

Anterior Cruciate Ligament (ACL) Injuries

• YELLOW

- **Intro:**
 - Stabilises the knee joint along w/ the PCL (forms a cross "X")
 - Prevent excessive forward or backward motion of the tibia relative to the femur during FX & EXT
 - **Nerve supply:** middle geniculate artery; **Innervation:** posterior articular n. (branch of tibial n.)
 - **Origin:** anteromedial aspect of tibial plateau; **Insertion:** Medial aspect of the lateral femoral condyle
 - Has 2 bundles (anteromedial & posterolateral)
 -
- **Aetiology (risk factors):**
 - Most commonly injured ligament in the knee (almost 1/2 of all knee injuries)
 - F>M (esp. F athletes 4.5:1)
 - Possible factors contributing to increased F risk include: weaker hamstrings, preferential recruitment of quads during deceleration, & weaker core stability
 - Biomechanics & landing factors:**
 - F landing mechanics mat ↑ injury risk, w/ ↑ valgus angulation & knee EXT
 - ↓ hip & knee FX & ↓ fatigue resistance also contribute to ↑ stress on the ACL
 - Other risk factors:**
 - Anatomical: high BMI, smaller femoral notch, impingement on the notch, smaller ACL, hypermobility, joint laxity, & previous ACL injury
 - Hormonal & genetic factors:**
 - Preovulatory phase, may affect coordination & predispose females to ACL injury
 - Females on OCP were noted less affected
 - Associated injuries w/ ACL ruptures:**
 - Both intra & extra-articular injuries can accompany acute ACL ruptures
 - Meniscal tears are common, w/ lateral meniscus injury more prevalent in acute cases, & medial meniscus more involved in chronic cases
 - Other ligaments (PCL, LCL, & PLC) could also be injured in conjunction w/ ACL
 - Chronic ACL deficiency effects:**
 - Detrimental effects on the knee
 - Development of chondral injuries & complex, unrepairable meniscal tears is observed (e.g bucket handle medial meniscus tears)

Anterior Cruciate Ligament (ACL) Injuries (cont)

- **Pathophysiology:**
 - Common in non-contact sports, esp. non-contact pivoting injuries
 - Tibia translation anteriorly during slight knee FX & valgus
 - Direct hits to the lateral knee can also cause ACL injuries
 - Injury occurs during activity/sports participation that involves sudden changes in the direction of movement, abrupt stopping or slowing down while running, or jumping & abnormal landing
- **Clinical presentation:**
 - Hx of injury mechanisms
 - Pt would complain of hearing/feeling a sudden "**pop**" w/ associated deep knee px
 - About 70% would experience immediate swelling due to haemarthrosis
 - Other Ssx: knee "giving way", difficulty ambulating, reduced knee ROM
- **Physical examination:**
 - Pt demonstrates quadriceps avoidance gait (no active knee EXT)
 - Varus knee malalignment should be noted as it increases risk of ACL re-rupture
 - Palpation: swollen knee, & potential joint line tenderness w/ an associated meniscal injury
 - Move: knee may be **locked** due to associated meniscal injury (other meniscal & ligamentous structures to be assessed)
 - Lachman test, Anterior drawer test, Pivot shift test
- **Diagnosis:**
 - **MRI** is the 1° modality to diagnose ACL pathology
 - Knee arthroscopy to differentiate complete from partial tears & chronic tears (gold standard test)
 - Radiography to rule out fractures & other osseous injuries



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Anterior Cruciate Ligament (ACL) Injuries (cont)

- **Complications:**
 - Surgical: tunnel malpositioning, posterior wall blowout, graft failure due to various other issues
 - Post op: infection & septic arthritis
 - Stiffness & arthrofibrosis
 - Infrapatellar contracture syndrome
 - Patella tendon rupture
 - CRPS
 - Patella fracture
 - Tunnel osteolysis
 - OA in the long term
 - Saphenous n. irritation
 - Cyclops lesion
- **Management:**
 - Non-operative management:**
 - **Indication:** when there's reduced ACL laxity on low-demand pts or athletes involved in no cutting or pivoting activities or partial ACL tears
 - RICE
 - Non-WB (crutches or wheelchair)
 - NSAIDs
 - Phase 1: acute symptomatic treatment
 - Phase 2: 12 weeks of supervised physiotherapy starting w/ regaining full ROM & progression to quad, hamstring, hip ABD & core strengthening
 - Operative management:**
 - **Indication:** complete ACL rupture in younger or older active, high-demand pts, & partial ACL rupture w. functional instability
 - Two options: ACL reconstruction or repair
- **Ddx:**
 - ACL tear
 - Epiphyseal fracture of femur/tibia
 - MCL injury
 - Meniscal tear
 - Osteochondral fracture
 - Patellar dislocation
 - Posterior cruciate ligament injury
 - Tibial spine fracture

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Lateral Collateral (LCL) & PLC Injuries

• **YELLOW**



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Lateral Collateral (LCL) & PLC Injuries (cont)

- **Intro:**
 - 1° resistor of varus stress
 - Provides posterolateral stability (preventing medial translation of the tibia)
 - LCL & PLT resist external tibial rotation in 0-30° of knee FLX
 - Minor role in tibial translation (stabilises anterior & posterior tibial translation when cruciate ligaments are torn)
 - **Origin:** lateral epicondyle of the femur, **Insertion:** fibular head
 - **Blood supply:** branches of popliteal artery, **Innervation:** common fibular n.
 - **Surrounding structures:** popliteus tendon (PLT) & iliotibial band (ITB)
- **Aetiology (risk factors):**
 - 40% of PLC (posterolateral corner) & LCL injuries result from contact sports
 - Other causes include trauma, motor vehicle accidents, & falls
 - F>M
 - High-contact sports
 - Sports involving high-velocity pivoting & jumping
 - Tennis & gymnastics are most specific for isolated LCL injuries
 - Prior knee, ankle, or hip injury increases the risk
- **Pathophysiology:**
 - LCL injuries rarely occur in isolation
 - High-energy blow to the antero-medial knee
 - Involves hyperEXT & extreme varus force
 - Non-contact hyperEXT & varus stressors can also cause LCL injuries
- **Clinical presentation:**
 - Acute event consistent w/ a medial blow to the knee while fully EXT, or extreme non-contact varus bending
 - Complain of sudden onset lateral knee px, swelling, & ecchymosis after the injury
 - May report thrust gait, inc. foot kicking in mid-stance
 - May complain of paresthesias over the lateral lower extremity, & weakness &/or a foot drop
 - Gain complete Hx inc. bleeding/clot disorders, previous surgeries, occupation, gait, ambulation-assisted devices, living situation (stairs at home)

Lateral Collateral (LCL) & PLC Injuries (cont)

- **Physical examination:**
 - Limited ROM
 - Lateral knee TTP
 - Ecchymosis, swelling, & warmth
 - Gait: classical *varus thrust* finding
 - **Special tests:** Varus stress test, EXT ROT recurvatum test, Posterolateral drawer test, Reverse pivot shift test, Dial test
- **Diagnosis:**
 - MRI is the gold standard
 - US useful for rapid diagnosis

Classification of injury:

 - **Grade 1:** Mild sprain - diagnosed w/ lateral knee tenderness, no instability mechanical Ssx
 - **Grade 2:** Partial tear - diagnosed w/ more severe localised lateral & posterolateral knee px, as well as swelling
 - **Grade 3:** Complete tear - px & swelling vary in pts, usually associated w/ PLC & other related injuries, & mechanical Ssx
- **Complications:**
 - Undiagnosed LCL & PLC injuries have several long-term complications
 - Continued knee instability & chronic px
 - 35% of PLC injuries may have an associated peroneal n. palsy (probs due to its proximity to the LCL)
 - Pts may develop long-term foot drop, as well as lower extremity weakness & decreased sensation
 - Post op: hardware irritation & stiffness
- **Management:**
 - Acutely, all grades treated w/ RICE & NSAIDs
 - Grade 1 & 2: non-operative, non-WB for 1 week for better px control; next 3-6 weeks, the pt should be in a hinged-knee brace while performing functional rehab
 - Grade 3: surgical reconstruction (best results), post op rehab and functional exercises
- **Ddx:**
 - ACL/PCL tears
 - Lateral meniscus tears
 - Popliteal injury
 - Bone contusion
 - ITB syndrome

