

Elderly Veteran With Diabetes and Foot and Ankle Swelling

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With a medical history of foot ulcers and current edema in both ankles and feet, an elderly veteran with diabetes needed a diagnosis for his foot pain.

A 62-year-old male veteran with poorly controlled type 2 diabetes presented with nontraumatic swelling about both his ankles and feet. He reported foot pain, fever, chills, and dyspnea on exertion. Chronic ulcers on his left 1st and 5th metatarsals plagued him over the prior year, occasionally necessitating antibiotics for cellulitis. One month prior to presentation, the patient developed a new ulcer under his right 5th metatarsal head; he was treated conservatively, because there were no signs of infection. His medical history also included hypertension, hyperlipidemia, osteoporosis, and dementia with a baseline Mini-Mental State Examination score of 20 out of 30. His medications included insulin, glipizide, amlodipine, atenolol, hydrochlorothiazide, enalapril, lovastatin, aspirin, donepezil, memantine, and fluoxetine. The patient lived alone and relied on his brother for ensuring medication compliance, blood pressure control, and blood sugar monitoring. His vitals were stable and a physical exami-

nation revealed edema in both ankles and feet. Peripheral neuropathy and diminished pedal pulses were also evident on physical examination. Routine 3-view radiographs of both feet were obtained (Figures 1a and 1b).

WHAT'S YOUR DIAGNOSIS?

The patient's diagnosis was diabetic calcaneal stress fracture, midbody compression type. Right and left lateral foot radiographs revealed diffuse osteopenia and a compacted right calcaneal fracture (white arrows) parallel to the subtalar joint (Figure 2). A key finding in calcaneal fractures is loss of Bohler's angle, which should fall between 20° and 40° and be directly compared with the opposite foot.

Bohler's angle is determined by measuring the angle created by drawing 2 intersecting lines: One line travels through the superior aspect of the calcaneal tuberosity and the superior aspect of the posterior calcaneal facet, and the second line travels through the superior aspect of the posterior calcaneal facet and the superior aspect of the anterior calcaneal process.

Close inspection of the patient's x-rays revealed that Bohler's angle was actually negative due to the mid-calcaneal depression. In the absence of serious trauma, this mid-calcaneal compression fracture is most ac-

curately described as an advanced stress fracture brought on by reduced weight bearing on the contralateral foot during an extended bout with plantar ulcers. Computed tomography (CT) was performed for surgical planning, but ultimately the patient was deemed a poor surgical candidate (Figure 3). At the time of this case report, the patient was being followed with periodic foot x-rays to document stability of the fracture with external bone stimulators being reserved for fracture progression.

DISCUSSION

According to the 2011 National Diabetes Fact Sheet, about 18.8 million Americans have been diagnosed with diabetes, incurring a total yearly cost of \$174 billion.¹ The future prevalence of diabetes in the U.S. is estimated to increase up to 3-fold by 2050.^{2,3} Specifically, within the VA system, the prevalence of diabetes rose from 16.7% in 1998 to 19.6% in 2000.⁴ In 1998, \$1.67 billion was spent on the treatment of veterans with diabetes, a figure that includes \$1.45 billion in inpatient care and \$214.8 million in outpatient care.⁵

Foot problems are common among older veterans with diabetes. These problems increase older veterans' risk of peripheral vascular

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Figures 1a and 1b. Lateral radiographs of the right (a) and left (b) foot.

disease (PVD), sensory neural impairment and subsequent infection, neuroarthropathy, and potential amputation.⁶ Indeed, roughly 60% of amputations in all VA hospitals between 1989 and 1998 were attributed to diabetes, and one study estimated the incidence of lower-extremity amputation over a 25-year period at 10% for patients with diabetes.⁷⁻⁹

Patients with type 1 and type 2 diabetes have an increased risk of fracture compared with people without diabetes.^{10,11} Diabetic neuropathy alters sensation and response to injury, leading to repetitive and progressive trauma that can result in deformity.¹²⁻¹⁴ Although controversial, some studies have found reduced bone mineral density (BMD) in patients with type 2 diabetes.¹⁵⁻¹⁷ The glitazones have been implicated in reducing BMD by stimulating adipocyte formation from mesenchymal stem cells at the expense of osteoblasts, leading to increased risk of fracture.¹⁸

