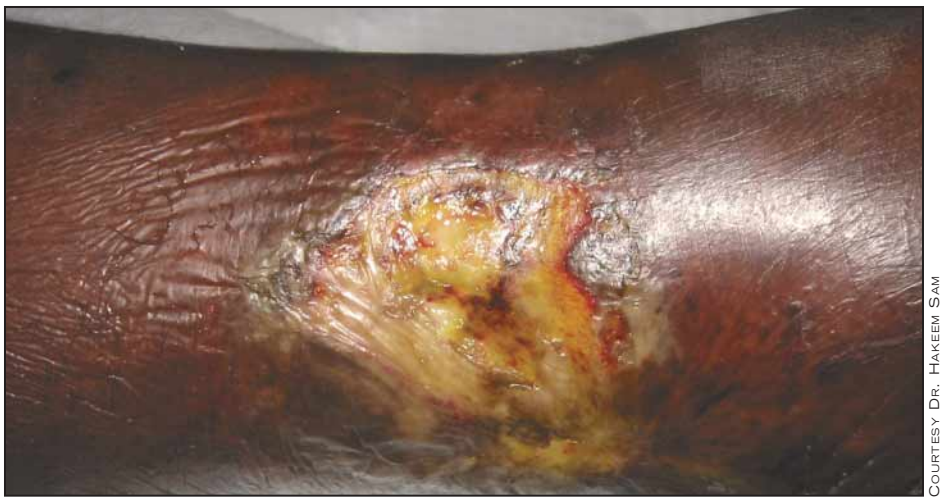
Derm Dx

A 29-year-old black man whose history included gallstones and a recurrent left leg ulcer presented with acute chest pain, fever, dyspnea, and a new and painful ulcer on his left leg. He was not a smoker and didn't drink alcohol. What's your diagnosis?



Courtesy Dr. Hakeem Sam

Toronto — A complete blood count showed an increased number of activated endothelial cells, said Dr. Hakeem Sam of McGill University, Montreal.

The leg ulcer was associated with sickle cell disease. The interaction between the activated endothelial and sickle cells led to increased expression of endothelial cell adhesion molecules, which promoted thrombotic vaso-occlusion, causing the ulcer.

Leg ulcers are not uncommon complications of sickle cell disease, especially in Jamaica and Nigeria. Data from a study in the British Journal of Haematology showed that 75% of 183 sickle cell patients in Jamaica had significantly more venous incompetence, compared with 39% of controls (Br. J. Haematol. 2002; 119:567-71).

Reported treatments for leg ulcers in sickle cell disease patients include wet dressings, hydroxyurea, transfusions, recombinant erythropoietin, zinc sulfate supplements, and surgical grafts and flaps.

In this case, the wound base was cleaned via autolytic and enzymatic debridement to avoid trauma to the wound bed. The wound was treated with a nonadherent dressing, and the patient received medication for local and systemic pain. Foam, Hydrofiber, and calcium alginate were used to manage wound exudate, according to Dr. Sam.

—Heidi Splete

Verbatim

'The fact is, when elderly people are depressed, they don't take their ACE inhibitors. It's as simple as that.'

Dr. William J. Hall, p. 51

New Drug Slows Cell Destruction In Sickle Cell

San Diego — A new drug helps slow accelerated red blood cell destruction that is a characteristic feature of sickle cell disease, Kenneth I. Ataga, M.D., reported at a press briefing during the annual meeting of the American Society of Hematology.

The drug, known as ICA-17043, is part of new class of drugs known as Gardos channel inhibitors. Potassium loss from red blood cells (RBCs) may occur via the Gardos channel, explained Dr. Ataga of the University of North Carolina, Chapel Hill. ICA-17043 blocks this channel and prevents accelerated potassium efflux, which in turn prevents dehydration of RBCs.

In a 12-week, multicenter, phase II study, he and his associates randomized 80 patients with sickle cell anemia to one of three groups: low dose ICA-17043 (6 mg/day), high dose ICA17043 (10 mg/day), or placebo. Total hemoglobin increased significantly from baseline among patients in the high-dose group and markers of RBC destruction decreased, compared with those in the placebo group.

—Doug Brunk