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CASE REPORT

Combination of Oral Hydroxychloroquine and Topical Pimecrolimus 1% for Linear Morphea in Breastfeeding: A Rare Case with Favorable Outcome

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Background: Linear morphea (LM) is a chronic autoimmune disease characterized by linear or band-like localized sclerosis that may involve the dermis, subcutaneous tissue, muscle, and underlying bone. While LM typically presents in childhood, adult-onset disease is considerably less common, and LM presenting during breastfeeding has not, to our knowledge, been previously documented. As a result, therapeutic guidance for managing LM in breastfeeding women remains extremely limited, highlighting the need for additional clinical evidence in this population.

Case Presentation: A 44-year-old breastfeeding woman presented with brownish-white skin lesions with a line configuration and hardened skin on the right shoulder, upper arm, forearm, hand, and fingers. The histopathological findings demonstrated epidermal thinning with flattened rete ridges, thickened fibrocollagenous connective tissue stroma extending into the subcutaneous fat, hyaline degeneration, a mild lymphocytic inflammatory, and absence of pilosebaceous unit. The laboratory analysis revealed a positive ANA test with a titer of 1:320 and the presence of anti-RNP and anti-Sm autoantibodies. The patient received hydroxychloroquine (HCQ) 200 mg twice daily and topical pimecrolimus 1% cream twice daily. After three months of observation, there was an improvement in the skin lesions. In addition, the modified localized skin index (mLoSSI) score and the localized scleroderma damage index (LoSDI) score decreased from 6 to 1 and 5 to 4, respectively. No adverse effects related to retinal toxicity were reported during the course of therapy.

Conclusion: This case suggests that the combination of oral HCQ and topical pimecrolimus may represent a safe and effective therapeutic option for LM in breastfeeding women, although further evidence is needed to confirm long-term outcomes.

Keywords: breastfeeding, hydroxychloroquine, linear morphea, pimecrolimus 1%

Introduction

Localized scleroderma, also known as morphea, is a chronic autoimmune disease characterized by depressed, fibrotic, and dyschromic cutaneous lesions.[1] Unlike systemic sclerosis, morphea is generally not associated with extensive internal organ abnormalities.[1,2] Morphea has an estimated incidence of 2.7 cases per 100,000 people, with a female to male ratio of 2.6:1. Twenty to thirty percent of morphea begins in childhood, but it can occur at any age. Morphea is currently classified into 5 putative subtypes, including generalized, linear, mixed, deep and circumscribed (plaque-type).[2,3]

Linear morphea (LM) is characterised by unilateral linear or band-like localized sclerotic lesions that may involve the dermis, subcutaneous tissue, muscle and underlying bone, often occurring on the extremities and following Blaschko's lines.[3,4] It is the most common pediatric subtype, but can occur at any age.[2,5] Two peaks of LM incidence are observed,

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one between 2 and 14 years of age, and a second in the fifth decade of life. The reported mean age of onset for juvenile and adult LM is 10 and 45 years, respectively. The disease also has a female preponderance, with an overall female-to-male ratio of 4:1.[4] Although the precise etiology remains uncertain, several risk factors have been proposed, including genetics, autoimmune processes, trauma, infections, medications, psychological stress, vascular abnormalities, and hormonal influences.[2,4,6]

Treatment options are classified into several modalities, including topical (eg, calcineurin inhibitor (CI)) and systemic treatments.[4] Calcineurin inhibitors, such as pimecrolimus at 1% and tacrolimus at 0.1%, have demonstrated substantial efficacy in addressing morphea lesions.[4] Adult-onset LM is less documented than childhood-onset disease, and treatment considerations become more challenging in special populations such as breastfeeding women.[1] Methotrexate (MTX), the primary systemic therapy for LM, is contraindicated during pregnancy and breastfeeding.[1,4] Published reports specifically describing LM occurring during breastfeeding are extremely limited, and to our knowledge, no previous cases have been documented. This represents a significant knowledge gap in the literature. Other agents, including hydroxychloroquine (HCQ), have been demonstrated to be efficacious in various case reports of morphea,[2] but data on LM remain scarce. HCQ is considered compatible with breastfeeding because only small amounts are excreted into breast milk and no adverse effects have been reported in breastfed infants.[7–9] In addition, topical pimecrolimus has minimal systemic absorption and is regarded as compatible with breastfeeding when used on limited body surface areas.[10] In this case report, we describe a 44-year-old breastfeeding woman diagnosed with LM and treated with a combination of systemic hydroxychloroquine (HCQ) and topical pimecrolimus 1%. The diagnosis was established following a comprehensive clinical evaluation that encompassed both histopathological and serological examinations.