A potentially serious foot injury is the calcaneal insufficiency stress fracture. Although calcaneal stress fractures are among the least common type of stress injury, they most commonly occur in military recruits as fatigue-type stress fractures, where abnormal stress is applied to normal bone.^{19,20} Insufficiency stress fractures, on the other hand, occur when abnormal bone is exposed to normal stress, typified by sacral and verte-



Figure 2. Lateral radiograph of the right foot demonstrating a midbody calcaneal compression fracture (white arrows) with loss of Bohler's angle and impaction of the inferior calcaneal fragment into the subtalar fragment.

bral insufficiency fractures in patients with osteoporosis.²⁰ Other factors predisposing to insufficiency fractures include chronic renal failure, parathyroid disease, steroid therapy, inflammatory arthritides, nutritional deficiencies, and diabetes.²¹ Delayed diagnosis and misdiagnosis are frequent problems with calcaneal insufficiency stress fractures, especially in patients with diabetes with poor sensory perception who may be asymptomatic. Prompt diagnosis and treatment are critical to avoid progression to irreversible damage, deformity, and possibly amputation.²²⁻²⁶

Calcaneal fractures are traditionally secondary to serious trauma and divided into intraarticular (75%) and extraarticular (25%).²⁷ Many classification schemes have been proposed for intraarticular fractures, but Hannover and Sanders are the 2 most widely used.²⁷ Hedlund proposed a

separate classification scheme for extraarticular calcaneal stress fractures in 25 patients with diabetes, calcaneal fracture, and no history of serious trauma.²⁸ The first type involves a coronal fracture through the posterior calcaneus along the apophyseal growth plate. The second type involves a fracture line that runs parallel to the subtalar joint but without involving the joint, described as a midbody compression fracture and seen in the study patient. The third type is associated with heel ulceration and involves an oblique coronal fracture extending from the calcaneal tubercle toward the subtalar joint. The calcaneal insufficiency avulsion fracture described by Kathol and colleagues is similar to Hedlund's type 3 and is most strongly associated with neuropathy, osteoporosis, diabetes, and steroid treatment (Figure 4).^{29,30} Soft tissue injury and infection can

complicate these avulsion fractures due to posterior skin compression and necrosis, often necessitating open reduction.^{30,31} If diagnosis and treatment are delayed, a more extensive surgical procedure may be required.²⁴

Insufficiency stress fractures in patients with diabetes differ from fatigue stress fractures in young healthy adults by their advanced state at presentation. Calcaneal fatigue stress fractures can occur in any location and in any orientation; however, they most commonly occur in the posterior calcaneus approaching

the vertical orientation (Figure 5).^{21,32} These fractures almost never progress to displacement or collapse in healthy individuals with appropriate treatment; whereas in patients with diabetes who have neuropathy and reduced sensation, calcaneal stress fractures often progress to displacement, collapse, disfigurement, and disability.^{22,33,34}

Early insufficiency fractures may be asymptomatic, especially in patients with diabetes with reduced sensation.^{12,23,35} Symptoms might include soft tissue swelling and heel or plantar pain with weight bearing.^{19,21,22} Soft tissue swelling associated with calcaneal stress fractures usually involves the retrocalcaneal bursa anterior to the Achilles tendon and may spread to involve the medial and lateral malleolus.^{19,21} Squeezing the calcaneus may elicit point tenderness over the fracture site, and advanced fractures can result in hind foot deformity if they are not properly treated.²¹⁻²³

Treatment involves wearing a protective rocker-bottom boot with



Figure 3. Sagittal CT of the right foot demonstrating a midbody calcaneal compression fracture with intact subtalar joints (asterisks). Talus, T; Calcaneus, C.



