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Single Case

Reticular Erythematous Mucinosis in an African Woman with HIV Infection: Case Report and Literature Review

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Keywords
Reticular erythematous mucinosis · Skin of colour · HIV infection · Photosensitivity · Lupus erythematosus · Case report

Abstract
Reticular erythematous mucinosis is a rare and persistent form of primary idiopathic mucinosis, often referred to as plaque-like cutaneous mucinosis or midline mucinosis. It presents with reticulate patches or erythematous plaques with predilection for the anterior and posterior trunk. Affected patients are frequently asymptomatic. Pruritus or burning sensations were reported after exposure to the sun. The aetiology remains obscure; its pathogenesis is poorly understood, particularly in immunocompromised patients such as HIV-infected patients. The disease associations are not uniformly documented. Antimalarial agents significantly improve and shorten the course of the disease. We report a case of a 31-year-old African woman with underlying HIV infection who displayed the classical clinical and histological features of reticular erythematous mucinosis. This condition is rare among the HIV-infected patients, particularly in those of African descent, in whom lichen myxoedematosus/scleromyxoedema variants and acral persistent papular mucinoses were most frequently reported. The higher incidence of photosensitivity in HIV-infected individuals including the patients with skin of colour may play a potential role in reticular erythematous mucinosis. Its relationship with lupus erythematosus and photosensitivity in the context of HIV infection is discussed. To the best of our knowledge, this is the first reported case of reticular erythematous mucinosis in an African HIV-infected patient. This case highlights the need for diagnostic awareness in cases presenting with erythematous plaques and patches in a net-like pattern developing on the midline and sun-exposed areas of the trunk.

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Introduction

Reticular erythematous mucinosis (REM) is an uncommon condition classified among midline primary cutaneous mucinoses. It presents with net-like confluent macules and patches or ill-defined plaques typically distributed on the anterior chest and upper back [1–3]. The lesions are essentially asymptomatic and tend to persist. REM has been reported more frequently in younger/middle-age women. The initial appearance of REM in children is uncommon [1–3]. The majority of reported cases were sporadic with only a few isolated familial reports described [4, 5].

The exact aetiology remains obscure. Exposure to heat, radiotherapy, hormonal status (menstruation, pregnancy, oral contraceptives), viral infections, and immunologic dysfunction may promote or exacerbate REM [6, 7]. The pathogenesis of REM remains poorly understood, particularly in immunocompromised patients such as HIV-infected patients [6, 7]. There is a gap in understanding the relationship between REM and inflammatory, autoimmune disorders. More consistently reported associations of REM were with autoimmune diseases such as Hashimoto thyroiditis, SLE/lupus tumidus, diabetes, idiopathic thrombocytopenic purpura [6, 7].

Antimalarial medications led to prompt resolution of the lesions in the majority of cases, shortening the course of the disease. Alternative treatment options continue to be investigated, yielding various results. Here we report, to the best of our knowledge, the first case of REM in a young African HIV-infected woman in whom clinical recognition and assessment may be difficult. The case report was prepared following the CARE guidelines. The CARE Checklist has been completed by the authors for this case report, attached as online supplementary material (for all online suppl. material, see https://doi.org/10.1159/000531464).

Case Report

A thirty-year-old African woman was referred to the Clinic of Dermatology for review of her skin condition as an accurate diagnosis had not been established, the disease had continued to progress for 2 years, and the lesions were not responsive to various attempted therapies. The cutaneous lesions were located on the right pectoral area, right shoulder, and right side of the neck, tending to spread towards the midline of the back (Fig. 1a, b).

