

- pharmacophore as defined by a series of reserpine analogs that modulate multidrug resistance. *Proc Natl Acad Sci USA* 1989;86:5128-5132.
11. Chan HLS, Haddad G, Thorne PS, et al. P-glycoprotein expression as a predictor of the outcome of therapy for neuroblastoma. *N Engl J Med* 1991;325:1608-1614.
  12. Morrow CS, Cowan KH. Mechanisms of antineoplastic drug resistance. In: DeVita VT, Hellman S, Rosenberg SA, eds. *Cancer, principles and practice of oncology*, 4th ed. Philadelphia: J.B. Lippincott; 1993:340-348.
  13. Piwnicka-Worms D, Chiu ML, Budding M, et al. Functional imaging of multidrug-resistant P-glycoprotein with an organotechnetium complex. *Cancer Res* 1993;53:977-984.
  14. Rao VV, Chiu ML, Kronauge JF, et al. Expression of recombinant human multidrug resistance P-glycoprotein in insect cells confers decreased accumulation of technetium-99m-sestamibi. *J Nucl Med* 1994;35:510-515.
  15. Dalton WS. Overcoming the multidrug resistant phenotype. In: DeVita VT, Hellman S, Rosenberg SA, eds. *Cancer, principles and practice of oncology*, 4th ed. Philadelphia: J.B. Lippincott; 1993:2655-2665.
  16. Miller TP, Grogan TM, Dalton WS, et al. P-glycoprotein expression in malignant lymphoma and reversal of clinical drug resistance with chemotherapy plus high dose verapamil. *J Clin Oncol* 1991;9:17-24.
  17. Goldstein LJ, Galski H, Fojo A, et al. Expression of a multidrug resistance gene in human cancers. *J Natl Cancer Inst* 1989;81:116-124.
  18. Liotta LA, Stetler-Stevenson WG. Principles of molecular cell biology of cancer: cancer metastasis. In: DeVita VT, Hellman S, Rosenberg SA, eds. *Cancer, principles and practice of oncology*, 4th ed. Philadelphia: J.B. Lippincott; 1993:340-348.
  19. Dimitrakopoulou-Strauss A, Strauss LG, Goldschmidt H, et al. Evaluation of tumor metabolism and multidrug resistance in patients with malignant lymphomas. *Eur J Nucl Med* 1995;22:434-442.
  20. Israel O, Front D, Lam M, et al. Gallium-67 imaging in monitoring lymphoma response to treatment. *Cancer* 1988;61:2439-2443.
  21. Wylie BR, Southee AE, Joshua DE, et al. Gallium scanning in the management of mediastinal Hodgkin's disease. *Eur J Haematol* 1989;42:344-347.
  22. Gasparini MD, Balzarini L, Castellani M, et al. Current role of gallium scan management of mediastinal Hodgkin lymphoma. *Cancer* 1993;72:577-582.
  23. Lin J, Leung W, Ho SKW, et al. Quantitative evaluation of thallium-201 uptake in predicting chemotherapeutic response of osteosarcoma. *Eur J Nucl Med* 1995;22:553-555.

## Scintigraphy of Posterior Tibial Tendinitis

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Our goal was to describe the typical scintigraphic pattern of posterior tibial tendinitis. **Methods:** Bone scintigraphs were reviewed to study the scintigraphic characteristics of posterior tibial tendinitis in nine patients with posterior tibial tendinitis related to generalized rheumatic disease and in eight patients with isolated posterior tibial tendinitis. **Results:** The scintigraphic pattern of posterior tibial tendinitis is elongated increased uptake in the blood flow and blood-pool phase along the anatomical course of the tibialis posterior tendon at the medial aspect of the ankle (malleolus region). Static images demonstrate increased focal abnormal uptake at the medial malleolus and in the navicular bone. **Conclusion:** Bone scintigraphy depicts a characteristic pattern of posterior tibial tendinitis. It is useful for the early diagnosis of idiopathic- or rheumatic-related posterior tibial tendinitis.

**Key Words:** posterior tibial tendinitis; bone scintigraphy

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**T**endinitis due to an inflammatory process, degenerative change, endocrine and metabolic disorder or trauma initiates a periosteal reaction with reactive new bone formation at the entheses (1,2). This will result in an increased uptake of <sup>99m</sup>Tc-MDP caused by the increased bone turnover at the site of tendon attachment. Recently, it has been recognized that inflammatory changes of the tibialis posterior tendon occur more frequently than previously believed (3,4). Bone scintigraphy is used to detect areas of abnormal bone turnover in various musculoskeletal diseases (1,5,6). The bone scintigraphic pattern in enthesopathies at the calcaneus (plantar fasciitis and achilles tendinitis), tibial tuberosity (patellar tendinitis), greater trochanter, inferior pubic ramus and anterior inferior iliac spine has been reported (7-12). However, the pattern of posterior tibial tendinitis is not yet recognized. The purpose of this study was to describe the scintigraphic characteristic findings of posterior tibial tendinitis.

