (4-6 weeks), then gentle ROM
  - Expected healing time for grade 3: 6-12 months

  Surgical repair:
  - If conservative management fails
  - Indications: large capsular avulsion, unstable joint, sesamoid issues, instability, hallux valgus deformity, chondral injury, intra-articular loose body, sesamoid fracture, failed conservative treatment{{nl}**Post-op management:**
  - Gentle passive motion at 7-10 days, then be non-WB in removable splint or boot w/ hallux protected for 4 weeks
  - At 4 weeks, increase active motion & allow ambulation in the boot
  - Pt wear modified shoe at 2 months & return to contact activity w/ protection from excessive DORSI at 3-4 months
  - Expect 6-12 months for a full recovery
- **Ddx:**
  - Hallux rigidus / limitus / valgus
  - Reverse turf toe
  - Soccer toe

link text

Hallux Valgus (HV)

- **GREEN**
- **Intro:**
  - Also known as bunion
  - Common forefoot deformity
  - Exact cause enot understood

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Hallux Valgus (HV) (cont)

• **Aetiology (risk factors):**	- F>M - Est. 23% of adults 18-65 - Est. 36% of adults >65 - Multi-factorial - Associated w/ connective tissue disorders, e.g. Marfan syndrome, Ehlers-Danlos syndrome, & Downs syndrome - Muscle imbalance due to conditions like stroke, cerebral palsy, or myelomeningocele - Slight increased risk in tight shoes & heels
• **Pathophysiology:**	- Interplay of various factors - Imbalance between extrinsic & intrinsic muscles, along w/ ligament involvement - 1st metatarsal alignment maintained by tension from *peroneus longus* laterally & *abductor hallucis* medially - Collateral ligaments prevent transverse plane movement at the 1st MTP joint - Increased pressure at 1st MT head lead to medial-dorsal movement, increasing hallux angle - Muscle stabilisation during walking worsens this condition - Forces pushing 1st MT medially & hallux laterally strain & eventually rupture medial collateral ligament & medial capsule - W/o medial stabilisation structures, lateral structures exacerbate HV deformity, inc. adductor hallucis muscle & lateral joint capsule ligaments

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Hallux Valgus (HV) (cont)	
• **Clinical presentation:**	- Commonly presents w/ a chronic progressive onset - Proximal phalanx pronation & lateral deviation, & medial deviation of the 1st MT head, often causing redness & px - Often sharp or deep px at the MTP joint exacerbated by walking - Aching px at the head of the 2nd MT may also be reported - Px, frequency, duration, & severity increases as the deformity progresses - Tingling or burning px at the dorsal part of the deformity may indicate medial dorsal cutaneous n. neuritis due to compression - Ssx are 1° due to pressure on the 1st MT, toes, & other MT bones - Additional Ssx inc. blisters, ulcerations, interdigit keratosis, & irritated skin, which can limit physical activities
• **Physical examination:**	- HV deformity severity more obvious in weight-bearing stance **Biomechanical exam** - Forefoot / rearfoot varus of valgus - 1st ray hypermobility - Subtalar joint stiffness - Midtarsal joint stiffness - Resting calcaneal stance position - Tibial torsion - Neutral calcaneal stance position **Non-weight bearing:** - Assess hallux position relative to the 2nd digit (under-riding, overriding, or w/o contact) - Evaluate lateral deviation of the MTP joint & medial prominence - Assess 1st MTP joint ROM & quality **Weight bearing:** - Evaluate for increased hallux abduction, medial prominence, 1st MTP joint dorsiflexion, hallux purchase, & metatarsus varus

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Hallux Valgus (HV) (cont)

