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Non-traumatic Muscle Pain in a Diabetic

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INTRODUCTION

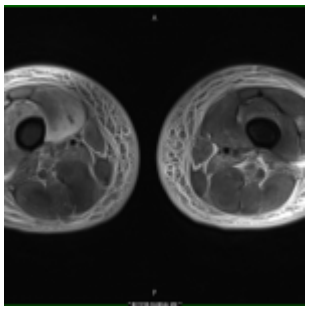
The prevalence of diabetes in the adult population in United States is approximately 10% and is expected to rise. The myriad of complexities of this entity, both microvascular and macrovascular is anticipated to follow suit, adding to the morbidity and mortality. Diabetic muscle infarction (DMI) is one such incompletely understood complication of long-standing uncontrolled diabetes and seems to play a crucial role in risk stratification in those with microvascular involvement. DMI has shown to be a poor prognosticator of long-term survival, a grim reality given the mean age of presentation is only 43 years. Further investigation into improving this outlook and whether tighter glycemic control changes outcome would be of significant interest and benefit.

CASE PRESENTATION

A 24-year-old male with hypertension and twenty-year history of uncontrolled type I diabetes was admitted with two months of pain near the right knee, acutely worsening over one week. Right knee effusion was recently diagnosed with synovial fluid twice negative for infection or crystals. He denied fever, chills, weight loss, trauma, injecting insulin in the area, or illicit drug use. Lower extremity paresthesias were chronic. He previously had a toe amputated due to osteomyelitis.

On exam, he was afebrile. Right knee had a small effusion but was without associated redness or pain. An area of significant tenderness to palpation with no erythema, warmth, swelling, or mass was present on the right medial thigh just proximal to the knee.

Electrolytes, renal function, leukocyte count and differential, and coagulation profile were normal. Blood glucose was 457 with glycosylated hemoglobin of 17. Synovial fluid was minimally inflammatory. Doppler was negative for DVT. Erythrocyte sedimentation rate (ESR) and c-reactive protein were 68 and 4.1 respectively. CPK was 166; ANA, ANCA and complements were normal. Ultrasound showed no abscess but diffuse edema suggested cellulitis for which Vancomycin was started; however, he did not improve. Ultimately, MRI of the thigh revealed findings consistent with Diabetic Muscle Infarction (DMI).



MRI: Axial view of mid-thigh image demonstrates edematous vastus medialis, intermedius and lateralis on the right and increased signal intensity in the vastus lateralis on the left.

That hospitalization was complicated by acute onset of hypoxia and he was treated with Vancomycin and Cefepime for possible hospital acquired pneumonia. He was discharged with pain medications and a walker shortly afterwards but readmitted three weeks later with systolic blood pressure in the 200s, shortness of breath, diaphoresis and chills. He unfortunately developed acute respiratory distress syndrome and died. Autopsy noted the cause of death as pan-lobar pneumonia. Post-mortem blood culture grew pseudomonas; thigh muscle culture remained negative.

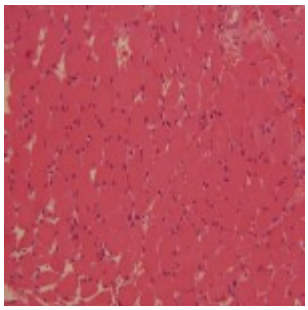
DISCUSSION

Diabetic Muscle Infarction (DMI), also called aseptic myonecrosis, ischemic myonecrosis and tumoriform focal muscle degeneration, is a poorly understood rare complication of long standing unchecked diabetes. First described in 1965, only 116 cases of DMI are documented in literature although its identification seems to be increasing.

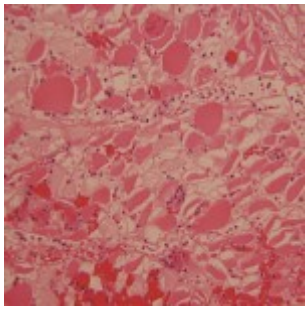
Pathogenesis remains unclear. Current hypotheses are that muscle infarction is caused by vascular disease such as arteriosclerosis and diabetic microangiopathy. Hypercoagulability due to alteration in the coagulation-fibrinolysis system may also contribute to ischemia.

Mean age of presentation for DMI is 43. The average duration of diabetes is 14 years, usually with accompanying microvascular complications: nephropathy (71%), retinopathy (57%), and neuropathy (55%). Localized pain and swelling is acute in onset, mostly involves the lower extremity (thigh 83%), bilateral in a third of the cases, and recurs in half of the patients.

There is no pathognomonic laboratory finding for DMI. Mild leukocytosis with elevated CPK and ESR can be present. MRI, the diagnostic modality of choice, demonstrates increased signal from the affected muscle in the T2-weighted and gadolinium enhanced images. Diffuse enlargement with ill- defined borders secondary to loss of normal fatty intramuscular septa may be noted. Grossly, the muscle has a pale, whitish appearance. Tissue sample is required only when the diagnosis is unclear. This patient developed ARDS and sepsis syndrome on readmission so underwent an ultrasound guided biopsy of the right vastus medialis which showed necrosis and edema with negative cultures, as expected in DMI.



Normal tissue with uniform caliber myocytes (H & E stain).



Affected tissue with areas of cell ischemia and dropped out muscle fibers and infiltration by inflammatory cells (H & E stain).

Because of the limited number of reported cases, optimal treatment remains uncertain. Average time to improvement is 8 weeks with rest, analgesics and continuation of routine activities. It may be shortened to 6 weeks with antiplatelet and/or anti-inflammatory drugs but their adverse effects must be considered in diabetics. Recovery from surgical excision takes 13 weeks; additionally, any invasive procedure can be further complicated by delayed wound healing, hematomas and wound infections. Glycemic control is suggested although direct benefit of this in regards to DMI has not been studied.

Although DMI is self-resolving, it is a poor prognosticator for survival with mean mortality rate of 10% in 2 years. Because of the significant presence of microvascular disease in those with DMI, it has been suggested that DMI may represent a new paradigm for risk stratification in diabetic patients based on the presence of microangiopathy. The cause of death is primarily macrovascular events such as MI or stroke. Curiously though on reviewing the literature, as with our patient, another article reported on a subject who also succumbed to pneumonia shortly after being diagnosed with DMI. This leads us to speculate if other unrecognized factors, such as difficulty in overcoming infections in this population with uncontrolled diabetes, are at play leading to their untimely deaths.

CONCLUSION

The challenge in DMI is to consider the diagnosis and avoid invasive procedures if possible. Infection (cellulitis, pyomyositis, osteomyelitis, necrotizing fasciitis) must be adequately ruled out. Other possibilities are neoplasms, DVT, non-infectious inflammatory conditions, or neurologic issues such as diabetic amyotrophy. In our patient, the long-standing uncontrolled diabetes, presence of microvascular disease such as peripheral neuropathy, location of the pain, paucity of signs and symptoms of infection, MRI findings, unresponsiveness to antibiotics but improvement over time with pain management and finally, tissue analysis which is not always required, confirmed DMI.

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