DIAGNOSIS

In our patient, three dissimilar skin lesions were present during a septic episode. On her ankle, a classical ecthyma gangrenosum lesion was observed. This lesion is pathognomonic for Pseudomonas Aeruginosa bacteremia and indeed, blood and urine cultures from this patient grew Pseudomonas Aeruginosa. Due to hematogenous spread, the organism invades the arteries and veins in the dermis and subcutaneous tissues leading to vasculitis and necrosis. Ecthyma gangrenosum lesions are commonly seen on the legs and are predominantly present in immunocompromised patients. Secondly, purple maculae were observed on her fingers. A previous report on patients with a few of these so called “nail fold lesions” has been published and these lesions are known to co-exist with rheumatoid arthritis/UCTD and are a sign of occlusive vasculitis. However, during this septic episode, the size and number of her nail fold lesions severely increased, which was interpreted as a local exacerbation of autoimmune associated occlusive vasculitis as a result of generalized systemic immune activation during sepsis. In addition, purpura were observed on her buttocks. However, in this case, the numerous and spread out lesions did not appear to be necrotic and were not consistent with the ecthyma lesion we observed on her ankle. A biopsy was taken and showed a granulocytic infiltration consistent with a leucocytoclastic vasculitis. No thrombi or necrosis were observed. Leucocytoclastic vasculitis is also known as “hypersensitivity vasculitis” and is most often idiopathic. It usually results from deposition of immune complexes at the vessel wall, although non-immune complex mediated mechanisms may be involved as well. There are many other triggers including, but not limited to, sepsis, infections, neoplasms, inflammatory disorders, and drugs.

Taken together, in this case several vasculitis skin eruptions appeared together during sepsis and in threes: an infectious necrotic vasculitis (ecthyma) on the ankle, an exacerbation of pre-existent occlusive autoimmune-related nail fold lesions and the occurrence of a cutaneous small vessel leucocytoclastic vasculitis near the buttocks. After several days of antibiotic treatment for Pseudomonas Aeruginosa, all three forms of skin lesions rapidly resolved indicating that the treatment of bacteremia as well as the decrease in systemic inflammatory activation may be all that is required to treat these skin abnormalities.

REFERENCES