Pregnancy-associated *de novo* systemic lupus erythematosus in people living with HIV/AIDS

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Abstract

Pregnancy with systemic lupus erythematosus (SLE) requires special attention in view of the enhanced risks to the fetus and the aggravation of SLE during pregnancy. Human immunodeficiency virus infection can further complicate the course of pregnancy as well as the outcome. We present a case of a 28-year-old primigravida who was diagnosed case of people living with HIV/AIDS and presented with SLE at 34 weeks of gestation. Subsequent evaluation of the patient revealed latent tuberculosis also. Cutaneous lesions responded well to oral corticosteroids, however, the outcome of pregnancy was hydrops fetalis. This report highlights the complex interplay of multiple comorbidities and their adverse impact on pregnancy outcome.

Key words: Human immunodeficiency virus, pregnancy, systemic lupus erythematosus

Introduction

Systemic lupus erythematosus (SLE) is a multi-organ system disorder that predominantly affects reproductive-aged women. SLE can flare during pregnancy, especially in those who have active disease at conception.[1] Concomitant presence of SLE and human immunodeficiency virus (HIV) is rare. The immunosuppression resulting from HIV generally improves lupus symptoms, but antiretroviral therapy (ART) may trigger flares.[2] An increased risk of tuberculosis (TB) infection in SLE is noted, secondary to the disease itself or as a consequence of immunosuppressive therapy.[3] The coexistence of multiple diseases such as SLE, HIV, and latent TB further jeopardizes maternal and fetal well-being. The present case emphasizes the significance of a high-risk approach to pregnancy in people living with HIV/AIDS (PLHA), the role of screening for autoimmune diseases, and concerted multispeciality antenatal care.

Case Report

A 28-year-old female, primigravida with period of gestation of 34 weeks, presented to the dermatology outpatient department with the complaint of red raised lesions over the face, trunk, back, and extremities for the last 5 months, and painless raw areas in the oral cavity for 4 months. She was a diagnosed PLHA, was on ART since 2011 (lamivudine 300 mg + nevirapine 200 mg + zidovudine 300 mg and tenofovir 300 mg + lamivudine 300 mg + dolutegravir 50 mg from 2020 onward). She also complained of diffuse hair loss over the scalp for the past 2 months.

There was no history of fever, photosensitivity, fluid-filled lesions, joint pains, any other connective tissue disease, or any systemic complaints.

On physical examination, erythematous scaly papules and plaques along the anterior hairline, eyebrows, eyelids, nose, cheeks, lips, chin, bilateral ears, and neck were present [Figure 1a-c]. Multiple discrete erythematous nonblanchable papules suggestive of vasculitis were found over the trunk, back, and extremities including palms and soles [Figure 2a and b]. Few annular plaques were present over the upper back and chest [Figure 3a and b].

Scalp examination revealed sparsening of hair at multiple places with yellowish-greasy adherent scaling. Erosive erythematous plaques were present over the hard palate, bilateral buccal mucosa, and left retromolar trigone. Other mucosae were normal. Nails were grossly normal. Nail fold capillaroscopy revealed dilated capillary loops, tortuous capillaries, avascular areas, hemorrhagic areas, and bushy capillaries [Figure 4a-d]. Systemic examination was unremarkable. A provisional diagnosis of pregnancy-triggered *de novo* SLE was made.

On laboratory evaluation, skin biopsy from the dorsum of the hand revealed thinning of epidermis with blunting of rete ridges, hydropic degeneration of basal cell layer and focal pigment incontinence, and moderate perivascular lymphocytic infiltrate in the papillary dermis along with evidence of vasculitis. Upper dermis showed mucin deposits on Alcian blue-periodic acid–Schiff stain [Figure 5]. Direct immunofluorescence revealed focal granular deposits of IgM, C3, and fibrinogen along the dermoepidermal junction. Connective tissue profile showed anti-nuclear antibody 3+ homogenous, 1+ nucleolar, dsDNA moderately positive, anti-Smith strongly positive, anti-Ro 4+, and beta 2 microglobulin were raised and C3 was low [Figure 6]. Erythrocyte sedimentation rate and C-reactive protein levels were raised. All other biochemical investigations including liver function tests and kidney function tests were normal.

Fetal scan revealed gross ascites, diffuse body wall and scalp edema, cardiomegaly, minimal pleural effusion, fetal brain edema, and anhydramnios. The patient also had a positive interferon-gamma release assay test and IgG positive for *cytomegalovirus*, Rubella, and herpes simplex virus 1 and 2. Other investigations for systemic examination were normal. Last CD4 count levels and viral load copies performed in March 2022 were 410 cells/mm^3 (normal range is 500–1500 cells/mm^3) and 1236 copies, respectively.

