

Muscle Strains

• GREEN

- **Intro:**
 - Muscle / tendon strain is equivalent to ligament sprain in terms of injury type
 - Happens when muscle fibres are overworked, resulting in fibre tearing

- **Aetiology (risk factors):**
 - ↑ incidence in athletes
 - Commonly occurs when there's sudden ↑ in duration, intensity, or frequency of activity
 - 3 types of muscles at risk:**
 - **Two-joint muscles:** motion at one joint can ↑ passive tension, leading to overstretching injuries
 - **Eccentric contractions:** common during deceleration phase, may change muscle tension & cause myofibril overload injuries
 - **Muscles w/ ↑ % of type II fibres:** fast-twitch muscles w/ high-speed contractions, making them more prone to injury (running & sprinting)
 - Hamstrings, gastrocnemius, quadriceps, hip flexors, hip adductors, ES, deltoids, & rotator cuff

- **Pathophysiology:**
 - Contraction induced injury caused by extensive mechanical stress
 - Often occurs due to powerful eccentric contractions or over-stretching of the muscle

Muscle lesions are classified as grade I, II, & III

Grade I (mild):

- Affect a limited number of muscle fibres
- No decrease in strength
- Full AROM & PROM
- Px & tenderness may be delayed to the next day

Grade II (moderate):

- Nearly half of muscle fibres torn
- Acute & significant px
- Accompanied by swelling
- Minor decrease in muscle strength

Grade III (severe):

- Complete rupture of the muscle
- Tendon separated from the muscle belly or muscle belly torn in 2 parts
- Severe swelling & px
- Complete loss of function



Muscle Strains (cont)

- **Sx & Ssx:**
 - Swelling, bruising, or redness
 - Px at rest
 - Inability to use the muscle at all
 - Weakness of muscle or tendons
- **Management:**
 - **First phase:** protection, rest, ice, compression, elevation (PRICE) & NSAIDs
 - **Second phase:** mobilisation should occur ASAP but gradually & within limits of px, Mobs/Drops, low impact exercises
 - 3-6 weeks for the muscle fibres to recover = full ROM, pain free, & 90% strength bilaterally
 - **Third phase:** proprioceptive & endurance, SMT / STW, TrPs

[link text](#)

AVN / Osteonecrosis

• YELLOW

- - Degenerative bone condition resulting from the death of bone cells due to disruption in the subchondral blood supply
- Intro**
- Also known as AVN, aseptic necrosis, & ischemic bone necrosis
 - Typically affects the epiphysis of long bones at WB joints, w/ severe cases potentially causing subchondral bone destruction or joint collapse
 - Common sites include femoral head, knee, talus, & humeral head
 - Less common occurrences in other bones like the carpus & jaw



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AVN / Osteonecrosis (cont)

- - 30-65 yrs
- Aetiology** - M>F
- (risk factors):** - Females more at risk w/ PMHx of lupus
- 6 groups of risk factors:**
 - **Direct cellular toxicity:** chemo/radiotherapy, thermal injury, smoking
 - **Extrasosseous arterial fracture:** hip dislocation, femoral neck fracture, iatrogenic post-surgery, congenital arterial abnormalities
 - **Extrasosseous venous:** venous abnormalities, venous stasis
 - **Intraosseous extravascular compression:** haemorrhage, elevated bone marrow pressure, fatty infiltration of bone marrow due to prolonged high-dose corticosteroid use, cellular hypertrophy & marrow infiltration (Gaucher disease), bone marrow oedema, displaced fracture
 - **Intraosseous intravascular occlusion:** coagulation disorders (thrombophilias & hypofibrinolysis), sickle cell crises
 - **Multifactorial**
- **Pathophysiology:**
 1. Reduction in subchondral blood supply
 2. Induces hypoxia
 3. Loss of cell membrane integrity
 4. Necrosis of cells (osteonecrosis)



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AVN / Osteonecrosis (cont)

