59 y.o. male with right thigh pain

Olesya Krivospitskaya, MD

Endorama 02/28/13

History of past illness:

- 59 y.o. male presented with R thigh pain for two weeks. He denied any trauma or injury to his thigh. Unable to bear weight and needed to use a walker. Denied any fevers, chills. Denied any prolonged immobilization of his leg.

Past medical history:

- DM2 (history of DM2 for at least 15 years, has been on insulin 70/30 since he was diagnosed, did not check his blood sugars at home, did not have glucometer)
- HTN
- s/p OHT in 07/2003 secondary to end stage CHF related to ischemic cardiomyopathy
- Anxiety
- Obesity

- Medications: amlodipine-benazepril, aspirin, lasix, cellcept, pravastatin, prednisone, tacrolimus, insulin 70/30 35 units BID.

- Family history: No diabetes or thyroid problems.

- Social history: lives by himself, mentally retarded, on disability, his mom comes twice a day to give him his insulin. Does not smoke, drink or use any illegal drugs. Diet: mostly junk food (potato chips, pasta, pizza, regular pop). Does not exercise.

Review of systems:

- Constitutional: No fevers. No weight loss. No fatigue.
- HEENT: No vision changes. No hoarseness. Neck: No neck swelling or pain.
- Cardiovascular: No chest pain. No palpitations.
- Respiratory: No dyspnea. No orthopnea.
- Gastrointestinal: No diarrhea. No constipation.
- Musculoskeletal: **+ R thigh pain for 1 week. + Mild edema of R thigh.**
- Skin: No rash. No skin changes. No hair loss.
- Neurologic: No tremor. No headache. No weakness.
- Psychiatric: No depression. **+ Anxiety. + Mental retardation.**
- Endo: No polyuria. No polydypsia.

Physical exam:

- Constitutional: Patient appears well-developed, well-nourished, in no acute distress.
- Eyes: Conjunctivae are not injected. Sclerae anicteric. Pupils are equal, round, and reactive to light. Extraocular movements are intact.
- ENT: Mucous membranes moist.
- Neck: Supple. No thyromegaly or nodules palpated.
- Cardiovascular: Regular rhythm and rate. No murmurs appreciated. Intact distal pulses.
- Respiratory/Chest: Normal respiratory effort. No wheezes or crackles.
- Gastrointestinal/Abdomen: Normoactive bowel sounds. Soft, nontender, nondistended.
- Musculoskeletal/extremities: No peripheral edema. **R thigh appears swollen, tender to palpation posteriorly.**
- Neurological: Alert and oriented to person, place, and date. Normal deep tendon reflexes.
- Skin: Skin is warm and dry. No acanthosis nigricans noted.
- Psychiatric: **Tearful during the exam.**
- Vitals: BP 134/86, Pulse 97, Temp 36 °C, Resp 18, Ht 190.5 cm, Wt 124.2 kg, BMI 34.22 kg/m2, SpO2 98%

Differential diagnosis:

- Pyomyositis
- Spontaneous gangrenous myositis
- Clostridial myonecrosis
- Necrotizing fasciitis
- Venous thrombosis
- Intramuscular hematoma
- Calciphylaxis
- Neoplasm

Labs:

136	103	16	173
3.6	25	1.1	

	10.6	
8.9		230
	32.8	

Ca 8.9 (8.4-10.2 mg/dL)
Mg 1.7 (1.6-2.5 mg/dL)
Phos 3.3 (2.5-4.4 mg/dL)

HA1C 9.6%

LFTs:
Total Protein 7.6 (6-8.3 g/dL)
Albumin 4.0 (3.5-6 g/dL)
Total Bilirubin 0.6 (0.1-1 mg/dL)
Alk Phos 99 (30-120 U/L)
AST 14 (8-37 U/L)
ALT 10 (8-35 U/L)

CK 550 (9-185 U/L)

ESR 67 (0-28 mm/hr)