The patient was admitted and started on hydroxychloroquine 200 mg twice a day and mid-potent topical steroids (mometasone) for twice a day application along with photoprotective measures and broad-spectrum chemical sunscreen. ART was continued. Subsequently, erythema over the lesions subsided and there were no new lesions. A week later, the patient delivered the fetus by normal vaginal delivery which was stillborn. After receiving 1 month of systemic steroids, lesions resolved with postinflammatory hypopigmentation [Figure 7a-e]. Isoniazid prophylaxis 300 mg a day was given for 6 months in view of latent TB along with pyridoxine 20 mg once daily. CD4 count levels after 1 month of treatment were 526 cells/mm^3 (November 2022).

Discussion

SLE is an autoimmune disorder affecting multiple systems with significant morbidity. Various diseases can be concomitantly present with SLE. These comorbidities have varying impacts on the presentation, course, management, and prognosis of SLE.

Pregnancy with SLE can result in flares of the disease. This results from the immunological imbalance in pregnancy in the form of a cytokine shift from Th1 to Th2 mediated. SLE being a Th2-mediated disease can thus flare at the time of pregnancy.[4] SLE adversely affects pregnancy in the form of impaired fertility which is seen in active phases and in renal dysfunction. Risk of premature delivery, fetal

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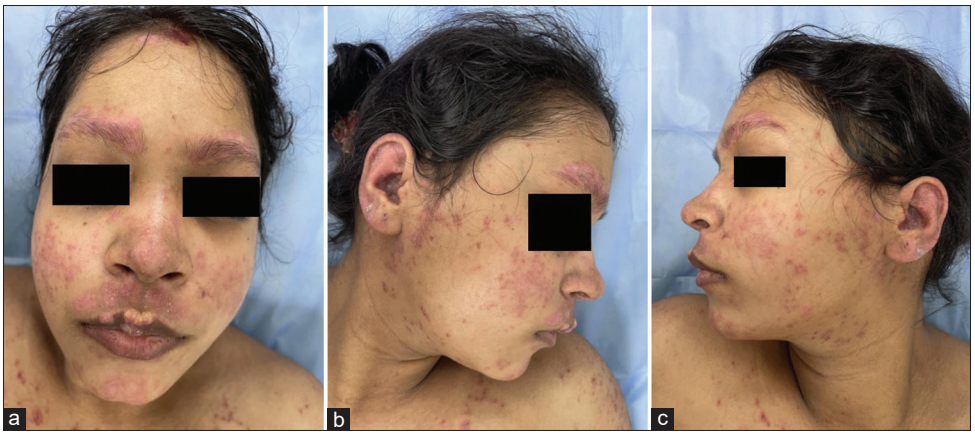

Figure 1: (a-c) Multiple discrete to coalescent, erythematous scaly papules and plaques along anterior hairline, eyebrows, eyelids, nose, cheeks, lips, chin, bilateral ears, and neck

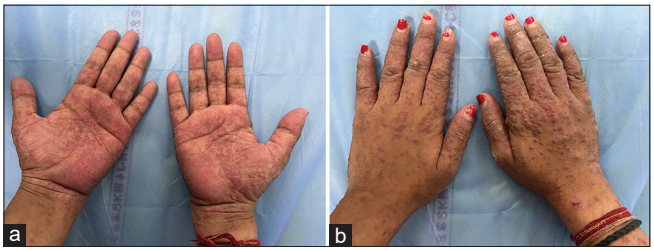

Figure 2: (a and b) Multiple discrete to coalescent, dusky red to violaceous, nonblanchable, tender, papules and plaques present over trunk, back, and extremities including palms and soles