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Posterior Cruciate Ligament (PCL) Injuries

• YELLOW

- **Intro:**
 - 1/4 major ligaments of the knee that function to stabilise the tibia on the femur
 - **Origin:** anterolateral aspect of the medial femoral condyle in the area of the intercondylar notch, **Insertion:** posterior aspect of the tibial plateau
 - Prevents posterior translation of the tibia on the femur
 - Lesser extent, the PCL functions to resist varus, valgus, & EXT ROT forces
- **Aetiology (risk factors):**
 - Caused by extreme anterior force applied to the proximal tibia of the FX knee
 - Dashboard injuries during car accident or falling forward onto a FX knee
 - M>F (2:1)
 - Motorcycle accidents (28%) & soccer-related injuries (25%) are the leading causes
- **Pathophysiology:**
 - Least common knee injury
 - Anterolateral portion is more commonly injured due to majority of injuries occurring in knee FX
 - Resists posterior translation w. the assistance of the posterolateral joint capsule, popliteus, MCL, & posterior oblique ligament
- **Clinical presentation:**
 - Pts often present w/ acute onset of posterior knee px, swelling, & instability
 - Hx includes mechanism of injury, ic. falling onto FX knee or recent vehicle accident
- **Physical examination:**
 - Pulses
 - SMR
 - Mild to moderate joint effusion
 - Swelling usually less than in ACL tear
 - Pt may present w/ antalgic gait & potential difficulty walking up & down stairs
 - Palpation: potential effusion, joint line for tenderness (suggestive of meniscal tears)
 - Muscle strength test: should be normal, but there may be weakness w/ knee EXT & FX 2° to guarding
 - Limited ROM
 - **Special tests:** Posterior drawer test, Quadriceps active test, Dial test or EXT ROT test, Varus/valgus stress



Posterior Cruciate Ligament (PCL) Injuries (cont)

- - MRI is the gold standard
- Diagnosis:**
- Initial imaging w/ plain X-rays
- Classification of injury:**
- Grade 1 (partial tear) - 1-5mm posterior translation, tibia remains anterior to femoral condyles
 - Grade 2 (complete isolated) - 6-10mm posterior tibial translation, complete tear of PCL w/o another injury, anterior tibia flush w/ femoral condyles
 - Grade 3 (complete PCL w/ combined capsular &/or ligamentous injury) - >10mm posterior tibial translation, tibia posterior to femoral condyles which may indicate a concomitant capsuloligamentous injury
- Complications:**
- Intra & postoperative complications of PCL surgery
 - Neuromuscular injury (e.g. popliteal artery injury)
 - Fracture
 - Residual instability
 - Osteoarthritic progression
 - Osteonecrosis
 - Stiffness
 - Failure of associated ligament reconstructions or meniscal repairs
 - Revision of PCL reconstruction



Posterior Cruciate Ligament (PCL) Injuries (cont)

- **Management:**
 - **Variables to consider:** Acute or chronic; isolated or combined
 - **Non-operative:**
 - Acute grade 1 & 2 injuries w/ posterior tibial translation (8-12mm)
 - Grade 3 injuries w/ mild Ssx or low-demand activities
 - Acute treatment inv. RICE, initial knee bracing, & crutches
 - Rehab focuses on knee EXT strengthening
 - Est. return-to-play in 2-4 weeks for Grade 1 & 2 injuries
 - Grade 3 may inv. knee immobilisation followed by rehab
 - **Operative:**
 - Acute injuries w/ tibial translation >12mm, associated meniscal tears, dislocation, bony avulsions, & combined injuries
 - Chronic injuries w/ posterior tibial translation >8mm, symptomatic cases, instability, & combined injuries
 - Arthroscopic procedures
 - Reconstruction
 - Graft fixations
 - High tibial osteotomy
- **Ddx:**
 - ACL injury
 - LCL injury
 - MCL injury
 - Meniscus injury
 - Talofibular ligament injury

[link text](#)

Baker's Cyst

- **GREEN**
- **Intro:**
 - Also known as popliteal or paramedical cyst
 - Fluid-filled sac, typically between semimembranosus & medial head of the gastroc
 - Common in adults & associated w/ degenerative knee conditions
 - Often linked to degenerative meniscal tears as one of the most common causes
 - In children, popliteal cysts more commonly arise as a 1° condition (resulting from herniated post knee joint synovium/capsule)



Baker's Cyst (cont)

• Aetiology (risk factors):	<ul style="list-style-type: none"> - Children aged 4-7 yo - Adults aged 35-70 - Most commonly found in adults w/ Hx of trauma, knee joint diseases (OA, RA, etc), or as incidental findings - They form due to accumulation & extrusion of synovial fluid between the semimebranosus & medial head of the gastroc - Popliteal cysts are located on the medial side of the popliteal fossa just below the crease at the posterior knee - Prevalence increases w/ age, likely due to an increase in knee-burial communication
• Pathophysiology:	<p>Several mechanisms:</p> <ol style="list-style-type: none"> 1. Joint-cyst communication 2. Sequestration of synovial fluid in popliteal fossa due to a valve-like effect between the joint & cyst (controlled by gastroc-s-emimembranosus m. w/ FX & EXT at the knee) 3. Negative intraarticular knee pressure during partial FX combined w/ a positive pressure during extension (as a result directing fluid flow towards the cyst from the suprapatellar bursa during FLX) 4. Gastroc-semimembranosus bursa enlargement resulting from micro-traumas to the bursa w/ muscle contractions 5. Herniation of the joint capsule into the popliteal fossa
• Clinical presentation:	<ul style="list-style-type: none"> - Sensation of tightness, discomfort, or px behind the knee - Swelling Moree noticeable when standing w/ full knee EXT - Swelling reduces or disappears when the knee is flexed to 45° (Foucher's sign) - Px worsens w/ increased activity & may limit full knee FX & EXT
• Physical examination:	<ul style="list-style-type: none"> - Compression of surrounding vessels - Lower extremity oedema due to venous obstruction - Enlargement into the calf m. (dissecting cyst) can cause swelling, erythema, distal oedema, & +ve Homan's sign - Venous obstruction can mimic Ssx of DVT or thrombophlebitis



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Baker's Cyst (cont)

• **Diagnosis:**

Diagnosis methods:

- Pt stand & full knee EXT
- Mass is most prominent in this position
- Mass often softens or disappears when the knee is FX to 45° (Foucher's sign)
- Supine exam: knee passively moved from full EXT to at least 90° FX

Imaging:

- Plain radiograph & US
- MRI is recommended, esp. if considering surgery

• **Complications:**

Complications & Ssx of ruptured Baker's cyst:

- Rapid fluid accumulation may cause cyst rupture
- Released fluid into surrounding tissues can lead to inflammation
- Ssx similar to thrombophlebitis: sharp pc in the knee & calf; swelling or erythema of the calf; sensation of water running down the calf

Complications of cyst rupture:

- Post tibial n. entrapment: posterior plantar numbness & calf px
- Popliteal artery occlusion: lower extremity oedema
- Anterior compartment syndrome: foot drop, oedema of anterolateral leg
- Posterior compartment syndrome: plantar dysesthesia, weakness of toes, calf swelling, px worsens w/ passive toe extension



Baker's Cyst (cont)

- **Management:**
 - Asymptomatic cases are managed through observation & reassurance alone
 - Essential to treat any underlying joint disorder in pts w/ symptomatic Baker's cysts
 - Helps reduce synovial fluid accumulation & cyst enlargement
- **Non-operative:**
 - Rest/activity modification
 - NSAIDs
 - Physical therapy & rehab regimes are effective for minimal Ssx & smaller degenerative meniscal tears
 - Aspiration & steroid injection
 - Lower recurrence in younger pts
 - Higher recurrence rates in older pts & those w/ degenerative meniscal tears
- **Operative:**
 - Arthroscopy
 - Open cyst excision (not recommended in case of underlying degeneration due to recurrence risk)
- **Ddx:**
 - Abscess
 - Arteriovenous fistula
 - DVT
 - Ganglion cyst
 - Hemangioma
 - Lipoma
 - Lymphadenopathy
 - Malignancy (e.g. fibrosarcoma, liposarcoma)
 - Popliteal (Baker's) cyst