• Clinical & physical exam presentation:	Non-traumatic cases: <ul style="list-style-type: none">- Mechanical px w/ variable onset & severity- Difficult to localise- Normal physical exam in early disease (causing delay in Dx)- Focused Hx considerations: recent trauma, steroid use, autoimmune disease, Sickle cell, alcoholism, tobacco use, manual labour, change in gait, connective tissue disorders, insidious onset px, decreased ROM AVN of the hip: <ul style="list-style-type: none">- Early stages often asymptomatic- Hip & groin px- Late-stage. progression indicated by px at rest- Associated Ssx: referred px in buttock & thigh, stiffness, changes in gait AVN of the knee: <ul style="list-style-type: none">- Acute onset knee px while WB & at night- Typical responses in Hx: osteoporosis or osteopenia, no recent trauma- Physical exam findings: px w/ palpation over medial femoral condyle, decreased ROM AVN of the talus: <ul style="list-style-type: none">- Associated w/ polyarticular disease & trauma- Complaints of px & difficulty ambulating beyond expected recovery time post-trauma
• Diagnosis:	MRI findings: <ul style="list-style-type: none">- Osteosclerotic changes- Decreased bone resorption due to disrupted osteoclast function- Low on T1 (fat is white)- High on T2 (fat is dark)
• Complications:	Postoperative complications: <ul style="list-style-type: none">- Surgical site infection- Prosthesis malfunctions- Neuromuscular compromise



AVN / Osteonecrosis (cont)

- **Management:**
 - Pharmacological therapy in early stages
 - Surgery
 - Exercises to maintain joint mobility & strengthen muscles around
 - Later in therapy implement endurance & coordination training
 - Post-surgery & recovery full conservative care
- **Ddx:**
 - OA
 - Osteoporosis
 - Osteomyelitis
 - Neoplastic bone conditions
 - Inflammatory synovitis
 - CRPS
 - Soft tissue trauma

[link text](#); [link text](#)

Bursitis

• YELLOW

- **Intro:**
 - Swelling or inflammation of a bursa
 - Bursae are found near bony prominences & between bones, muscles, tendons, & ligaments (approx. 150 facilitate MSK movement)
 - Bursitis causes the bursa to enlarge w/ fluid, resulting in pain w/ movement & pressure
 - Not all forms of bursitis are due to 1° inflammation, some result from swelling due to a noxious stimulus
- **Aetiology (risk factors):**
 - Overuse of the joint
 - Repetitive strain: picking up & lifting heavy loads
 - Trauma: falling / bumping against things
 - Pressure: "student's elbow" & "housemaid's knee"
 - Bacterial infection: unattended wound (causing septic bursitis)
 - Other inflammatory disease: e.g. Gout (crystals can form in the bursa & cause inflammation)
 - Immunocompromised individuals: diabetes, rheumatological disorders, alcoholism, or HIV, are at risk of septic bursitis



Bursitis (cont)

- **Pathophysiology:**
 - Bursa is a synovial lining sac
 - Collapses upon itself until triggered, leading to irritation & filling with synovial fluid
 - Px occurs when the inflamed bursa is compressed against bone, muscle, tendon, ligaments, or skin
 - Not all bursitis is linked to an overt inflammatory process
 - Subacromial bursa examination shows ↑ inflammatory mediators
- **Clinical presentation:**
 - ↓ ROM due to px in involved joint
 - Px with AROM, but not w/ PROM in some cases
 - Two forms of bursitis: acute & chronic
 - Acute:**
 - Caused by trauma, infection, or crystalline joint disease
 - Pts experience px on palpation of bursa
 - Px w/ FX, but no px w/ EXT in certain types (e.g. prepatellar & olecranon bursitis)
 - Chronic:**
 - Often results from inflammatory arthropathies & repetitive pressure/overuse
 - Often painless
 - Bursa has had time to expand to accommodate increased fluid, resulting in significant swelling & thickening of the bursa
- **Physical examination:**
 - Evaluate skin for trauma, erythema, & warmth
 - Temperature increase of 2.2°C over affected bursa compared to unaffected indicative of *septic bursitis*
 - Deep bursitis may not show tenderness or obvious skin changes
 - Normal ROM in septic bursitis
- **Diagnosis:**
 - **Plain radiography:** recommended w/ Hx of trauma, concern for foreign body, or fracture causing swelling or px
 - **MRI:** for evaluating deeper bursa
 - **US:** helpful in differentiating cellulitis from infectious bursitis
 - **Bursa fluid puncture:** can rule out infections



Bursitis (cont)