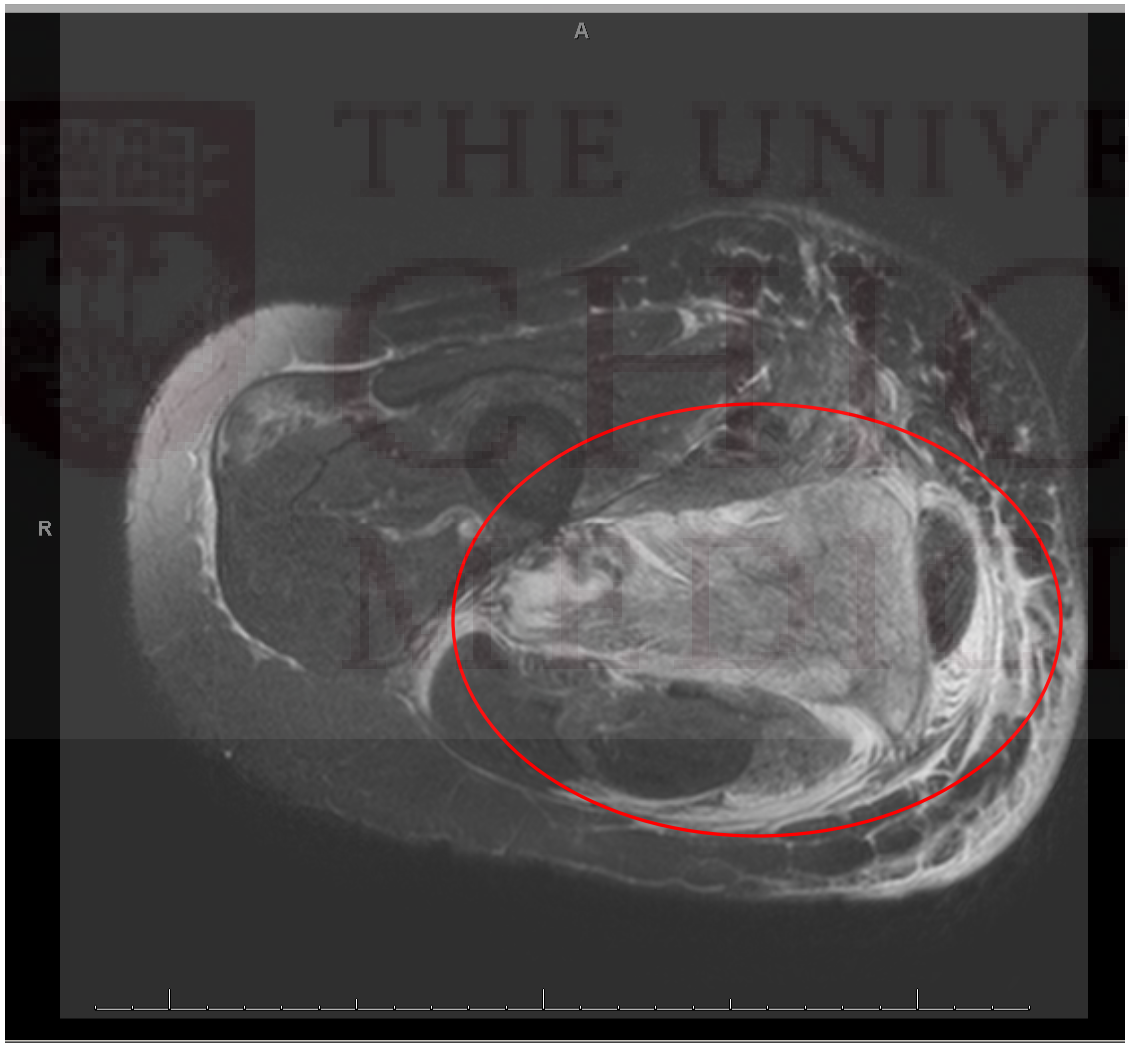
TSH 1.36 (0.3-4.0 mcU/ml)

Free T4 1.6 (0.9-1.7 ng/dL)

Blood cx: no growth in 6 days

Doppler US of lower extremities: No evidence of DVT or venous obstruction in the right and left lower extremity

MRI:



Diabetic myonecrosis, most severely affecting the right adductor magnus muscle adductor magnus muscle, with areas of nonenhancing tissue. No discrete fluid collection is seen to suggest abscess.

- What is diabetic myonecrosis?
- What is the diagnosis?
- How it should be managed?
- How it should be prevented?

- Spontaneous ischemic necrosis of skeletal muscle, unrelated to atheroembolism or occlusion of major arteries

- Muscle infarction is caused by vascular disease such as arteriosclerosis and diabetic microangiopathy

- Two main hypothesis:

- There is severe distal peripheral vascular disease in the muscles, suggesting that the underlying process is arteriosclerosis obliterans. Initial ischemia could cause tissue swelling that, through a pressure effect, might compromise blood flow.

- Hypercoagulability, with increased factor VII activity, impaired response of tissue plasminogen activator to venous occlusion, and increased plasma levels of both plasminogen activator inhibitor and thrombomodulin.