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Chondrocalcinosis (pseudogout)

• YELLOW



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Chondrocalcinosis (pseudogout) (cont)

• Intro:

Overview:

- Calcium pyrophosphate deposition disease (CPPD)
- Crystal deposition arthropathy involving synovial & periarticular tissues
- Asymptomatic to acute or chronic inflammatory arthritis

Phenotypes & terminology:

- Various terms used for different phenotypes
- Acute CPP deposition arthritis: *"Pseudogout"*
- Chronic CPP deposition arthritis: informally called *"pseudo-rheumatoid arthritis"*
- Characterised by a waxing & waning clinical course, resembling RA

Radiological findings:

- Term: **chondrocalcinosis**
- Describes intra-articular fibrocartilage calcification

Commonly affected joints:

- Hips
- Knees
- Shoulders

Underlying factors:

- Often associated w/ underlying joint disease or metabolic abnormalities
- Predisposing factors: OA, trauma, surgery, RA

• Aetiology (risk factors):

- Often pts >65 yo, w/ 30-50% >85 yo
- M>F
- Rare <60 yo
- High prevalence of radiographic **chondrocalcinosis** in the general population

Comorbidities associated w/ CPPD:

- Hyperparathyroidism
- Gout
- OA
- RA
- Hemochromatosis

Other related comorbidities:

- Osteoporosis
- Hypomagnesium
- Chronic kidney disease
- Calcium supplementation



Chondrocalcinosis (pseudogout) (cont)

- **Pathophysiology:**
 - Caused by an imbalance between pyrophosphate production & pyrophosphate levels in diseased cartilage
 - Pyrophosphate deposits in the synovium & adjacent tissues combine w/ calcium to form CPP
 - Deposition of calcium pyres-hate can activate the immune system, leading to inflammation & soft tissue injury
- **Clinical & physical presentation:**
 - Acute cases:**
 - Typically, self-limiting, & inflammation resolves within days - weeks w/ treatment
 - Similar to acute rate arthropathy
 - Joint oedema, erythema, & tenderness
 - Up to 50% may have a low-grade fever
 - Most commonly affected joint: knee
 - Other affected joints: hip, shoulders
 - Chronic cases:**
 - May show Ssx of RA inc. morning stiffness, localised oedema, & ↓ ROM
 - Waxing & waning episodes of non-synchronous, inflammatory arthritis
 - Affects multiple non-WB joint: wrist & MCP joint
 - Causes "*crowned dens syndrome*" (deposition of CPP around C2)
 - Mostly asymptomatic
- **Diagnosis:**
 - Confirm diagnosis through synovial fluid analysis
 - Imaging:**
 - For involved joints is recommended
 - Presence of *chondrocalcinosis* in imaging supports CPPD Dx
 - Absence of *chondrocalcinosis* doesn't rule out CPPD
 - US may reveal early signs like cartilage abnormalities
 - Radiographic imaging may show joint cartilage calcification
 - MRI is useful - can evaluate crystal deposition in joint cartilage

Chondrocalcinosis (pseudogout) (cont)

- **Complications:**
 - Potential of triggering inflammatory responses
 - Presence of *chondrocalcinosis* has associations w/ degradation of menisci & synovial tissue
 - Pts rarely present w/ palpable nodules (resembling gout) that may lead to further joint degradation
 - Rare spinal involvemen, causing clinical manifestations like spine stiffness & bony ankylosis (resembling AS)
 - Some pts present w/ manifestations similar to DISH w/ PLL calcification leading to spinal cord compression Ssx
- **Management:**
 - 1st step reduce inflammation & addressing underlying metabolic conditions
 - NSAIDs
 - Acute flares inv. 1-2 joints often treated w/ joint aspiration
 - Medication
 - Low-alkaline diet
- **Ddx:**
 - Tenosynovitis w/ carpal or cubital tunnel s. can occur (multiple joints affected)
 - RA
 - AS
 - Erosive OA
 - Gout

[link text](#)

Acute Compartment Syndrome

- **RED**
- **Intro:**
 - Increased pressure in closed osteofascial compartment
 - Leads to impaired local circulation
 - Surgical emergency
 - Untreated ACS can lead to ischemia & necrosis
- **Compartments:**
 - Lg has 4 compartments: anterior, lateral, deep posterior, & superficial posterior
 - Anterior compartment is most common ACS
 - Contains extensor m., tibialis anterior m., deep peroneal n., & tibial artery
- ***Open fractures:**
 - Skin laceration doesn't relieve compartment pressure
 - ACS is still predictable, esp. in open Gustily type 2 & 3 lesions in proximal intra-articular tibia fractures

Acute Compartment Syndrome (cont)

- - M>F (7.3:0.7)
- Aetiology** - Majority of cases result from trauma, w/ tibial shaft fracture being the most common
- (risk factors):** - More common in males <35 yo, possibly due to larger muscle mass & high-energy trauma involvement
- Pts w/ bleeding diathesis (e.g. hemophilia) are at high risk
- ACS reported w/o trauma in paediatric leukaemia cases
- Pts w/o fractures at high risk of complications & delayed treatment
- Other causes:** soft tissue injuries, burns, vascular injuries, crush injuries, drug overdoses, reperfusion injuries, thrombosis, bleeding disorders, infections, improperly placed casts or splints, tight circumferential bandages, penetrating trauma, intense athletic activity, & poor positioning during surgery
- In children, supracondylar # of the humerus & ulnar/radial # are associated w/ compartment syndrome
- - Caused by ↓ intracompartmental space OR ↑ fluid volume, making the surrounding fascia non-compliant
- Pathophysiology:** - ↑ compartment pressure impairs hemodynamics, disrupting the equilibrium between venous outflow & arterial inflow
- Elevated compartment pressure leads to reduced venous outflow, ↑ venous capillary pressure
- If intracompartmental pressure surpasses arterial pressure, arterial inflow ↓, causing tissue ischemia
- Reduced venous outflow & arterial inflow result in ↓ tissue oxygenation, potentially leading to irreversible necrosis
- Normal compartment pressure is <10mmHg, while reading of 30mmHg or higher indicates ACS
- **Clinical presentation:** - Can occur within few hours to up to 48h after trauma
- Px is severe & disproportionate to the injury; may be felt as a burning sensation or deep ache
- Initially, px may only occur w/ passive stretching but can be absent in advanced cases
- Paresthesia, hypoesthesia, or poorly localised deep muscular px may be present
- The "5 P's" (px, pulselessness, paresthesia, paralysis, & pallor) are classic signs, but they're typically late findings
- Paresthesia may occur earlier
- In some cases, a pulse may still be present, even in a severely compromised extremity



Acute Compartment Syndrome (cont)

- **Physical examination:**
 - Earliest objective physical finding is the **tense, or 'wood-like' feeling in the involved compartment**
 - Focus on neurovascular exam.**
 - Observe skin for lesions, swelling, or colour change
 - Palpate compartment, noting °C, tension, & tenderness
 - Check pulses in the affected area
 - Evaluate two-point discrimination & sensation
 - Assess motor function
 - Due to potential rapid progression, serial exam should be performed to monitor changes over time
- **Diagnosis:**
 - Radiographs are recommended if # suspected
 - Measurement of intracompartmental pressure (not required), can aid in Dx
 - Normal pressure: 0-8mmHg
 - Abnormal: exceeding 30mmHg indicates compartment s. & necessitates intervention
 - Pressure within 10-30mmHg of diastolic blood pressure suggests inadequate perfusion & relative ischemia, prompting clinical attention
 - **DUS** can be used to detect occlusion or thrombus
 - Elevated CPK levels may suggest muscle breakdown from ischemia, damage, or rhabdomyolysis
 - Pre-operative studies: CBC & coagulation studies
- **Complications:**
 - Px
 - Contractures
 - Rhabdomyolysis
 - N. damage & associated numbness &/or weakness
 - Infection
 - Renal failure
 - Death
- **Management:**
 - Immediate surgical consult
 - Keeping extremity at heart level
- **Ddx:**
 - DVT
 - Cellulitis
 - Peripheral vascular injury