• **Diagnosis:**	- Labs if there's suspected metabolic or systemic disease (rheumatoid factor, CRP, ESR, uric acid, CBC) - MRI & radionuclide imaging for suspected osteomyelitis - Plain radiographs help determine the extent of damage to the 1st MTP joint **Lateral callus deviation, w/ normal angles:** - Hallux valgus angle (HVA) less than 15° - Intermetatarsal angle (IMA) less than 9° **Classification of hallux valgus severity:** - Mild: HVA 15-30° / IMA 13-20° - Moderate: HVA 30-40° / IMA 13-20° - Severe: HVA over 40° / IMA over 20° **Imaging views:** - WB A-P - Lateral oblique - Lateral - Sesamoid axial
• **Complications:**	- Bursitis (most common) - Second toe hammertoe deformity - Degenerative disease of the metatarsal head - Central metatarsalgia - Medial dorsal cutaneous n. entrapment - MTP joint synovitis
• **Management:**	- Good prognosis - First a trial of conservative treatments (Ssx management): shoe modification, orthoses, analgesics, icing, bunion pads, & stretching - Surgery if px & functionality isn't improved - Post-op care varies based on the procedure, commonly involves limited WB, ROM exercises, & long-term monitoring
• **Ddx:**	- OA - Freiberg disease - Hallux rigidus - Morton neuroma - Turf toe - Gout - Septic joint

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Meidal Tibial Stress Syndrome (MTSS)

• GREEN	
• **Intro:**	- Also known as *shin splints*, *Jogger's foot*, & *Medial plantar nerve s.* - Common overuse lower extremity injury in athletes & military - Exercise-induced px over the anterior tibia - Early stress injury in the continuum of tibial stress #
• **Aetiology (risk factors):**	- 13.6-20% in runners - Up to 35% in military - Overdue condition - Tibial bony overload injury w/ associated periostitis **Causes & predisposing factors:** - Significant increasing loads, volume, & high impact exercises - F gender - Previous Hx of MTSS - High BMI - Navicular drop - Ankle PLANTAR ROM - Hip EXT rot ROM - Vitamin D deficiency
• **Pathophysiology:**	- Results from accumulation of unprepared micro damage in the cortical bone of the distal tibia - Periostitis, inflammation of the periosteum, is typically present at the site of bony injury - Affected area correlates w/ tendinous attachments of the soleus, flexor digitorum longus, & posterior tibialis muscles - Sharpey's fibres, connective tissue fibres linking periosteum to bone, play a role - Repetitive muscle traction is believed to contribute to periostitis & cortical microtrauma - Uncertain whether periostitis or cortical microtrauma occurs 1st in the development
• **Clinical presentation:**	- Presence of exercise-induced px along the distal 2/3s of the medial tibial border - Presence of px provoked during or after physical activity, which reduces w/ relative rest - The absence of cramping, burning px over the posterior compartment &/or numbness/tingling int he foot

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Meidal Tibial Stress Syndrome (MTSS) (cont)

• **Physical examination:**	- Presence of recognisable px reproduced w/ palpation of the postero-medial tibial border >5cm - Absence of other findings not typical of MTSS (e.g severe swelling, erythema, loss of distal pulses, etc)
• **Diagnosis:**	- Dx though clinical & physical findings - Imaging is often used when uncertain of the cause or to rule out other lower extremity injuries - Plain radiographs normal for MTSS & early stress fractures, but a "dreaded black line" indicates # - MRI is the preferred imaging for MTSS & higher grade bone stress injuries (e.g tibial stress #) - Evaluation for vitamin D deficiency may be necessary, especially for persistent cases
• **Complications:**	- Px leading to decreased performance &/or time away from training/participation - Presumption: MTSS may progress to a tibial stress # - Cortical microtrauma may evolve into cortical # - Not every pt experiencing MTSS develops a tibial stress # - Several tibial stress # may necessitate surgical intervention
• **Management:**	- Full recovery expected w/ adequate rest & activity modification - SMT & STW - Foot arch support/orthotic Exercises phase 1: Ant. tib. stretch - sitting, Post. tib. stretch - standing, Dynamic gastroc stretch, standing soleus stretch - Exercises phase 2: Semi-stiff dead lift, Resisted post. tib. strengthening - Optimising calcium & vitamin D - Gait training
• **Ddx:**	- Tibial stress # - Chronik exertional compartment s/ (CECS) - Vascular ethologies (e.g. functional popliteal artery entrapment s., peripheral arterial disease, etc) - FPAES & PAD both manifest as claudications