- **Management:**
 - Bursitis w/o infection:**- Most often self-limiting
 - RICE
 - NSAIDs
 - Injections
 - Mobs
 - Gradual ↑ in exercise
 - Immobilising is a risk towards adhesive capsulitis

Septic bursitis:

- Antibiotics
- Aspiration (needle)
- NEVER inject w/ steroids
- Surgical removal of bursa (in case of tuberculous bursitis)
- Surgical incision & drainage

- **Ddx:**
 - OA
 - RA
 - Can mimic other conditions in specific locations (e.g. shoulder - rotator cuff / labral tear)
 - Pathologies can coexist w/ or precipitate bursitis (e.g. gout)
 - Ischial bursitis can mimic sciatica (sitting-induced px distinguishes it from sciatica)
 - Trochanteric bursitis differs from ITB syndrome, w/ tenderness in IT band more distal compared to proximal location of trochanteric bursa
 - Iliopsoas bursitis can resemble arthritis, overuse injuries, synovitis, labral tears, or AVN
 - Knee bursitis typically doesn't cause effusion, aiding in differentiation from other knee pathologies
 - Retrocalcaneal bursitis may initially resemble achilles tendinitis, enthesopathy, px from bone spurs, or plantar fasciitis

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Calcific Tendonitis

• GREEN

- **Intro:**
 - Self-limiting disorder characterised by deposition of calcium in the tendon / muscle
 - Leads to px & reduced ROM



Calcific Tendonitis (cont)

- **Aetiology (risk factors):**
 - 30-50yrs
 - F>M
 - Occupational risk (construction, agriculture, certain sports)
 - Metabolic conditions (e.g. diabetes)
 - Mechanical stress: repetitive microtrauma or overuse of tendons
 - Vascular factors: poor blood supply (reduced clearance of metabolic waste)
 - Genetic predisposition
- **Pathophysiology:**
 - Repetitive trauma → tendon degeneration → calcification
 - Tendon necrosis → intracellular calcium accumulation
 - Active process mediated by chondrocytes arising from metaplasia → calcium deposition
 - Phagocytosis of metaplastic areas reforms normal tendon
- **Clinical presentation:**
 - Px w/ or w/o loss of ROM
 - Stiffness, usually after periods of inactivity (morning)
 - Swelling
 - TTP
- **Physical examination:**
 - Limited ROM
 - Crepitus
 - Muscle weakness
 - Warmth & redness
 - Palpable calcium deposits
- **Diagnosis:**
 - **X-ray:** identify calcifications in the tendon or adjacent soft tissue
 - **US:** visualise extent & characteristics of calcifications & assess tendon thickness
 - **MRI:** may be used to evaluate soft tissue involvement & inflammation
- **Complications:**
 - Chronic px
 - Tendon rupture
 - Compression of adjacent structures
 - Bursitis
 - 2° OA
 - Functional impairment
 - Psychosocial impact
 - Recurrence



Calcific Tendonitis (cont)

- **Management:**
 - Pts w/ chronic calcific tendonitis often don't respond to conservative care
 - Anti-inflammatory NSAIDs (ibuprofen)
 - Injections
 - Surgery
- **Ddx:**
 - Adhesive capsulitis
 - Tendinopathy
 - Bursitis
 - Arthritis
 - Ossifying tendinitis

Osteoarthritis

• GREEN

- **Intro:**
 - Non-inflammatory, degenerative joint disease
 - Characterised by loss of articular cartilage & marginal hypertrophy of bone
 - Accompanied by px & stiffness that is aggravated by prolonged activity
 - Most prevalent type of arthritis
- **Aetiology (risk factors):**
 - Higher age
 - F>M
 - Hx of joint trauma
 - Obesity
 - **1° OA:** most common subset of OA; absence of predisposing trauma or disease
 - **2° OA:** occurrence w/ pre-existing joint abnormality; Ssx
 - Modifiable environmental factors: repetitive movements, obesity, metabolic syndrome, smoking, vitamin D deficiency, muscle weakness, low bone density
 - Commonly affects hands, knees (most common), feet, facet joints, & hips



Osteoarthritis (cont)