[link text](#)

Chronic (Exertional) Compartment Syndrome

- RED



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Chronic (Exertional) Compartment Syndrome (cont)

- **Intro:**
 - Often a Dx of exclusion characterised by ↑ pressures in a muscular compartment , leading to ischemia & px
 - CECS inv/ recurrent, reversible ischemic episodes after activity cessation, leading to predictable ↓ in fascial compartment pressures
 - Rare condition w/ delayed diagnosis, resembling ACS
 - Requires surgical emergency intervention through fasciotomies to prevent irreversible m. ischemia & neurovascular injury
 - Usually occurs in the LL but can also affect forearm, thigh, or hand
- **Aetiology (risk factors):**
 - Relatively common among young adult athletes (running, endurance training, soccer, field hockey, & lacrosse)
 - Anterior compartment most commonly affected (70% of cases), then deep posterior
 - B limb in 37-82% of symptomatic cases
 - 20-25 yo, M>F, & often B
 - Associated w/ sports like running or skating, & higher activity intensities
 - Can result from overuse injuries, repetitive mechanisms causing tissue degeneration, scar formation, & military training
 - Pts w/ decreased fascial elasticity may be at risk for nerve entrapment & quicker rises in pathological pressures
- **Pathophysiology:**
 - Has multiple etiologies
 - Muscle compartment swelling during exercise, leading to increased pressure within musculofascial compartments
 - Rise in pressure compromises blood flow, causing px, motor weakness, & paresthesia, in corresponding neurovascular distributions
 - Specific Ssx depend on the affected compartment: e.g. anterior & lateral compartment involvement in the LL may present w/ px & tingling on the dorsum of the foot
 - Increased intracompartmental pressure results in reduced myocyte oxygenation, leading to myonecrosis & neurological damage



Chronic (Exertional) Compartment Syndrome (cont)

- **Clinical presentation:**
 - Pts often present following sport-related activity ro exertion w/ non-specific leg px & persist after strenuous or repetitive activity
 - Ssx will predictably abate following activity cessation
 - Particular attention to pts characterisation of px during strenuous activity, well-localised to a specific compartment, & the px/Ssx disappear quickly after the cessation of activity
 - Pts will generally complain of discomfort described as squeezing, cramping, aching, or burning that typically begins within 15-20 minutes of activity
 - Discomfort resolves completely w/ rest, although the duration may vary
- **Physical examination:**
 - In 70-95% of cases B px
 - Physical exam often unremarkable, esp. if not done during or immediately after exercise
 - Suspected cases should undergo pre- & post-exercise physical exams
 - After exercise, the affected compartment may feel tender, bulge, or be tight, & passive stretching may cause px
 - Focal neuro findings may inc. ↓ sensation, paresthesia, or weakness

Stryker pressure monitoring system:

 - Baseline measurements w/ pt at rest
 - Pts then perform controlled exercise until severe Ssx occur
 - After 5-minute rest, compartment pressure measured again
- **Diagnosis:**

Pedowitz criteria:

 - Rule out a Dx of CECS
 - Resting pressure $\geq 15\text{mmHg}$ &/or a pressure of $\geq 30\text{mmHg}$ at 1 min post-exercise in any compartment, &/or;
 - Post-exercise pressure greater than 20mmHg at 5 min post-exercise
- **Complications:**
 - Benign condition characterised by resolution of Ssx w/ rest



Chronic (Exertional) Compartment Syndrome (cont)

- **Management:**
 - Conservative treatment generally ineffective, inc. rest, activity modification, stretching, orthotics, & physical therapy
 - Non-operative modalities inc. NSAIDs, injections, gait training (forefoot strike patterns)
 - Open fasciotomy is the predominant surgical technique
- **Ddx:**
 - Initially gets misDx as shin splints or medial tibial stress syndrome (MTSS)
 - Vascular pathologies (intermittent claudication, popliteal artery impingement)
 - Tibial stress #
 - Tendon pathologies (tendinitis, tendinosis, or tendon rupture)
 - N. entrapment

[link text](#)

Fibular Nerve (Peroneal N.) entrapment

- **GREEN**



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Fibular Nerve (Peroneal N.) entrapment (cont)

- - Branches off the sciatica n. in the distal posterior thigh & receives fibres from L4-S2 nerve roots
- Intro:**
- Runs down the thigh, posterior to the biceps femoris m., & crosses laterally to the head of the lateral gastroc m.
 - Provides sensory innervation to the lateral leg via the lateral sural n.
 - **Two branches:** *superficial* which innervates the lateral compartment of the leg, & *deep* which innervates the anterior compartment of the leg & foot dorsum
 - Both have roles in foot eversion & dorsiflexion
- Innervation of superficial**
- Motor:**
- Lateral compartment
 - Peroneus longus
 - Peroneus brevis
- Sensory:**
- Anterolateral leg
- Innervation of deep**
- Motor:**
- Anterior compartment
 - Tibialis anterior
 - Extensor hallucis longus
 - Extensor digitorum longus
 - Peroneus tertius
- Sensory:**
- First dorsal webspace



Fibular Nerve (Peroneal N.) entrapment (cont)

- **Aetiology (risk factors):**
 - Most common mononeuropathy in the LL & 3rd most common focal neuropathy overall (after carpal tunnel s. & ulnar neuropathies)
 - Common in traumatic injuries in young athletes (e.g. football, soccer) & following high energy trauma (car accidents) in adults
 - Occurs in about 16-40% of knee dislocations
 - Trauma or injury to the knee:**
 - Knee dislocation
 - Direct impact, penetrating trauma, or lacerations
 - Fibula #, esp. proximal fibula
 - External compression sources:**
 - Tight splint/cast
 - Compression wrapping/bandage
 - Habitual leg crossing
 - Prolonged bed rest
 - Positioning during anaesthesia & surgery (important to pad bony prominences)
 - Systemic causes:**
 - Diabetes mellitus
 - Inflammatory conditions
 - Anorexia nervosa
 - Others:**
 - Intramural ganglion
 - Peripheral nerve tumour
 - Iatrogenic injury following surgery to the hip, knee, & ankle
- **Clinical presentation:**
 - Varies based on location, severity, & anatomic variations
 - Commonly presents w. weakness in ankle dorsiflexion
 - Classic result is *foot drop* or *catching toes* while walking
 - Development of acute or gradual, complete or partial
 - Numbness or paresthesia along lateral leg, dorsal foot, &/or first toe webspace
 - Possible px in traumatic cases



Fibular Nerve (Peroneal N.) entrapment (cont)

- **Physical examination:**
 - Gait: significant for chronic peroneal nerve palsy w/ foot drop, high stoppage gait weakened dorsiflexors to prevent toe dragging
 - Localisation of lesions:**
 - Proximal lesions (e.g. knee dislocations) may present w/ numbness in both superficial & deep n. distributions
 - Upper lateral leg numbness indicates a lesion proximal to fibular head (possibly inv. sciatic n. or lumbosacral n. roots)
 - Lower lateral leg & dors of the foot involvement suggests superficial peroneal n.
 - Altered sensation in the dorsal aspect of the first web space implicates the deep peroneal n.
 - Motor involvement testing:**
 - Assess foot eversion (superficial n.) & foot/toe dorsiflexion (deep n.)
 - Weakness in both suggests common Peroneal n. involvement
 - Proximal lesion may result in both distributions
 - Detailed examination of dorsiflexion ability is crucial
 - Tinel sign:**
 - Tapping along the nerve course, esp. around the fibular neck
 - +ve test = tingling or paresthesia distally
- **Diagnosis:**
 - CT can be used to assess osseous abnormalities
 - MRI/US suitable for evaluating soft-tissue sources or masses (es. in cases of traumatic knee dislocations)
 - Electrodiagnostic studies (inc. NCV & EMG) are used to Dx peroneal nerve palsy
 - They evaluate motor & sensory axons of the peroneal n. aiding in localisation of the nerve injury
 - Useful in post-operative setting of a known traumatic injury for long-term management planning & pt care
- **Management:**
 - Full physical therapy
 - Ankle-foot orthoses, even for foot-drop when surgery isn't warranted
 - Surgical indicators: rapid deterioration & no signs of improvement within **3 months** & open injuries w. suspected nerve laceration
 - Open lacerations should undergo exploration & surgical repair within 72h

