Dermatology Practical & Conceptual

Reactive Infectious Mucocutaneous Eruption (RIME): Narrative Review and Proposed Management Algorithm

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ABSTRACT

Introduction: RIME is an emerging dermatologic condition marked by prominent mucositis with minimal skin involvement, often mimicking Stevens-Johnson syndrome. While initially linked to Mycoplasma pneumoniae, a broader range of infectious triggers is now recognized.

Objectives: We present a narrative review of this entity. A proposed diagnostic and therapeutic algorithm is also provided to aid clinicians in clinical practice.

Methods: We performed a narrative review of English-language literature on RIME and MIRM indexed in PubMed through 2024, supplemented by clinical insights from the experience of our centre.

Results: Evidence regarding treatment is lacking, with systemic steroids and/or cyclosporine A showing the most benefit. RIME usually portends a good prognosis, with recovery within seven to 21 days, although complications can occur. Recurrences are rare, and subsequent episodes can be triggered by different microorganisms.

Conclusions: RIME represents a distinct clinical entity with a broadening range of infectious triggers beyond Mycoplasma pneumoniae. Prompt recognition, accurate differentiation from mimickers, and a structured diagnostic and therapeutic approach are essential for effective management and improved patient outcomes.

Introduction

RIME is a parainfectious inflammatory syndrome that primarily affects multiple mucous membranes, with minimal (<10% body surface area [BSA]) or no cutaneous involvement. Initially identified as mycoplasma-induced rash with mucositis (MIRM), the condition was renamed RIME to encompass a broader range of etiological agents [1]. RIME is most frequently observed in pediatric patients and young adults, with a notable male predominance. However, RIME has also been reported in older patients [2,3]. This syndrome tends to occur more frequently during the months from October to February [4]. The diagnosis of RIME is clinical, focusing on the characteristic mucosal involvement and the exclusion of other potential causes. Despite its distinct clinical presentation, RIME is likely underreported, with many cases potentially misclassified as incomplete Stevens-Johnson syndrome [5,6].

Objectives

This manuscript aims to provide an updated overview of Reactive Infectious Mucocutaneous Eruption (RIME), focusing on its clinical presentation, pathophysiology, and differential diagnoses. It also seeks to identify infectious triggers beyond *Mycoplasma pneumoniae*, propose a practical diagnostic and therapeutic algorithm, and highlight treatment strategies and prognostic considerations to support clinical decision-making.

Methods

We conducted a narrative literature review of English-language case reports, case series, and systematic reviews related to RIME and its former designation, MIRM, published up to 2024 and indexed in PubMed. Data were collected on clinical features, histopathology, diagnostic workup, management strategies, and outcomes. In addition, we incorporated insights from our own clinical experience managing RIME cases to complement and contextualize the findings from the literature.

Results

Etiological Agents

Originally thought to be exclusively associated with MP, RIME is now known to be triggered by several agents (Table 1). Bacterial agents include MP, *Chlamydophila pneumoniae* (CP) [3,7], group A beta hemolytic *Streptococcus* [8], and *Chlamydia psitacci* [9]. Regarding viral infection, influenza A [10], influenza B [11], SARS-CoV-2 [10,12–19], common coronavirus [20], norovirus [21], metapneumovirus [6], parainfluenza 2 [6], enterovirus, rhinovirus [16], herpes simplex virus 6 [22], and adenovirus [23] have been reported.

Table 1. Etiological Agents of RIME.

Bacteria	*Mycoplasma pneumoniae*, *Chlamydophila pneumoniae*, group A hemolytic *Streptococcus*, *Chlamydia psitacci*.
Virus	Influenza A, influenza B, SARS-CoV-2, common coronavirus, norovirus, metapneumovirus, parainfluenza 2, enterovirus, rhinovirus, herpes simplex virus 6, adenovirus.

Abbreviations: RIME: reactive infectious mucocutaneous eruption; SARS-CoV-2: severe acute respiratory syndrome coronavirus 2.

Pathophysiology

The pathophysiology of RIME remains unclear. B-cell proliferation, immune complex deposition due to antibodies against MP, and other agents have been proposed, as has complement activation. Resemblance of an unknown keratinocyte antigen and Mycoplasma P1 adhesion molecules in patients with a genetic predisposition has also been suggested [7]. Interestingly, a genetic proneness, especially in human leukocyte antigen (HLA)-B27 and B51 carriers has been hypothesized [6,24].