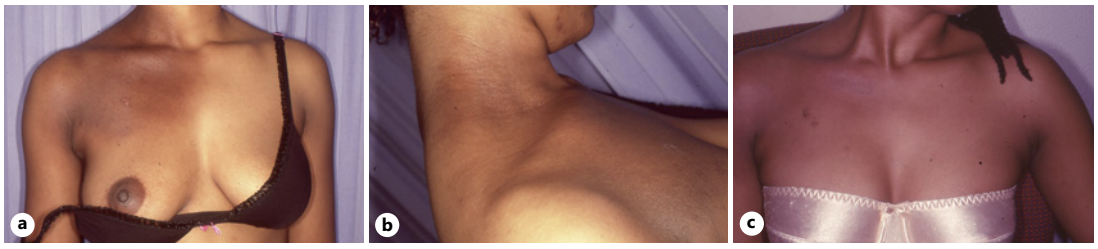

Fig. 1. a Poorly defined erythematous plaques with a vague reticular pattern in a characteristic midline distribution. **b** Spreading of the erythema to the neck and interscapulo-vertebral area. **c** Remission of the skin lesions following treatment with antimalarial agents.

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They consisted of blotchy erythematous ill-defined plaques and patches in a vague reticulate pattern. No changes in the skin surface were noted. The lesions had a tendency to persist, without seasonal incidence and were essentially asymptomatic, although exposure to the sun appeared to aggravate them.

General physical examination revealed acrocyanosis, a vague livedo reticularis over her thighs, and discrete punctate red macules of the pulps of the fingers resembling vasculitis. No adenopathy or visceromegaly were palpable. Neither the patient's siblings nor parents were similarly affected. Her medical history was relevant for HIV infection for several years prior to presentation. She was not on HAART or any other chronic medication. The patient used only her usual products for personal hygiene and cosmetic purpose.

Taking into account the in-depth history and clinical findings, several skin conditions were considered in the differential diagnosis: lupus tumidus, scleredema, dermatomyositis, REM, and Jessner's benign lymphocytic infiltrate of the skin.

A complete evaluation and work-up were conducted to establish a final diagnosis and to start appropriate treatment. The results of routine laboratory studies (FBC, coagulation profile, UEC, thyroid function, glucose, calcium, and urine analysis) were within reference range. ESR was raised (75 mm-1 h), liver enzymes showed increased values (AST-60 U/L, ALT-66 U/L, GGT-149 U/L, LDH-308 U/L, ALP-142 U/L). Screening for porphyrins was negative, and hepatitis studies indicated immunity to hepatitis B and C viruses. Protein electrophoresis demonstrated a polyclonal elevation of gammaglobulins without discrete bands or spikes as seen in infections and inflammatory processes.

HIV test confirmed positivity with CD4+ T-cell readings varying from 788 to 1,082 cells/μL and CD3+ T-lymphocyte counts ranging from 1,272 to 1,934 cells/μL at different evaluations in time. HIV-RNA viral load was 19,736 copies at the initial evaluation and undetectable afterwards.

The following laboratory studies gave normal or negative results: IgM, IgG, IgA, complement, EBV, CMV, HTLV-1 and 2, syphilis serology (TPHA). The antibody panel was negative (ds-DNA, anti-Ro/SSA, anti-La/SSB, anti-RNP1, anti-Sm, anti-thyroglobulin), and complement studies were normal. Serum immunoelectrophoresis ruled out the presence of paraprotein.

Microscopic examination of the biopsy collected from the lesional skin showed a well-preserved epidermis without thickening of the basal membrane and no interface dermatitis. A prominent feature was the thickening of the reticular dermis with extensive deposition of basophilic material in the superficial and mid-dermis (Fig. 2a), highlighted with Alcian Blue stain at pH 2.5 (Fig. 2b). A moderate perivascular and patchy periadnexal lymphocytic infiltrate was demonstrated in the superficial and mid-reticular dermis without features of

