Leprosy with Necrosis in Granulomatous Reaction

Case 1: A 43-year-old, white man presented with a four-month history of six plaques well-circumscribed. Laboratory results, including several serological tests, as well as a radiological investigation, were either negative or normal. The Mitsuda reaction was positive (9 mm). He was put on prednisone therapy and multi-drug therapy (MDT) with subsequent healing of the cutaneous lesions and improvement of the neuritis in about six months. The diagnostic hypothesis was reactional tuberculoid leprosy (Figure 1).

Case 2: A 23-year-old, mulatto man with a two-year history of prednisone treatment for bouts of nodules appearing over a macula who had been on treatment for tuberculosis leprosy with MDT-PB. Laboratory data, which included a search for other infectious diseases (HIV, HTLV-1 and 2, hepatitis B and C, syphilis, tuberculosis, and chest X-ray) was normal/negative. However his tuberculosis skin test (PPD) was strongly positive (30 mm), and the Mitsuda reaction was also positive (7 mm). A biopsy revealed a chronic granulomatous dermatitis with caseous necrosis. Although a search for M. tuberculosis was negative by immune-histochemistry and PCR, he received conventional anti-TB therapy, with improvement of the nodules and the infiltrative lesions. Hypotheses: Tuberculoid leprosy in one reaction with caseous necrosis or Tuberculosis and Tuberculoid leprosy?

Discussion: Various infectious/inflammatory diseases are associated with necrosis [1-3]. Caseous lesions are frequently seen in tuberculosis but are rare in leprosy granulomas [4]. In case 1, the corticotherapy may have accelerated the healing of the ulcers and limited the fibrinoid necrosis, avoiding the deleterious effects of exacerbated host cell-mediated immunity [5]. In case 2, the two-year persisting caseous necrosis could have been due to a re-infection partially controlled by rifampicine. The strongly-positive PPD might be due to a cross reactivity between M. tuberculosis and M. leprae. However, neural involvement, the histopathology, and the lack of other pathogens, point to a leprosy granulomatous reaction [6-8]. These cases illustrate the difficulties in establishing a definitive diagnosis of a chronic infectious disease, when it is accompanied by a strong cellular immunity response, usually resulting in complete elimination of the invading organisms [9-10]. This difficulty emphasizes the need for careful anamnesis and dermato-neurological examination, looking for areas of anesthesia and neural thickening, to establish the diagnosis of leprosy, while we await improvement in the laboratory diagnostic techniques for mycobacteria.

References

Maria Ângela Bianconcini Trindade, Brandt H., Teixeira R., Sotto M.N. and Fleury R.N.
1Dermatology, Clinical Hospital, Medicine Faculty of São Paulo University; 2Health Institut and 3Lauro Souza Lima Institut, Department of Health State of São Paulo; São Paulo, SP, Brazil
E-mail: angelatrindade@aol.com.br