

Introduction

Compartment syndrome (CS): intra-compartmental pressures exceed to a point where arterial, venous and lymphatic circulation of local tissues, muscles and nerves is compromised¹

- Most common after a traumatic injury². Usually occurs in the leg or forearm and less commonly in the thigh³
- Thigh compartment syndrome (TCS) is rare due to its larger size and more compliant borders. If affected, it usually occurs in anterior compartment over others⁴

Rhabdomyolysis^{5,6}: muscle damage resulting in pain and the release of muscular cell contents into the circulation. Common etiologies include drugs/alcohol, crush injuries, medications and muscle disease

- Usually presents with triad of myalgia, weakness, tea color urine
- A high creatinine kinase, five times the normal limit (198 u/L) is diagnostic and myoglobinuria is pathognomonic

Patient Presentation and Work Up

38 yo man presented to ED at 4:40am complaining of sudden onset **severe left groin and anterolateral thigh pain** that woke him from sleep. Also reported numbness radiating down his thigh.

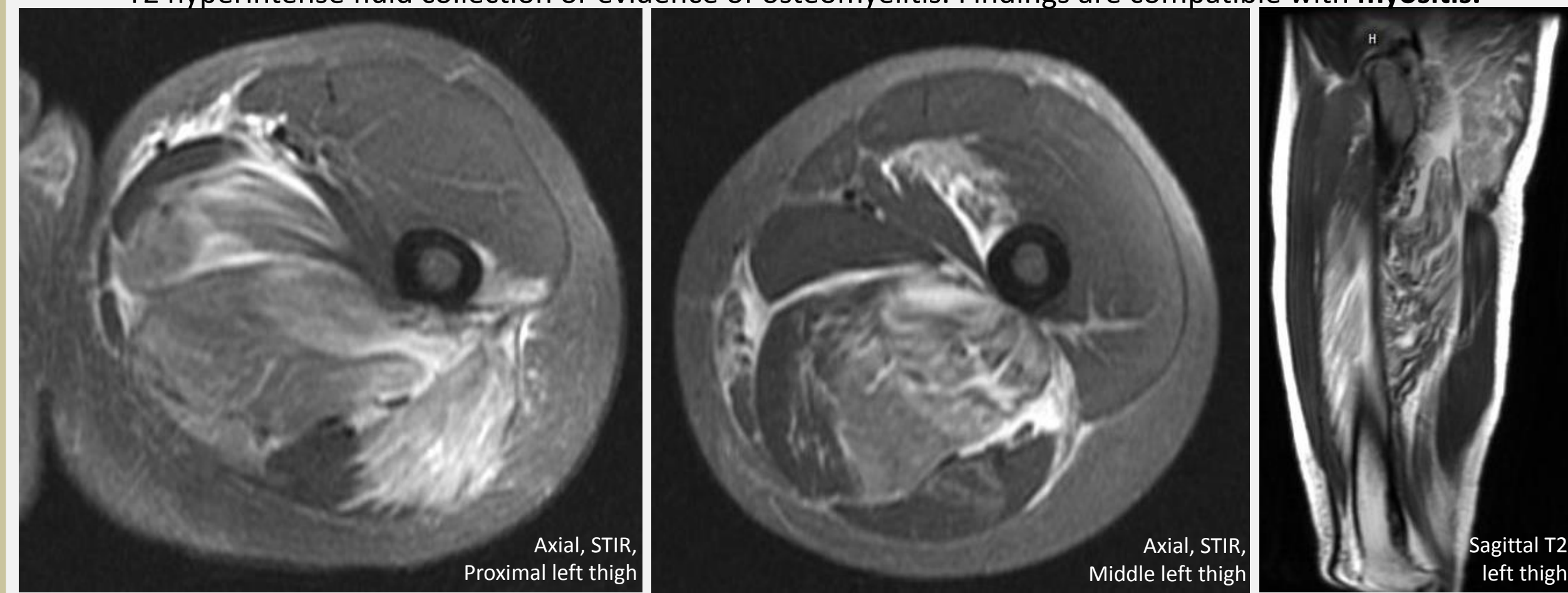
- No recent trauma or muscle overuse. Diarrhea for past 1 month.
- Reported past IV opioid abuse but had been clean for 8 months, no alcohol use but smoked 15 cigarettes/day.

