Generalized anetoderma: An unusual manifestation of secondary syphilis treated with injection penicillin

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Abstract

Anetoderma also called macular atrophy is a rare, benign disorder characterized microscopically by the pan-dermal loss of elastic fibers in the dermis and presenting clinically as circumscribed, skin-colored or gray-white atrophic macules and/or patches on the trunk and/or extremities. Lesions are described as having a "sac-like" appearance, since they bulge or herniate upon palpation. It is a rare benign condition of diverse etiology; whose characteristic is the diminution or absence of the dermal elastic fibers. Anetoderma is divided into primary (idiopathic) and secondary anetoderma, with the former occurring in areas of previously normal skin and the latter developing in areas of prior skin pathology. Both may occur in association with underlying systemic conditions and warrant evaluation for associated disorders. There are no effective treatment options for anetoderma at present. We report here an unusual case of generalized anetoderma occurring in association with secondary syphilis treated with injection benzathine penicillin.

Key words: Anetoderma, elatolysis, macular atrophy, penicillin, syphilis

INTRODUCTION

Anetoderma is a benign but rare condition with focal reduction of dermal elastic tissue resulting in localized areas of flaccid or herniated sac-like skin.[1] Anetoderma is classified as either primary or secondary. Primary anetoderma is an idiopathic condition where lesions appear on previously normal skin, whereas secondary anetoderma occurs in areas of the previous inflammatory, infectious, neoplastic skin lesions of varying types. Both types may be associated with systemic diseases.[2]

We present here the unusual case of generalized anetoderma and review the clinical presentation, possible etiology, and associated conditions.

CASE REPORT

A 39-year-old, human immunodeficiency virus (HIV) reactive male patient was referred from the ART center for evaluation of generalized skin lesions present for 4 years.

Clinical examination revealed multiple, hypopigmented to skin-colored, well-circumscribed, wrinkled, centrally depressed, 0.5–2.5 cm flesh-colored, oval-to-round atrophic sac-like lesions which protruded on forward bending. Hundreds of lesions were present over the trunk, abdomen, chest, and proximal extremities bilaterally symmetrical with sparing of the scalp, palms, soles, face, and mucous membranes [Figures 1 and 2]. Lesions were asymptomatic in nature, were present for 4 years.

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The patient gave a history of asymptomatic genital ulcer which healed on its own before 4 years followed within a month by the development of generalized papular, erythematous, inflammatory skin lesions; which were diagnosed as secondary syphilis on the basis of mucocutaneous features and positive serology for syphilis (rapid plasma regain test - RPR1: 64). The patient was treated with single dose of injection benzathine penicillin 2.4 mega units at that time by a private practitioner as the patient was HIV nonreactive at that time. His inflammatory skin lesions healed with atrophic scars and atrophic skin lesions were status quo since then. The patient sought no further treatment for skin lesions. The patient had no history of varicella, insect bites, prurigo nodularis, Lyme disease, or molluscum contagiosum or any other skin lesions. The patient was recently diagnosed as HIV reactive; 1 month before presentation to us. He was on standard tenofovir, lamivudine, efavirenz regimen, and prophylactic oral sulfamethoxazole-trimethoprim for 1 month. His CD4 count was 65 cells/mm^3 (9%) at the time of presentation. No lymphadenopathy, oral or genital lesions were observed. Other hematological investigations were within the reference range except low total WBC count with lymphocytes 11%. Serum treponema pallidum hemagglutination assay was positive. The serum RPR test was 1:2.

An atrophic macule from the lower back was biopsied. H and E section showed mildly effaced epidermis with mild interstitial edema and perivascular lymphocytic infiltrate with loss of elastic fibers in the papillary and mid-reticular dermis [Figure 3]. Masson-trichrome stain confirmed fragmented elastic fibers and loss of elastic fibers at places in the dermis [Figure 4].