- **Pathophysiology:**
 - Multifactorial & involves 3 major processes: mechanical degeneration (wear & tear), structural degeneration, & joint inflammation
 - Overuse & aging of the joint are believed to be the main contributors, but inflammatory processes indicated by ↑ cytokines are also present
 - Firstly, OA involves cartilage damage, including surface fibrillation, irregularity, & focal erosions
 - Then, cartilage damage prompts chondrocyte proliferation, & outgrowths can ossify, forming osteophytes
 - Later, subchondral bone sclerosis & bone cyst formation occur, potentially increasing joint stiffness & px
 - Advanced OA may lead to episodic synovitis, & in rare cases, bony erosions can occur in erosive OA
- **Clinical presentation:**
 - Joint px worse w/ use & improves w/ rest
 - Px peaks in late afternoon or early evening, also present in the early morning
 - Two types of px:**
 - Dull, aching, throbbing px (predictable & constant over time)
 - Intense, unpredictable px for short periods
 - Classified into three stages based on px types:**
 - **Early OA:** sharp, predictable px limiting high-impact activities
 - **Mid OA:** constant px, unpredictable joint px or locking, affecting ADLs
 - **Advanced OA:** constant dull-aching px w/ intermittent intense episodes, limiting recreational activities
 - Additional joint Ssx:**
 - Tenderness, stiffness, crepitus
 - Limited ROM
 - Joint swelling, deformity, or instability



Osteoarthritis (cont)

- **Physical examination:**
 - Bony enlargement (commonly in DIP & PIP joint of fingers & toes)
 - Crepitus
 - Effusions (non-inflammatory)
 - Joint line tenderness
 - Limited ROM due to px, swelling, or joint deformity

Specific bony enlargements:

 - *Heberden's nodes*: posterolateral bony swelling of DIP joints
 - *Bouchard nodes*: posterolateral bony swelling of PIP joints
 - OA involving the base of the thumb is described as a "shoulder appearance" or "squaring"
- **Diagnosis:**

Plain radiographs to grade OA:

 - **0**: no OA
 - **1**: Doubtful narrowing of joint spaces &/or possible osteophytes
 - **2**: Definite osteophytes & possible narrowing of joint spaces
 - **3**: Multiple osteophytes, definite narrowing of joint space & some sclerosis & deformity of bone ends
 - **4**: Large osteophytes, marked narrowing of joint space, severe sclerosis & definite deformity of bone ends
- **Complications:**
 - Chronic px
 - Long-term analgesics use
 - Reduced joint mobility
 - Decreased stability & increased fall risk
 - Joint malalignment
 - Deformity
 - Stress fractures
 - Hemarthrosis
 - Osteonecrosis
 - Joint infection
 - Gout & pseudogout
 - Depression
- **Management:**
 - Exercise program
 - STW
 - Mobs/drops
 - SMT
 - NSAIDs (preferably topical)



Osteoarthritis (cont)

- Ddx:
 - RA
 - Gout
 - Pseudogout
 - Septic arthritis
 - Hemochromatosis
 - Fibromyalgia
 - Lyme disease
 - Ankylosing spondylitis
 - Psoriatic arthritis
 - Neuropathic arthropathy
 - Parvovirus-associated arthritis

[link text](#)

Inflammatory arthropathies

• YELLOW

- Intro:
 - Painful inflammation & stiffness of the joints
 - Inflammatory arthritis is typically associated w/ classic Ssx of inflammation
 - Can have various factors - inc. infectious / non-infectious factors
 - Inflammatory arthritis may or may not be associated w/ systemic features related to the underlying condition causing the inflammation

- Aetiology (risk factors):
 - Increasing age
 - F > M (autoimmune inflammatory arthritis)
 - M>F (gout & seronegative spondyloarthritis)
 - Smoking (strongest environmental factor)
 - Caucasian, FHx
 - W/ juvenile idiopathic arthritis presenting before 10 yrs of age



Inflammatory arthropathies (cont)