Figure 4. Lateral radiograph of the ankle in a different patient demonstrating a calcaneal insufficiency avulsion fracture. Note the retracted and thickened Achilles tendon (asterisk) and the arterial calcification commonly seen in patients with diabetes (white arrow).

cushioning material, physical therapy, and nonsteroidal antiinflammatory drugs for pain relief. The boot redistributes pressure and limits impact on the fracture site. More advanced fractures may be treated with surgical techniques or bone stimulators. Preventive measures such as tight glycemic control, aggressive treatment and prevention of vascular complications, visual assessment, and regular exercise help combat osteoporosis and fall risk. Bisphosphonates have also been shown to reduce bone turnover and symptoms in patients with diabetes with Charcot arthropathy and may reduce future fracture risk.³⁶

The differential diagnosis for heel swelling in patients with diabetes also includes plantar fasciitis, retrocalcaneal bursitis, tarsal tunnel syndrome, tendonitis, nerve entrapment, infection, and tumors. Imaging plays a central role in differentiating between these etiologies when the history and physical examination are equivocal. Plain radiographs should be the initial imaging study, although they will often

be negative. Early findings of stress fractures include faint intracortical striations known as the gray cortex sign, discrete sclerotic bands, and subperiosteal or endosteal bone formation.^{37,38} These may appear from 1 week to several months after the onset of symptoms. As seen in this study patient, late plain radiographic findings included a definite lytic fracture line or osseous collapse.³⁸ Three-phase bone scintigraphy has a high sensitivity for stress fracture approaching 100% but provides low specificity, because tumor, infection, infarction, and periostitis can have similar scintigraphic findings.^{32,39}

Depending on availability, CT or magnetic resonance imaging (MRI) should be the second study performed if plain radiography is unrevealing. Computed tomography will demonstrate a linear fracture line with surrounding sclerosis and is useful for defining anatomy when surgical intervention is considered.³⁸ An MRI demonstrates reduced bone marrow signal on T1 weighted sequences and increased



Figure 5. Coned down lateral radiograph of the foot demonstrates a sclerotic line in the posterior calcaneus approaching the vertical orientation (asterisks).

bone marrow signal on T2 and proton density weighted sequences. A distinct cortical break may also be seen, especially on T1 weighted sequences.³² An MRI is helpful in that it can identify many other entities in the differential for heel swelling in patients with diabetes, especially in the soft tissues, such as tendonitis, bursitis, plantar fasciitis, and osteomyelitis.

Early diagnosis and treatment of stress injuries in patients with diabetes are crucial in preventing long-term complications and restoring function. At least, plain radiographs should be obtained in any patient with diabetes with foot pain or swelling.³ Ultrasound can be useful in the evaluation of soft tissues; however, ultrasound is not useful for detecting early bone marrow edema indicative of a stress injury. Bone scintigraphy is sensitive but lacks specificity. In patients with diabetes with radiographically occult stress injuries visible by MRI, off-loading and immobilization

result in reduction in bone marrow edema and progression of bone destruction.¹³ Therefore, the authors advocate using MRI in any patient with diabetes with unexplained foot or ankle pain or swelling in order to detect early signs of stress injury or an alternative etiology for the symptoms.

Preventive strategies will play a major role in the future to help reduce the disability and cost related to diabetic foot pathology. “Comprehensive foot care programs—ones that include risk assessment, foot-care education and preventive therapy, treatment of foot problems, and referral to specialists—can reduce amputation rates by 45% to 85%.”¹ Indeed, the VHA practice guidelines for diabetes recommend risk stratifying patients with diabetes according to their risk for lower-extremity ulcer and amputation, followed by appropriate referral to a foot specialist for high-risk patients.⁴⁰ Features of high risk include lack of sensation using a monofilament; evidence or history of lower-extremity arterial disease; foot deformity, such as hammer toe, Charcot foot, or bunions; and a history of foot ulcer or nontraumatic amputation at any level.⁴⁰

Although most lower-extremity amputations in patients with diabetes occur secondary to PVD and infection, calcaneal insufficiency fractures remain an important cause of deformity that may require amputation if it is left untreated. Early diagnosis and treatment of lower-extremity pathology is crucial in reducing long-term disability and health care utilization in patients with diabetes. ●

Author disclosures

The authors report no actual or potential conflicts of interest with regard to this article.