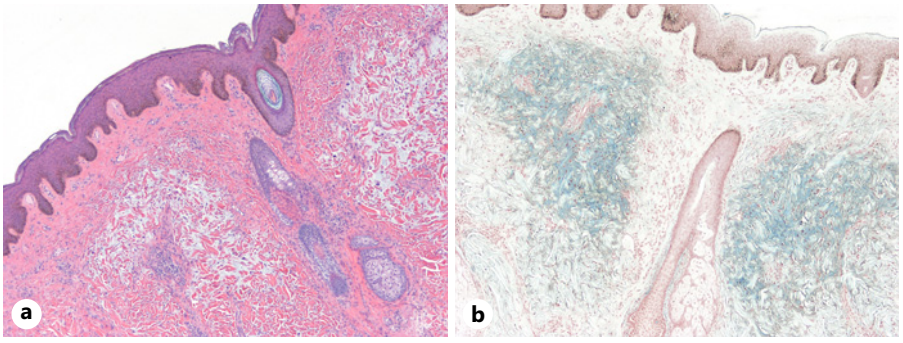

Fig. 2. **a** Regular epidermis with mild predominantly lymphocytic inflammation in perivascular and periadnexal location (H&E, original magnification, ×40). **b** Alcian blue stain highlights the area of mucin deposition in the superficial/mid-dermis. Mucin splaying collagen fibres (original magnification, ×100).

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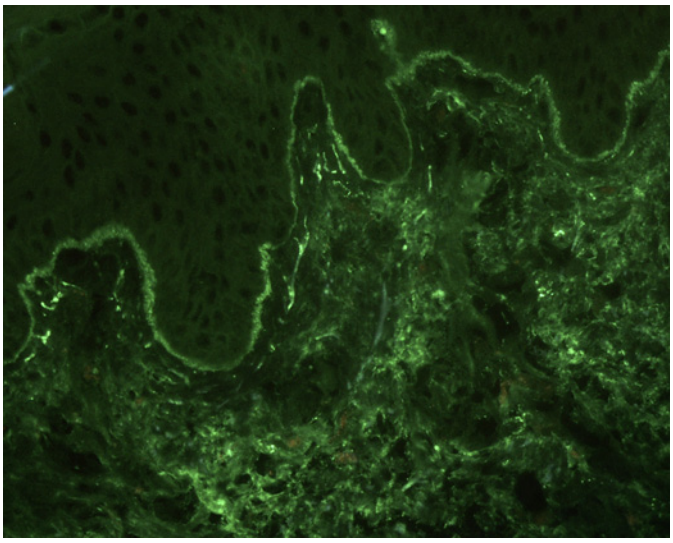

Fig. 3. DIF showing faint granular positivity for IgM along the basal layer.

vasculitis. Immunohistochemical stains with anti-CD3, anti-CD20, anti-CD4, anti-CD8, and anti-CD68 antibodies showed that the infiltrate was composed predominantly of T-helper lymphocytes. CD44 epidermal expression of keratinocytes was unaltered despite the accumulation of mucin in the dermis. We examined HLA types in our patient, and this revealed polymorphism: HLA-A26,-29; HLA-B35,-44; HLA-Cw4,-7; HLA-DRB1 (04, 13); and HLA-DQB1 (03, 06).

Direct immunofluorescence (DIF) examination demonstrated a faint granular positivity for IgM in the basement membrane zone and negative staining for C3, IgG, and IgE (Fig. 3). Chest roentgenogram, skeletal radiological studies, and abdominal ultrasound were within normal parameters.

Based on the history and clinical presentation, supported by histopathologic findings and DIF results, a diagnosis of REM was favoured. Following a normal ophthalmologic examination and counselling regarding the risk of toxicity and rationale for screening, she was commenced on hydroxychloroquine 200 mg twice daily for 2 weeks, tapered to 200 mg daily, combined with topical tacrolimus 0.1% and topical steroids. The treatment led to prompt resolution of the lesions after 3 months of initiating the antimalarial therapy (Fig. 1c). The patient followed up regularly for a period of 6 years with a good outcome. Her clinical condition has remained stable without recurrence; her antibody profile stayed negative; liver functional tests have normalized; and CD4+, CD8+, and CD3+ T-cell counts have maintained within a normal range. The patient was not considered a candidate for commencing HAART, and she stayed free of antiretrovirus treatment throughout the entire period of follow-up.

Discussion

REM is a rare disease, the nosology and distinct characteristics of which are controversial due to the scanty number of case reports (95 case reports in English literature) [6, 7].