- **Pathophysiology:**
 - Varies depending on the underlying etiology
 - External or self-antigens trigger an immune-mediated inflammatory response
 - Inflammatory cells migrate from the bloodstream into synovial membrane
 - Hyperplasia of synovial fibroblasts is associated with inflammatory response
 - Cartilage & bone damage can occur, leading to joint destruction in some cases
 - Infectious arthritis results from the direct invasion of the joint by infectious organisms
 - Infectious agents may trigger an immune response, leading to inflammatory arthritis
 - Autoimmune inflammatory arthropathies involve an interplay of environmental & genetic factors activating the immune system
 - Crystalline arthropathies involve crystals in the synovial acting as antigens, triggering a neutrophil-mediated inflammatory cascade
- **Clinical presentation:**
 - Disease progression can be chronic & progressive, leading to joint damage, deformity, & disability if left untreated
 - Joint px
 - Joint stiffness
 - Joint swelling
 - Warmth & redness
 - Fatigue
 - Malaise
 - Low-grade fever
 - Inflammatory arthritis often follows a pattern of flares & remissions
- **Physical examination:**
 - Inflammatory eye conditions: uveitis or iritis, common in various types of IA
 - Skin rashes: commonly in connective tissue diseases (systemic lupus or psoriatic arthritis)
 - Enthesitis: inflammation in the sites where tendons & ligaments insert into bones (common in seronegative spondyloarthropathies)
 - Dactylitis: swelling of an entire digit (common of certain spondyloarthropathies)
 - Symmetry: common in RA



Inflammatory arthropathies (cont)

- **Diagnosis:**
 - Labs:**
 - Elevated inflammatory markers: erythrocyte sedimentation rate (ESR) & C-reactive protein (CRP)
 - Positive autoantibodies: such as rheumatoid factor (RF) & anti-cyclic citrullinated peptide (anti-CCP) antibodies in RA
 - Plain radiographs:**
 - Initially normal in early inflammatory arthritis
 - May show periarticular osteopenia at the disease progresses
 - Periarticular erosions are seen in inflammatory arthritis like RA
 - Gout erosions are typically juxta-articular/rat-bite erosions w/ overhanging edges
 - Chondrocalcinosis or CPPD deposition can be easily visualised
 - Axial spondyloarthropathies can later show 'bamboo' spine & SIJ fusion & erosions
 - MRI:**
 - Beneficial, especially when radiographs are nondiagnostic
 - More sensitive than plain film in evaluating synovitis, erosions, sacroiliitis
 - Higher sensitivity than X-rays
- **Complications:**
 - Arise from delay in treatment or mass Dx
 - May lead to aggressive & permanent joint damage
 - Associated conditions w/ joint damage: chronic gout, RA, seronegative spondyloarthritis
 - Erosive changes in joints
 - Interference w/ ADLs
- **Management:**
 - Inflammation is reversible, while joint destruction is not
 - Antibiotic therapy
 - NSAIDs
 - Education
 - Manual therapy
 - Diet
 - Lifestyle modification



Inflammatory arthropathies (cont)

- **Ddx:**
 - RA
 - AS
 - Psoriatic arthritis
 - Lupus arthritis
 - Gout
 - Juvenile idiopathic arthritis
 - Reactive arthritis
 - Spondyloarthritis

[link text](#)

Septic arthritis

- **YELLOW** - Same day referral
- **Intro:**
 - Inflammation of joints 2° to an infectious etiology
 - Usually monoarticular, however polyarticular also occurs
 - Although rare, septic arthritis is considered an orthopaedic emergency



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Septic arthritis (cont)

• **Aetiology (risk factors):**

In children:

- Diverse casues
- Staphylococcus is the 1° pathogen
- Kingella king affects children under 2-3 yrs
- Neonates face streptococcus, staphylococcus, gonorrhoea
- Adolescents: gonorrhoea concern
- Salmonella links w/ sickle cell disease
- Prolonged antibiotic use raises fungal infection risk
- Pseudomonas from puncture wounds/drug use
- Hip joint commonly affected in children

In adults:

- Staphylococcus major in adults
- Streptococcus pneumonia significant
- Gonorrhoea causes non-traumatic mono-arthritis
- Fungal/mycobacterial organisms challenging to Dx
- SCJ/SIJ infections involve pseudomonas, common in IV drug abusers
- Damaged joints, especially in RA, prone to infection w/ cartilage damage, effusions, & px

Epidemiology:

- Incidence peaks at 2-3 yrs & elderly
- M>F (2:1)

Risk factors:

- Age >80
- Diabetes mellitus
- RA
- Recent joint surgery
- Joint prosthesis
- Previous intra-articular injection
- Skin infections & cutaneous ulcers
- HIV infection
- OA
- Sexual activity (gonorrhoea)



Septic arthritis (cont)