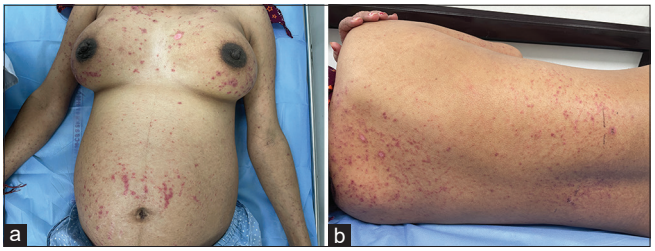

Figure 3: (a and b) Few annular plaques present over the upper back and chest

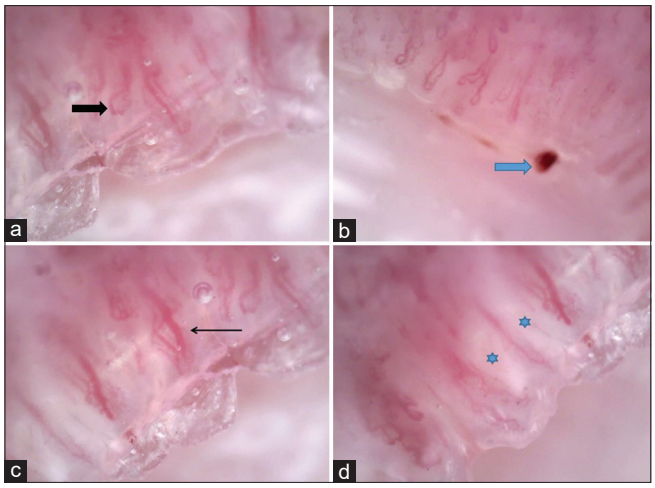

Figure 4: Nail fold capillaries showing tortuous capillaries (a), hemorrhagic area (b), dilated capillary loops (c) and bushy capillaries (d)

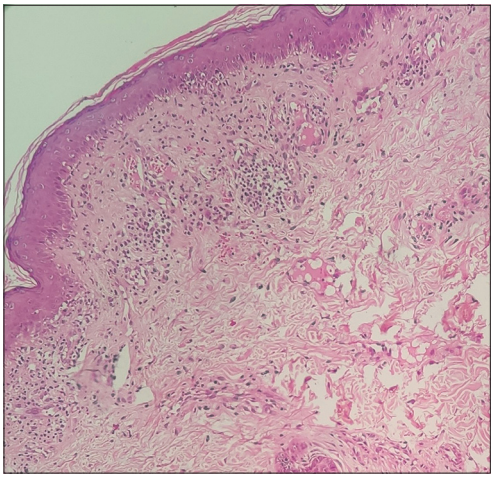

Figure 5: Histopathology from skin lesion

loss, and perinatal mortality increases in SLE, especially in cases of high disease activity, anti-phospholipid antibody positivity, and lupus nephritis. Risk of neonatal LE is approximately 5%, esp in anti-Ro + mother. Rare fetal outcomes in SLE include hydrops fetalis, hematological abnormalities, and hepatosplenomegaly.[1]

Poorest outcomes of pregnancy are seen with active nephritis or irreversible organ damage. Safe treatment options in pregnancy include hydroxychloroquine (Category C), sulfasalazine (Category A), azathioprine (Category D), cyclosporine (Category C), tacrolimus (Category C), and low-dose steroids (Category C).[1]

The effect of SLE on HIV is unclear, some authors paradoxically suggest that the presence of autoimmune diseases such as SLE may be protective against the progression of HIV disease. Others believe that SLE also affects the immune system, compromising the ability to combat infection. Therefore, coexistence may predispose to opportunistic infections. LE symptoms usually improve with immunosuppression, although ART may trigger flares.[2]

Concomitant SLE and HIV become a diagnostic challenge as both may present with similar symptoms such as constitutional symptoms including fever and malaise; dermatologic findings including alopecia, oral ulcers, and facial rash; musculoskeletal involvement such as arthralgias, arthritis, and myalgias; renal abnormalities,

including hematuria and proteinuria; central nervous system disorders, including seizures and psychosis; hematologic alterations, including anemia, leukopenia, lymphopenia, and thrombocytopenia; and immunologic features including hypergammaglobulinemia and positive ANA.[3] Hydroxychloroquine is a safe, cost-effective oral once-daily (200 mg) treatment option for HIV–SLE coexistence. ART should be continued.[5]

There is an increased risk of TB infection under the conditions of immunosuppression which can be a result of the disease itself or a consequence of immunosuppressive therapy. The risk of TB infection is high in this case due to HIV infection as well. Patients with SLE are more susceptible to the risk of dissemination of TB. Among SLE patients, extrapulmonary TB is more common while pulmonary involvement will be severe and relapses may occur frequently.[6] The effect of TB on SLE is not well understood. It is postulated that *Mycobacterium tuberculosis* being an immunomodulatory agent can precipitate SLE. Management of SLE patients with TB is the same as that of non-SLE patients. In cases of latent TB, isoniazid prophylaxis 300 mg/day for 6 months is preferred.[7]