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Metatarsalgia

• GREEN	
• **Intro:**	- Px & inflammation of the ball of the foot - Commonly occurs in runners & jumpers - Other causes inc. foot deformities & ill-fitting footwear - Rest & ice can alleviate Ssx - Proper footwear w/ shock-absorption insoles or arch support
• **Aetiology (risk factors):**	- Participating in high-impact sports (running & jumping - Ill-fitting shoes (especially heels) - Obesity - Other foot problems (hammertoe & calluses on the bottom of the foot) - Inflammatory arthritis (RA & gout)
• **Pathophysiology:**	- Intense training or activity - Certain foot shapes: high arch, 2nd toe that's longer than the big toe - Foot deformities: hammertoe & bunions - Excess weight - Poorly fitting shoes - Stress fractures: can change WB distribution - Morton's neuroma: noncancerous growth usually occurs between 3-4th MT head, causes Ssx similar to metatarsalgia & can also contribute to metatarsal stress
• **Clinical & physical findings:**	- Sharp, aching or burning px of the ball of the foot - Px that worsens when standing, running, flexing the foot or walking - Improves w/ rest - Sharp or shooting px, numbness, or tingling in toes - Feeling of having a pebble in the shoe - Tender on palpation - Mulder sign (squeeze test) helps Dx conditions like Morton's neuroma, which can present similar to to metatarsalgia
• **Diagnosis:**	- X-ray to rule out stress fractures & other conditions

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Metatarsalgia (cont)

• **Management:**	- Rest & ice - NSAIDs - Wear proper shoes - Use metatarsal pads - Consider arch supports - STW (gastroc & soleus) - SMT - Exercises: toe curls & spreads, active arch, single limb heel raise
• **Ddx:**	- Morton's neuroma - Stress fracture - Capsulitis - Freiberg's disease - Sesamoiditis - Arthritis - Bursitis - Tendonitis

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Morton's Neuroma

• **GREEN**

• **Intro:**	- Compressive neuropathy of forefoot interdigital n. - Compression, irritation at plantar aspect of transverse inter-MT lig. - Not a true neuroma: degenerative, not neolpastic - Also known as *Morton metatarsalgia / entrapment*, *Interdigital neuritis / neuralgia / neuroma / n. compression s.*, & *InterMT neuroma* - Most common location: between 3 & 4th MT head (termed Morton neuroma)
• **Aetiology (risk factors):**	- Common in middle - aged F (F>M, 5:1) - Rarely B, & rare to have 2 neuromas on the same foot **Common causes:** - Narrow toe-box footwear - HyperEXT of toes in high-heeled shoes - Deviation of toes - Inflammation of interMT bursa - Thickening of transverse MT lig - Forefoot trauma - High-impact sporting activities - MTP joint pathology - Lipoma

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Morton's Neuroma (cont)