- **Pathophysiology:**
 - Bacterial invasion of synovial, followed by inflammatory processes
 - Common pathogens change throughout lifetime (viz. aetiology)
 - Damaged joints through RA are highly susceptible to infection
 - Synovium lacks limiting basement membrane → systemic infection can spread to bones
 - Contagious spread from osteomyelitis (hip & shoulder are prone)
- **Clinical presentation:**
 - In children**
 - Local Ssx:*
 - Px, joint swelling, warmth
 - Limited ROM
 - Limp, refusal to use or move the affected joint (pseudoparalysis)
 - Systemic Ssx:*
 - Ill appearance
 - Fever
 - Tachycardia
 - Fussiness/irritability
 - Decreased appetite
 - In adults:**
 - Acute onset monoarticular (large joints) joint px
 - Fever (40-60% of pts), swelling, reluctance or refusal to move the affected joint
 - LL (hip, knees, ankles) commonly affected
 - Knee most affected joint
- **Physical examination:**
 - Palpation may elicit px
 - Limited ROM
 - Effusions are common



Septic arthritis (cont)

- **Diagnosis:**
 - Synovial WBC count
 - ESR & CRP

Imaging

Plain radiographs:

 - May reveal widened joint spaces
 - Soft tissue bulging
 - Subchondral bony changes (late finding)
 - Normal plain radiograph doesn't rule out septic arthritis

US:

 - Useful for identifying & quantifying joint effusion
 - Aids needle aspiration

MRI:

 - Sensitive for early detection of joint fluid
 - Reveals abnormalities in surrounding soft tissue & bone
 - Cartilaginous involvement
- **Complications:**
 - Osteomyelitis
 - Chronic px
 - Osteonecrosis / AVN
 - Leg length discrepancies
 - Sepsis
 - Death
- **Management:**
 - Same day referral to GP
 - Antibiotics
 - Aspiration of joints
- **Ddx:**
 - Infections
 - CPPD
 - OA
 - Fractures
 - AVN/osteonecrosis
 - Other inflammatory arthropathies
 - Systemic infections
 - Tumour

[link text](#)

Tendinopathies

• GREEN

- **Intro:**
 - Tendinopathy is an umbrella term to describe tendon px, w/ unknown cause
 - **Tendinosis** describes the degenerative state of tendons (more applicable)



Tendinopathies (cont)

- **Aetiology (risk factors):**
 - Not fully understood
 - Mechanical stressors, repetitive overloading, or toxic chemical exposure ca initiate tendinosis
 - Age, genetic predisposition, &/or comorbidities can increase susceptibility to healing failure leading to tendinosis
- **Pathophysiology:**
 - Decreased simplistically in 3 stages, but actually occurs on a continuum
 - Stage 1:**
 - Begins w/ tendon experiencing the initial insult, stress, or injury
 - Causes: acute overload, repetitive stress, or chemical irritation
 - Linked to the death of tenocytes (tendon cells)
 - Stage 2:**
 - Characterised by failed healing of the tendon
 - Unclear cause, but believed to result from an altered tendon environment (steroids/NSAIDs may alter natural healing cascade)
 - Improper cell recruitment & a cascade of healing issues may occur
 - Stage 3:**
 - Characterised by apoptosis (death) of cells, disorganisation of the matrix, & neovascularisation
 - Pts often present at this stage, experiencing mechanical weakness or increased px
 - Neovascularisation theorised to supply neonerves, contributing to px (neurogenic inflammation)
- **Clinical presentation:**
 - Identify potential stressors
 - Impact on ADLs
 - Recent changes in medication, inc. antibiotics (may influence treatment)
- **Physical examination:**
 - Tenderness
 - Swelling
 - Other abnormalities
 - Special tests based on the specific tendon
- **Diagnosis:**
 - Labs: CRP & ESR (aid in identifying inflammatory processes)
 - **X-ray:** if bone injury is suspected
 - **US:** increased spacing of fibrillar lines, reduced echogenicity, tendon thickening, neovascularisation (via colour Doppler)
 - **MRI:** valuable for evaluating tendinosis



Tendinopathies (cont)