Case Presentation

A 44-year-old female presented to the Dermatology Allergy and Clinical Immunology Clinic with a chief complaint of brownish-white patches and plaques with hardened skin on the right shoulder, upper arm, forearm, right hand and fingers. There was no itching or pain. For the past three months, the skin lesions had spread to the wrist and fingers of the right hand, with some areas becoming harder. The patient then visited the clinic for further evaluation.

The initial complaint was documented 18 months prior to the first consultation. The patient observed a blackish-brown macule on her right upper arm, approximately the size of a small coin. Notably, the lesion had not exhibited any pruritus or discomfort. At the time of the consultation, she was in the final month of her third trimester of pregnancy. Two months after delivery, the patches increased in size and number, accompanied by hardening of the skin over the lesions. The skin lesions lasted for ten months before spreading to the right shoulder and forearm. Some patches were reported to be brownish–white. The patient had been intermittently applying an over-the-counter hydrocortisone cream to the affected areas for a month. However, seeing no improvement, the patient decided to stop using the medication. Over the three months leading up to the consultation, it was observed that the skin lesions had started to spread to the right hand and fingers. Upon examination, she reported an absence of muscle or joint pain and indicated that there were no limitations in her range of motion (ROM). Furthermore, she did not present with any symptoms indicative of wounds, cold-induced cyanosis of the fingertips and toes, finger swelling, digit lesions, dysphagia, dyspnea, nausea, vomiting, or paresthesia in the hands. No history of infection, radiation exposure, chemical exposure, trauma, or use of certain medications prior to the initial skin lesion was reported. There was no history of alcohol consumption or smoking. A review of her medical history showed no previous occurrences of similar symptoms during either her first or second pregnancy. She also had no history of hypertension, diabetes, liver or heart disease, and there was no family history of morphea or any autoimmune disorders.

A physical examination revealed multiple irregular lesions on the right shoulder, upper and lower arms, hand, and fingers, with notable skin hardening. The borders of the lesions were partially indistinct, and the lesions resembled hyperpigmented, hypopigmented, and depigmented macules, patches, and plaques with line configuration (Figure 1). There were also areas of epidermal atrophy with cigarette paper skin changes. No limitations in ROM or other associated organ disorders were identified during the physical examination.

The laboratory analysis revealed a positive ANA test with a titer of 1:320 and the presence of anti-RNP and anti-Sm autoantibodies. Complete blood count, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), urea, creatinine,

