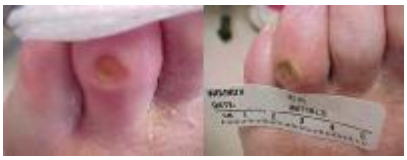
Diabetic foot ulcers, as shown in the images below, occur as a result of various factors, such as mechanical changes in conformation of the bony architecture of the foot, peripheral neuropathy, and atherosclerotic peripheral arterial disease, all of which occur with higher frequency and intensity in the diabetic population.

Diabetic ulcer of the medial aspect of left first toe before and after appropriate wound care.



Diabetic ulcer of left fourth toe associated with mild cellulitis.

Nonenzymatic glycosylation predisposes ligaments to stiffness. Neuropathy causes loss of protective sensation and loss of coordination of muscle groups in the foot and leg, both of which increase mechanical stresses during ambulation. (See Pathophysiology and Etiology.)

Diabetic foot lesions are responsible for more hospitalizations than any other complication of diabetes. Diabetes is the leading cause of nontraumatic lower extremity amputations in the United States, with approximately 5% of diabetics developing foot ulcers each year and 1% requiring amputation. (See Epidemiology.)

Physical examination of the extremity having a diabetic ulcer can be divided into examination of the ulcer and the general condition of the extremity, assessment of the possibility of vascular insufficiency,[1] and assessment for the possibility of peripheral neuropathy. (See Clinical Presentation.)

The staging of diabetic foot wounds is based on the depth of soft tissue and osseous involvement.[2, 3, 4] A complete blood cell count should be done, along with assessment of serum glucose, glycohemoglobin, and creatinine levels. (See Workup.)

A vascular surgeon and/or podiatric surgeon should evaluate all patients with diabetic foot ulcers so as to determine the need for debridement, revisional surgery on bony architecture, vascular reconstruction, or soft tissue coverage. (See Treatment and Management.)

Cilostazol is contraindicated in patients with congestive heart failure. See Medication regarding the product's black box warning.

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