Psoriasiform Sarcoidosis Presenting in Pregnancy and Treatment Considerations

Christian A Albornoz¹, Jordan V Wang²* and Matthew Keller²

¹Lewis Katz School of Medicine, Temple University, Philadelphia, PA, USA
²Department of Dermatology and Cutaneous Biology, Thomas Jefferson University, Philadelphia, PA, USA

*Corresponding author: Jordan V Wang, MD, MBE, MBA, Department of Dermatology and Cutaneous Biology, Thomas Jefferson University, 833 Chestnut Street, Suite 740, Philadelphia, PA, 19107, USA, Tel: (215)-955-6680, E-mail: jordan.wang@jefferson.edu

Abstract

Cutaneous sarcoidosis is a common presentation for patients with sarcoidosis. Rarely, patients can present with psoriasiform lesions mimicking chronic plaque psoriasis. Here, we present a case of psoriasiform sarcoidosis in a pregnant patient. Pregnancy represents a unique challenge to systemic treatments if topical management fails. Tumor necrosis factor alpha inhibitors warrant special consideration during pregnancy.

Keywords
Sarcoidosis, Psoriasis, Tumor necrosis factor-alpha, High-risk pregnancy

Introduction

Sarcoidosis is a systemic inflammatory disease characterized by granuloma formation in multiple organs of the body, with a predilection for the lungs, skin, and eyes. Cutaneous sarcoidosis presents in myriad forms, appearing in about 25% of patients, while psoriasiform lesions are a rare manifestation in only about 0.9% [1]. We report the case of a 40-year-old woman at 10-weeks' gestation with psoriasiform sarcoidosis.

Case Report

A 40-year-old African-American woman at 10-weeks gestation with a history including active pulmonary sarcoidosis and diabetes was referred for several-year history of skin lesions by maternal-fetal medicine, which recently became scalier. Her medications included hydroxychloroquine 200 mg qday, prednisone 20 mg qday, and insulin. Physical examination revealed large, well-demarcated, hyperpigmented plaques with micaceous scale on her upper extremities, knee, neck, and back (Figure 1). There were also thin hyperpigmented plaques on her temple, nose, cheeks, and chin. A punch biopsy of the arm showed granulomatous inflammation consisting of histiocytes and lymphocytes in discrete aggregations and abutting the hyperkeratotic and psoriasiform epidermis (Figure 2). Clinical pathologic correlation was consistent with psoriasiform sarcoidosis.

The patient was started on topical corticosteroids and continued on hydroxychloroquine. The prednisone was to be tapered. At follow-up, there was moderate improvement of skin lesions with near complete resolution of scaling, but still with significant classic sarcoidal appearance. Due to active pulmonary lesions, her...
treatment was to be discussed with pulmonology and maternal-fetal medicine regarding the decision to increase hydroxychloroquine or start a tumor necrosis factor alpha (TNF-α) inhibitor.

Discussion

While the etiology of sarcoidosis has yet to be fully elucidated, environmental exposures and infectious agents have been suspected [2]. The presence of non-caseating granulomas is firmly established as the key pathologic finding. These characteristic sarcoidal granulomas consist predominantly of macrophages and epithelioid cells with peripherally surrounding lymphocytes. The pathological process begins when an unknown sarcoidal antigen is phagocytosed by antigen-presenting cells. This subsequently stimulates CD4+ T cells into secreting Th1-associated cytokines, such as IL-12, IL-15, IL-18, interferon gamma, and TNF-α, leading to the formation of the characteristic sarcoidal granulomas [3]. Many of these cytokines are also elevated in psoriasis, producing irregular hyperproliferation and differentiation of keratinocytes with histopathology demonstrating parakeratosis, intraepidermal neutrophilic infiltrations, and hypogranulosis [4].

Investigations into the overlap between sarcoidosis and psoriasis have focused heavily on the similar pathologic roles of Th1 cells. Both disorders have been associated with specific gene polymorphisms, such as IL23R, and psoriasis itself has been linked with 14 other autoimmune conditions, further strengthening the argument for shared pathways [5,6]. Paradoxically, sarcoidosis and psoriasis have both been reported after the initiation of TNF-α inhibitors, hinting again at homologous pathologic factors [7]. A previous report theorized that overexpression of TNF-α from both diseases may be responsible for the psoriasiform lesions arising in areas of cutaneous sarcoidosis [8].

For now, the goal of treating sarcoidosis focuses on suppressing symptoms, as the underlying cause remains elusive. First line therapy consists of topical and intrale-}

Figure 2: Granulomatous inflammation consisting of histiocytes and lymphocytes in discrete aggregations and abutting the hyperkeratotic and psoriasiform epidermis. (A) H&E, original magnification x4; (B) H&E, original magnification x20.
References


