Sickle cell anemia is one of the most common genetic diseases and was first described by Herrick (1), in 1910. It is frequent but not exclusive in black individuals, and originates from the replacement of the amino acid valine (Val) by glutamine (Glu) at position 6 in the beta globin chain, resulting in hemoglobin S. In hypoxic conditions, the erythrocytes with predominantly hemoglobin S (HbS) take the shape of a sickle – hence the name of the disease – due to polymerization of hemoglobin S (2-3).

The hemoglobin S gene is highly frequent in tropical Africa and among black people from countries involved in slavery traffic (4). In Brazil, approximately 0.1 to 0.3% of the black population is affected by the disease (5-6) and it is estimated that at least two million individuals are HbS carriers (heterozygotes). The clinical manifestations of the disease are present as from the first year throughout life and vary much (7).

One of the complications of sickle cell anemia is leg ulcers, which usually initiate as lesions that are small or related to external factors. The ulcers are difficult to treat and heal, and require a perfect and coordinated cascade of cell and molecular events interacting to result in reconstruction of tissues (8). Such events comprise a dynamic process, involving biochemical and physiologic phenomena that act in a harmonious manner to ensure tissue restoration (Figure 1A and B).

The ulcers have a significant social and economic impact due to their recurrent nature and to the prolonged period between onset and healing. Hence, due to the need of prolonged therapies, ulcer patients often require care delivered by diverse healthcare professionals, they are frequently on sick leave and usually retire earlier (9). All these factors result in a marked burden to health and welfare systems due to high costs of treatment, absenteeism and unemployment, in addition to interfering in the quality of life of patients from having less pleasant daily activities.
REFERENÇAS