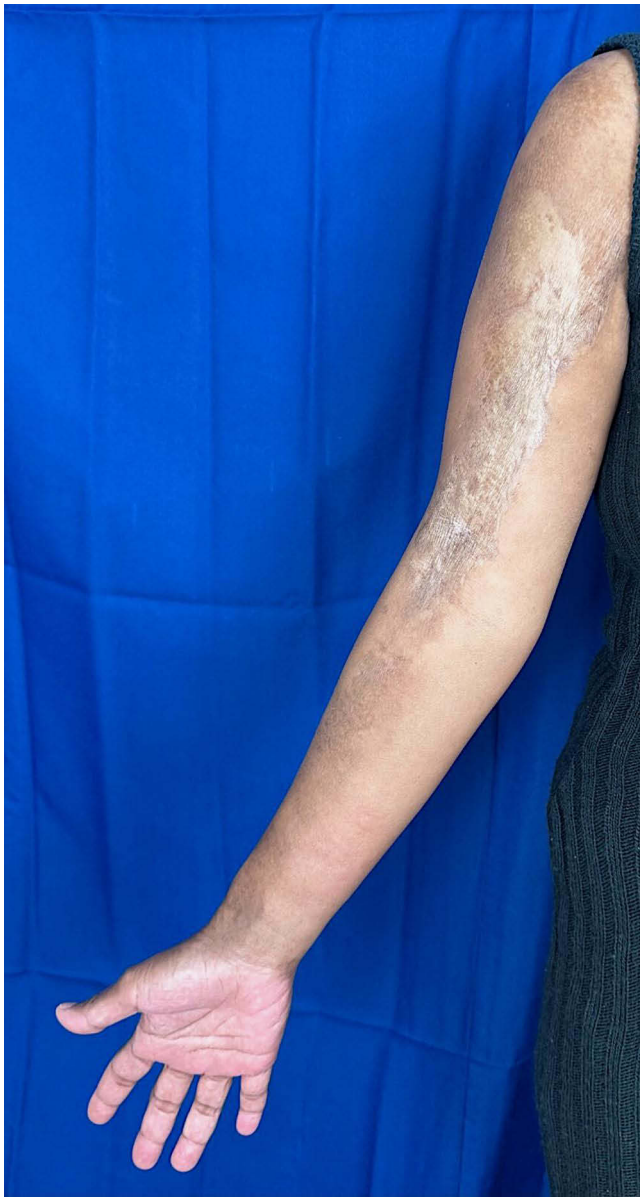

Figure 1 Multiple irregular lesions were observed, characterized by a linear configuration, alongside with skin hardening and atrophy manifestations.

and liver enzymes were all within normal limits. The patient's rheumatoid factor (RF) was reported as non-reactive. The histopathological examination of the skin biopsy specimen, obtained from the patch exhibiting an indurated border on the upper arm, demonstrated epidermal thinning with flattened rete ridges. The dermis layer was characterized by partial hyalinization, lymphocyte inflammatory cells, and a thickened fibro collagenous connective tissue stroma extending to the subcutaneous fat layer. The pilosebaceous units were not visible, and there were no indications of malignancy (Figure 2).

In this case, the treatment involved administering HCQ 200 mg twice daily. Additionally, pimecrolimus 1% cream was applied to the skin lesions twice daily. The patient was then followed up once a month to monitor the progress. After three months of observation, the skin hardening had begun to soften. Furthermore, there was a significant decrease in the size of some hyperpigmented macules, patches, and plaques, as well as a concomitant repigmentation of some depigmented lesions (Figure 3). The mLoSSI and the LoSDI scores decreased from 6 to 1 and 5 to 4, respectively. Furthermore, there was no evidence of new skin lesions, and no adverse effects related to retinal toxicity such as cystoid macular edema; damage of retinal pigment epithelium, photoreceptor complex, vascular, ganglion cell or optic nerve; and