ED Exam: left thigh tender to palpation, no swelling or firmness. Diffuse pain with ROM, reduced EF on echo, negative LE dopplers

• **Vitals:** 106/40 mmHg, 125 bpm, 98.5°F, 18 breaths/min, 97% on RA
 39 | 16 | 289 | 137 | 100 | 30 | U/A: Amber, +blood, -RBCs. **CPK of 36,954 iu/L**
 49.2 | 7.3 | 20 | 2.7 | Neg tox screen and BAL. Pulses, cap refill intact

- Pt was given IV antibiotics, fluids, electrolyte repletion and Lasix

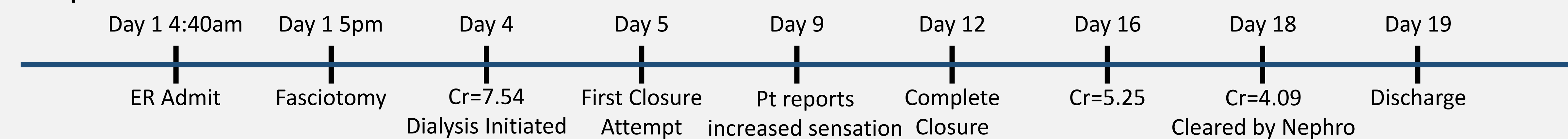
MRI: Extensive fluid signal within musculature of left thigh with interfascial edema. Most prominent in medial superior and posterior compartments. Fluid noted surrounding sciatic nerve along its course. No drainable T2 hyperintense fluid collection or evidence of osteomyelitis. Findings are compatible with **myositis**.



Orthopedic Exam at 3:30 pm: extremely firm and tender posterior compartment, soft medially and anteriorly. Edema from the groin to knee. Decreased sensation along left lateral thigh, entire leg and foot. Muscle strength in Hamstrings and Quads 4/5, Plantarflexion 3/5 and Dorsiflexion and Extensor Hallucis 0/5. Repeat CPK was 127,503 iu/L.

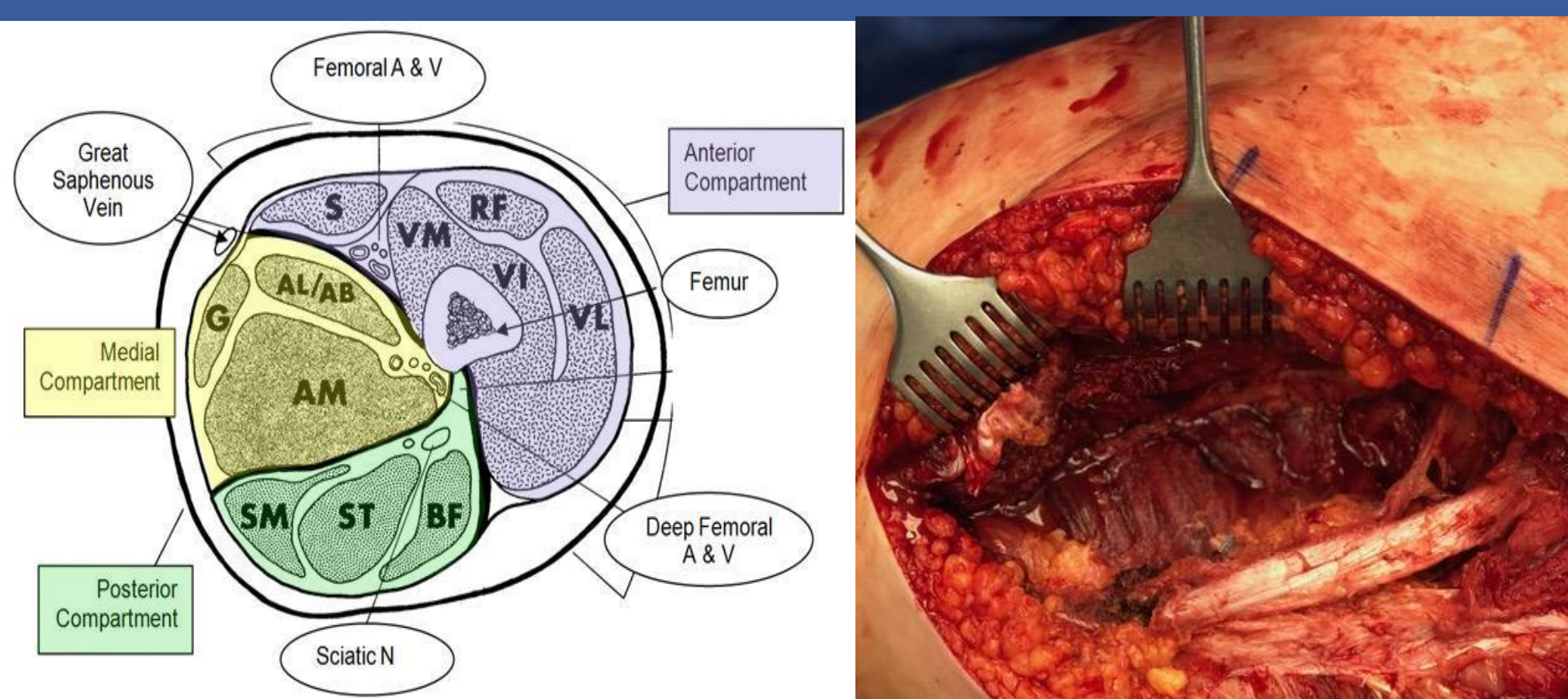
- **Stryker Intra-Compartmental Pressure Monitoring System** found Pressures of **70 and 75 mmHg** in two different posterior locations.

