The publication about diffuse lepromatous leprosy and lucio’s phenomenon, by lucio and alvarado (1852) is remarkable. This work preceded the definition of leprosy as an infectious disease, however, the clinical observation is perfect, describing rigorously the main characteristics of this manifestation of leprosy. Very recently, more than 150 years later, xiang et al. In 2008 published a paper in which they have used modern and complex molecular techniques and concluded that the agent of diffuse lepromatous leprosy would be a new described mycobacteria, mycobacterium lepromatosis.

In this regard, we would like to make some comments in the pure clinical aspects of this condition, taking into consideration that in their paper we found pathological and clinical concepts very diverse from our experience at the lauro de souza lima institute. One of them is that the lucio’s phenomenon would be a vasculitis. In diffuse lepromatous leprosy we observe massive bacillary invasion (much more intense than in the common lepromatous leprosy) in the skin, nerves and viscera, usually involved. It stands out the involvement of arteries and veins, in the dermis and subcutis (by lepromatous infiltrate and bacilli), besides intense parasitism of all cutaneous microcirculation. The skin biopsies of lucio’s phenomena show thrombosis on arteries and veins in the deep dermis and subcutis, and also in the cutaneous microcirculation. Based on general pathology concepts, this doesn’t characterize acute vasculitis. It is possible that ischemic necrosis of dermal structures and specific infiltrate, as well as secondary neutrophilic aflux with nuclear fragmentation suggest the occurrence of a leucocitoclastic vasculitis. On the other hand, the occlusion of all microcirculation with fibrin thrombus is also not a feature of acute vasculitis. The coagulation alterations could be related to massive and generalized endothelial bacillary parasitism. In 3 out of 4 autopsies of individuals who died in the course of lucio’s phenomenon we found vascular thrombosis in several localization in the bodies, in addition to skin. This suggests that in the genesis of lucio’s phenomenon there would be some type of blood coagulation alteration.

Until recently we had the impression that diffuse lepromatous leprosy would develop in individuals extremely anergic to m. lepra, allowing the mentioned massive bacillary infiltration. The work by xiang et al., which is based mostly in mycobacteriological grounds, suggests that a different species of mycobacteria would cause this different manifestation of leprosy, despite the fact that the mycobacteria remains invading nerves, which is a unique characteristic of m. lepra.

The diffuse lepromatous leprosy and lucio’s phenomenon are rare occurrences in brazil, as opposed to mexico and central america. However, occasionally characteristics cases are reported without any specific relation with geographic location suggesting a possible random distribution of mycobacterium lepromatosis among brazilian leprosy cases, if, indeed, this mycobacteria is the agent for diffuse lepromatous leprosy. In this context, it would be interesting to engage in a study using the methodology proposed in xiang’s et al. To elucidate the distribution of the new species of mycobacteria.
REFERÊNCIAS

1 Lúcio, r., Alvarado y. – Opúsculo sobre el mal de san lázaro o elefanclasis de los griegos. M. Murguia y cia. México, 1852:53.