- **Complications:**
 - Tendon rupture (if untreated)
 - Contractures of the tendon, w/ reduced tendon liability
 - Tendon adhesions
 - Atrophy of muscles
 - Loss of functionality, even up to & including disability
- **Management:**
 - Healing as long as 3-6 months
 - STW
 - SMT
 - Mobs/drops
 - RICE
 - NSAIDs
- **Ddx:**
 - Acute compartment syndrome (ACS)
 - Ankle injury
 - Bursitis
 - Carpal tunnel syndrome
 - Gout & pseudogout
 - Hand infections
 - Reactive arthritis
 - Rotator cuff injuries
 - Soft tissue knee injury

[link text](#)

LL nerve entrapments / Tunnel syndromes

• GREEN

- - Umbrella term for conditions characterised by compression or entrapment of nerves, blood vessels, or tendons within anatomical tunnels in the LL

Intro: - Can lead to various Ssx inc. px, numbness, tingling, & weakness, affecting the function & sensation of the LL

Common LL tunnel syndromes inc.:

- Sciatica
- Tarsal tunnel s.
- Common peroneal n. entrapment
- Anterior compartment s. (ACS)
- Popliteal artery entrapment s. (PAES)



LL nerve entrapments / Tunnel syndromes (cont)

- **Aetiology (risk factors):**
 - Age-related degeneration
 - Obesity
 - Sedentary lifestyle
 - Trauma
 - Arthritis
 - Tumours
 - Diabetes
 - Pregnancy
 - Tight clothing
 - Occupation-related factors
- **Pathophysiology:**
 - Increased pressure within confined spaces leads to nerve or vascular compression
 - Inflammation, fibrosis, or space-occupying lesions contribute to compression
 - Repetitive use or trauma can exacerbate Ssx
 - Individual anatomical variations may predispose individuals to specific tunnel syndromes
- **Clinical presentation:**
 - Px or discomfort in the affected LL
 - Numbness or tingling
 - Weakness in the muscles of the affected area
 - Altered sensation or hypersensitivity
 - Px may worsen w/ specific activities or movements
 - Swelling in the affected area
 - Impaired coordination or balance
- **Physical examination:**
 - Tenderness or px upon palpation of the affected nerve pathway
 - Muscle atrophy in severe or chronic cases
 - Limited ROM in affected joint
 - Positive **Tinel's sign**: tingling or px elicited by tapping on the nerve
 - Positive **Nerve tension tests**: straight leg raise / slump test to assess nerve mobility & irritation
 - Changes in reflexes, such as diminished or exaggerated reflexes
- **Diagnosis:**
 - **MRI**: provides detailed soft tissue visualisation, helping identify nerve compression & surrounding structures
 - **US**: useful fro dynamic imaging, assessing nerve movement during various joint positions
 - **CT**: may be used in specific cases, especially fro bony abnormalities contributing to nerve compression



LL nerve entrapments / Tunnel syndromes (cont)

- **Complications:**
 - Nerve compression & damage
 - Ischemia (reduced blood supply)
 - Muscle atrophy & weakness
 - Chronic px
 - Motor dysfunction
 - Sensory abnormalities
 - Functional limitations
 - Trophic changes (skin, hair, nail condition)
 - Risk for 2° injuries (risk of falls)
 - Psychosocial impact
 - Surgical complications
- **Management:**
 - Rest & activity modification
 - NSAIDS & neuropathic px medication
 - Injections
 - Occupation & ergonomic modifications
 - Weight management
 - Surgery
 - Pt education
 - SMT & STW
 - Strengthening & mobilising
- **Ddx:**
 - Peripheral neuropathy
 - Lx radiculopathy
 - MFPD
 - Complex regional px s. (CRPS)
 - Tarsal tunnel syndrome
 - Morton's neuroma
 - Stress fractures
 - Compartment s.
 - MSK injuries (ligament sprains, tendonitis etc.)
 - Inflammatory arthropathies (RA etc.)
 - Infectious causes (cellulitis, osteomyelitis etc.)
 - Neoplastic conditions (tumours)
 - Systemic conditions (diabetes, hypothyroidism etc.)
 - Iliotibial band s.
 - Popliteal entrapment s.