Fibular Nerve (Peroneal N.) entrapment (cont)

- **Ddx:**
 - Peroneal tendon pathology
 - Other compressive neuropathies (tarsal tunnel s., anterior tarsal tunnel s., non-specific tendinitis affecting lower limb m./t.)
 - Chronic ankle px

[link text](#)

Meniscal Tears

• YELLOW

- - Lateral & medial menisci function in load transmission & shock absorption in the tibiofemoral joint
- Intro:**
 - Inner 2/3 (white zone) of the menisci is avascular, likely receiving nutrition through synovial fluid diffusion
 - Peripheral 1/3 (red zone) is well-vascularised, supplied by branches of the medial & lateral vehicular arteries
 - Medial meniscus is less mobile than the lateral one, firmly attached to the joint capsule & deep fibres of the MCL
 - Lateral meniscus doesn't connect w/ the LCL & has looser attachments w. the joint capsule
 - Anterior margins of the menisci are connected by the transverse inter meniscal ligament
 - Peripheral 2/3 of the menisci contain nociceptive free endings (pain perception), while mechanoreceptors are in the anterior & posterior horns, suggesting a proprioceptive function
 - Posterior horn of the lateral meniscus connects to the femur via menisiofemoral ligaments & the adjacent popliteus tendon



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Meniscal Tears (cont)

- **Aetiology**
 - 61/100,000 in general population (USA), 9/1000 in military population
- (risk factors):
 - 15% of sports injuries
 - M>F
 - Age >40 yo
 - ACL deficient knees, esp. if ACL reconstruction is delayed beyond 1 year from initial injury
 - Medial>lateral meniscal tears
 - **Increased risk factors:** infantry-related duties, frequent squatting/kneeling, & participation in sports like soccer, rugby, football, basketball, baseball, skiing, & wrestling
 - Traumatic impacts to the knee can lead to isolated meniscal tears or tears concomitant w/ bony lesions or damage to primary stabilising ligaments (ACL & MCL)
 - Less force is required for tears in individuals w/ degenerative changes of the menisci, typically seen in adults >40 w. concomitant OA
 - **Isolated meniscal tears** result from rotational or shearing forces across the tibiofemoral joint, esp. during activities w/ increased closed kinematic chain FX, heavy lifting, rapid acceleration/deceleration, change of direction, & jumping



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Meniscal Tears (cont)

- **Pathophysiology:**
 - Characterised by shape & location on MRI
 - **Horizontal** (cleavage) run parallel to the tibial plateau, associated w/ degenerative changes in people >40 w/o specific causes
 - **Longitudinal** run perpendicular to the tibial plateau & parallel to the meniscus axis
 - **Radial** run perpendicular to both the tibial plateau & the meniscus axis, originating from the inner free edge
 - **Complex** involve combinations of horizontal, longitudinal, or vertical tears
 - **Displaced** involve complete detachment or flipping of a piece still attached to the meniscal body
 - **Bucket-handle** are complete longitudinal tear fragments that migrate centrally
 - **Parrot-break** are radial tears w/ partially detached fragments
 - **Flap** are partially detached fragments of horizontal tears
 - Tears in the outer 1/3 vascular zone are "red-red"; those extending into the inner 2/3 avascular zone are "red-white", & tears within the inner 2/3 avascular zone are "white-white"
 - Tears in the red zone have the highest potential for spontaneous healing w/ conservative management or successful outcomes after meniscal repair
- **Clinical presentation:**
 - **"POP"** sensation w/ immediate knee effusion suggests ACL tear w/ possible medial meniscal involvement
 - Gradual effusion over 24h indicates an isolated meniscal tear
 - Ssx can be insidious, featuring low-grade effusion& stiffness w/o a specific triggering event
 - Px commonly reported along the anteromedial or anterolateral joint line
 - **Additional Ssx:** locking, clicking, catching, intermittent inability to fully EXT the knee, & a sense of the knee giving way

Meniscal Tears (cont)

- **Physical examination:**
 - Inspection of edema, palpation of joint line, standing & supine ROM, muscle strength testing, special testing
 - Anteromedial & anterolateral joint line tenderness at 90° FX
 - Px & deficits in FX or EXT ROM may vary based on tear type & effusion extent
 - Deficits in open kinetic chain knee FX/EXT strength testing are unlikely
 - Antalgic gait or increased px w/ squatting may indicate meniscal issues due to compressive forces
 - **Special tests:** Thessaly test, McMurray's test, Apley's compression test
- **Diagnosis:**
 - Begin w/ radiographs - AP, lateral, oblique, sunrise, & WB views to assess concomitant bony pathologies, loose bodies, & OA
 - Arthroscopy is the gold standard
 - MRI is the best mode of imaging to Dx & characterise tears
- **Management:**
 - RICE
 - NSAIDs
 - Early px-free knee & ankle ROM exercise (help limit motion loss & aid edema)
 - Bracing/sleeves (protection & compression)

Simple tears (outer 1/3 of the meniscus) & degenerative tears:

 - 4-6 weeks relative rest & physical therapy
 - Despite conservative management, pts w/ persistent px, swelling, & mechanical Ssx should be evaluated for surgical intervention

Surgical tears:

 - Meniscal repair is preferred over meniscectomy (risk of accelerated OA)
 - Factors ↑ success: tears that occur in red zone of the meniscus, shorter than 2cm, vertical longitudinal tears, & acute tears

Rehab:

 - First 6 weeks inc. restrictions in knee FX ROM & WB status (depending on tear & repair type)
 - Strengthening
 - Mobs



Meniscal Tears (cont)

- Ddx:
 - ACL injury
 - Contusions
 - ITB syndrome
 - Knee osteochondritis
 - LCL injury
 - Lumbosacral radiculopathy
 - MCL injury
 - Medial synovial plica irritation
 - Patellofemoral joint syndrome
 - PCL injury

[link text](#)

Osgood-Schlatter Disorder (OSD)

• GREEN

- Intro:
 - Common cause of anterior knee px in skeletally immature athletes
 - Also known as *osteochondrosis* or *traction apophysitis of the tibial tubercle*
 - Common in sports like basketball, volleyball, sprinting, gymnastics, & football
 - Self-limiting & results from repetitive stress on the extensor mechanism (jumping/sprinting)
 - While benign, OSD can lead to prolonged recovery & absence from sports
- Aetiology (risk factors):
 - Leading cause of knee px in adolescent athletes
 - Onset typically aligns w/ growth spurts: 10-15 M & 8-13 F
 - M>F
 - 9.8% of adolescents 12-15 yo (11.4% M; 8.3% F)
 - B Ssx observed in 20-30% of pts
 - Overuse injury due to repetitive strain from patellar tendon
 - Force increases w/ higher activity levels, after rapid growth
 - **Predisposing factors:** poor flexibility of quadriceps & hamstrings, extensor mechanism misalignment



Osgood-Schlatter Disorder (OSD) (cont)