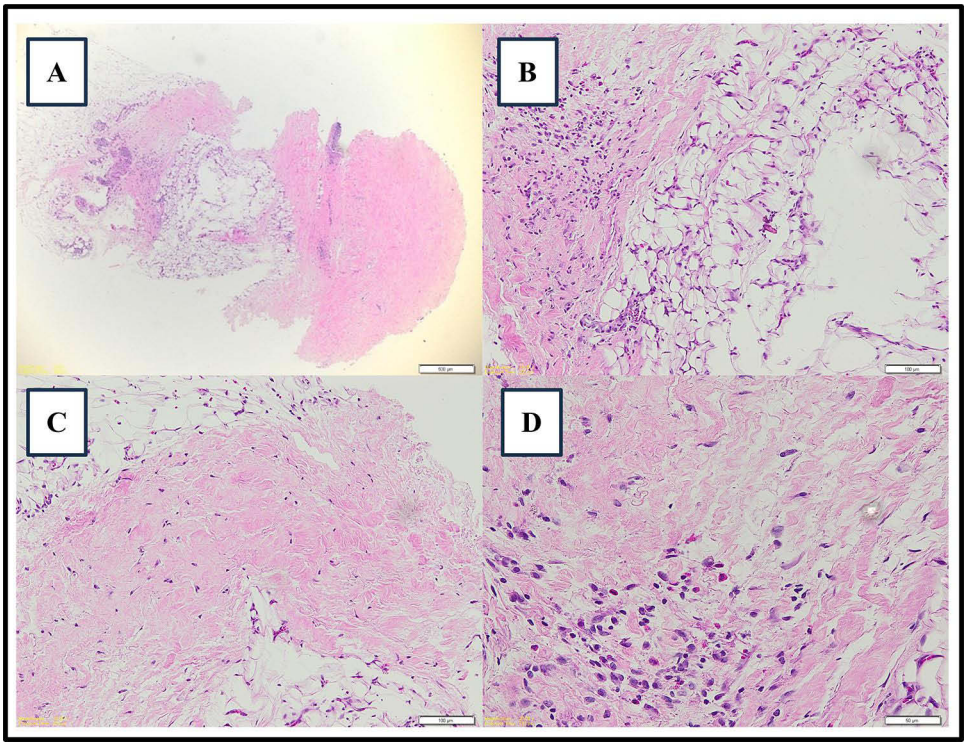

Figure 2 (**A**–**D**) Histological findings from the skin lesion, stained with hematoxylin and eosin (H&E) (20x magnification [A]; 100x [B]; 200x [C–D]), demonstrated thinning and flattening of the overlying epidermis (**A**). The dermis consisted of a fibrocollagenous connective tissue stroma (**B**), that showed significant thickening extending into the subcutaneous fat layer (**C**), This region also exhibited hyaline degeneration and mild lymphocytic inflammatory infiltrates (**D**). Pilosebaceous units were markedly absent, and no features suggestive of malignancy were identified.

other miscellaneous effects were reported. This was corroborated by an ophthalmologist following a thorough ophthalmoscopic examination. Written informed consent for publication of clinical details and images was obtained from the patient.

Discussion

Morphea results in cutaneous morpho-structural alterations with considerable aesthetic impact on the daily life of patients.[1] It has been observed to manifest in various clinical presentations, including LM.[11,12] LM usually arises during childhood; however, in some cases, it may appear in adulthood with a prevalence of 32.6% cases.[1,13] The reported mean ages of disease onset for juvenile and adult LM were 10 and 45 years, respectively, with an overall female-to-male ratio of 4:1.[4] Research on morphea and pregnancy is still limited, with approximately 30% of morphea reactivations occurring during pregnancy, and the linear subtype has rarely been documented in this context.[14] Moreover, to our knowledge, there have been no previous reports describing LM developing or progressing during the breastfeeding period, which highlights the clinical relevance and novelty of this case. This case involved a 44-year-old female patient who was diagnosed with LM during her pregnancy, and the disease continued to develop during the subsequent period of breastfeeding.

The pathogenesis of morphea, including LM, remains incompletely understood. It is thought to involve an interplay between genetic predisposition (such as HLA-DRB1*04:04 and HLA-B37) and triggering factors, such as mechanical trauma, infections, insect bite, psychological stress, drugs, X–irradiation, and hormonal influences.[15,16] At the molecular level, the development is hypothesized to be driven by three major mechanisms, including microvascular injury, T-cell-mediated inflammation, and altered connective tissue production by fibroblasts.[15]

Microvascular injury, which leads to the recruitment of immune cells, may provide a pro-fibrotic microenvironment.[2,16] Vascular damage results in the upregulation of adhesion molecules and recruitment of inflammatory cells, particularly T lymphocytes.[2] Th1 responses dominate the early active phase,[2,12] while Th2 lymphocytes