Table 1. Coagulation factors observed in patients with diabetic muscle infarction

Reactants	Normal	Case 1	Case 2
Global tests			
aPTT	24.7–34.5 s	21.2	54.8*
PT	9.0–10.4 s±	9.3	10.3
Thrombin time	Control ± 1 s	13.1	13.3
Euglobulin lysis time	90–300 min	255	185
Bleeding time	2–9 min	8.5	7.0
Coagulation			
Factor VII	0.60–1.4 U/ml	2.0*	2.0*
Factor VIII	0.60–1.5 U/ml	–	1.92*
Factor IX	0.50–1.4 U/ml	–	1.8*
Factor XI	0.60–1.3 U/ml	–	1.28
Factor XII	0.50–1.5 U/ml	–	0.76
Fibrinogen	170–410 ml/dl	254	980*
Anticoagulation			
Protein C			
Activity	0.69–1.34	1.52	1.04
Antigen	0.70–1.30 U/ml	1.38	1.02
Protein S			
Free	0.50–1.50 U/ml	0.90	0.64
Total	0.70–1.34 U/ml	1.02	1.12
Antithrombin III	0.80–1.05 U/ml	1.18	0.94
Fibrinolytics			
Tissue plasminogen activator			
Pre-VO	0.00–10.0 ng/ml	4.50	14.5*
Post-VO	0.00–10.0 ng/ml	9.20*	18.5*
Plasminogen activator inhibitor			
Pre-VO	0.00–10.0 IU/ml	21.0*	22.0*
Post-VO	0.00–10.0 IU/ml	24.0*	22.0*
Plasminogen	0.70–1.26 U/ml	1.20	1.30
Platelet aggregation	Normal	Normal	
Thrombomodulin	<38.2 ng/ml	71.5	466

*Represents abnormal values in this profile.
VO = venous occlusion.

Bjornskov EK, Carry MR, Katz FH, Lefkowitz J, Ringel SP. Diabetic muscle infarction: a new perspective on pathogenesis and management. Neuromuscul Disord. 1995 Jan;5(1):39-45.

Clinical features:

Table 1—*Clinical features of patients with DMI*

Number of patients	Number of episodes	Episodes/ patient	Age (range) (years)	Sex (male/female)	Diabetes type	Duration of diabetes (range) (years)	Complications (*n*/reported)
115	166	1.44	42.63 (19–81)	61/54 (38.47%)/(61.53%)	Type 1: 68 (59.1%) Type 2: 27 (23.8%) Not indicated 20 (17.1%)	14.35 (0–50)	Nephropathy: 74/104 (71.1%) Retinopathy: 56/99 (56.6%) Neuropathy: 54/99 (54.5%)

Muscle involved	Weeks from onset (range)	Clinical features	CK level	Leucocytosis	ESR increased
Thigh: 139 (83.7%) Calf: 32 (19.28%) Both: 4 (2.41%) Bilateral: 14 (8.43%)	3.98 (1 day to 40 weeks)	Pain: 133 (80.12%) Swelling: 126 (75.9%) Mass: 56 (33.73%) Fever: 3 (10.71%)	High: 27/56 (48.2%) Normal: 29/56 (52.7%) Not reported: 110	7.01 (8%)	28 (52.8%)

