Compartment Syndrome

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Historical Perspective

- **Volkmann (1881)** – related hand contracture (cx) to ischemic process in forearm following supracondylar humerus fx attributed to tight dressing, venous stasis with arterial insufficiency
- **Petersen (1888)** – described surgical tx. with release of contracted scarred tissue with some improvement in function
- **Hildebrand (1906)** – coined term “Volkmann’s ischemic cx.” to describe the end point of an untreated compt. Syndrome. Ist to suggest that elevated tissue pressure might be casually related to the ischemic cx.
- **Thomas (1909)** – extensive review of 112 cases (107 in UE) of ischemic cx. Fractures m/c factor in most end-stage ischemic dx. Other causes included arterial injury, emboli, tight bandaging
- **Rowlands (1910)** – Ist to suggest that reperfusion following ischemic event could result in post-ischemia congestion and edema of muscle and nerve and lead to an acute compt. Sx.
- **Bardenheuer (1911)** – Ist to report on fasciotomy of the forearm
- **Murphy (1914)** – Ist to suggest prophylactic fasciotomy
- **Griffiths (1940)** – described painless onset, then pain with passive stretch, pallor and pulselessness
- **WWII – Ischemic cx.** From compt. Sx. Secondary to fractures from high velocity GSW. Many still thought arterial spasm/damage as primary cause. Exploration frequently led to inadvertent fasciotomy and improvement/reversal of impending compt. Sx.
- **Chandler/Knapp (1967)** – Vietnam – fasciotomies routinely performed after vascular repair
- **Seddon (1966)** and Kelly **Whitesides (1967)** – described 4 separate compartments in the leg and hence the need to decompress more than just the anterior compt.

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Etiology

- Condition in which increased tissue pressure within a limited space compromises the circulation and the function of the contents of that space.
  - This results in reduced capillary blood perfusion and worsening neuromuscular function.
- Most common causes include:
  - Fractures
  - Crush injuries
  - Prolonged limb compression (i.e., drug overdose)
  - Arterial injury (bleeding into compart., inadequate perfusion with post-ischemic swelling after circulation restored)
  - Burns
  - Snakebites

Pathophysiology

- Decreased Perfusion
- Increased pressure
- Muscle/nerve ischemia
- Edema/Hemorrhage
- Capillary endothelium and basement membrane damage
- Increase interstitial fluid

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Acute Compartment Syndrome (ACSx)

- Most common in the leg as the result of tibia fractures...
  - Other less common causes include muscle rupture, crush injuries, and circumferential burns
  - Direct pressure, circumferential dressings and casts can also significantly increase compartment pressures...
  - Bi-valving a cast can reduce pressures by up to 85%
- Fortunately uncommon in athletic population
- Resting pressures of greater than 30mmHg represent a level that can be associated with interruption of the capillary circulation and thus can lead to muscle and nerve ischemia
- Once recognized, treatment is emergent (fasciotomy)

Indications for Surgical Decompression of ACSx (Rorabeck 1983)

- Absolute
  - Clinical signs of ACSx with motor or sensory loss
  - Elevated tissue pressure greater than 35 mm Hg or a Delta P within 30 mm Hg of diastolic
  - Interrupted arterial circulation for greater than 4 hrs
- Relative
  - Circumferential full-thickness burns
  - Muscle overuse syndrome
  - Limb compression syndrome
Chronic Exertional compartment syndrome (CECSx) or Exercised Induced Compartment Syndrome (EICSx)

- Exact underlying pathophysiology unclear, however it is clear that abnormal pressures are present
  - Significant pressure elevations of up to 80mmHg and beyond can occur with muscle contractions alone
  - Muscle weight can increase by as much as 20% owing to increased tissue perfusion with exercise
    - This increase in muscle weight and size can lead to increased intracompartmental pressure which leads to transudation of fluid into interstitial space.
    - As tissue pressures approach the level of diastolic blood pressure, the micro-circulation is impeded
  - Has been reported in hand, forearm, leg, thigh, gluteus, and foot.
    - In rare untreated case, an EICSx may progress to an ACSx and can result in myonecrosis, causing release of myoglobin into vascular circulation which can lead to renal failure.
  - Tx consists of prompt hydration, restoration of fluid deficits, and concomitant diuresis and bicarbonate therapy/spironolactone...
  - M/C compts affected are the lower leg, thigh, and forearm