- **Pathophysiology:**
 - Tibial tubercle develops as a 2° ossification centre for patellar tendon attachment
 - Bone growth surpasses muscle-tendon-bone-attachment, susceptible to injury from repetitive stress
 - Repeated quadriceps muscle contraction, esp. in sports involving running & jumping, can cause apophyseal ossification centre softening & partial avulsion, resulting in osteochondritis
 - Tibial tubercle development sequence:**
 - Entirely cartilaginous before age 11
 - Apophysis forms between 11-14
 - Apophysis fuses w/ proximal tibial epiphysis between 14-18
 - Proximal tibial epiphysis & tibial tubercle apophysis fuse w/ the rest of the proximal tibia after age 18
 - **Prevailing theory:** repeated traction over tubercle causes microvascular tears, fractures, inflammation
- **Clinical presentation:**
 - Common Ssx: anterior knee px
 - Presentation: w/ or w/o swelling, unilateral or bilateral
 - Onset: typically insidious, w/o preceding trauma
 - Nature of px: dull ache localised over tibial tubercle
 - Px progression: gradually increases w/ activity
 - Px relief: typically improves w/ rest
 - Duration of relief: subsides minutes to hours after stopping activity or sport
 - Exacerbating factors: running, jumping, direct knee trauma, kneeling, & squatting
- **Physical examination:**
 - Enlarged prominence at the tibial tubercle
 - Tenderness over the patellar tendon insertion site
 - Reproduction of px: resisted knee EXT & active/passive knee FX can reproduce px



Osgood-Schlatter Disorder (OSD) (cont)

- **Diagnosis:**
 - 1° Dx clinically, radiographic evaluation typically not necessary
 - Consider comparing B images to help delineate normal vs abnormal in the pt
- **Radiographic use:**
 - Plain radiographs may be employed in severe or atypical presentations
 - Used to rule out additional conditions like fractures, infections, or bone tumours
 - Assessment for avulsion injury or other traumas may necessitate radiographic evaluation
- **Classic findings:**
 - Elevated tibial tubercle w/ soft tissue swelling
 - Fragmentation of the apophysis
 - Calcification in the distal patellar tendon
- **Complications:**
 - Prominence of tibial tubercle
 - Ongoing pain
 - Sx continue to adulthood if treatment isn't provided or poor compliance w/ recommended treatment
- **Management:**
 - Excellent prognosis
 - Self-limiting but time to resolution can take up to 2 yrs until apophysis fuses
 - Surgery rarely indicated, low benefit & high complication risk
 - Relative rest & activity modification based on pain levels
 - Participation in sports allowed if pain resolves w/ rest & doesn't limit activities
- **Px management:**
 - Ice & NSAIDs
 - Protective knee pad recommended over tibial tubercle to prevent direct trauma
 - Hamstring & quadriceps stretching, & strengthening
 - In severe cases, short knee immobilisation might be considered
- **Refractory cases:**
 - In up to 10%, Sx may persist >1-2 yrs beyond skeletal maturity
 - Ossicle excision may be performed in skeletally mature pts w/ persistent Sx

Osgood-Schlatter Disorder (OSD) (cont)

- Ddx:
 - Patella tendonitis
 - Osteomyelitis of the tibia
 - Perthes disease
 - Synovial place injury
 - Infectious apophysitis

[link text](#)

Osteochondritis Dissecans (OCD)

• YELLOW

- Intro:
 - Rare condition affecting the knee, categorised as a form of osteonecrosis in the subchondral bone
 - 1° occurs in school-aged children & adolescents, w/ manifestations of the dysfunction & px
 - Juvenile OCD occurs in pts w/ open growth plates, while adult OCD applies to skeletally mature pts
 - If left untreated, OCD can lead to degenerative changes, chronic px, & mechanical Ssx such as 'locking' & 'clicking'
- Aetiology (risk factors):
 - Highest incidence 12-19 yo
 - 9.5-29 / 100,00
 - M>F
 - 75% of affected pts have knee lesion, w/ 64% localised in the medial femoral condyle
 - 32% of knee lesions are found in the lateral condyle, while other cases localise to the trochlea, patella, & tibial plateau
 - Usually unilateral, but 7-25% of pts have B disease
 - Theories inc. micro-trauma, ischemia, & genetic predisposition
 - Pts w. extreme obesity & elevated BMI face an increased risk of developing OCD
 - Repetitive trauma is widely considered the 1° cause of OCD of the knee
 - Adult form of OCD is believed to result from vascular insult



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Osteochondritis Dissecans (OCD) (cont)

- **Pathophysiology:**
 - Disruption of epiphyseal vessels, leading to ischemia & necrosis at trauma site
 - Softening, tearing, fissuring, & erosion of hyaline cartilage follow as a consequence of the disrupted blood supply
 - Advancement of the affected area result in focal demineralisation & repeated shear forces, causing detachment of bone & overlying cartilage
 - Repetitive axial loading, esp. w/ increased valgus & varus stress, is suggested by experts as a contributing factor tot he condition
 - OCD lesions can introduce irregularities in the articular surface, potentially leading to degenerative arthritis
- **Clinical presentation:**
 - Vague, poorly localised knee px that worsens w/ activity
 - Stiffness & occasional swelling may occur during or after activity as the disease progresses
 - Advanced stages may be indicated by **locking or catching**, suggesting the presence of a sizeable loose body in the knee
 - Hx of trauma, recent increase in activity level, previous knee injuries, & the presence of mechanical Ssx
 - Approx. 80% of pts report px when WB
 - Juvenile: intermittent, activity-associated px poorly localised around the anterior aspect of the joint
 - Adult: more likely effusion, limited ROM, or mechanical Ssx such as 'catching or locking'
 - Depending on chronicity of the lesion, pts may report quadriceps dysfunction & intermittent knee instability



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Osteochondritis Dissecans (OCD) (cont)

- **Physical examination:**
 - Genu varus, associated w/ lesions at medial femoral condyle
 - Genu valgus, associated w/ lesions at lateral femoral condyle
 - Quadriceps atrophy or weakness may be evident
 - Foreign body may be palpable
 - FX of knee during joint palpation can reveal effusion or bony tenderness along the femoral condyles
 - ROM may be restricted due to px, swelling, or the presence of a loose body
 - Antalgic gait or lateral rotation of the foot on the affected side may indicate efforts to alleviate WB px

Wilson sign:

- Identifies lesions of the lateral aspect of the medial femoral condyle
- +ve test: px w/ INT ROT, relieved by EXT ROT, indicating impingement of the OCD lesion
- Absence of the Wilson sign does not rule out OCD

- **Diagnosis:**
 - Arthroscopy is the gold standard for assessing lesion stability
 - Plain radiographs used to locate the lesion, assess growth plates, & rule out other conditions
 - Initial radiographs may appear normal in OCD

Lesion characteristics:

- Distinct Lucent areas w/ varying density levels
- Calcifications & Lucent lines may or may not be present, depending on lesion severity
- B comparison

Classification:

- Lesion location can provide important prognostic info
- Atypical locations like trochlea or patella may not respond effectively to conservative management

MRI evaluation:

- Useful for assessing unstable lesions presenting w/ mechanical Ssx or knee effusion
- Unstable lesions on mRI may exhibit increased T2 signal, destruction of overlying articular cartilage, or multiple cyst-like foci
- Gadolinium contrast may be necessary for assessing blood supply & stability uncertainties
- Line of high signal intensity between fragment & underlying bone is a sensitive prognosticator

Osteochondritis Dissecans (OCD) (cont)

- **Complications:**
 - If left untreated, adults often progress to arthritis
 - Degenerative articular changes over time
 - A non-union & dissociation of the bony fragment
 - Chronic px & mechanical Ssx
 - Surgical complications inc. postop infection, pneumonia, haemorrhage, & reactions to anesthesia
 - Venous thrombosis due to immobility
- **Management:**
 - Prognosis influenced by age, location & appearance of lesion

Non-operative:

 - Recommended for juvenile pts w/o a displaced fragment OR stage 1-3 disease
 - Immobilisation & protected WB for 4-6 weeks
 - Physical therapy initiated after immobilisation & continued until pain-free, achieving full ROM, strength, power, & mobility
 - NSAIDs for px & edema

Operative:

 - Surgery recommended if conservative treatment ineffective after 3-6 months or if not suitable
 - 1° treatment for Ssx related to OCD in adults, stage 2 disease, or expanding lesions on radiographs
 - Surgical intervention warranted in juveniles w/ stage 4 disease, loose bodies, unstable lesions, or impending physeal closure



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Osteochondritis Dissecans (OCD) (cont)