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REFERENCES

- Centers for Disease Control and Prevention. National diabetes fact sheet: National estimates and general information on diabetes and prediabetes in the United States, 2011. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention; 2011.
- Narayan KM, Boyle JP, Geiss LS, Saaddine JB, Thompson TJ. Impact of recent increase in incidence on future diabetes burden: U.S., 2005-2050. *Diabetes Care*. 2006;29(9):2114-2116.
- Boyle JP, Honeycutt AA, Narayan KM, et al. Projection of diabetes burden through 2050: Impact of changing demography and disease prevalence in the U.S. *Diabetes Care*. 2001;24(11):1936-1940.
- Miller DR, Safford MM, Pogach LM. Who has diabetes? Best estimates of diabetes prevalence in the Department of Veterans Affairs based on computerized patient data. *Diabetes Care*. 2004;27(suppl 2):B10-B21.
- Maciejewski ML, Maynard C. Diabetes-related utilization and costs for inpatient and outpatient services in the Veterans Administration. *Diabetes Care*. 2004;27(suppl 2):B69-B73.
- Holewski JJ, Moss KM, Stess RM, Graf PM, Grunfield C. Prevalence of foot pathology and lower extremity complications in a diabetic outpatient clinic. *J Rehabil Res Dev*. 1989;26(3):35-44.
- Mayfield JA, Reiber GE, Maynard C, Czerniecki JM, Caps MT, Sangeorzan BJ. Trends in lower limb amputation in the Veterans Health Administration, 1989-1998. *J Rehabil Res Dev*. 2000;37(1):23-30.
- Mayfield JA, Reiber GE, Maynard C, Czerniecki JM, Sangeorzan B. The epidemiology of lower-extremity disease in veterans with diabetes. *Diabetes Care*. 2004;27(suppl 2):B39-B44.
- Sahakyan K, Klein BE, Lee KE, Myers CE, Klein R. The 25-year cumulative incidence of lower extremity amputations in people with type 1 diabetes. *Diabetes Care*. 2011;34(3):649-651.
- de Liefde II, van der Klift M, de Laet CE, van Daele PL, Hofman A, Pols HA. Bone mineral density and fracture risk in type-2 diabetes mellitus: The Rotterdam Study. *Osteoporos Int*. 2005;16(12):1713-1720.
- Vestergaard P. Discrepancies in bone mineral density and fracture risk in patients with type 1 and type 2 diabetes—a meta-analysis. *Osteoporos Int*. 2007;18(4):427-444.
- Chantelau E, Richter A, Ghassem-Zadeh N, Poll LW. “Silent” bone stress injuries in the feet