The condition currently accepted as REM was initially described by Perry et al. [8] in 1960 in 3 patients as plaque-like cutaneous mucinosis (PCM). Steigleder and colleagues coined the term of REM in 1974 for net-like blotchy erythematous plaques having similar distribution and histopathologic features with plaque-like cutaneous mucinosis [9]. Subsequent reported cases

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from different groups of investigators have concluded that REM and PCM are both midline mucinoses with different clinical presentations and transitional forms of the same disease spectrum [10–12]. Certain distinct features, however, make REM a recognizable disease: typical localization on midline aspect of the trunk, persistent reticulate pattern, benign, self-limited course with lack of systemic consequences, variable response to sun exposure.

To what extent REM can be called mucinosis and how far the disease is related to other inflammatory autoimmune skin diseases is a matter of debate. The fact that it shares several clinical, histological, and DIF features with lupus erythematosus tumidus (LET) prompted some authors to consider REM as a subset of lupus erythematosus [12–14]. Different studies [12–14] showed that apart from the distinct clinical presentation of REM, the histological examination revealed minimal epidermal changes with discrete pigment incontinence, while in LET, the epidermis was more involved, showing atrophy, focal basal vacuolar changes, and thickened basal membrane. Mucin deposition, highlighted by Alcian blue stain, was seen in the superficial and mid-reticular dermis in the majority of cases of REM and had a deeper distribution in LET.

The inflammatory infiltrate composed of T-helper lymphocytes was sparse and limited to the superficial interstitial and perivascular dermis in REM. The infiltrate extended to the deeper dermis and had a tropism for hair follicles in LET besides the perivascular distribution. DIF positivity was not discriminatory between the two conditions in terms of findings (granular deposits of IgM and C3 along the dermal-epidermal junction), but it was reported more frequently in LET than in REM [13, 14].

Results of DIF suggested that immunoglobulins and complement have a more important role in mediating tissue damage in LET than in REM. DIF studies from the lesional skin have not been performed in all reported cases of REM, and their value is uncertain. The DIF findings in our case were in agreement with most other reports of REM in whom this investigation has been performed (Fig. 3). Whether the positive immunofluorescence finding in our patient is related to REM or whether it is a secondary phenomenon cannot be discerned. Overall, REM and LET show some overlapping features, but the presence of some differences at histopathological level justifies the distinction between the two diseases and makes REM a distinct entity [12–14].

Different types of mucin deposits are frequently found in all subsets of lupus erythematosus including papulonodular dermal mucinosis and reticular mucinosis [14–16]. Papular mucinosis (lichen myxoedematosus) is most likely to be confused with LE-associated cutaneous mucinosis. It is the most common type of mucinosis encountered in HIV-infected African patients in our setting, either as an entity or as part of clinical manifestations of SLE. Rongioletti et al. [16] previously reported that lupus erythematosus is systemic in the majority (80%) of cases, suggesting that papulonodular mucinosis may herald severe systemic disease [14, 15].

Studies conducted in patients with REM to highlight the exact nature of the mucinous deposition, the stimulus for increased production, and its significance have pointed to the abnormal response of fibroblasts to exogenous IL-1b with subsequent accumulation of hyaluronan [16, 17]. The overexpression of hyaluronic acid was shown to appear at an early stage of the disease (oedematous, inflammatory), while fibroblasts from the late skin lesions may cease to express hyaluronic acid [17]. These findings suggest that fibroblasts at different stages of the disease express different phenotypes.

Another study demonstrated that accumulation of hyaluronan in the lesional skin was approximately 2.9-fold higher than in unaffected skin [17, 18]. The number of hyaluronic acid synthase HAS2+ cells was significantly increased in the involved skin, suggesting that these Factor XIIIa+/HAS2+ dermal dendrocytes might actually be responsible for the increased synthesis of hyaluronan [17, 18]. CD 44, a widely distributed transmembrane glycoprotein that is expressed in many isoforms, functions as a cell adhesion molecule, binding several ligands.

