

Cutaneous Leukocytoclastic Associated to Pulmonary Tuberculosis

To the Editor: The causes of leukocytoclastic vasculitis are varied and among them one can find those associated to an infectious cause such as infection by *Mycobacterium tuberculosis*¹; however, there are very few cases described²⁻⁵ and the mechanism is still uncertain.²

We describe the case of a young man with lung tuberculosis whose initial presentation was that of a lower limb leukocytoclastic vasculitis.

A 39-year-old male who was a smoker and had no other important aspects to his history, had presented in the past 2 weeks with pain and swelling of the knees, ankles, and metacarpophalangeal joints of the hands. He also presented skin lesions on the lower limbs (Figure). He mentioned that in the previous months he had also had cough and expectoration, without fever or dyspnea. Arthritis had improved with anti-inflammatory drugs. Upon physical examination the only finding was the presence of non-palpable purpuric lesions that did not change color under pressure, on both legs, without signs of arthritis or other findings. The hemogram and coagulation panel did not show any alterations. The blood chemistry showed: glucose, 143 mg/dL; cholesterol, 223 mg/dL; GGT, 82 U/L; alkaline phosphatase, 293 U/L; LDH, 484 U/L; the rest without alterations. Immunoglobulins, proteins, and complement were normal. Autoantibodies (ANA, ANCA) were negative. Erythrocyte sedimentation rate was 78 mm, and C-reactive protein, 54.20 mg/dL. Serology for *Coxiella burnetii*,

quantitative RPR, hepatitis, HIV, and Q fever were negative. On the chest x-ray there was atelectasia of the right superior lobe with a central cavitation and an air-water level probably secondary to a tuberculous abscess. The Ziehl-Nielsen stain of sputum showed abundant acid-alcohol resistant bacilli and *M tuberculosis* was isolated in the culture. A biopsy of the skin lesions was carried out. The pathological analysis showed neutrophilic vasculitis of the small superficial vessels (leukocytoclastic vasculitis) and immunofluorescence did not show the deposit of immunoglobulin's, complement, or fibrinogen. Given the findings of the complementary tests, antituberculosis treatment with isoniazide, rifampin, and pirazinamide was decided upon, with a good response and tolerance. The skin lesions presented reduction in size until disappearing after 2 weeks of treatment.

Tuberculosis produces 2 types of skin lesions: through a direct mechanism, where the microorganism is present (inoculation from the exterior, skin mycobacteriosis of endogenous origin, or through hematogenous dissemination) or through hypersensitivity vasculitis, where the microorganism cannot be found and the proposed pathogenic mechanism is the deposit of immunocomplexes formed by antibodies against proteins from *M tuberculosis* on the walls of small caliber vessels, though this mechanism is not clear yet.^{1,3-5} As happens with other granulomatous diseases, such as sarcoidosis or Crohn's disease, immunocomplexes have been found in active tuberculosis.⁴ The titer of these immunocomplexes is related to the activity of the disease and tends to fall after antituberculosis treatment has been started.^{2,4} Very few cases of skin small vessel vasculitis have been associated to lung tuberculosis, one of which is Schoenlein-Henoch's purpura and the rest are typical leukocytoclastic vasculitis.²⁻⁶ There are also reports of cases associated to treatment with rifampin.⁴ Vasculitis can be the first symptom of tuberculosis, as in our case, or appear after the diagnosis. The association of tuberculosis and vasculitis occurs more frequently in patient with normal immunity and chronic, untreated tuberculosis. It can affect the small vessels of the lower limbs.² Treatment of vasculitis is that of the underlying infection and only in exceptional cases is the use of corticosteroids necessary.^{2,3,5,6}

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Figure. Macroscopic vision of skin vasculitis lesions on a foot.

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