- Ddx:
 - Juvenile:**
 - Patellofemoral syndrome
 - Patellar tendonitis
 - Osgood-Schlatter disease
 - Sinding-Larsen-Johansson syndrome
 - Fat pad impingement
 - Symptomatic discoid meniscus
 - Symptomatic synovial plica
 - Adult:**
 - Patellofemoral px
 - Knee OA
 - Chondromalacia
 - Patellar tendonitis
 - Meniscal tear
 - Fat pad impingement
 - Symptomatic synovial plica
 - Adult w/ more severe Ssx:**
 - E.g. atraumatic edema & mechanical Ssx
 - Meniscal tear
 - Osteochondral loose body
 - Neoplasm

[link text](#)

Patellofemoral Pain Syndrome (PFPS)

• GREEN

- Intro:
 - Also known as *Chondromalacia patella (CMP)* & *Runner's knee*
 - Softening of hyaline cartilage on articular surfaces of bones
 - CMP specifically refers to the softening, tearing, fissuring, & erosion of the patellar cartilage
 - Can occur in any joint, but common in joints w/ trauma & deformities



Patellofemoral Pain Syndrome (PFPS) (cont)

- Aetiology (risk factors):
 - F>M
 - Increased Q angles in F (lateral positioning of the patella)
 - No hormonal cause has been identified
 - Active young adults (esp. running sports), & workers who stress their patellofemoral joint (stairs, kneeling)
 - Often multifactorial
 - LL malalignment & patellar maltracking play a significant role
 - Foot & ankle variances: pes planus can lead to increased lateral wear of the patellofemoral joint
 - Miserable malalignment syndrome, w/ femoral ante version, genu valgum, & pronated feet
 - Muscular weakness: vastus medialis & core m.
 - Patellar lesions from injuries, immobilisation, or surgical procedures causing quadriceps atrophy
 - Abnormal wear & tear of the patellofemoral joint's hyaline cartilage
 - Iatrogenic factors: injecting chondrotoxic medications into joints



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Patellofemoral Pain Syndrome (PFPS) (cont)

- **Pathophysiology:**
 - Pathological process:**
 - Hyaline cartilage composed of chondrocytes, type 2 collagen, proteoglycans, & water
 - Avascular w/ nutrients diffusing from synovial fluid
 - Poor repair due to lack of blood supply, devoid of lymphatic & neural tissue
 - Factors leading to hyaline cartilage degeneration:**
 - Destruction by chondrotoxic substances, cytokinins, & proteolytic enzymes
 - Microtrauma from wear & tear
 - Repeated compressive stress or increased loads on patellofemoral joint
 - Aging-related decrease in chondrocytes, proteoglycan production, & water content
 - Cross-linking of collagen fibrils leads to loss of elastic properties
 - Superficial zone of hyaline cartilage is the 1st to degenerate in aging process
 - Px generation:**
 - Anterior fat pad & joint capsule commonly involved in generating px signals
 - SUBchondral bone less likely to cause px signals
 - Initiation of CMP pathology:** begins w/ softening, swelling, & edema of articular cartilage
- **Clinical presentation:**
 - CC: anterior knee px
 - Pts may report insidious onset of diffuse retropatellar or pre patellar px, exacerbated by activities stressing the patellofemoral joint
 - Aggravating factors: stair ascending or descending, squatting, kneeling, running, & prolonged sitting (theatre px)
 - Additional Ssx: effusion, quadriceps wasting, & retropatellar crepitus (not specific to CMP)
 - Hx evaluation: previous trauma, comorbid conditions, joint stability, foot & ankle issues, & activity levels
- **Physical examination:**
 - Px is usually sharp & achy
 - Examine quadriceps appearance, foot & ankle orientation, & specific evaluation of the patellofemoral joint
 - Patella malt racking signs: increased femoral anteversion, EXT tibial torsion, lateral patella subluxation, loss of medial patellar mobility
 - +ve patellar apprehension test
 - +ve Clark's test



Patellofemoral Pain Syndrome (PFPS) (cont)

- **Diagnosis:**
 - Reliable Dx requires excluding other conditions causing anterior knee px
 - Arthroscopy is the most efficient (invasive, so non-invasive methods essential for initial Dx)
 - Plain radiographs: lower sensitivity in earlier stages
 - CT: measures TT-TG distance & detects torsional deformities of the LL
 - MRI: modality of choice for articular cartilage, esp. T2 sequence
- **Complications:**
 - 2° to NSAID usage (e.g. GI Ssx)
 - Bracing may cause dermatological reactions
- **Management:**
 - **Min. 12 months** of conservative management before considering surgery
 - May be reversible
 - Could progress to patellofemoral OA
 - Pts often fully recover (can take months to yrs)
 - **Conservative 1st phase:**
 - Activity modification
 - RICE
 - NSAIDs
 - **Conservative 2nd phase:**
 - Knee + hip exercise to increase strength, mobility & function
 - Patella taping
- **Ddx:**
 - Patellofemoral OA
 - Osgood-Schlatter
 - Plica syndrome
 - Bursitis
 - Saphenous neuritis
 - Quadriceps tendinopathy
 - Patellar tendinopathy
 - Referred px from hip/back

[link text](#)

Medial Tibial Stress Syndrome (MTSS)

- **GREEN**
- **Intro:**
 - Early stress injury leading to tibial stress fractures
 - Known also as *shin splints*
 - Common overuse injury in athletes & military personnel
 - Involves exercise-induced px along the anterior tibia



Medial Tibial Stress Syndrome (MTSS) (cont)

- **Aetiology (risk factors):**
 - 13-20% incidence in runners
 - Up to 35% in military
 - **Factors contributing:** significant increasing loads, volume, & high-impact exercises
 - **Intrinsic risk factors:** F gender, previous MTSS Hx, high BMI, navicular drop, ankle plantar FX range, hip EXT ROT range
 - Overuse condition, specifically a tibial bony overload injury w/ associated periostitis
 - **Common for:** recurrent impact exercise, such as running, jumping, & military personnel
 - Suggested link between vitamin D & increased risk of stress injury
- **Pathophysiology:**
 - Involves accumulation of unprepared microdamage in the cortical bone of the distal tibia
 - Overlying periostitis is typically present at the site of bony injury
 - Periostitis correlates w/ tendinous attachments of soleus, flexor digitorum longus, & posterior tibialis
 - Sharpey's fibers, perforating connective tissue linking periosteum to bone, play a role in the mechanical connection
 - Repetitive muscle traction is believed to be the underlying cause of periostitis & cortical microtrauma
- **Clinical presentation:**
 - Presence of exercise-induced px along the distal 2/3 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 in the foot
- **Physical examination:**
 - Presence of recognisable px reproduced w/ palpation of the posteromedial tibial border >5cm
 - The absence of other findings not typical of MTSS (e.g. severe swelling, erythema, loss of distal pulses, etc)
- **Diagnosis:**
 - Dx through clinical & physical findings
 - Imaging done when uncertain about cause or to rule out other exercise-induced LL injuries
 - Plain radiographs are normal in MTSS & early stress fractures
 - "*dreaded black line*" indicates a *stress fracture*
 - MRI is preferred for identifying MTSS & higher-grade bone stress injuries like tibial stress fractures



Medial Tibial Stress Syndrome (MTSS) (cont)

- **Complications:**
 - Px leading to decreased performance &/or time away from training/participation
 - May progress to tibial stress fracture
 - Severe tibial stress fractures may require surgical intervention
- **Management:**
 - Full recovery is expected
 - Rest & activity modification w/ less repetitive, load-bearing exercise
 - **Additional therapies:** iontophoresis, phonophoresis, ice massage, US therapy, periosteal pecking, & extracorporeal shockwave therapy
 - **No benefit:** low-energy laser therapy, stretching, strengthening, LL braces, & compression stockings
 - **Slow response cases:** optimising calcium & vitamin D status & gait retraining may improve recovery & prevent further progression
- **Ddx:**
 - Tibial stress fracture
 - Compartment syndrome
 - Functional popliteal artery entrapment syndrome

[link text](#)