Clinical Aspects

RIME predominantly affects the mucous membranes extensively, while causing minimal involvement of the skin [1]. The clinical presentation of RIME typically begins with a prodromal phase lasting 7–9 days (1-21 days) characterized by upper respiratory tract infection symptoms or conjunctivitis [21,25]. However, the prodromal phase can be absent [21]. Mucosal involvement is extensive, presenting with edema, erosions, friable mucosa, and hemorrhagic crusts [7]. The oral mucosa is affected in 94–100% of cases, followed by the ocular mucosa in 82%, manifesting as bilateral purulent or seromucous conjunctivitis and eyelid edema. Urogenital mucosal involvement appears in 63% of cases and can lead to permanent sequelae [1]. Involvement of the nasal and anal mucosa is rare or possibly underreported, warranting thorough examination [14,26].

Ocular signs include conjunctival injection, conjunctival ulcers, erythema and ulceration of the eyelid margin, pseudomembrane formation, corneal epithelial involvement, and superficial punctate keratitis [27]. Though ocular sequelae are uncommon, they can occur in up to 13.3% of cases [27].

Cutaneous involvement occurs in 34% of patients and is characterized by erythematous-violaceous plaques, vesiculobullous lesions (77%), atypical targetoid lesions (48%), papules (14%), macules (12%), and morbilliform exanthems

(9%) [1]. The palms and soles are typically spared. In the absence of cutaneous lesions, the condition is referred to as RIME *sine rash* [28]. Conversely, atypical presentations with moderate-to-severe skin involvement have also been published [1,21]. General symptoms include malaise, fever, and joint pain [29], often presenting with feeding difficulties.

Histopathology

The histopathological findings of RIME are nonspecific and can be indistinguishable from those of erythema multiforme/Stevens-Johnson syndrome (SJS) or even toxic epidermal necrolysis (TEN) [21]. The condition is characterized by subepidermal separation and necrosis of keratinocytes within the epidermis. A perivascular lymphocytic infiltrate is usually present, accompanied by vacuolar or lichenoid interface dermatitis, affecting both the epidermis and the follicular epithelium [20]. Additionally, neutrophils and cells with a myeloid and histiocytoid appearance (CD68+, CD163+) can be present within the infiltrate [17]. Importantly, the presence of these cells has not been proven to imply an underlying malignant hematopathology.

Diagnosis

Diagnosis of RIME is clinical. In 2015, Canavan et al. proposed a comprehensive set of diagnostic criteria for classic RIME based on a systematic review of 202 published reports [1]. These criteria include (i) skin detachment involving less than 10% of the BSA, (ii) involvement of two or more mucosal surfaces, (iii) few vesiculobullous or targetoid lesions, the latter most frequently presenting as atypical targets, and (iv) clinical or laboratory evidence of respiratory infection, such as atypical pneumonia, elevated IgM antibodies against MP, positive polymerase chain reaction (PCR), and/or serial cold agglutinins for MP in oropharyngeal swabs or blister fluid. It is crucial to closely monitor cases where only one mucosal surface is affected as well as cases of RIME *sine rash*. Notably, these criteria do not include the involvement of nasal (as in our case) and anal mucosa or microbiological evidence of other etiological agents, which have been identified in subsequent reports.

Differential Diagnosis

Differential diagnoses include conditions with mucocutaneous involvement and epidermal detachment. The most important and time-dependent differential diagnosis is the SJS-TEN spectrum, usually induced by drugs or infections. SJS-TEN (or the overarching term drug-induced epidermal necrosis – DEN) presents a greater extent of detachment and worse prognosis. Viral exanthems, like those caused by EBV, enterovirus (like hand, foot, and mouth disease), and cytomegalovirus (CMV) can present with mucosal involvement. Notably, primary infection by herpes simplex virus (HSV) can be very difficult to distinguish from RIME, especially in pediatric patients. It manifests with gingivostomatitis, pharyngitis, and feeding difficulties, usually accompanied by fever and lymphadenopathy. Autoimmune blistering diseases should also be ruled out. These include pemphigus vulgaris, paraneoplastic pemphigus, and bullous lupus erythematosus. Laboratory testing, histopathology, and immunofluorescence are cornerstone methods in this diagnostic process. Kawasaki disease usually presents in patients younger than 5 years of age and can manifest with targetoid lesions and pulmonary involvement. However, lymphadenopathy is usually present, mucosal involvement most frequently presents with strawberry tongue and cracked lips (non-painful hemorrhagic erosions or ulceration), and other signs such as acral edema and coronary aneurysms may be present [23].

