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 tuberculoid 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?

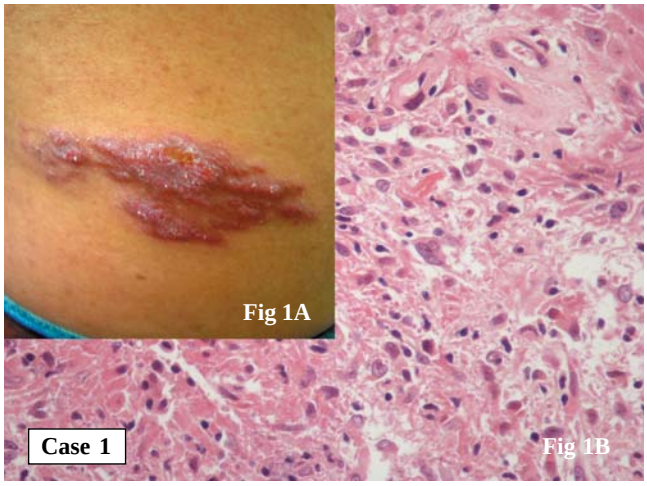

Figure 1 A. A well-circumscribed ulcerated and infiltrated patch, with altered temperature sensitivity; **Figure 1B.** HE staining shows a chronic granulomatous (CG) dermatitis with fibrinoid necrosis and a thickened nerve. Faraco and Grocott negative.

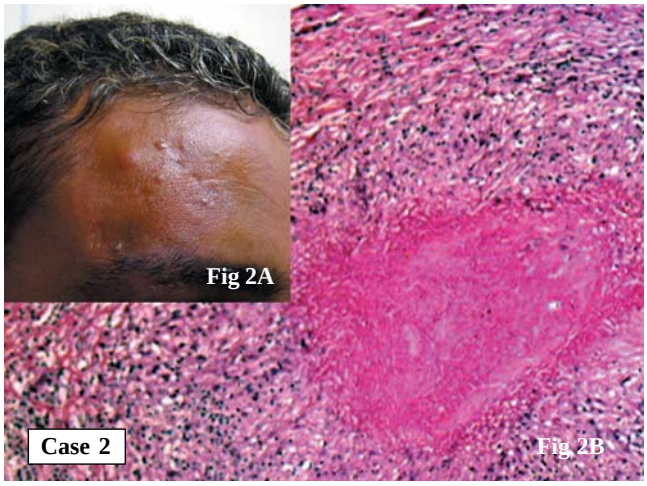

Figure 2A. A fronto-temporal erytemathous and infiltrative 4 cm patch, with well-defined borders and nodulo-cystic lesions larger than 0.5 to 2 cm, with purulent secretion; **Figure 2B.** HE: Chronic granulomatosus reaction with extensive caseous necrosis and neuritis on dermis and hypodermis.

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 reinfection 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.

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