CECSx

- History: usually seen in runners who are completely asx in the off season but gradually develop an aching in the lower leg with training
  - Pain initially dull and occurs following runs
  - As sx progress the pain may persist into next day
  - Paresthesias may be seen in plantar aspect foot with deep posterior compt and dorsum foot with anterior compt sx
  - Most frequent location in leg are anterior and posterior which acct for up to 80% of all cases

CECSx

- The average delay in treatment from the onset of sx is 22 months
- Gold standard for clinical dx is still elevated resting intracompt pressure post-exercise
- Normally pressures will return to normal within 2 minutes with rest
  - Post exercise delay is prolonged in CECSx
  - Remember that pressures are affected by knee and ankle positioning so standardize approach
- Bone scan, x-rays, and CT scans are useful only in ruling OUT associated injuries or dx
- MRI may show prolonged relaxation in T1 and T2 imaging
- US may show differences in compt with between joggers and non-joggers but are not necessarily correlated with sx

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EICsx
- Elevated comp. resting pressure both before and after exercise
- Hallmark is delayed return to normal tissue pressure after exercise
- Rorabeck and colleagues include a resting intramuscular pressure above 10 mmHg and abnormally elevated tissue pressure in excess of 15 mm Hg measured 15 minutes after exercise
- Pedowitz et. al. include any one of the following...
  - Pre-exercise pressure greater than 15 mm Hg
  - Post-exercise pressure at 1 minute greater than 30 mm Hg
  - Post-exercise pressure at 5 minutes greater than 20 mm Hg
  (Note that these are static not dynamic readings)

CECSx (cont’d)
- Roscoe, Roberts, Hulse AJSM 2014 suggested new and improved Dx criteria...
  - Level 2 Cohort study
    - 40 pts... 20 controls and 20 with symptoms of anterior compt sx.
    - Rigorous inclusion criteria to r/o other diagnoses
    - Treadmill wearing identical footwear
    - CONTINUOUS monitoring pre, during, and post exercise
      - Equal and normal pressures supine, but higher immediate pressures immediately standing at rest with greatest difference corresponding to period of maximal tolerable pain (68.7 mm Hg vs 114 mm Hg)
      - Recommended using a cutoff of 105 mm Hg in phase 2 with better dx accuracy than Pedowitz criteria (sens 63%, specificity 95%)
      - Shorter subjects higher incidence than taller ax controls
      - Elongated stride length an forced changes to walking and running biomechanics also noted

Differential Diagnosis of chronic leg pain in the athlete
- Bone/periosteum
  - Medial tibial stress syndrome (“shin splints”)
  - Stress fracture
- Vascular
  - Popliteal artery entrapment syndrome
  - Intermittent claudication
- Nerve entrapment
  - Peripheral or spinal/radiculopathy
- Knee or Hip abnormality (especially young athletes)
- Muscle/tendon
  - Chronic Exertional Compartment Syndrome
  - Muscle strains
  - Tendinitis, tendinosis
  - Neoplasm
  - Infection

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Medial Tibial Stress Syndrome
- Hx – variable but usu occurs late in a season after prolonged activity
- PE – pain along the posteromedial border of the distal 1/3 of the tibia
- Neurologic and Vascular exam normal
- Dx studies – Xrays usu normal but Bone Scan usu longitudinal uptake along distal 1/3 of tibia

Stress fractures
- Hx – sudden increase in activity, M/C seen in military recruits, females with menstrual disorders, previous hx stress fx.
- PE – focal tenderness, warmth, occas swelling. Usually midshaft or proximal (occas distal in runners)
- Dx studies – Xrays early may be nl but bone scan and MRI very sensitive

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Nerve entrapment
- Hx – usu pain brought on by activity and exacerbated by continued exercise
- PE – able to localize along distribution
- PE – pain with direct compression
- Dx studies – Xrays normal, EMG may be useful.
- Dx injection with anesthetic helpful
- MRI to rule out mass effect or tumor

Popliteal Artery Entrapment Syndrome
- Caused by congenital anomaly where the artery runs in abnl course in the popliteal fossa usu medial gastro
- M/C seen males under 30, usu unilateral
- PE at rest usu normal.
- Palpat pulse with ankle in passive DF or active PF with knee in extension to place tension on gastro leading to extrinsic compression of popliteal artery
- Dx studies – x-rays and bone scans usu normal, MRI or MRA as screening test then if positive consider arteriogram with limb in position of provocation.
Incidence of causes of chronic leg pain in the athlete