Management

No standardized protocol exists to address the diagnosis of RIME nor its therapeutic management. This work intends to propose a diagnostic and therapeutic algorithm (Figures 1 and 2, respectively), based on the information available in the recent literature, with the aim of facilitating the management of this entity.

Laboratory evaluation should include a complete blood count with differential, as commonly reported hematologic abnormalities include anemia, leukocytosis or leukopenia, neutrophilia, monocytosis, and reactive thrombocytosis. Notably, the presence of anemia should prompt screening of autoimmune hemolysis mediated by cold-agglutinin antibodies related to MP (cold agglutinin syndrome) [30], which tends to be mild. Liver and renal biochemistry and acute phase reactants, such as CRP and erythrocyte sedimentation rate (ESR), should also be included in the study.

Serological testing for MP is crucial at admission, after seven days, and at two months. It is important to note that only IgG may be detected as IgM responses are not always present during primary infection. IgA anti-MP has proven to be a more reliable marker due to its early rise, rapid peak, and decline before IgM and IgG, though its availability is limited [31]. Furthermore, IgG levels have significant fluctuations, and IgM may remain elevated for months if reinfections occur, making it difficult to discriminate between recent and remote infections. For CP, IgM levels exceeding the positivity laboratory threshold or a fourfold increase in IgG of paired sera is necessary for diagnosis [3]. Serological tests should also include HSV-1, HSV-2, varicella-zoster virus (VZV), and CMV antibodies. Serological status of hepatitis B virus (HBV), hepatitis C virus (HCV), and Human Immunodeficiency Virus (HIV) should be investigated, especially if immunosuppressive drugs are used. Anti-desmoglein 1 and 3 and anti-bullous pemphigoid (BP) 180 and anti-BP230 antibodies are useful to discern autoimmune blistering diseases.