### MATERIALS AND METHODS

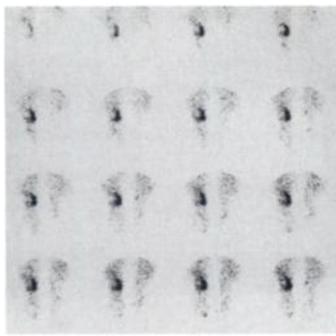
Seventeen patients with posterior tibial tendinitis were studied. Three men and five women, aged 30-68 yr, had idiopathic posterior tibial tendinitis and in five men and four women, aged 21-75 yr, posterior tibial tendinitis was associated with systemic inflammatory disease (two rheumatoid arthritis, two undifferentiated spondyloarthropathy, one fibromyalgia, one reactive arthritis, one psoriasis arthritis, one pseudogout and one patient with gout and pseudogout). Diagnosis of posterior tibial tendinitis was based on clinical signs and symptoms which include pain, swelling and tenderness on palpation of the tendon, presence of low or flat longitudinal arch (pes planus deformity) and weakness during inversion of the foot. In the idiopathic posterior tibial tendinitis patients, the duration of symptoms was 3-48 mo. Response was achieved with systemic anti-inflammatory treatment in five patients and with systemic and local anti-inflammatory treatment in one. Two patients did not respond to systemic or local anti-inflammatory treatment and underwent surgery, and tenosynovitis was confirmed on histology. MRI diagnosis of posterior tibial tendinitis was obtained in two patients. In the posterior tibial tendinitis associated with systemic inflammatory disease, the duration of symptoms was 4-12 mo with response to systemic anti-inflammatory treatment.

Bone scintigraphy was performed after intravenous injection of 20-25 mCi <sup>99m</sup>Tc-MDP. A digital gamma camera with an all-purpose, low-energy collimator was used. A blood flow study was obtained in the anterior or plantar view of the feet with dynamic acquisition of 1 frame/2 sec for 32 sec. Immediate blood-pool and delayed (2-4-hr) planar scans of the feet in the anterior, medial, lateral and plantar views were obtained using static acquisition with 400,000 counts.

### RESULTS

The scintigraphic pattern of posterior tibial tendinitis found in all patients was an elongated increased uptake in the blood-flow (Fig. 1) and blood-pool phase (Fig. 2) of the study in the anatomical course of the tibialis posterior tendon at the medial malleolus region. The delayed static images demonstrated

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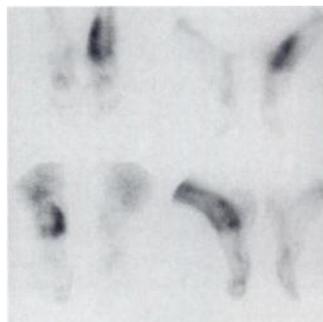
**FIGURE 1.** Blood flow images in plantar view showing elongated increased uptake along the anatomic course of the tibialis posterior tendon at the medial aspect of the left ankle.

increased focal abnormal uptake at the medial malleolus and in the navicular bone (Fig. 3). Patients with systemic inflammatory disease showed the same pattern in the posterior tibial tendon affected associated with sites of increased  $^{99m}\text{Tc}$ -MDP uptake at other entheses and/or joints involved (Fig. 4).

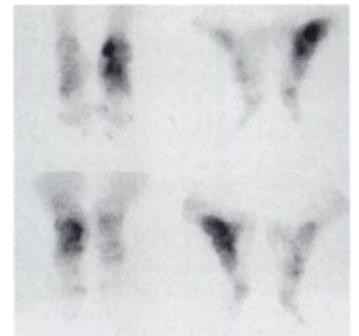
## DISCUSSION

Posterior tibial tendinitis is an inflammatory process in the tendon and tendon sheath of the tibialis posterior muscle. The tibialis posterior tendon starts at the distal third of the leg passing behind the medial malleolus and is mainly attached to the tuberosity of the navicular bone and into the medial cuneiform bone. There are other minor insertions at the intermediate cuneiform bone and the bases of the second, third and fourth metatarsal bones (2,4). The tibialis posterior muscle executes plantar flexion and inversion of the foot and contributes to the preservation of the longitudinal arch of the foot. Dysfunction of the tibialis posterior tendon causes planovalgus deformity of the foot and inability to supinate and invert the foot while the toes are being bended (4,13).