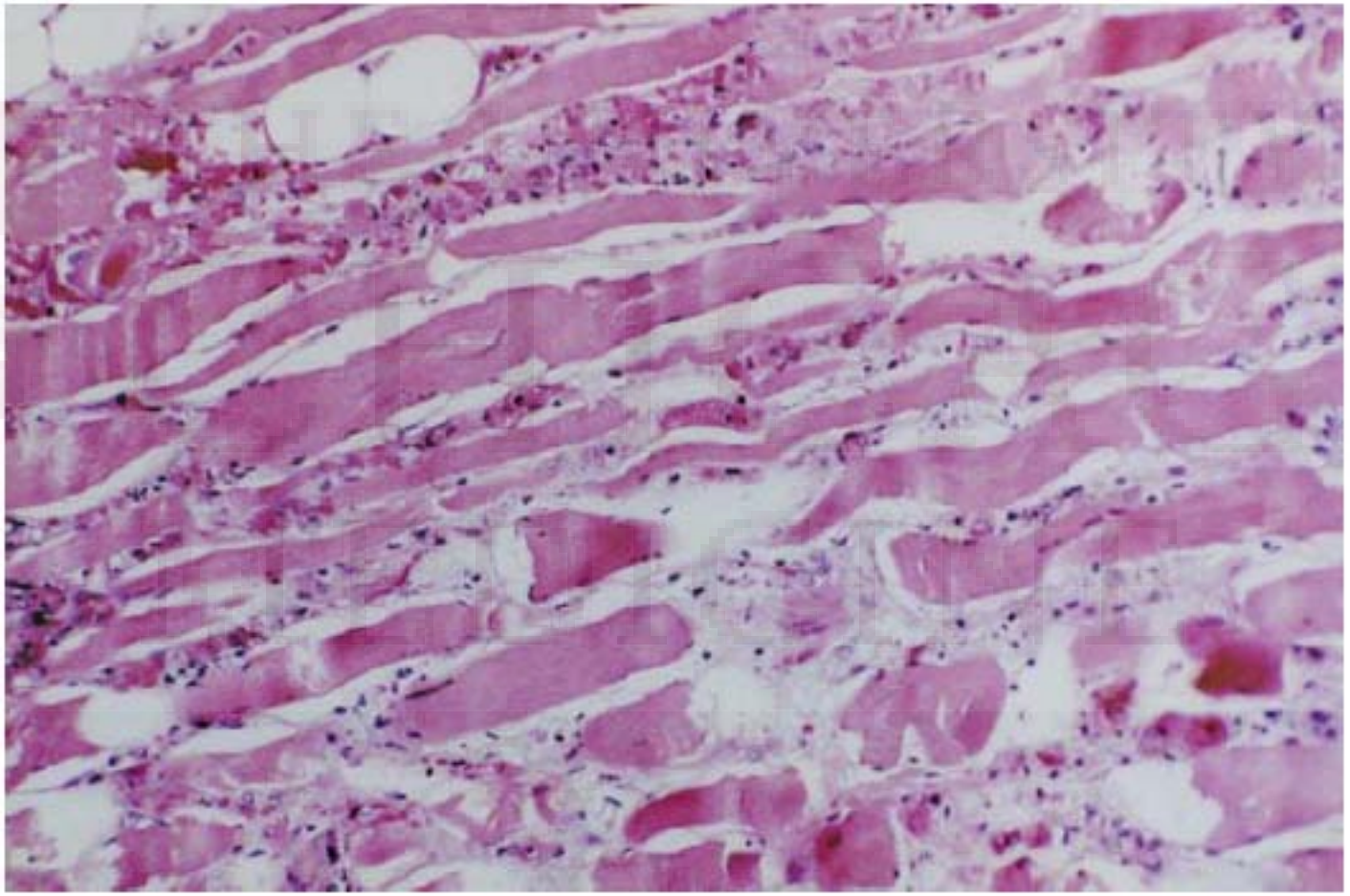
Biopsies	Treatment	Weeks until resolution (range)	Recurrences
Fine-needle: 39 Open: 29 Total: 95 Type not reported: 27	Bed rest: 32 (19.28%) Analgesics: 48 (28.92%) Physical therapy: 23 (13.86%) Not indicated: 36 (21.69%)	4 (2–17)	Same muscle: 10 (8.69%) Another muscle: 45 (39.13%)

Trujillo-Santos AJ. Diabetic muscle infarction: an underdiagnosed complication of long-standing diabetes. Diabetes Care. 2003 Jan;26(1):211-5

Diagnosis:

- MRI (increased signal from the affected muscle area (intramuscular and perimuscular tissues) in T2-weighted, inversion-recovery, and gadolinium-enhanced images and isointense or hypointense areas on T1-weighted images, secondary to increased water content from edema and inflammatory changes that accompany the infarction)

- Muscle biopsy (should be reserved for cases in which the clinical presentation is atypical or the diagnosis uncertain or when appropriate treatment fails to elicit improvement)



Management and prognosis:

- Rest and analgesics
- Antiplatelet agents and/or antiinflammatory drugs
- Surgical excision

- Diabetic muscle infarction resolves spontaneously over a few weeks to months in most patients.

Take home points:

- Diabetic muscle infarction is a rare disorder that affects patients who have relatively longstanding diabetes, many of whom have other micro or macrovascular complication.
- Clinical manifestations include acute or subacute onset of muscle pain, swelling, and associated tenderness. The muscles of the thigh and calf are most commonly affected.
- Definitive diagnosis of diabetic muscle infarction requires biopsy of the affected area of muscle that demonstrates ischemic necrosis and excludes infection. A clinically based diagnosis of muscle infarction may be appropriately made for patients with compatible MRI findings.
- Tight glycemic control might prevent developing this complication.

References:

- Trujillo-Santos AJ. Diabetic muscle infarction: an underdiagnosed complication of long-standing diabetes. Diabetes Care. 2003 Jan;26(1):211-5

- Bjornskov EK, Carry MR, Katz FH, Lefkowitz J, Ringel SP. Diabetic muscle infarction: a new perspective on pathogenesis and management. Neuromuscul Disord. 1995 Jan;5(1):39-45

- Kattapuram TM, Suri R, Rosol MS, Rosenberg AE, Kattapuram SV. Idiopathic and diabetic skeletal muscle necrosis: evaluation by magnetic resonance imaging. Skeletal Radiol. 2005 Apr;34(4):203-9. Epub 2005 Feb 8.