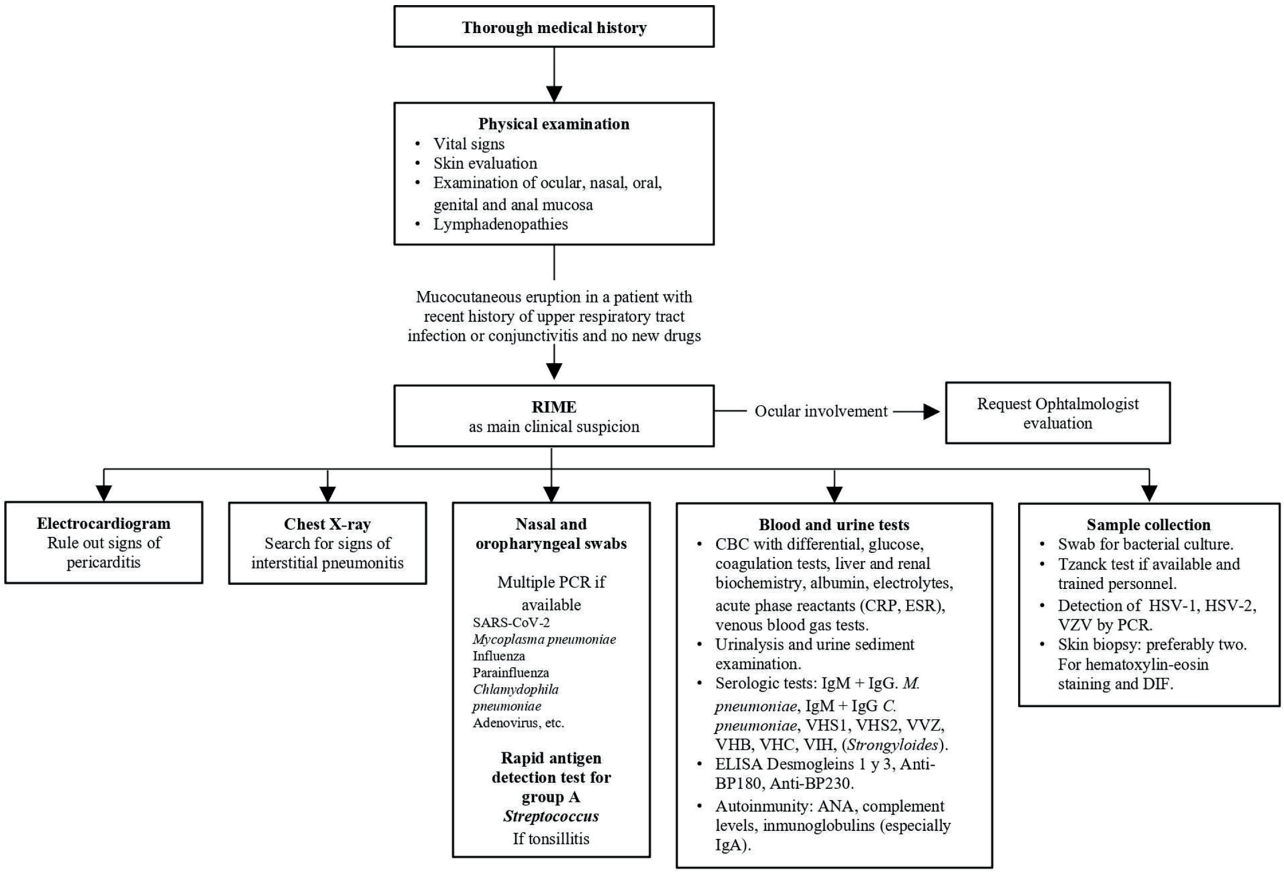

Figure 1. Proposed diagnostic algorithm. (ANA = anti-nuclear antibodies; CBC = complete blood count; CRP = C-reactive protein; DIF = direct immunofluorescence; ESR = erythrocyte sedimentation rate; HSV = herpes simplex virus; PCR = polymerase chain reaction; RIME = reactive infectious mucocutaneous eruption; SARS-CoV-2 = severe acute respiratory syndrome coronavirus 2; VZV = varicella-zoster virus.)

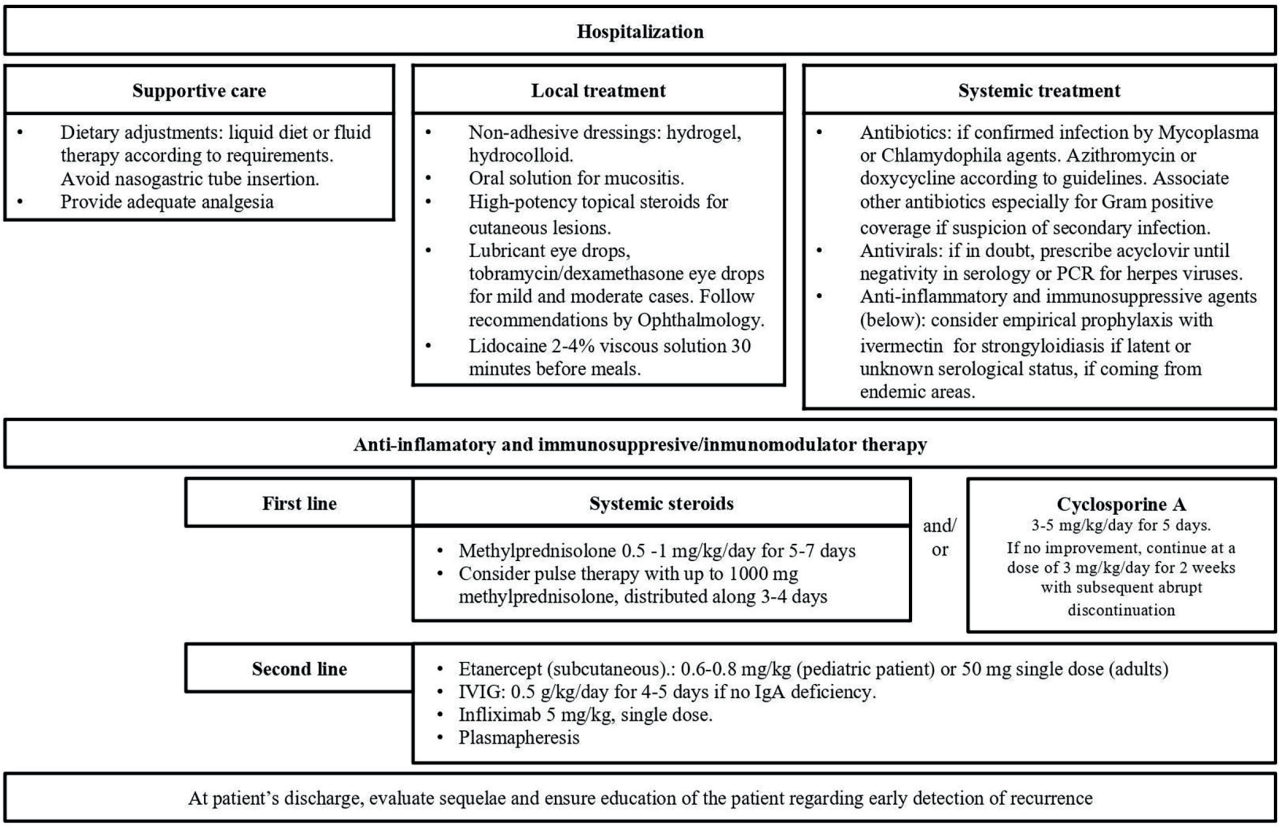

Figure 2. Proposed therapeutic algorithm. (IVIG = intravenous immunoglobulin therapy; PCR = polymerase chain reaction.)