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It is thought to be the principal cell surface receptor for hyaluronate [19]. Our case, similar to the results of other studies, confirmed that CD44 epidermal expression of keratinocytes was not affected by the accumulation of mucin in the dermis [19].

There are limited data on photosensitivity in HIV-infected African individuals. It may appear with the recovery of the immune system, from prolonged subclinical UV-light-induced inflammation, or after taking potentially photosensitizing drugs [20, 21].

An increased prevalence and severity of cutaneous photosensitivity have been recognized in association with HIV infection but rarely demonstrated [20, 21]. The role of sunlight, reported to be controversial in REM, appears to be more relevant in HIV-infected patients with REM. Skin lesions in our case, similar to previous reports, were aggravated by sun exposure. Studies have confirmed the photosensitive nature of REM by provocative irradiation in UVA cabin that reproduced the lesions [22]. Test reactions appeared after a relatively long interval of up to 7 days, explaining the absence of photosensitivity in some patients with REM [22].

The heterogeneity of clinical features of photosensitive reactions in HIV-positive patients and the significant overlap of clinical presentations as well as the subtle histological features make the interpretation challenging. The pathogenesis of HIV-associated photosensitivity in REM is poorly understood.

Photosensitivity may be related to depletion of endogenous scavengers, which results in oxidative stress and, consequently, disruption of intracellular homoeostasis [21, 23, 24]. Alterations in DNA, cell membrane, enzyme structural proteins, and RNA induced by UV-light exposure led to changes in antigenic expression, neoantigen formation, and sensitization with predisposition to autoimmune phenomena [23, 24].

REM has not been well characterized immunologically. An increase in circulating immune complex (CIC) levels has been reported in patients with REM at the initial assessment and during recurrence [25]. In different studies, measurements of CIC were undertaken before, during, and after therapy [25]. The patient presented herein has had normal complement and CIC values throughout the entire period of follow-up of 6 years. A significant depression of NK cell immune cytolytic activity has been described in patients with REM, suggesting an alteration in immunoregulatory chains that may correlate with the predisposition to autoimmunity, neoplasms, and endocrine disorders reported in these patients [23–25].

Cytokines such as IL-1, IL-6, IL-10, TGF-beta, and TNF-alpha, elevated by UV-light exposure, are also elevated in HIV disease. It is reasonable to postulate that these immune defence mechanisms might be related to the pathogenesis of REM in HIV-infected individuals. An association with specific HLA constellations is suspected but has not been proven to date.

HLA comparison study with sporadic cases of REM reported in the literature was not possible since similar analyses have never been carried out. Caputo et al. [4] found polymorphisms in HLA types in a familial case with REM. It is not clear whether any HLA allele represents susceptibility genes for this disease as there are a limited number of reported cases and HLA typing was not consistently explored.

Studies have demonstrated that antimalarial agents significantly improved and cleared the skin lesions in patients with REM, but relapses were common and the adverse side effects must be considered [26, 27]. Antimalarial agents should not be withheld from patients because of tobacco use. The patient reported herein showed remission to hydroxychloroquine within several months combined with topical tacrolimus 0.1% ointment and topical steroids (Fig. 1c). Alternative therapeutic options continue to be investigated, with various results: systemic corticosteroids, cyclosporine, topical calcineurin inhibitors as monotherapies, UVA1 phototherapy, and pulsed-dye laser [28–31].

Conclusions

This case highlights the need for diagnostic awareness and early recognition among the African population in cases presenting with plaques or patches in a reticular pattern on the midline of the trunk. There is a gap in understanding the relation between REM and LET as these two entities share some clinical, histological, and DIF similarities, but REM is more benign, having fewer, if any, systemic involvement.

There are a scanty number of case reports of REM and small case studies that prevent us from drawing conclusions on the potential role of photosensitivity in REM in the context of HIV infection. Further study to address this possibility seems warranted.