[link text](#)

Stress fractures

- **RED**
 - - Fractures that occur due to an imbalance between the bone strength & the chronic mechanical stress placed upon the bone (→ overuse s.)
- Intro:** - *spondylolisthesis* is also classified as stress fracture



Stress fractures (cont)

• **Aetiology (risk factors):** - Abrupt increase in activity or training patterns

Intrinsic factors:

- Poor physical conditioning
- F>M
- Hormonal disorders
- Menstrual disorders
- Poor bone density
- Reduced muscle mass
- Genu valgum kness
- Short leg

Extrinsic factors:

- High-impact sports
- Abrupt increase in physical activity
- Irregular or angled running surface
- Poor footwear
- Running shoe wear older than 6 months
- Vitamin D & calcium deficiency
- Smoking

Common risk factors:

- Abrupt increase in activity
- Females
- PMHx of stress fractures

Common sites:

- Metatarsals, tibia, tarsals, femur, fibula, & pelvis (in decreasing order)
- Pelvic & metatarsal stress # common in females
- UL stress # rare but reported in gymnasts, weightlifters, & throwing sports

Sports-specific risks:

- Runners: tibia & metatarsal stress # (F may also experience pelvic stress #)
- Long-distance runners: association w/ femoral neck & pelvic injuries
- Hurdlers: patella #
- Gymnasts, female soccer players, certain American football positions & weightlifters: increased risk of spondylolysis (unique stress # related to repeated hyperEXT of the spine)



Stress fractures (cont)

- **Epidemiology:**
 - Stress # are 20% of all sports injuries
 - Common along military
 - Runners (16% of injuries): tibia (23.6%), tarsal navicular (17.6%), metatarsals (16.2%), femur (6.6%), pelvis (1.6%)
 - F>M
 - Neuromuscular factors play a role (muscle loss / fatigue decreases ability to absorb forces)
 - Rapid weight loss, particularly muscle loss, is associated w/ stress #
 - Overtraining / relative energy deficiency s. contribute (esp. in females w/ disordered menstruation & hormonal imbalances)
 - Male endurance athletes w/ high training volumes & restricted calorie intake (low testosterone), leading to osteoporosis & stress #
- **Pathophysiology:**
 - Wolff's law: applied force on a normal bone leads to remodelling for increased strength
 - Osteocytes: most common bone cells, orchestrate osteoclastic & osteoblastic functions
 - Osteocyte's dendritic network: responds to biomechanical stress, secretes mediators regulating bone activities
 - Cycling loading impact: compromises osteocyte signaling, hinders physiological repair mechanisms
 - Repetitive loading effect: stimulates osteoclasts for faster resorption, outpacing osteoblasts in new bone formation
 - Normal remodelling cycle: takes 3-4 months
- **Clinical presentation:**
 - Insidious onset of pain after activity
 - Progressive increase in pain duration post-exercise
 - Pain present upon waking up following training activity
 - Important factors in Hx: recent changes in training, nutrition, intrinsic & extrinsic risk factors, PMHx, & medications



Stress fractures (cont)

- **Physical examination:**
 - Focal TTP
 - Occasional edema at the suspected stress fracture site
 - Limited clinical diagnostic tests
 - Px percussion & vibration
 - Commonly used tests: "hop test" & fulcrum test
 - Bony tenderness on palp, often at distal to middle third junctions of the tibia or over 3rd & 4th metatarsal shafts
- **Spondylolysis & spondylolisthesis:**
 - Require high index of suspicion
 - *Spondylolysis* may be asymptomatic & found incidentally on Lx films
 - Lx extension increases px in *spondylolysis*, Stork test or single leg hyperextension test is common
 - *Spondylolisthesis* occurs when pars defect does not heal, leading to anterior migration of the vertebral body
- **Diagnosis:**
 - First is plain radiography
 - CT
 - MRI
- **Complications:**
 - Small for low-risk stress fractures
 - Occasional residual px
 - Higher for high-risk stress fractures
 - More likely to progress to non-union & thus require surgery
- **Management:**
 - NSAIDs
 - Splinting
 - Resting / non-WB
 - Supplementation of vitamin D, magnesium
 - Potential surgery
 - Post-surgery / healing rehab
 - Strengthening / mobs



Stress fractures (cont)

- Ddx:
 - Cellulitis
 - Osteomyelitis
 - Tendonitis
 - Tendinopathy
 - Exertional compartment syndrome
 - Tumours (benign & malignant)
 - Nerve entrapment
 - Arterial entrapment
 - Coagulation disorders
 - Compartment s.
 - Neuropathic px
 - CRPS

link text



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