Autoimmunity panels, including antinuclear antibodies, anti-double stranded DNA, antineutrophil cytoplasmic antibodies, complement levels, immunoglobulins, and rheumatoid factor, are also useful in the diagnostic process.

Venous blood gas analysis may be of value in the emergency room to detect acidosis or alkalosis, given that patients with RIME usually have difficulty in solid and liquid intake prior to consultation.

A urinalysis can also be informative, since the presence of hematuria and/or abundant epithelial cells in the sediment can be an indirect sign of inflammation of the urethral mucosa [26].

Nasopharyngeal and oropharyngeal swabs for MP, CP, SARS-CoV-2, influenza, and other agents are also recommended. Multiplex PCR, if available, is a rapid and efficient diagnostic tool. If tonsillitis is present, rapid test for group A *Streptococcus* is indicated. The performance of the Tzanck smear for cytological evaluation can be helpful to detect signs of herpetic infection or blistering disease when used by trained personnel.

An electrocardiogram is a cheap and valuable diagnostic tool which should be performed if there are signs or symptoms of pericarditis, since pericarditis is a recognized extrapulmonary manifestation of MP [32]. Diffuse concave upward ST segment elevation and/or PR segment depression are the most prominent electrocardiographic findings in early phases. Chest X-ray is recommended to identify signs of atypical pneumonia (patchy infiltrates or interstitial patterns), although it should be remembered that DEN might also present with interstitial pneumonitis [33].

Sample collection is also fundamental. This should include bacterial cultures to rule out secondary infection as well as swabs for PCR test for ulcerative diseases (HSV-1, HSV-2, VZV). Ideally, two skin biopsies for hematoxylin-eosin study and direct immunofluorescence should be performed.

Since many patients may have used analgesics or non-steroidal anti-inflammatory drugs, among other medications, prior to consultation, further testing to identify any potential drug hypersensitivity reaction (immunofluorescence, lymphocyte activation assay, patch test, intradermal test, or drug provocation testing) could be performed if in doubt or if overlapping features exist [34]. However, it should be noted that the sensitivity of these tests can vary significantly.

The available evidence on the therapeutic management of RIME, sparse and limited in quality, is based on case reports, case series, and a systematic review published by Vujic et al. addressing solely MIRM [31]. The approach to RIME includes supportive care, which usually requires hospitalization. Dietary adjustments are essential, including the implementation of a liquid diet or fluid therapy as needed and avoiding nasogastric tube insertion. Adequate analgesia is also key in supportive care.

Local treatment includes the use of non-adhesive dressings for cutaneous lesions, prioritizing hydrogel or hydrocolloid dressings. For pain relief in mucositis, specific compounds can be used. The authors prescribe an oral solution which contains 20 mg/mL methylprednisolone, 2% mepivacaine, 40 mg/mL gentamicin, 100 000 UI/mL nystatin, and 1/6 molar bicarbonate. Furthermore, in our experience, adding viscous lidocaine 2%–4% 30 minutes before meals decreases pain and feeding difficulties.

Cutaneous lesions can be managed with high-potency topical corticosteroids such as clobetasol 0.05% ointment. If there is ocular involvement, it is imperative to request evaluation by an ophthalmologist. Fluorescein staining may help assess the severity of the disease. Prescribed treatments will often include lubricants in mild cases and a combination of antibiotic/ corticosteroid eye drops such as tobramycin 0.3% and dexamethasone 0.1% if the ocular involvement is moderate [35]. Topical cyclosporine A (CsA) eye drops have also been used in combination with systemic therapy [21]. If there is progression despite the aforementioned therapies, amniotic membrane transplantation and other advanced therapies might be necessary in a subset of patients [15,21,35].