Hydroxychloroquine remains the leading therapeutic agent in REM. It has been associated with substantial improvement of skin lesions including in our patient. Further studies are needed to unravel the pathogenic mystery of REM and optimize and expand the therapeutic possibilities.

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Statement of Ethics

The Ethics Committee approval was not required for this case study in accordance with national guidelines. Written and informed consent was obtained from the patient for publication of the details of her medical case and accompanying images.

Conflict of Interest Statement

The authors declare that there is no actual or potential conflict of interest in relation to this article.

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Author Contributions

Daniela Tenea performed the clinical work and the case management and drafted the article (conception and design). Cinzia Campaini analysed the histopathology slides (H&E, immunohistochemical stains, and DIF), provided the microphotographs, and made comments for Figures 2a, b and 3.

Data Availability Statement

All data generated or analysed in this case report are included in the article and its online supplementary material. Further inquiries can be directed to the corresponding author.

References

1 Kucukunal A, Altunay I, Demirci GT, Sarikaya S, Sakiz D. Reticular erythematous mucinosis on the midline of the back. Cutis. 2014;93(6):294–6.
2 Cohen PR, Rabinowitz AD, Ruszkowski AM, DeLeo VA. Reticular Erythematous Mucinosis Syndrome: review of the world literature and report of the syndrome in a prepubertal child. Ped Dermatol. 1990;7(1):1–10.
3 Sidwell RU, Francis N, Bunker CB. Hormonal influence on reticular erythematous mucinosis. Br J Dermatol. 2001;144(3):633–4.
4 Caputo R, Marzano AV, Tourlaki A, Marchini M. Reticular Erythematous Mucinosis occurring in a brother and sister. Dermatol. 2006;212(4):385–7.
5 Fühler M, Ottmann K, Tronnier M. Reticular erythematous mucinosis (REM syndrome) in twins. J Dtsch Dematol Ges. 2009;7(11):968–70.
6 Wriston CC, Rubin AI, Martin LK, Kossard S, Murrell DF. Plaque-like cutaneous mucinosis: case report and literature review. Am J Dermatopathol. 2012;34(4):e50–4.
7 Thareja S, Paghdal K, Lien MH, Fenske NA. Reticular erythematous mucinosis: a review. Int J Dermatol. 2012;51(8):903–9.
8 Perry HO, Kierland RR, Montgomery H. Plaque-like form of cutaneous mucinosis. Arch Dermatol. 1960;82:980–5.
9 Steigleder GK, Gartmann H, Linker U. REM syndrome: reticular erythematous mucinosis (round-cell erythematosis), a new entity? Br J Dermatol. 1974;91(2):191–9.
10 Quimby SR, Perry HO. Plaquelike cutaneous mucinosis: its relationship to reticular erythematous mucinosis. J Am Acad Dermatol. 1982;6(5):856–61.
11 Rongioletti F, Merlo V, Riva S, Cozzani E, Cinotti E, Ghigliotti G, et al. Reticular erythematous mucinosis: a review of patient's characteristics, associated conditions, therapy and outcome in 25 cases. Br J Dermatol. 2013;169(6):1207–11.
12 Almohssen AA, Suresh RV, Schwartz RA. Reticular erythematous mucinosis: a rare cutaneous. Acta Dermatovenerol Croat. 2019;27(1):16–21.
13 Cinotti E, Merlo V, Kempf W, Carli C, Kanitakis J, Parodi A, et al. Reticular erythematous mucinosis: histopathological and immunohistochemical features of 25 patients compared with 25 cases of lupus erythematosus tumidus. J Eur Acad Dermatol Venereol. 2015;29(4):689–97.