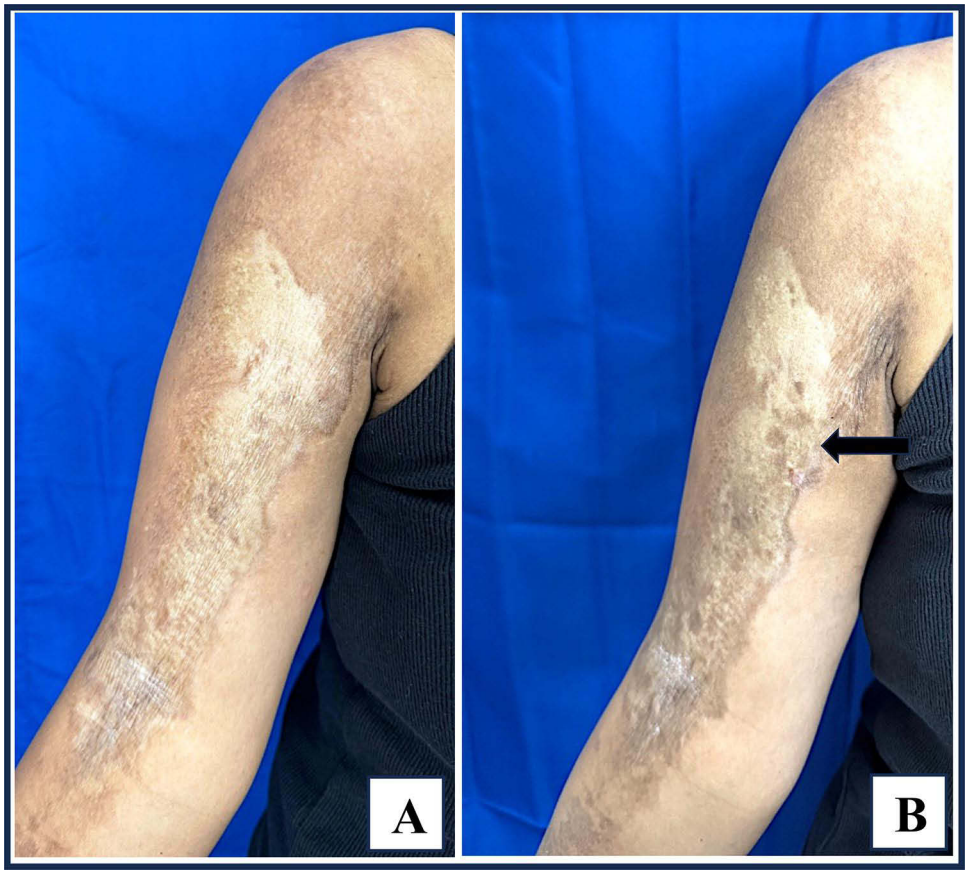

Figure 3 (**A**–**B**) Clinical improvement of the skin lesions was documented. Certain areas exhibited partial fading (yellow arrow), while repigmentation was observed in previously depigmented lesions (black arrow). Furthermore, a degree of skin softening was noted in specific regions. This evaluation was conducted prior to the combination therapy of HCQ and topical pimecrolimus 1% (**A**) and three months following the initiation of this combination therapy (**B**).

significantly predominate during the sclerosing phase of morphea. Th2-related cytokines, particularly IL-4, induce the production of transforming growth factor-β (TGF-β), which stimulates collagen production and myofibroblast differentiation, culminating in dermal fibrosis.[2,12,17]

Pregnancy has also been reported as a potential trigger for morphea in several cases. Proposed mechanisms include mechanical injury, maternal microchimerism, and hormonal shifts involving TGF-β.[6,16,18] A case by Anh Khoa Pham et al described morphea during pregnancy that resolved rapidly after delivery.[16] The underlying mechanism is related to the changes in the maternal immune system that promote tolerance to paternal alloantigens, which lead to the production of the pro-fibrotic cytokine TGF-β, which has been implicated in the pathogenesis of morphea.[15,16] Nevertheless, it has been established that progesterone increases 17β-estradiol levels, which, via skin receptors, regulate fibroblast proliferation and tissue-degrading matrix metalloproteinase (MMP) synthesis. Liliana et al demonstrated that estrogens may stimulate the production of essential fibroblast growth factors and increase the secretion of TGF-β1.[15]

In contrast, our patient developed lesions in late pregnancy with progression into the postpartum breastfeeding period. It was found to be in contrast with previously documented cases of morphea in pregnancy, as the progression of the lesion ceased following delivery.[16] Although no study to date has specifically reported LM in breastfeeding, a potential contribution of elevated prolactin (PRL) levels may be hypothesized. La Montagna et al documented mild hyperprolactinemia in a subset of systemic sclerosis patients, suggesting an association between PRL and autoimmunity.[18] The PRL receptor belongs to the type 1 cytokine/ hematopoietic receptor superfamily and is extensively expressed throughout the immune system. Consequently, the binding of PRL to its receptor activates downstream signalling pathways that regulate immune cell proliferation, differentiation, survival, and cytokine production, with immunostimulatory effects on

both Th1 and Th2 responses.[19,20] Further investigation is required to determine whether this phenomenon is attributable to elevated levels of the hormone PRL.