According to the literature and based on the authors' experience, all patients will need systemic treatment. If MP or CP infection is demonstrated, azithromycin, tetracyclines, or quinolones can be prescribed. However, their impact on RIME disease progression is uncertain [6]. Azithromycin can be administered at a dose of 500 mg daily for three days, either orally or intravenously. Doxycycline is a valid alternative if suspicion of resistant MP (more prevalent in Asia) in patients over 8 years of age. Additionally, if herpetic infection cannot be ruled out, prescription of acyclovir until confirmed negativity in serology or PCR might be a prudent conditional recommendation.

Anti-inflammatory and immunosuppressive agents are the keystone of systemic treatment. If *Strongyloides stercolaris* serology is pending, unavailable, or the patient comes from or travels to endemic areas, empirical prophylaxis with ivermectin can be considered. Most published cases have been treated with systemic corticosteroids, which are thus considered the first line of treatment [36]. Prednisone or methylprednisolone 0.5–1 mg/kg/day for 5–7 days [20] or hydrocortisone at 100 mg every 8 hours for three days have been described with successful outcomes [12]. Pulse therapy with methylprednisolone, up to 1000 mg distributed over 3–4 days, may also be used.

CsA at a dose of 3–5 mg/kg/day for 5–8 days can be used as either a first- or second-line treatment, in monotherapy or in association with systemic steroids [21]. The dose can be continued at 3 mg/kg/day for 1–2 weeks, followed by abrupt discontinuation. CsA serves as a steroid-sparing agent, and as it can shorten hospitalization by 6–7 days compared to

corticosteroids, it is therefore considered as first-line treatment by some authors [13,25].

Other alternative treatments include anti-tumor necrosis factor alpha (TNFα) such as infliximab and etanercept. These have been used with limited evidence, extrapolated from data on DEN. Infliximab may be considered, administered at a single 5 mg/kg dose. Etanercept at 0.6–0.8 mg/kg (children) or 50 mg (adults), with a second possible dose if no improvement is seen after 48 hours, has also been described [21,29]. Intravenous immunoglobulins (IVIG) at 0.5–2 g/kg/day for 4–5 days may also be administered, ensuring there is no IgA deficiency prior to the administration [20,23,35]. Lastly, plasmapheresis has been described in a single case [37].

Prognosis

RIME generally portends a favorable prognosis, with a typically benign course leading to complete recovery within seven to 21 days. However, certain complications can arise, such as secondary bacterial superinfections by Gram-positive skin flora. There is a report of a patient presenting both *Staphylococcus* bacteremia and disseminated HSV infection as complications of RIME [38]. Gram-negative superinfection can also arise [22].

Long-term sequelae, though relatively uncommon, may include ocular and genital synechiae, post-inflammatory hyperpigmentation, and lymphopenia. Recurrences occur in approximately 8–38% of the patients [4], with presumable higher frequency in HLA-B51 and B27 carriers [6,24]. Recurrences have been reported following infections with both MP (more frequently) and CP, among many. Epstein-Barr virus reactivation has been described in a patient with recurrent RIME, with uncertain clinical significance [39]. Interestingly, recurrent episodes are often milder and may affect only one mucosa [8]. However, more severe recurrences can occasionally occur. While different agents may trigger recurrences, MP is usually responsible for the first episode [10,16].

Conclusions

RIME is an underreported and probably misdiagnosed entity that presents predominantly in pediatric and young adult patients, although some cases in older adults have been described. Its distinctive clinical feature is extensive mucosal involvement accompanied by minimal cutaneous manifestations. Initially attributed solely to MP, the list of etiological agents is gradually becoming broader, including bacterial and viral infections. Diagnosis can be challenging due to overlapping features with other entities with prominent mucositis, such as DEN, herpetic gingivostomatitis, and autoimmune bullous diseases. There is a paucity of knowledge on the management of RIME, but hospitalization is usually necessary for supportive care. Systemic steroids and/or CsA are the most reported effective treatments. The role of antibiotics is unclear, but macrolides or tetracyclines are often used if there are signs of pneumonia or demonstration of MP or CP infection. Other therapies include anti-TNFα agents (infliximab or etanercept), IVIG, or plasmapheresis. Prognosis is generally favorable, but complications can occur. These include secondary bacterial superinfection, disseminated HSV infection, and ocular sequelae. Recurrences, though uncommon, seem more frequent in HLA-B51 and B27 carriers. They can be caused by different agents, but the first episode is generally related to MP. Further research is needed to establish diagnostic and therapeutic protocols to enhance patient care in RIME cases.

Ethics Statement: The clinical images included have been provided by the authors. Informed consent was retrieved.

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