- of diabetic patients with polyneuropathy: A report on 12 cases. *Arch Orthop Trauma Surg.* 2007;127(3):171-177.
13. Chantelau E, Richter A, Schmidt-Grigoriadis P, Scherbaum WA. The diabetic charcot foot: MRI discloses bone stress injury as trigger mechanism of neuroarthropathy. *Exp Clin Endocrinol Diabetes.* 2006;114(3):118-123.
 14. El-Khoury GY, Kathol MH. Neuropathic fractures in patients with diabetes mellitus. *Radiology.* 1980;134(2):313-316.
 15. Isaia G, Bodrato L, Carlevatto V, Mussetta M, Salamano G, Molinatti GM. Osteoporosis in type II diabetes. *Acta Diabetol Lat.* 1987;24(4):305-310.
 16. Levin ME, Boisseau VC, Avioli LV. Effects of diabetes mellitus on bone mass in juvenile and adult-onset diabetes. *N Engl J Med.* 1976;294(5):241-245.
 17. Nuti R, Righi G, Signorini AM. Bone mineral content value (single americium 241 energy source) in diabetes mellitus. *Radiol Med.* 1980;66(10):756-758.
 18. Bodmer M, Meier C, Kraenzlin ME, Meier CR. Risk of fractures with glitazones: A critical review of the evidence to date. *Drug Saf.* 2009;32(7):539-547.
 19. Leabhart JW. Stress fractures of the calcaneus. *J Bone Joint Surg Am.* 1959;41(7):1285-1290.
 20. Daffner RH, Pavlov H. Stress fractures: Current concepts. *AJR Am J Roentgenol.* 1992;159(2):245-252.
 21. Weber JM, Vidt LG, Gehl RS, Montgomery T. Calcaneal stress fractures. *Clin Podiatr Med Surg.* 2005;22(1):45-54.
 22. Andersen LB, Dipreta J. Charcot of the calcaneus. *Foot Ankle Clin.* 2006;11(4):825-835.
 23. Chantelau E. The perils of procrastination: Effects of early vs delayed detection and treatment of incipient Charcot fracture. *Diabet Med.* 2005;22(12):1707-1712.
 24. Itokazu M, Matsunaga T, Yang W, Wada E. A neglected avulsion fracture of calcaneal tuberosity associated with diabetes mellitus: A case report. *Changgen Yi Xue Za Zhi.* 1996;19(3):277-280.
 25. Nade S, Monahan PR. Fractures of the calcaneum: A study of the long-term prognosis. *Injury.* 1973;4(3):200-207.
 26. Thompson RC Jr, Clohisy DR. Deformity following fracture in diabetic neuropathic osteoarthropathy. Operative management of adults who have type-1 diabetes. *J Bone Joint Surg Am.* 1993;75(12):1765-1773.
 27. Daftary A, Haims AH, Baumgaertner MR. Fractures of the calcaneus: A review with emphasis on CT. *Radiographics.* 2005;25(5):1215-1226.
 28. Hedlund LJ, Maki DD, Griffiths HJ. Calcaneal fractures in diabetic patients. *J Diabetes Complications.* 1998;12(2):81-87.
 29. Kathol MH, el-Khoury GY, Moore TE, Marsh JL. Calcaneal insufficiency avulsion fractures in patients with diabetes mellitus. *Radiology.* 1991;180(3):725-729.
 30. Hess M, Booth B, Laughlin RT. Calcaneal avulsion fractures: Complications from delayed treatment. *Am J Emerg Med.* 2008;26(2):254.e1-4.
 31. Biehl WC 3rd, Morgan JM, Wagner FW Jr, Gabriel R. Neuropathic calcaneal tuberosity avulsion fractures. *Clin Orthop Relat Res.* 1993(296):8-13.
 32. Dodson NB, Dodson EE, Shromoff PJ. Imaging strategies for diagnosing calcaneal and cuboid stress fractures. *Clin Podiatr Med Surg.* 2008;25(2):183-201.
 33. Coventry MB, Rothacker GW Jr. Bilateral calcaneal fracture in a diabetic patient. A case report. *J Bone Joint Surg Am.* 1979;61(3):462-464.
 34. Johnson JT. Neuropathic fractures and joint injuries. Pathogenesis and rationale of prevention and treatment. *J Bone Joint Surg Am.* 1967;49(1):1-30.
 35. Gill G, Benbow S, Tesfaye S, Kaczmarczyk E, Kaye L. Painless stress fractures in diabetic neuropathic feet. *Postgrad Med J.* 1997;73(858):241-242.
 36. Jude EB, Selby PL, Burgess J, et al. Bisphosphonates in the treatment of Charcot neuroarthropathy: A double-blind randomised controlled trial. *Diabetologia.* 2001;44(11):2032-2037.
 37. Darby RE. Stress fractures of the os calcis. *JAMA.* 1967;200(13):1183-1184.
 38. Krestan C, Hojreh A. Imaging of insufficiency fractures. *Eur J Rad.* 2009;71(3):398-405.
 39. Santi M, Sartoris DJ. Diagnostic imaging approach to stress fractures of the foot. *J Foot Surg.* 1991;30(1):85-97.
 40. United States Department of Veterans Affairs. VA/DoD Clinical practice guideline. Management of diabetes mellitus (DM). Version 4.0. United States Department of Veterans Affairs Website. http://www.healthquality.va.gov/diabetes/DM2010_FUL-v4e.pdf. Updated August 2010. Accessed April 11, 2013.