• **Pathophysiology:**	- Compression & repetitive trauma to the n. results in vascular changes, edoneurial edema, & excessive burial thickening leading to perineural fibrosis **4 main hypotheses:** - Chronic trauma theory: walking causes chronic micro-traumas to interMT plantar digital n., compressed between MT heads & MTP joints - Entrapment theory: interdigital neuromas occur due to compression of interdigital n. against deep transverse MT lig & plantar soft tissue structures - InterMT bursa theory: bursitis in interMT region causes compression, inflammation, & subsequent fibrosis of affected common plantar digital n. - Ischemic theory: based on histopathological findings of common plantar digital artery exhibiting degenerative changes before n. thickening
• **Clinical & physical findings:**	- Plantar px located between MT heads - Aggravation of px by walking & wearing tight-fitting, high-heeled shoes - Relief when resting & removing shoes - Described as burning, stabbing, or tingling, sometimes w/ electric sensation - Sensation akin to walking on a stone or marble - Numbness between the toes is present in <50% of pts - Prolonged walking may lead to px radiating to the hind foot or leg, possibly causing cramps - Palpation may reproduce px - Compression of the forefoot mediolaterally can cause "Mulder's click"
• **Diagnosis:**	- Dx based mainly on clinical & physical findings - Plain WB radiograph to tule out various conditions - US aids in Dx - MRI, especially to rule out other Dx
• **Complications:**	- Chronic px (CRPS) - Recurrence of the deformity due to inadequate excision or converting a Morton neuroma into a true neuroma - Surgical complications (infection, px, bleeding) - Corticosteroid injection complications (skin/fat pad atrophy, skin discolouration)

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Morton's Neuroma (cont)

• **Management:**	- Good prognosis if proper protocol followed - NSAIDs - MT pads - SMT & STW - Nerve release - Exercises: Resisted flexor hallucis longus, Standing gastroc stretch, Plantar fascia - towel & golf ball
• **Ddx:**	- MT stress fracture - Hammertoe - RA or OA - Malignancy - Ganglion cyst

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Plantar Fasciitis

• **GREEN**

• **Intro:**	- Results from degenerative irritation at the origin of the plantar fascia - Overuse stress is a 1° cause, leading to sharp localised px at the heel - Heel spurs may occasionally accompany plantar fasciitis - Treatment is challenging, w/ pt dissatisfaction common despite various approaches - Non-surgical management is typical but often results in recurring px

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Plantar Fasciitis (cont)

• **Aetiology (risk factors):**	- Lead cause of heel px - 10% of general population - 40-60 yrs - 10% of runner-related injuries & 11-15% of foot Ssx needing medical care - Often B in 1/3 of cases - F>M (especially w/ higher BMI) - 1° an overuse injury causing micro-tears in the plantar fascia, but trauma or other causes can contribute - Predisposing factors include pes planus, pes cavus, limited ankle dorsiflexion, prolonged standing or jumping, & excessive pronation or supination - Tightness in posterior leg muscles can alter ambulation biomechanics - Risk factors include obesity, aging, occupations w/ prolonged standing, & certain medical conditions - Linked to some spondyloarthropthies
• **Pathophysiology:**	- Multifactorial cause - Believed to start w/ microtears due to repetitive trauma → stretching of plantar fascia → chronic degeneration of fascia **Histological findings:** - Granulation tissue - Micro-tears - Collagen disarray - Lack of traditional inflammation

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Plantar Fasciitis (cont)

• **Clinical & physical findings:**	- Pts typically report progressive px at the inferior & medial heel - Px can radiate proximally in severe cases - Sharp, worse in the morning, exacerbated by prolonged standing or sitting - Px decreases w/ ambulation but increases throughout the day - Px reproducible by palpating the plantar medial calcaneal tubercle or passive dorsiflexion of foot / toes - +ve windlass or Jack test: px elicited w/ passive dorsiflexion of 1st MTP joint - 2° findings: tight Achilles heel cord, pes planus, pes cavus - Assessment of gait to evaluate biomechanical factors or predisposing factors - Consider Ddx including fat pad contusion or atrophy, stress fractures, & nerve entrapments (e.g tarsal tunnel s.)
• **Diagnosis:**	- Usually Dx clinically - X-rays or US used if other injuries suspected or pt doesn't improve conservatively - MRI considered to check for tears, fractures, or defects **X-ray findings:** - Calcifications - Heel spurs - Thickening - Swelling **MRI findings:** - Thickening - Increased signal on specific images
• **Complications:**	- Rupture of the tendon, 1° if corticosteroid injections are employed - Fat pad necrosis - Flattening of the arch

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Plantar Fasciitis (cont)

