The Medicine Forum

Volume 14 Article 6

2013

An Interesting Case Report of Diabetic Myonecrosis

Manjula Nagaraja, MD
Thomas Jefferson University Hospitals, Manjula.Nagaraja@jefferson.edu

Jim Zhang, MD
Thomas Jefferson University Hospitals, Xing.Zhang@jefferson.edu

An Interesting Case Report of Diabetic Myonecrosis

Manjula Nagaraja, MD and Jim Zhang, MD

Case Presentation

A 49-year-old male with a history of ischemic cardiomyopathy, New York Heart Association class II heart failure with an ejection fraction of 35% status post biventricular implantable cardiac defibrillator (ICD), end stage renal disease on dialysis, diabetes mellitus II, and pancreatitis complicated by pseudocyst presented with a sudden onset of left thigh pain with a palpable mass. He denied trauma to the site, numbness, tingling, weakness, fevers, or chills. Review of systems was otherwise negative. Vital signs at presentation were as follows: temperature 98.8°F, pulse 77 beats per minute, blood pressure 150/88 mm Hg, respiratory rate 18 breaths per minute, and oxygen saturation of 96% on 2 liters of nasal cannula. He was notably uncomfortable on exam. The left thigh was edematous on the lateral side and there was tenderness over the distal, lateral portion of the anterolateral left thigh. No tenderness in the calf was noted. He had full range of motion at the hip, ankle, and knee. Straight leg test was negative and pinprick sensation was decreased.

Laboratory tests revealed leukocytosis with a white count of 23.1, slight anemia with a hemoglobin of 9.4. Chem 7 revealed BUN of 33 and creatinine of 4.1. Hemoglobin A1c was 8.0 and CPKwas 82. Coags were normal but ESR was elevated at 98.

Radiographic studies included x-ray of the left femur, which revealed no fracture or dislocation. Chest x-ray on admission demonstrated a right dialysis catheter, low lung volumes with mild background pulmonary edema and cardiomegaly. Lower extremity ultrasound was negative for deep vein thrombosis. MRI of the left thigh was not obtained due to his ICD. CT scan of the left thigh showed heterogeneously decreased enhancement of the quadriceps particularly within the vastus lateralis; skin thickening with infiltration of the subcutaneous fat, possibly representing cellulitis in the appropriate clinical setting; diffuse atherosclerotic disease with moderate stenoses of the left femoral artery at the level of the adductor hiatus and the left popliteal artery (Figure 1).

Differential Diagnosis

The patient presented with a left thigh mass containing inflammatory and infectious features. The differential includes: superficial and deep vein thromboses, pyomyositis, myositis ossificans, traumatic muscle rupture, muscle hemorrhage, fasciitis, osteomyelitis, abscess, soft tissue neoplasm (primary lymphoma or sarcoma), granulomatous lesions involving TB or sarcoid, calciphylaxis. A rare but important diagnosis on the differential should include diabetic myonecrosis, which is what our patient had.

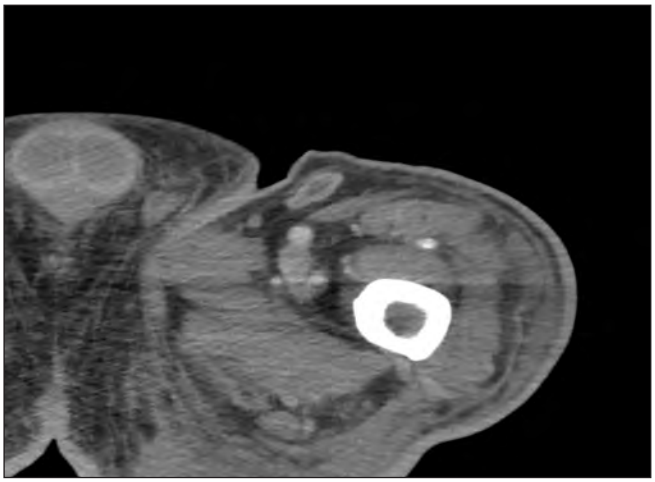

Figure 1. CT Scan of Left Thigh

Biopsy showed increased variation in the fiber diameter, increased interstitial fibrosis, and relative type II fiber atrophy. There was no evidence of a neurogenic process or malignancy.