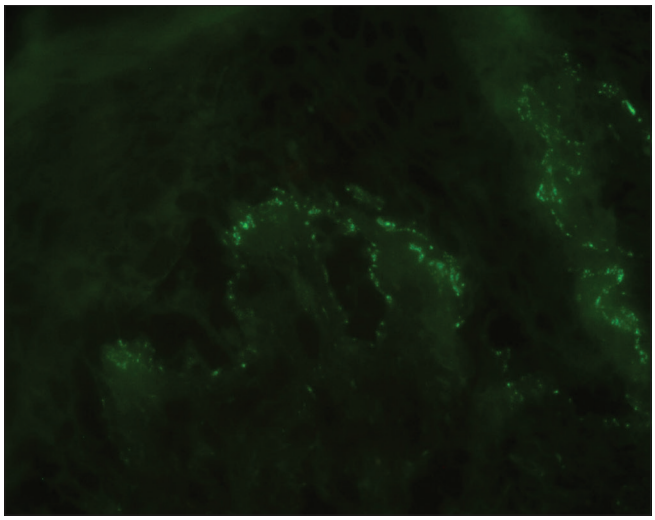

Figure 6: Direct immunofluorescence

Gayed *et al*. reported a 37-year-old woman of African origin who developed SLE on a background of well-controlled human immunodeficiency virus (HIV) infection who conceived 2 months after the diagnosis of SLE was made. Disease became more active at the start of pregnancy. She was treated with oral prednisolone 20 mg once daily, hydroxychloroquine 200 mg once daily along with azathioprine. ART was continued. Disease control was achieved and the pregnancy was uneventful.[8]

Conclusion

This case highlights an unusual association of acute SLE with pregnancy, HIV, and latent TB. The need of extensive clinical as well as laboratory evaluation is necessary in such patients, followed by an appropriate safe choice of therapy as treatment options are limited by coexisting comorbidities.

Declaration of patient consent

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

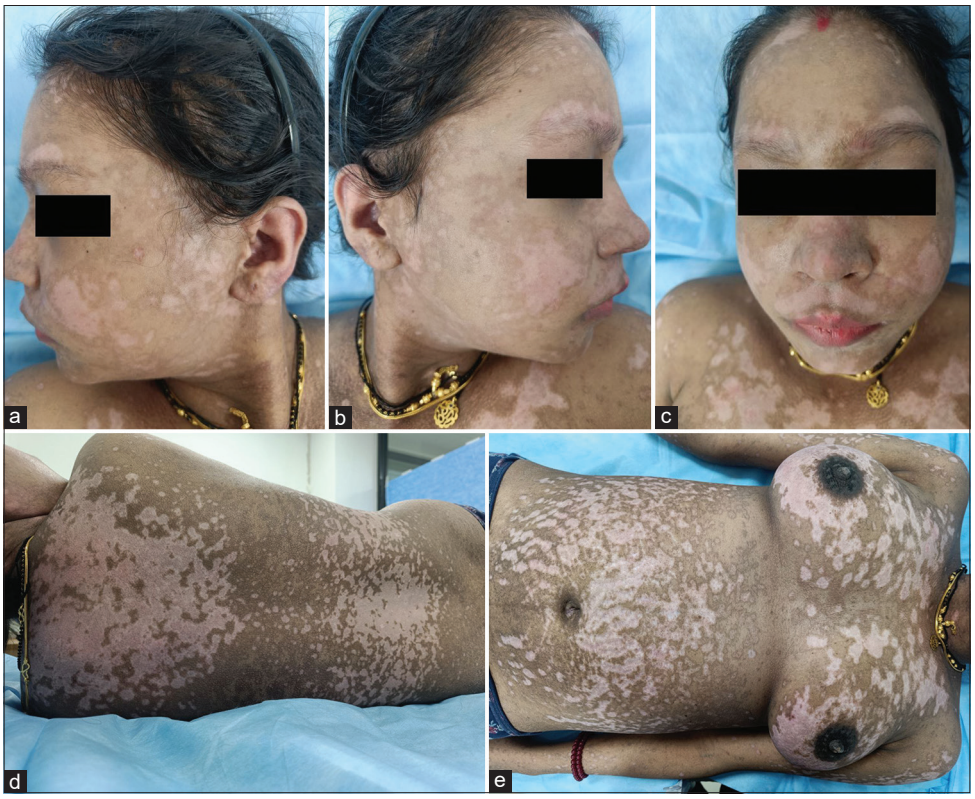

Figure 7: (a-e) Lesions resolving after treatment with postinflammatory hypopigmentation

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Conflicts of interest
There are no conflicts of interest.

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