• **Management:**	- 75% resolve spontaneously within. 12 months - 5% need surgery (not consistently +ve) - SMT - STW & IASTM/TFM - Foot arch taping - Support brace (Strassberg sock) - **Exercises Phase 1:** Hamstring doorway stretch, golf ball, standing gastroc stretch on step, flexor digitorum brevis strengthening, plantar fascia stretch - **Exercises Phase 2:** Resisted post tib strengthening, Vele's, Eccentric achilles strengthening
• **Ddx:**	- Calcaneus injury - Infection - Sickle cell bony px - Bone contusion - Neuropathic px - Tendinitis - Osteoporosis - Malignancy

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Sesamoiditis

• **GREEN**	
• **Intro:**	- Inflammation of the sesamoid bones in the ball of the foot & the tendons they are embedded in
• **Aetiology (risk factors):**	- Athletes get it from over-practicing movements that transfer weight to the ball of the foot **Population at risk:** - Dancers - Runners - Athletes - High-heel shoes - High arches - Flat feet - Overpronated feet - People w/ gout
• **Pathophysiology:**	- Sesamoid bones are only connected to tendons - These bones endure stress from movement & interact w/ tendons during motion - Bear additional stress from shock absorption during walking - Activities that frequently transfer weight to the ball of the foot can overstress these tendons & bones, causing inflammation & px

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Sesamoiditis (cont)

• **Clinical & physical findings:**	- Px under the big toe - Struggle to flex big toe - Struggle to WB or walk - Swelling - Redness - Bruising - TTP - Passive axial compression test +ve
• **Diagnosis:**	- X-ray, CT, US, or MRI to rule out conditions
• **Ddx:**	- Stress fracture - Turf toe - Hammertoe - OA - Gout - Hallux rigidus (especially if previous big toe injury)

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Sinus Tarsi Syndrome (STS)

• **GREEN**	
• **Intro:**	- Persistent anterolateral ankle px 2° to traumatic injuries to the ankle - Recent theories: 1° an instability of the subtalar joint due to ligamentous injuries that results in a synovitis & infiltration of fibrotic tissue into the sinus tarsi space
• **Aetiology (risk factors):**	- Associated w/ ankle sprains, potentially leading to talocrural joint instability - Est. 10-25% of pts w/ chronic talocrural joint instability also have subtler joint instability

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Sinus Tarsi Syndrome (STS) (cont)

• **Pathophysiology:**	- Arises from a single traumatic event or multiple ankle sprains causing significant damage to ligaments - Injuries 1° affect the talocrural interosseous & Cx ligaments, leading to subtler joint instability & excessive supination / pronation movements - Excessive movement in the subtalar joint applies increased forces on the synovium & sinus tarsi tissues - Resultant forces induce subtalar joint synovitis, chronic inflammation, & fibrotic tissue infiltration in the sinus tarsi, causing anterolateral ankle px characteristic of STS - Traumatic injuries may also harm ligaments in the tibiotalar & talocalcaneal joints, increasing rearfoot & mid foot mobility & instability - Athletes w/ heightened mobility in the talocrural & subtalar joint are at higher risk of instability following an ankle injury
• **Clinical & physical findings:**	- Deep sinus tarsi px - Swelling, bruising, TTP - Feeling of instability - Px at end of PF + SN in sinus tarsi - Ankle instability + px over sinus tarsi indicates STS - **Tests:** foot hyperpronation cluster, anterior drawer, ROMs, subtler instability test, standing ankle torsion test
• **Diagnosis:**	- Used to assess soft & bony tissue
• **Management:**	- NSAIDs - Ice - Support brace - **Exercises Phase 1:** Lower extremity Y-balance, Single leg stance, Active arch, Wobble board - **Exercises Phase 2:** Semi-stiff dead lift, Resisted ankle inversion/eversion w/ band, Eccentric post tib
• **Ddx:**	- Instability of talocrural & subtalar joints - Cuboid subluxation - Fracture

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