DISCUSSION

Anetoderma, first described by Jadassohn in 1892, is a rare cutaneous disorder characterized by localized areas of elastolysis. Historically, idiopathic lesions were classified as Jadassohn–Pellizzari type when preceded by inflammatory skin lesions and Schweninger–Buzzi type when noninflammatory in

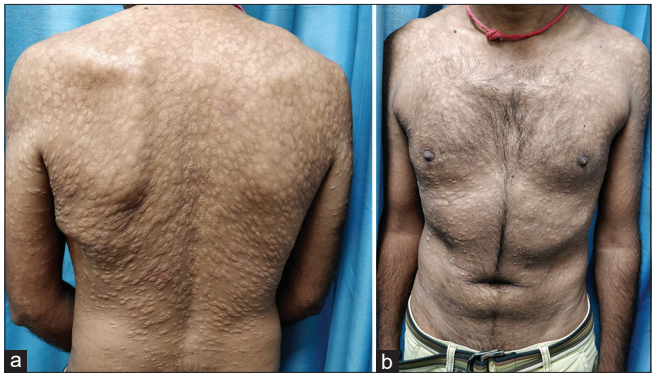

Figure 1: (a and b) Multiple, well-circumscribed, wrinkled, hypopigmented, outpouching appearing lesions on the trunk

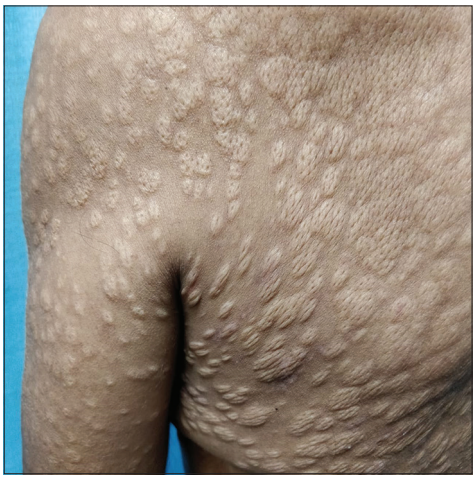

Figure 2: Close up view of lesions

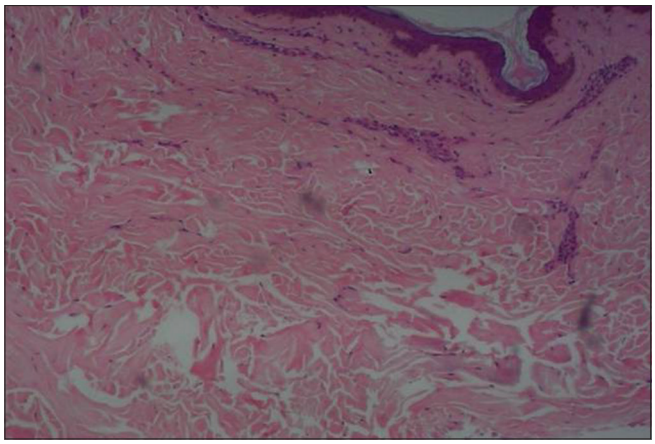

Figure 3: Mildly effaced epidermis with mild interstitial edema and perivascular lymphocytic infiltrate with loss of elastic fibers in the papillary and mid-reticular dermis (H and E, ×10)

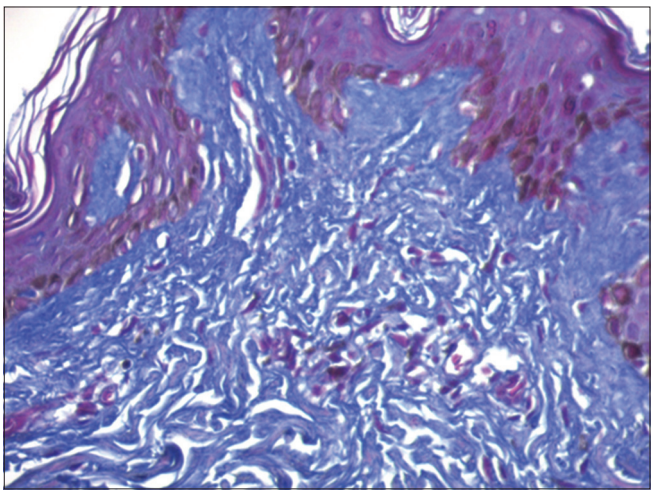

Figure 4: Normal collagen with absent and fragmented of elastic fibers in papillary and mid dermis (Masson's trichrome, ×10)

onset. Now, anetoderma is classified as primary or secondary.