Varicose Veins

• YELLOW

- **Intro:**
 - Characterised by subcutaneous dilated, tortuous veins of $\geq 3\text{mm}$
 - Age & FHx are important risk factors
 - Common clinical manifestations of chronic venous disease
 - Evaluating associated superficial axial venous reflux is crucial
 - Manifestations can range from limited leg discomfort to swelling & non-healing ulcers
- **Aetiology (risk factors):**
 - Up to 30% of general population
 - F>M
 - **Risk factors:** F gender, multiparity, high BMI, constipation, Hx of venous thrombosis, smoking, & circulating iron levels
 - Both genetic & environmental factors



Varicose Veins (cont)

- **Pathophysiology:**
 - Valve dysfunction leads to increased pressure in veins
 - Elevated pressure causes vein walls to weaken & dilate
 - Ironic pressure causes the vein walls to stretch & lose elasticity
 - Weakened walls contribute to the development of varicosities
- **Clinical presentation:**
 - Leg heaviness
 - Itching
 - Cramps
 - Mild tenderness
 - Skin discoloration
 - Exercise intolerance
 - Leg fatigue
- **Physical examination:**
 - Visible distended veins from thigh to ankle
 - Discolouration most prominent around ankle & calf
 - **Special test:** Trendelenburg test - assesses deep venous valve competency
- **Diagnosis:**

CEAP classification:

 - C0: no visible, palpable signs
 - C1: spider veins
 - C2: varicose veins
 - C3: edema
 - C4a: pigmentation, eczema
 - C4b: lipodermatosclerosis
 - C5: healed ulcer
 - C6: active ulcer
 - Colour duplex venous US exam is recommended for suspected venous reflux
- **Complications:**
 - Venous ulcers
 - Pain
 - Poor cosmesis
 - DVT
 - PE (rare)
 - Superficial thrombophlebitis might be complicated w/ prolonged bleeding & px
 - Superficial vein thrombosis
- **Management:**
 - No cure
 - Long-term graduated compression stockings, leg elevation, & oral px medication
 - Surgery (recurrence is likely)

Varicose Veins (cont)

- Ddx:
 - Lymphedema
 - DVT
 - Cellulitis
 - Dermatological disorders (e.g. stasis dermatitis)

[link text](#)

Vasculitis

- RED
- Intro:
 - Heterogenous group of over 30 different kinds of vasculitis. presenting either as a 1° process or 2° to another pathology
 - Clinical & pathological manifestation vary based on the affected blood vessels' type & location
- Aetiology (risk factors):
 - Incidence of 20-40 million / year
 - Gender dominance depending on type of vasculitis
 - Giant cell arteritis is the most common form
 - **Risk factors:** Behcet disease (ancient Silk route), Takayasu disease (South Asian), Kawasaki disease (children <5), hepatitis B/C
- Pathophysiology:
 - Unknown specific cause
 - Immune system activation: becomes overactive
 - Inflammation of blood vessels: immune system mistakenly identifies blood vessels as foreign invaders
 - Attack on endothelium: attacks the endothelium (inner lining of blood vessels)
 - Adhesion molecules & leukocyte activation: cytokines. signalling molecules in the immune system, cause changes in adhesion molecules on the endothelium → inappropriate activation of leukocytes & their adherence to the blood vessel walls
 - Vessel damage: combined effect of immune cells sticking to the blood vessel walls & the inflammatory response damages the vessels
 - Formation of immune complexes or antibodies: different forms of vasculitis may involve the formation of immune complexes or the production of antibodies targeting specific components in the blood vessels
 - Granuloma formation (in some cases): in certain vasculitis types. granulomas may form. contributing to tissue damage
- Clinical presentation:
 - Ssx dependent on location of vasculitis
 - Potential organ damage from vasculitis
 - Fevers, unexplained weight loss
 - Nose bleeding, hemoptysis, hematuria



Vasculitis (cont)

- **Physical examination:**
 - Upper airway disease
 - Ocular inflammation
 - Limb claudication
 - +ve sensory/motor neuropathy, purpura, change of pulses
- **Diagnosis:**
 - Chest x-ray or high-resolution CT for respiratory Ssx
 - Vascular imaging (MRI, MRA, CTA, vascular US, PET) to detect large artery lesions in vasculitis cases
 - **Labs:** CBC, kidney & liver function, ESR, serologies, & urinalysis w/ urinary sediment
- **Complications:**
 - Depend on the type of vessel involved
 - **Large vessel involvement:** acute MI, stroke, mesenteric ischemia, aortic s., critical extremity ischemia
 - **Life threatening complications of small vessels:** alveolar haemorrhage, renal failure, intestinal ischemia
- **Management:**
 - Long-term survival highly depends on the Dx, response to treatment, & adverse effects of drugs
 - Managed w/ medication
 - 3 main components: remission induction, remission maintenance, & monitoring
- **Ddx:**
 - Infections, neoplasms & certain drug toxicities can mimic vasculitis
 - Coagulopathies can present w/ similar Ssx to vasculitis

[link text](#)

Venous Thrombo-embolism (VTE)

- **RED**
- **Intro:**
 - Significant complication of hospitalisation
 - 3rd leading CV Dx, following heart attacks & strokes
 - VTE encompasses DVT & PE
 - Growing public health concern
 - Need for increased awareness among the public & healthcare providers



Venous Thrombo-embolism (VTE) (cont)

• Aetiology (risk factors):	<ul style="list-style-type: none"> - Global annual burden of VTE is in millions - Significant morbidity & mortality associated w/ VTE cases worldwide - Majority of VTE cases are hospital-related or acquired (60%) - Leading preventable cause of death in hospitalised pts - Risk factor: >40 yrs, obesity, varicose veins, immobility, oral contraceptive, smoking, hypercoagulability, pregnancy, & pelvic/hip/long-bone fractures - Disease states ↑ risk: malignancies, spinal cord injury, nephrotic s., congestive heart failure, IBD, & recent MI
• Pathophysiology:	<ul style="list-style-type: none"> - Venous thrombosis involves the formation of a clot made of platelets & fibrin within blood vessels - Clinically significant thrombi typically form in large-lumen vessels, such as deep veins in the legs, pelvis, & arms - Clots can propagate & extend proximally, leading to clinical Ssx when vascular flow is obstructed - Dislodged clots may embolise to distant sites, w/ the pulmonary vasculature being a common location - Obstruction in pulmonary vascular flow can result in impaired gas exchange, alveolar edema, & pulmonary alveolar necrosis - Chronic repetitive pulmonary embolisation can increase pulmonary vascular resistance, ultimately causing pulmonary hypertension - In the presence of cardiac abnormalities like a patent foramen ovale or atrial septal defect, paradoxical embolism may occur, leading to systemic arterial vascular involvement
• Clinical & physical findings of DVT:	<ul style="list-style-type: none"> - Unilateral limb px is a common complaint - Physical signs may include swelling, warmth, & tenderness to touch - Physical exam signs for DVT have low Dx yield



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Venous Thrombo-embolism (VTE) (cont)

- **Clinical & physical findings:**
 - Sudden onset of dyspnea (most common presenting complaint)
 - Pleuritic chest px, cough, & hemoptysis
 - Massive PE can lead to syncope, hypotension, & shock
 - Physical examination findings for PE are variable & often nonDx
 - Tachypnea (resp. rate >18/min) is common
 - In older pts, new-onset atrial fibrillation may be a presenting Ssx
- **Established PE physical findings:**
 - Tachypnea (resp. rate >18/min) is common
 - Rales may be present in up to 50% of cases
 - Tachycardia (HR >100/min) & fever occur in about 45%
 - Diaphoresis & S3 or S4 gallop may be audible in about 30%
 - Pleural friction rub may indicate peripheral PE w/ pulmonary necrosis
- **Diagnosis:**
 - Chest radiography
 - D-dimer assay
 - US & serial US for DVT
 - CTPA & VQ for PE
- **Complications:**
 - Bleeding
 - Heparin-induced thrombocytopenia
 - Warfarin-induced skin necrosis
- **Management:**
 - Treatment is based on associated conditions
 - 1° treatment is anticoagulation
- **Ddx:**
 - Localised Ssx of DVT can be similar to cellulitis, arterial insufficiency, lymphedema, & hematoma
 - PE: congestive heart failure, acute respiratory distress s., pneumonia, & MI

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