The diagnosis of LM is primarily made through a clinical approach, which necessitates a comprehensive medical history and a meticulous skin examination. A skin biopsy may be performed in cases requiring further diagnosis, followed by imaging studies to assess severity.[2] Furthermore, biological indicators, such as antinuclear antibodies (ANA), have been observed in approximately 30% of patients, primarily among those with LM.[6,12] Clinical manifestation, consideration of histopathology, and serology tests concluded the diagnosis of LM in this case.

The clinical features of LM typically involve progressive loss of subcutaneous fat with pigmentation changes that appear as shiny, thick patches of the skin. It usually presents with at least one linear streak of cutaneous induration, which may involve the dermis and extend to underlying structures such as subcutaneous tissue, muscle, and bone, leading to cosmetic and functional disability.[2,6] The patient initially presented with a blackish-brown patch that changed to brownish-white in color, spread in a linear configuration, and followed by skin hardening.[2] There were also areas of shiny epidermal atrophy with cigarette paper skin changes, without any joint complaints or muscle pain.

The histopathological examination should be performed to exclude malignancy and other sclerosing conditions.[11] Biopsies should be taken from the inflammatory or indurated edge or sclerotic center, including subcutaneous fat.[2] The inflammatory phase is characterized by an interstitial and perivascular inflammatory cell infiltrates in the dermis and rarely in the subcutaneous tissue, accompanied by edema, enlarged tortuous vessels, and thickened collagen bundles.[2,12] In sclerotic lesions, the papillary dermis is homogenized with a flattened dermo-epidermal junction, thickened collagen bundles, and sclerosis extends to the reticular dermis (or a deeper layer).[2,12,21] The atrophic phase is characterized by a decrease in inflammatory cells and the absence of appendageal structures.[2] The skin biopsy was obtained from a brownish-white patch with an indurated border on the upper right arm region. The histopathological findings showed subcutaneous involvement, epidermal thinning with a flattened dermo-epidermal junction. The dermis consisted of a fibro collagenous connective tissue stroma thickened to the subcutaneous fat layer, with hyaline degeneration and mild lymphocytic inflammatory cells. Pilosebaceous units were not seen and there was no evidence of malignancy.

Indeed, several autoantibodies have also been identified in individuals with LM, including ANA, anti-single-stranded DNA (ssDNA), anti-ribo-nucleoprotein (RNP), and anti-Sm. Positive ANA titers were found in 34% to 80% of LM patients, being associated with a higher risk for extracutaneous, deeper involvement and disease relapse.[2] The laboratory results showed a positive ANA test with a titer of 1:320, and the ANA profile suggested the presence of specific autoantibodies, particularly anti-RNP and anti-Sm. These findings are interesting, as they may raise suspicion for overlap connective-tissue disease; however, the patient did not exhibit clinical features suggestive of mixed connective-tissue disease or systemic sclerosis (such as Raynaud phenomenon, sclerodactyly, internal organ involvement, or characteristic nailfold capillary changes). Therefore, the overall clinicopathologic picture remained most consistent with isolated LM. The results of further baseline laboratory tests were within the normal range.

Furthermore, differential diagnoses also considered in this case included linear cutaneous lupus erythematosus, lichen sclerosus, linear atrophoderma of Moulin, eosinophilic fasciitis, and localized forms of systemic sclerosis.[4,22–24] These entities were excluded based on the absence of interface dermatitis, genital or intertriginous involvement, predominant atrophy without sclerosis, peripheral eosinophilia, deep fascial involvement, or systemic manifestations, in addition to the histopathologic features described above.