14 Del Pozo J, Peña C, Almagro M, Yebra MT, Martínez W, Fonseca E. Systemic lupus erythematosus presenting with a reticular erythematous mucinosis-like condition. Lupus. 2000;9(2):144–6.
15 Lowe L, Rapini RP, Golitz LE, Johnson TM. Papulonodular dermal mucinosis in lupus erythematosus. J Am Acad Dermatol. 1992;27(2 Pt 2):312–5.
16 Rongioletti F, Ghigliotti G, De Marchi R, Rebora A. Cutaneous mucinoses and HIV infection. Br J Dermatol. 1998;139(6):1077–80.
17 Izumi T, Tajima S, Harada R, Nishikawa T. Reticular erythematous mucinosis syndrome: glycosaminoglycan synthesis by fibroblasts and abnormal response to interleukin-1 beta. Dermatology. 1996;192(1):41–5.
18 Tominaga A, Tajima S, Ishibashi A, Kimata K. Reticular erythematous mucinosis syndrome with an infiltration of factor XIIIa+ and hyaluronan synthase 2+ dermal dendrocytes. Br J Dermatol. 2001;145(1):141–5.
19 Kaya G, Augsburger E, Stamenkovic I, Saurat J-H. Decrease in epidermal CD44 expression as a potential mechanism for abnormal hyaluronate accumulation in superficial dermis in lichen sclerosus et atrophicus. J Invest Dermatol. 2000;115(6):1054–8.
20 Bilu D, Mamelak AJ, Nguyen RH-N, Queiroz PC, Kowalski J, Morison WL, et al. Clinical and epidemiologic characterization of photosensitivity in HIV-positive individuals. Photodermatol Photoimmunol Photomed. 2004;20(4):175–83.
21 Vin-Christian K, Epstein JH, Maurer TA, McCalmont TH, Berger TG. Photosensitivity in HIV-infected individuals. J Dermatol. 2000;27(6):361–9.
22 Adamski H, Le Gall F, Chevrant-Breton J. Positive photobiological investigation in reticular erythematous mucinosis syndrome. Photodermatol Photoimmunol Photomed. 2004;20(5):235–8.
23 Dauden E, Peñas PF, Buezo GF, Fraga J, García-Diez A. Reticular Erythematous mucinosis associated with human immunodeficiency virus infection. Dermatol. 1995;191(2):157–60.
24 Smith KJ, Skelton HG, Yeager J, Tuur S, Angritt P, Wagner KF. Histopathologic features seen in cutaneous photoeruptions in HIV-positive patients. Military medical consortium for the advancement of retroviral research (MMCARR). Int J Dermatol. 1997;36(10):745–53.
25 Braddock SW, Kay HD, Maennle D, McDonald TL, Pirruccello SJ, Masih A, et al. Clinical and immunologic studies in reticular erythematous mucinosis and Jessner's lymphocytic infiltrate of skin. J Am Acad Dermatol. 1993;28(5 Pt 1):691–5.
26 Kreut er A, Scola N, Tigges C, Altmeyer P, Gambichler T. Clinical features and efficacy of antimalarial treatment for reticular erythematous mucinosis: a case series of 11 patients. Arch Dermatol. 2011;147(6):710–5.

27 Marmor MF, Kellner U, Lai TYY, Lyons JS, Mieler WF; American Academy of Ophthalmology. Revised recommendations on screening for chloroquine and hydroxychloroquine retinopathy. Ophthalmology. 2011;118(2):415–22.
28 Rubegni P, Sbano P, Risulo M, Poggiali S, Fimiani M. A case of reticular erythematous mucinosis treated with topical tacrolimus. Br J Dermatol. 2004;150(1):173–4.
29 Mansouri P, Farshi S, Nahavandi A, Safaie-Naraghi Z. Pimecrolimus 1 percent cream and pulsed dye laser in treatment of a patient with reticular erythematous mucinosis syndrome. Dermatol Online J. 2007;13(2):22.
30 Amherd-Hoekstra A, Kerl K, French LE, Hofbauer GFL. Reticular Erythematous Mucinosis in an atypical pattern distribution responds to UVA1 phototherapy. J Eur Acad Dermatol Venereol. 2014;28(5):672–3.
31 Erceg A, De Jong EMJG, Van de Kerkhof PCM, Seyger MB. The efficacy of pulsed dye laser treatment for inflammatory skin diseases: a systematic review. J Am Acad Dematol. 2013;69(4):609–15.e8.