Originally related to rheumatoid arthritis patients presenting with foot pain and planovalgus foot deformity, inflammation and rupture of the posterior tibial tendon occur more frequently than previously believed (3,4). Inflammatory changes in the tibialis posterior tendon can be caused by mechanical macro- or microtrauma or as a local manifestation of a systemic inflammatory process such as rheumatoid arthritis, gout or seronegative spondyloarthropathy (3,4). The role of rheumatic conditions in the inflammatory and degenerative process found in tendinitis is well-recognized (3). Idiopathic posterior tibial tendinitis unrelated to rheumatic disease is believed to be due to trauma or mechanical process (3,4,13). Repetitive trauma may lead to microtears of the tendon and eventually produces an inflammatory response. Mechanical compression and constriction of the tendon beneath the flexor retinaculum or a sharp angle turn behind the medial malleolus may create excessive frictional forces that degenerate the tendon (3). It seems that the combination of predisposing mechanical factors and trauma initiates the inflammatory process (3). The severity of involvement of the tibialis posterior tendon and surrounding structures



**FIGURE 2.** Immediate blood-pool images in the anterior (top left), medial (top right), plantar (bottom left) and lateral (bottom right) views showing elongated increased uptake along the anatomic course of the tibialis posterior tendon.

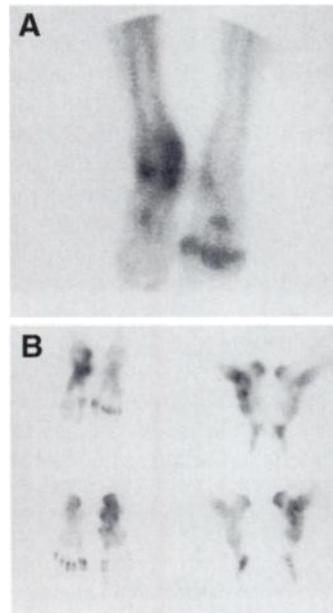


**FIGURE 3.** Delayed static images in the anterior (top left), medial (top right), plantar (bottom left) and lateral (bottom right) views showing focal increased abnormal uptake at the left malleolus and navicular bone.

vary from longitudinal splits in the deep surface of the tendon with a normal outer surface and sheath to complete rupture of the tendon with adherence of the proximal and distal portions of the tendon to subjacent bone (4). Symptoms may be present from a few months to several years and vary from minimal swelling, mild tenderness, slight weakness during inversion of the foot with minimal valgus deformity of the hindfoot to severe swelling, pain and tenderness with hindfoot dissociation (3,4).

Early recognition and appropriate management of tibialis posterior tendinitis can reduce or eliminate symptoms and can help to avoid late complications such as tendon rupture and hindfoot dissociation (3,13). CT and MRI are the techniques commonly used to evaluate the tibialis posterior tendon. These methods are useful in detecting morphological changes such as degeneration, inflammation, calcification and disruption of the tibialis posterior tendon and navicular bone avulsion (4).

Bone scintigraphy is used to detect areas of abnormal bone turnover in a variety of conditions and reflects the activity of a disease process, the inflammatory focus, rather than its morphological consequences (1,5,6,12). Tendons and ligaments are attached to the bone by the Sharpey's fibers that are metabolically active and can act like the periosteum of bone at their insertion sites (1). The scintigraphic appearance in entesopathies seems to be very characteristic. However, there is a paucity of articles about the scintigraphic findings in this disorder. Some reports concerning entesopathies at the calcaneus (plantar fasciitis and achilles tendinitis), patellar tendon, greater trochanter, inferior pubic ramus and anterior inferior iliac spine have shown the usefulness of bone scintigraphy in detecting disease activity (7-12).



**FIGURE 4.** (A) Blood-pool images in the anterior view in a patient with spondyloarthritis showing increased uptake along the anatomic course of the tibialis posterior tendon in the right foot as well as increased uptake at the metatarsal-phalangeal joints of the left foot. (B) Delayed static images in the anterior (top left), medial (top right), plantar (bottom left) and lateral (bottom right) views showing focal increased abnormal uptake at the right malleolus and navicular bone as well as increased uptake at the metatarsal-phalangeal joints of the left foot.