Primary anetoderma represents an idiopathic occurrence of atrophic lesions in areas of the skin that appear normal before the onset of atrophy.[2] Secondary anetoderma occurs in areas of previous or current skin pathology and has been described with diverse conditions including acne, acrodermatitis chronica atrophicans, insect bites, varicella, syphilis, leprosy, tuberculosis, granuloma annulare, and urticaria pigmentosa. Secondary anetoderma may be associated with tumors, inflammatory and infectious diseases.[3] Anetoderma lesions generally appear on the upper arms, trunk, and thighs while the scalp, palms, and soles are usually spared.[1]

The exact etiology of anetoderma is not known. The loss of elastic tissue could be the result of defective elastin synthesis, uncontrolled production of elastolytic enzymes, loss of elastolytic enzyme inhibitors, elastophagocytosis, or degeneration of elastic fibers secondary to local ischemia induced by microthrombosis in dermal vessels.[4] Extracellular matrix integrity is largely maintained through a careful balance of two enzymatic families: matrix metalloproteinases (MMPs) and tissue inhibitors of metalloproteinases (TIMPs). Focal imbalance of MMPs and TIMPs favors an elastic fiber breakdown.[2] Furthermore, a recent study has shown significantly decreased dermal expression of fibulin-4 involved in elastic fiber assembly; suggesting that in addition to elastolytic overactivity, altered reassembly of elastic fibers may also play a significant role in the pathogenesis anetoderma.[5]

Secondary syphilis has been reported as one of the common causes of anetoderma and there is a report of generalized anetoderma developing after injection benzathine penicillin in a patient with secondary syphilis and HIV infection.[2] Two distinct clinical subtypes of syphilitic anetoderma are described: one characterized by numerous, subtle, atrophic lesions in a widespread, pityriasiform distribution, and the other fewer, well-defined, convex, atrophic lesions.[6]

Our patient developed generalized anetoderma after secondary syphilis treated with injection benzathine penicillin. Lesions of anetoderma in our patient were much more numerous and generalized in comparison to lesions of secondary syphilis. It is possible that intense inflammation associated with secondary syphilis produced extensive elastolysis and extensively distributed anetoderma in our patient. Our patient was diagnosed with HIV infection only recently, but it is likely that patient might have been infected sometime before as he never consulted any doctor in the past few years. It is difficult to establish the role of HIV infection in causing anetoderma in our patient.

We are reporting this case because generalized anetoderma is a rare entity. Only few cases of generalized anetoderma are reported in the literature. As anetoderma can develop in the context of infectious diseases, a diagnosis of anetoderma should trigger a thorough examination and evaluation for treatable concomitant illnesses. No satisfactory treatment option is available, but recently, resurfacing with ablative or newer fractional laser or RF systems has been shown to provide some benefit.[2] Owing to the small number of reported cases, it is difficult to develop strong conclusions regarding the exact correlation of anetoderma in the secondary syphilis and treatment with benzathine penicillin.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Aghaei S, Sodaifi M, Aslani FS, Mazharinia N. An unusal presentation of anetoderma: A case report. BMC Dermatology 2004;4:9.
2. Emer J, Roberts D, Sidhu H, Phelps R, Goodheart H. Generalized anetoderma after intravenous penicillin therapy for secondary syphilis in an HIV patient. J Clin Aesthet Dermatol 2013;6:23-8.
3. Kineston DP, Xia Y, Turiansky GW. Anetoderma: A case report and review of the literature. Cutis 2008;81:501-6.
4. Weinstein S, Piette W. Cutaneous manifestations of antiphospholipid antibody syndrome. Hematol Oncol Clin North Am 2008;22:67-77.
5. Gambichler T, Reininghaus L, Skrygan M, Schulze HJ, Schaller J, Colato C, *et al*. Fibulin protein expression in mid-dermal elastolysis and anetoderma: A study of 23 cases. Acta Derm Venereol 2016;96:708-10.
6. Clement M, du Vivier A. Anetoderma secondary to syphilis. J R Soc Med 1983;76:223-4.