Discussion

Skeletal muscle infarction is a rare and unusual complication of diabetes mellitus. Most patients have advanced end organ damage by the time of presentation. Typical clinical presentation of diabetic myonecrosis includes abrupt onset of pain and swelling in the affected muscle. Patients have subsequent partial resolution and appearance of a palpable painful mass. The quadricep muscle group is most commonly affected; calf involvement is rare. Bilateral involvement has been reported in a few cases. The muscles most frequently affected are the vastus lateralis (24%) and vastus medialis (22%).[1]

Most laboratory values are nonspecific. Elevated body temperature was not always reported. Creatinine kinase enzymes were often normal in reported cases. About one-third of the patients had leukocytosis; three quarters had ESR > 50.[2] Of known cases, the majority (75%) had a hemoglobin A1C greater than 7%, suggesting poor glycemic control. An ultrasound should be performed to evaluate for deep vein thrombus and abscess collection. MRI is most sensitive and is the diagnostic modality of choice. In approximately 57% of cases, however, diabetic myonecrosis was confirmed by biopsy. Currently, because of complications of biopsy in diabetic patients, physicians tend to eschew invasive methods. Biopsy should be reserved for cases with an atypical presentation.

MRI can show an increased signal from the affected muscle area in T2 images that are hypointense areas on T1 images. Other features can include diffuse enlargement with foci of hemorrhage. Pathologic features most commonly showed muscle fiber necrosis and inflammatory infiltrates. Findings at later stages include replacement of necrotic muscle fibers by fibrous tissue. Microvascular abnormalities can include luminal narrowing and intramural calcifications or perhaps fibrinoid occlusion. The etiology of diabetic myonecrosis remains controversial. There are two proposed mechanisms, microvascular disease and hypercoagulability. Extensive arterial occlusive disease has been identified in many patients. The theory suggests diabetic microangiopathy and/or arteriosclerosis lead to ischemia of muscle, which results in an intense inflammatory response, edema and hyperemia. Some authors suggest that acquired hypercoagulability might be to blame. This latter theory is supported by detection of antiphospholipid antibodies in several cases.

Optimal treatment of diabetic myonecrosis is uncertain. Most patients received bed rest and analgesics. Therapeutic modalities that may be beneficial include antiplatelet agents and anti-inflammatory agents. Some authors recommend anticoagulation therapy. Short term prognosis is good as symptoms usually resolve within 8 to 12 weeks.[3] Approximately 42-48% of cases re-occur usually on the same side. Contralateral recurrence is associated with a poorer prognosis. Most of the patients diagnosed with diabetic myonecrosis will eventually die from long-term complications of diabetes. The mean mortality rate was 10% within two years from diabetic myonecrosis onset.[4]

References

1. Kapur S, Brunet JA, McKendry RJ. Diabetic muscle infarction: case report and review. *Journal of Rheumatology* 2004:31: 190-4
2. Trujillo-Santos AJ. Diabetic muscle infarction: an underdiagnosed complication of long-standing diabetes. *Diabetes Care* 2003:26:211-15.
3. Reyes-Balaguer J, Solaz-Morena E, Morata-Aldea C, Elorza-Montesinos P. Spontaneous Diabetic Myonecrosis. *Diabetes Care* 2005:28:980-1
4. Khan S, Al-Riyami D, Al-Ghailani M, Al-Dhuhli H. An Under-recognized Complication: Diabetic myonecrosis. *Oman Medical Journal* 2009:24:228-30.

"Bench"
photograph by Rajan Singla