CASE REPORT

Generalized anetoderma in a patient with HIV and dual mycobacterial infection

ARUN C. INAMADAR, APARNA PALIT,
S. B. ATHANIKAR, V. V. SAMPAGAVI &
N. S. DESHMUKH
Department of Dermatology, Venereology and Leprology,
BLDEA’s SBMP Medical College, Hospital & Research Centre,
Bijapur 586103, Karnataka, India

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Summary A middle-aged HIV infected man receiving treatment for pulmonary tuberculosis, presented with a febrile illness along with evanescent, erythematous nodular lesions all over the body. On examination, he had features suggestive of lepromatous leprosy with lesions of erythema nodosum leprosum. In addition, there were multiple small, circumscribed areas of slack skin, clinically and histopathologically suggestive of anetoderma. Both leprosy and HIV infection are known to give rise to lesions of anetoderma. Pathogenesis of anetoderma in these infectious conditions is discussed.

Introduction

Anetoderma is a clinical entity with localized areas of slack skin, manifested as circumscribed areas of bulging, histopathologically characterized by loss of dermal elastic tissue.1 Sometimes the lesions are primary, and the predisposing factors remaining obscure. Many dermatoses are known to produce secondary anetoderma, in which lesions are produced at the sites of inflammation. Association with several infectious diseases such as syphilis, borreliosis, varicella, pityriasis versicolor, leprosy and HIV infection have been described.1 The pathogenesis of selective destruction of dermal elastic tissue in these infectious conditions remains elusive.

Case report

A 35-year-old male labourer attended the dermatology outpatient department for recurrent crops of erythematous, painful, nodular lesions over extremities and back of 15 days’
duration. This was associated with fever, joint pain, redness of eyes and swelling of both feet. He had been diagnosed as a case of pulmonary tuberculosis 3 months previously and received rifampicin, INH, pyrazinamide and ethambutol. At the time of presentation, he was on rifampicin and INH daily. He was promiscuous in nature and had had multiple unprotected sexual exposure to commercial sex workers in the past.

On clinical examination, he was found to have mild pallor, generalized lymphadenopathy and marked pitting pedal oedema extending up to mid-legs. Cutaneous examination revealed multiple, dusky red, tender, nodular lesions over back, upper chest and face. There was diffuse infiltration of skin over face, upper back, chest and arm. Superciliary madarosis was present and ear lobules were thickened. In addition to these, numerous 2–3 cm circumscribed, thin, shiny, wrinkled, loose areas of skin with pouch-like appearance were seen all over the body, especially over the back, chest and proximal extremities (Figures 1 and 2). There was bilateral, symmetrical, thickening of the peripheral nerves with gloves and stockings type of hypeaesthesia. Marked congestion was seen at the corneoscleral junction of both eyes and both the testicles were acutely tender. Systemic examination revealed diminished breath sounds in the right upper zone. A provisional diagnosis of LL leprosy with type 2 lepra reaction and pulmonary tuberculosis on therapy was made.

Investigations revealed mild anaemia, peripheral blood leucocytosis and raised ESR. There was mild albuminuria. Chest X-ray revealed right upper zone fibrosis. Sputum was negative for acid-fast bacilli. VDRL test was non-reactive. ELISA for HIV was positive and CD4+ T cell count was 300. Slit skin smear from both ear lobules revealed a BI of 5+ and MI 0. Skin biopsy was taken from infiltrated skin on the back and also from one of the
circumscribed areas of slack skin. In both the specimens, flattening of the epidermis, a grenz zone and diffuse dermal cellular infiltrate composed of lymphocytes, histiocytes, plasma cells and foamy macrophages were seen. There was destruction of the dermal appendageal structures. Fite Faraco stain demonstrated numerous red stained fragmented bacillary structures. Features were consistent with lepromatous leprosy. The second tissue specimen, on Van Giesson stain, showed greatly reduced, shortened and fragmented elastic fibres at the periphery with total loss of elastic tissues at the centre. Collagen fibres were reduced. These histopathological features of the lesion were consistent with anetoderma.

The patient’s antituberculous drugs were continued. Daily dapsone (100 mg) and clofazimine were added. Prednisolone (40 mg) was started, tapering gradually over 6 weeks, and the patient’s erythema nodosum leprosum (ENL) lesions and other systemic symptoms improved. He was prescribed antiretroviral therapy.

**Discussion**

The primary defect in anetoderma is damage to the dermal elastic tissue.\(^1\) The underlying pathogenic mechanism in primary anetoderma is not clearly defined as yet, though involvement of an autoimmune mechanism is suspected.\(^2\) Some granulomatous disorders like leprosy are also known to cause anetoderma,\(^3\) the precise mechanism being still unknown.

Several suggestions have been made regarding the pathogenesis of anetoderma in HIV-infected patients. Different autoimmune disorders, both cutaneous and systemic, have been reported in association with HIV infection.\(^2\) Probably, primary anetoderma represents one of
the autoimmune disorders associated with HIV infection. The lesions of primary anetoderma in HIV infected patients occur before a significant fall in the CD4+ T cell count. In one reported series of patients, the occurrence of anetoderma was reported at a relatively early stage of immunosuppression in HIV infected patients. The authors proposed this as an early marker of HIV infection. Other dermatoses occurring in association with HIV infection can give rise to secondary anetoderma.

Some authors have found increased anticardiolipin antibodies in HIV infected patients with anetoderma. It has been proposed that these autoantibodies and immune complexes potentiate the development of secondary anetoderma by direct endothelial damage or complement activation and immune complex formation. Another proposal involves the role of vitronectin, which normally promotes integration of dermal elastic fibres with other components of the matrix and protects elastic fibres from complement mediated tissue destruction. Decreased hepatic production of vitronectin in HIV infected patients results in loss of this protective function, in addition to decreased binding of free plasminogen activator inhibitors and other serine protease inhibitors to tissues. This leads to increased serine protease activities, including elastase, which affect the dermal elastic tissue.

The coexistence of histopathological features of LL leprosy and loss of dermal elastic tissues in the same lesion in this patient suggests that these are secondary anetoderma for which leprosy may be the initial predisposing cause. The patient was also on treatment for pulmonary tuberculosis. Co-occurrence of tuberculosis and anetoderma has been reported. However, there is no clear relationship between the two entities.

It is possible that the intense inflammation associated with ENL had precipitated dermal elastolysis and the earlier ENL lesions had healed with clinical features of anetoderma. In routine practice, such extensive generalized lesions as this patient had are not usually encountered in leprosy patients with ENL. The co-existent HIV infection might have induced the severity of anetoderma in the present case.

References