A wide variety of measurement tools have been validated in LM, including ultrasonography (USG), magnetic resonance imaging (MRI), and a morphea-specific outcome measure, the localized scleroderma cutaneous assessment tool (LoSCAT).[25] USG and MRI assessments are becoming increasingly valuable for determining lesion activity and depth; however, this is not yet standard practice without associated clinical manifestations.[2,25] LoSCAT is a validated practical tool that encompasses two LM domains: the modified localized skin index (mLoSSI) and the localized scleroderma damage index (LoSDI).[2,12] Both have been demonstrated to have excellent reliability and validity in previous studies of LM. An additional study by Kelsey et al shows that LoSCAT is a significant and sufficient measure to assess LM therapeutic response, which helps monitor disease activity.[26] The LoSCAT assesses 18 cutaneous anatomic sites, focusing on both disease activity (mLoSSI) and damage (LoSDI) parameters. Serial photographs are useful in clinical practice.[12,26] mLoSSI is calculated as the sum of three distinct activity scores: (1) erythema (ER) based on the

colour of the lesion's edge (score 0–3); (2) skin thickness (ST) (score 0–3); (3) new lesion/ lesion extension (N/E) within the past month (score of 3).[2,26] LoSDI is calculated by accumulating the scores from three domains of cutaneous damage; (1) dermal atrophy (DAT) (score 0–3), (2) subcutaneous atrophy (SAT) (score 0–3), (3) dyspigmentation (DP) (score 0–3).[26] In this case, both mLoSSI and LoSDI scores were initially recorded as 6 and 5, respectively.

The management of LM is fundamentally divided into two principal categories: non-pharmacological strategies and pharmacological therapies, including topical and systemic medications. The utilization of topical medications comprising corticosteroids and CI (eg, tacrolimus 0.1%, pimecrolimus 1%) constitutes a significant proportion of pharmacological therapies, in addition to the administration of immunosuppressive agents, such as MTX, corticosteroids, and HCQ.[1,12] Local application of glucocorticosteroids has proven effective for active lesions; however, its use should be limited due to the potential risk of atrophy. CI has been demonstrated to suppress the immune response and inflammatory factors by impeding T lymphocyte activation and reducing cytokine production. Both experimental and clinical studies comparing topical corticosteroids to topical CI have demonstrated that CI does not cause skin atrophy and has a lower incidence of adverse effects.[27]

MTX is frequently the initial therapeutic option and the preferred medication for LM, with an average dosage of 1 mg/kg/week or 15–25 mg/week.[1,6,28] Methotrexate (MTX) may have teratogenic effects during pregnancy, which can lead to typical embryopathy characterized by craniofacial and central nervous system anomalies, as well as malformations of the extremities. Additionally, MTX is known to be excreted in breast milk. Given its mechanism as a folic acid antagonist and the associated risk of immunosuppression, breastfeeding should be avoided while using MTX.[9,29] In cases where MTX is contraindicated, HCQ may be a suitable alternative. Despite an absence of prior data concerning the effectiveness of LM, it has been shown to be effective in various case reports of morphea with a low rate of adverse effects.[8,22,30] Hydroxychloroquine (HCQ) has been shown to act as an immunomodulatory drug rather than an immunosuppressant. It inhibits immune activation at various cellular levels by potentially interfering with TLR7 and TLR9 ligand binding as well as disrupting TLR signaling through lysosomal inhibition. This mechanism ultimately prevents TLR-mediated cell activation and the production of cytokines. Moreover, in antigen-presenting cells (APCs), such as dendritic cells (DCs) and B cells, HCQ has been observed to inhibit antigen processing and subsequent MHC class II presentation to T cells, thereby preventing T cell activation and reducing the production of cytokines by both T cells and B cells.[31] There is growing evidence to suggest that HCQ is also a safe option for women during lactation. The safety of HCQ has been extensively documented by Elliot et al, showing that doses up to 400 mg daily are well tolerated.[7] While there are concerns about the potential toxic effects of HCQ, particularly regarding retinal toxicity, no such complication has been reported during breastfeeding. This is likely because the levels of HCQ excreted into breast milk are minimal, generally below 0.2 mg/kg per day, which is not considered toxic.[7] Additionally, in contrast to treatment with immunosuppressant medications such as MTX, the use of HCQ is not associated with an elevated risk of infectious complications. The effectiveness of therapy is characterized by the resolution of erythema, typically observed over a period of 2–3 months, as well as softening of lesions, cessation of lesion growth, and the absence of new lesions.[2,7] In this case, the patient was treated with HCQ 200 mg twice daily (total 400 mg/day) in combination with topical pimecrolimus 1% applied twice daily. The outcomes were then monitored on a monthly basis. After an average of three months period of observation, there was a noticeable softening and reduction of the skin lesion, particularly on the shoulder and the forearm. No evidence of new lesion formation or progressive skin hardening were observed, and the mLoSSi and LoSDI scores decreased to 1 and 4, respectively. Furthermore, neither the patient nor the infant exhibited any ophthalmic disturbances, and the retinal examination findings were within normal limits.