Hospital Course



Surgical Intervention

- **Single incision fasciotomy approach:** started at greater trochanter and carried distally
- **Anterior Decompression:** iliotibial fascia released anterior to the lateral intermuscular septum
- **Posterior Decompression:** vastus lateralis retracted anteriorly to expose and release intermuscular septum
- Gluteus fascia was noted to be extremely tight so it was also released
- **Iliotibial band fascia was pie crusted** to prevent laceration of the underlying vastus lateralis
- There was no gross purulence or necrosis in tissue. No constriction/collection around sciatic nerve
- Muscle **biopsy** and tissue **cultures** were taken
- Wounds were copiously irrigated with saline and then partially closed with placement of a wound vacuum



Patient Progression

- Reported significant pain relief after fasciotomy
- Developed **Acute Renal Failure** and required catheter placement for hemodialysis (thrice/wk) by Day 4
- 1st closure attempt limited by **persistent tissue tension**.
- Blood and tissue cultures negative for bacterial growth
- Eventually kidneys began recovering around Day 16
- **10 week follow up:** patient reported no pain. Scar was well healed, sensation was intact and equivocal, muscle strength was 5/5 in left LE. No longer needed dialysis.

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Discussion

What Came First? We propose the Rhabdomyolysis

- Serum CK rises within 2-12 hrs of muscle injury⁶. Presenting CK was 185x the normal limit, indicating it had been increasing beforehand
- CK continued to rise demonstrating ongoing muscle damage possibly due to the increasingly building pressures and progression of CS.
- A *'second wave phenomenon'* of persistently elevated CPK is an indicator of CS development in setting of rhabdomyolysis^{5,6,7}
- Pt developed characteristic CS signs³ of pain out of proportion, paralysis, paresthesias & firmness to palpation hours after admission

How Low is Too Low?

- A delta pressure less than 30 mmHg or a direct intra-compartmental pressure greater 30 mmHg is the gold standard for CS diagnosis⁸
- Pain develops and capillary blood flow becomes compromised at compartment pressures within 20-25 mmHg of MAP⁹
- Therefore hypotension may cause tissue compromise to occur prior to an intra-compartmental pressure of >30mmHg

To Loop or Not to Loop?

- Loop diuretics increase urinary flow and reduce precipitation of myoglobin, but also acidify urine and exacerbate its nephrotoxicity¹⁰
- pH of 6.5 is recommended minimum to reduce acidosis induced damage^{10,11}. Pt urinary pH six hours after admission was 6.0
- Lasix was given in ED to help to address the reduced EF, but it may have contributed to worsening of patient's kidney injury

Proposed Pathogenesis

- In light of one month history of diarrhea we propose a viral myositis as initial trigger. One of two muscle biopsies showed focal necrosis and chronic inflammation, consistent with myositis¹². Influenza is the most common viral cause, but enteroviruses have also been linked¹³

Why is this unique?

- Systematic review² found that 90% of TCS cases are from trauma.
- Of an analysis of 35 TCS cases¹⁴, only 5 are of solely posterior compartment. Only two other cases^{15,16} of posterior TCS also had sciatic nerve palsy; one had ARF but underlying trigger was alcohol¹⁶.

Conclusions

- Initiate rapid treatment for both rhabdomyolysis and CS to avoid dire outcomes of chronic kidney disease, neuro deficits, amputation or death¹⁷
- Avoid loop diuretics in rhabdo; exercise caution, monitor closely if used¹⁸
- CS can occur in posterior thigh in the absence of trauma. Consider the diagnosis when patient presents with an acute sciatic nerve palsy