SKIN MANIFESTATIONS OF INTERNAL DISEASE

PYODERMA GANGRENOSUM WITH SPLEEN INVOLVEMENT AND IGG KAPPA PARAPROTEINAEMIA

S Mai [1] - S Mansouri [1] - M Meziane [1] - N Ismaili [1] - L Benzekri [1] - K Senouci [1] - B Hassam [1]

Ibn Sina University Hospital, Dermatology, Rabat, Morocco [1]

Background: Pyoderma gangrenosum (PG) is a rare, non-infectious ulcerative neutrophilic dermatosis. In exceptional cases, the aseptic infiltrate in PG can touch other organs than the skin such as lungs and spleen. More than 50% of PG cases are associated with an underlying systemic disease, such as inflammatory bowel disease, inflammatory arthritis, hematological disorder or malignancy. The diagnosis of PG is based on the clinical criteria, in conjunction with compatible histology.
We report a case of PG associated with IgG- kappa paraproteinaemia with a history of aseptic splenic abscesses.

Observation: A 56 year-old woman with a rich medical history: protein c and s deficiency under oral anticoagulant, a splenectomy 4 years ago for splenic abscesses and also a collection of the the right cheek 2 years ago, surgically drained. Both splenic and cheek collections were sterile. Histological examination of the spleen showed large numbers of neutrophils.
The patient presented with a one month history of painful rapidly growing ulceration on the right leg. The rest of examination was normal. Histological findings were consistent with a PG.
Serum protein electrophoresis, showed an IgG- kappa paraproteinaemia. Standard radiograms were performed, without signs of multiple myeloma. Cytology and histology of the bone marrow were normal.
The ulcer started to heal two weeks after treatment with prednisolone was initiated.

Key points: PG is an auto-inflammatory condition that can reveal many systemic hematological or neoplastic disorders. This entity does not only affect the skin. In fact, few cases of authentic spleen or lung involvement have been reported. The particularity of our case is that the spleen and oral mucosa abscesses preceded the skin lesions by many years making it almost impossible to set the diagnosis until the onset of the skin lesions and the monoclonal gammapathy that confirmed the diagnosis years later.