- CECSx 34%
- Stress fractures of tibia 25%
- "Shin splints / medial tibial stress syndrome 13%
- Popliteal artery entrapment syndrome
- Nerve entrapment (peripheral or central/spinal)
- Muscle herniation (seen in approx 40% of CECSx vs. 5% in controls)
  - Seen in up to 80% anterior compartment
  - Referred from Hip or Knee

ACSx Diagnosis

- Passive muscle stretch testing should be directed to each compartment.
- It is a clinical dx. Based on muscle and nerve ischemia
  - Muscles can tolerate ischemia for 4 hours, but by 6 hours the effect is uncertain and after 8 hours the damage is irreversible
  - Peripheral nerves can survive 4 hours of ischemia with only neuropraxic damage but suffer irreversible damage after 8 hours
- Unresponsive/obtunded pts. and those with a CNS/PNS deficit warrant a high index of suspicion with liberal compartment pressure measurements
ACSx Diagnosis

- Manifestations: Progressive, persistent pain out of proportion to the primary problem/injury, pain with passive muscle stretch, hypesthesia/paresthesia in distribution of traversing nerve, muscle weakness, “pig swollen”.
- Pallor/Pulselessness are usually NOT seen in comp. Sx. (less than 10%) but rather are indicative of arterial injury
- If it looks like a brick, and feels like a brick, then it’s probably a brick...

Measurement of Intracompartmental pressure

- Indicated whenever the diagnosis cannot be made or ruled out by clinical findings alone. Especially valuable in uncooperative/unresponsive pts and those with neurovascular injury
- Normal comp joint pressure should be in the range of 0 to 8 mmHg
- Compt pressures vary with proximity to fracture site (highest nearer the fracture)
- Intrinsic muscle compjs (hand/foot) readings are technically feasible but many times unreliable. Normal intrinsic pressures are stated to be less than 15mm Hg
There are many reported techniques, including needle, continuous infusion wick catheter, slit catheter, and Stryker or STIC.

Slit catheter technique – (Rorabeck et al.) later refined by Mubarak et al. Five 3mm slits cut in tip of polyethylene tubing to maintain continuity between tissue fluids and saline within the catheter without saline injection or flushing. Less prone to coagulation problems.

Stryker (STIC) technique – (Whitesides)– hand held pressure monitor which has an auto-zeroing device and side ported needle, sterile disposable.

Most impt info – Where is it kept?

There is not an absolute increased compartment pressure that warrants fasciotomy.

When the pressure approaches 30 to 45mm Hg, or within 30 mm Hg of the diastolic pressure, with concordant physical exam findings, then decompressive fasciotomies should be performed.

Don’t forget that you can use the normal side for comparison if necessary…

Also don’t forget that the position of the limb affects pressure (i.e., knee flexed or extended, active muscle contraction, passive flexion or extension).
Treatment

› In general, for ACC... fasciotomy should be performed whenever there is a high clinical suspicion by examination, regardless of pressure...

› Treatment for CECSx is also surgical
  • Should have at least 90% probability of significant improvement if not complete recovery
  • Many of cases of “failure” due to inadequate fasciotomy or missed dx / wrong compt
  • Anterior or lateral compt involvement better outcomes for some reason compared to posterior compt

Treatment

› Acute fasciotomy can be thru single incision laterally or combined medial and lateral
  • Easier to get to deep posterior compt thru combined approach
  • These are performed open under direct vision with secondary skin closure once swelling subsides
  • CECSx may do well with subcutaneous fasciotomies with primary skin closure
  (exception are those pts that have an associated muscle herniation (usually located at the site of penetration of the superficial peroneal nerve)... do those under direct visualization
  • Fascial defects should never be closed

Pertinent Anatomy

› 4 well described fascial compts within leg: Anterior, Lateral, Superficial posterior and Deep posterior
  • The tibialis posterior behaves as if it were within it’s own compt and has been shown to be vulnerable itself to a chronic compt syndrome
  Lying within the 4 major compts are 4 major nerves with sensory components...
  › Superficial posterior – Sural nerve
  › Deep posterior - tibial nerve
  › Anterior – deep peroneal
  › Lateral – superficial peroneal
Post operative rehabilitation

- Outpt surgery with subq fasciotomy
- Soft dressing and elevation for 24-48 hours then WBAT and d/c crutches within 5-7 days
- Gentle stretching/strengthening
- Return to running activities around 2 weeks
- Subsequent rehab much like that used for medial tibial stress syndrome
- Return to sports when no pain, full return strength/flexibility...

Thank You 😊

References

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