In the present report, a scintigraphic pattern of posterior tibial tendinitis is recognized on bone scintigraphy. Hyperemia and local abnormalities of permeability associated with the inflammatory process cause the increased uptake observed in the early phase of bone scintigraphy (vascular and extracellular fluid phase). The focal increased uptake of  $^{99m}\text{Tc}$ -MDP in the delayed phase of the study at the medial malleolus and navicular bone are probably due to increased osteoblastic activity caused by periosteal reaction at these sites.

Patients with systemic-related disease showed increased MDP uptake at multiple entheses and joints affected by the generalized disease process in addition to the tibialis posterior tendon. So, when related to a generalized inflammatory disease, additional information about the extent of disease is obtained by bone scintigraphy.

## CONCLUSION

Bone scintigraphy shows characteristic findings in posterior tibial tendinitis. This pattern should be recognized when a bone scan is performed in patients with rheumatic disease and in the evaluation of ankle and foot pain unrelated to rheumatic disease.

## REFERENCES

1. Matin P. Basic principles of nuclear medicine techniques for detection and evaluation of trauma and sports medicine injuries. *Semin Nucl Med* 1988;18:90-112.
2. Resnick D, Niwayama G. Entheses and enthesopathy. Anatomical, pathological and radiological correlation. *Radiology* 1983;146:1-9.
3. Supple KM, Hanft JR, Murphy BJ, Janecki CJ, Kogler GF. Posterior tibial tendon dysfunction. *Semin Arthritis Rheum* 1992;22:106-113.
4. Resnick D. Internal derangements of joints. In: Resnick D, ed. *Diagnosis of bone and joint disorders*, 3rd ed. Philadelphia: W.B. Saunders; 1995:3168-3182.
5. Zwas ST, Frank G. The role of bone scintigraphy in stress and overuse injuries. In: Freeman LM, Weissmann HS, eds. *Nuclear medicine annual 1989*. New York: Raven Press; 1989:109-141.
6. Merrick MV. Investigation of joint disease. *Eur J Nucl Med* 1992;19:894-901.
7. Sewell JR, Black CM, Chapman AH, Statham J, Hughes GRV, Lavender JP. Quantitative scintigraphy in diagnosis and management of plantar fasciitis (calcaneal periostitis): concise communication. *J Nucl Med* 1980;21:633-636.
8. Scuderi AJ, Datz FL, Valdivia S, Morton KA. Enthesopathy of the patellar tendon insertion associated with isotretinoin therapy. *J Nucl Med* 1993;34:455-457.
9. Intenzo CM, Wapner KL, Park CH, Kim SM. Evaluation of plantar fasciitis by three-phase bone scintigraphy. *Clin Nucl Med* 1991;16:325-328.
10. Aburano T, Yokoyama K, Taki J, Nakajima K, Tonami N, Hisada K. Technetium-99m-MDP bone imaging in inflammatory enthesopathy. *Clin Nucl Med* 1990;15:105-106.
11. Baumgarten DA, Taylor AT. Enthesopathy associated with seronegative spondyloarthropathy:  $^{99m}\text{Tc}$ -methylene diphosphonate scintigraphic findings. *Am J Roentgenol* 1993;160:1249-1250.
12. Dasgupta B, Bowles J. Scintigraphic localization of steroid injection site in plantar fasciitis. *Lancet* 1995;346:1400-1401.
13. Michelson J, Easley M, Wigley FM, Hellman D. Posterior tibial tendon dysfunction in rheumatoid arthritis. *Foot Ankle* 1995;16:156-160.

## Scatter

(Continued from page 3A)

respite from the assault and battery of everyday affairs. "Yes?," you answer with a tinge of hope that it will be something lighthearted, something pleasant. And you learn that a friend of many years died suddenly last night while having a meal in a local restaurant.

It is lunch time. I put on my coat and head to the Journal office. I pick up a salad along the way and bury myself in manuscripts and faxes. The daily ration of complaints awaits: "I have been a member for many years..."

A note to call a colleague in another city. He has an idea for a "special" article. "I wonder if you would be interested in some work I've been doing... You don't have to commit now but do you think that it would interest the readership...?" And he goes on to describe an absolutely delightful intellectual adventure. As I listened, the muscles in my scalp and face relaxed. My pulse must have slowed too. My preoccupation with all that is wrong with the world faded. I was once again listening and seeing how good things are and could be. There is sunshine behind the clouds.

What a day! Surely tomorrow will be a better one.

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