Morphea is rarely life-threatening, although it can be functionally and aesthetically disfiguring. Depending on the subtype, adjacent areas such as muscles, bones, and joints may be affected.[3] It causes limited range of motion, limb length discrepancy, joint deformity, and contractures (45% to 56% of LM cases). Schoch et al found that 23 patients (45%) had at least one joint contracture in a study of 51 patients with LM.[5] In some cases, LM can be self-limiting, but it often has a remitting-relapsing or chronic course, leading to a significant burden of disease over time. Recurrences occur in about 25% of patients. A higher risk of recurrence (31% of patients) was reported for LM of the extremities compared to other subtypes.[2]

Limitations of this report include its single-case nature, the relatively short follow-up period of three months, the absence of adjunctive imaging modalities such as USG or MRI to further characterize lesion depth, and limited generalizability to other breastfeeding patients. Larger case series and longer-term follow-up are needed to better define the natural history and optimal management of LM occurring during breastfeeding.

Conclusion

LM is a localized fibrosing disorder that may extend into deeper tissues, leading to both cosmetic and functional impairment. Management can be challenging in breastfeeding women, as standard first-line systemic therapies, such as methotrexate, are contraindicated, making the identification of safe and effective alternatives particularly important. In this case, the combination of oral HCQ 200 mg twice daily and topical pimecrolimus 1% was associated with clinical improvement and reductions in mLoSSI and LoSDI scores. Nevertheless, as this observation is derived from a single patient, the therapeutic implications remain preliminary, and the relatively short follow-up period limits conclusions regarding long-term durability of the response. HCQ in combination with pimecrolimus may represent a potential therapeutic option for LM when standard systemic agents are contraindicated during breastfeeding; however, additional cases and longer-term studies are needed to substantiate its efficacy and safety in this population.

Abbreviations

LM, linear morphea; CI, calcineurin inhibitor; MTX, methotrexate; HCQ, hydroxychloroquine; ROM, range of motion; ESR, erythrocyte sedimentation rate; CRP, c-reactive protein; RF, rheumatoid factor; ICAM, intercellular adhesion molecule; VCAM, vascular cell adhesion molecule; IFN-γ, interferon-γ; IL, interleukin; TGF- β, transforming growth factor-β; MMP, matrix metalloproteinase; PRL, prolactin; ANA, antinuclear antibodies; RF, rheumatoid factor; ssDNA, single-stranded DNA; dsDNA, double-stranded DNA; RNP, ribo-nucleoprotein; USG, ultrasonography; MRI, magnetic resonance imaging; LoSCAT, localized scleroderma cutaneous assessment tool; mLoSSi, modified localized skin index; LoSDI, localized scleroderma damage index; ER, erythema; ST, skin thickness; DAT, dermal atrophy; SAT, subcutaneous atrophy; DP, dyspigmentation; APC, antigen-presenting cells; DC, dendritic cells.

Ethical Approval and Consent to Participate

The publication of images was included in patients' consent for the publication of the case. Institutional approval was obtained to publish the case details from Dr. Hasan Sadikin Hospital Ethical Committee.

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Disclosure

The authors report no conflicts of interest in this work.

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